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MORTALITY OF COMMON LOONS IN NEW ENGLAND, 1987 TO 2000

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ABSTRACT: Diagnostic findings are presented on 522 common loons (*Gavia immer*) found dead or moribund in New England (Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont, USA) between 1987 and 2000. Common loon numbers and range in New England have decreased from historic levels over the last century due to a number of proposed factors. Goals of this study were to identify and categorize causes of mortality and quantify natural versus anthropogenic causes. The majority of identifiable mortality in chicks was from intraspecific aggression (25%) and other causes of trauma (32%). Death in immature loons was primarily from fungal respiratory disease (20%) and trauma (18%). Causes of adult loon mortality differed significantly in breeding and wintering habitats. Wintering adults primarily died of trauma (17%) and infection (11%) and had significantly poorer body condition than breeding loons. In breeding adults, confirmed and suspected lead toxicosis from ingested fishing weights accounted for almost half of all mortality. Direct anthropogenic factors accounted for 52% of loon mortality in this study. Because of high carcass recovery rates, we believe these data are a good representation of loon mortality in New England. Results highlight the importance of human influences on conservation and management of the common loon in New England.

Key words: Anthropogenic factors, *Aspergillus,* common loon, *Gavia immer,* lead toxicosis, New England, trauma.

INTRODUCTION

Common loons (*Gavia immer*) have been proposed as indicators of aquatic health in the northern lake ecosystems they inhabit (Scheuhammer et al., 1998) and are the subjects of intense study. Loons are listed as a threatened or endangered species in several northeastern states. Because of a variety of environmental and human factors, their numbers and range in New England have decreased from historic levels over the last century (McIntyre and Barr, 1997). Despite recovery over the last few decades due to management of these populations, mortality has increased in some areas (New Hampshire Loon Preservation Committee, unpubl. data). Previous studies of loon mortality have documented lead toxicosis, oil spill contamination, respiratory aspergillosis, botulism, trauma, net or monofilament fishing line entanglement, and an emaciation syndrome of unknown etiology as leading causes of death (Brand et al., 1988; Pokras et al., 1991; Forrester et al., 1997; Augspurger et al., 1998; Daoust et al., 1998). Environmental contaminants, such as mercury and organochlorines, are elevated in some populations as well, though it is debated whether this is linked to increased mortality (Frank et al., 1983; Forrester et al., 1997; Barr, 1986; Augspurger et al., 1998).

This mortality study of New England's common loons was begun in 1987 after a case of lead toxicosis due to fishing sinker ingestion was found in New Hampshire. The goals were to identify and quantify natural and anthropogenic causes of mortality in this population. This report summarizes results of 12 yr of collection and analysis of loons from across New England and compares these results with those of previous studies.

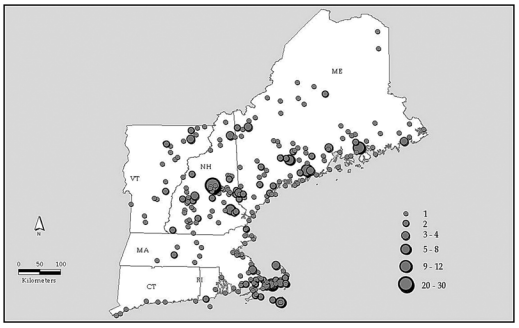

FIGURE 1. Location of common loon carcasses examined from New England, 1987–2000. The number of loons collected per site is indicated by the size of the corresponding circle. No site had between 13 and 19 loons collected.

MATERIALS AND METHODS

From 1987 to 2000, 522 dead or moribund loons were submitted to the Wildlife Clinic at Tufts University School of Veterinary Medicine (TUSVM) in North Grafton, Massachusetts. Loons were collected from both interior and coastal regions in Connecticut (n=6), Maine (n=173), Massachusetts (n=118), New Hampshire (n=184), Rhode Island (n=11), and Vermont (n=30) (between 47°25′N, 66°55′W and 41°0′N, 73°45′W) by state and federal wildlife agencies, wildlife rehabilitators, veterinarians, and non-profit organizations. Geographic distribution of loons collected for this study is shown in Figure 1. Sick or injured loons deemed non-releasable were euthanized by intravenous injection of sodium pentobarbital (Beuthanasia-D Special Euthanasia Solution, Schering-Plough Animal Health Corp., Union, New Jersey, USA), according to currently accepted veterinary protocol (American Veterinary Medical Association, 2000). Carcasses collected from remote areas were frozen as soon as possible after collection, and kept frozen until the time of examination, usually 3–6 mo postmortem. Each bird was weighed and radiographed and complete gross necropsy was performed. Age class categories were chick (prefledging, on natal lake), immature (fledged, just before migration away from the natal lake or on saltwater after migration), or adult, as differentiated by plumage. Breeding and wintering adult populations were distinguished by the season and site of collection and plumage. Body condition was subjectively scored as good, thin, or emaciated, as assessed by the abundance of subcutaneous, mesenteric, and pericardial fat stores and amount of pectoral muscle mass. Birds scored as good had abundant fat stores and pectoral muscle mass obscuring a barely palpable keel. Thin loons had adequate to scant amounts of fat and a palpable keel. Emaciated loons had no fat depots, severely atrophied pectoral muscle mass, and a prominent keel. Sex was determined by direct visualization of the gonad if carcass condition allowed positive identification (n=451). The entire liver was collected for lead and other contaminant analysis, and stored in glass jars (I-Chem, Division of Nalge, New Castle, Delaware, USA) at −70 C.

Liver lead was determined by inductively coupled plasma-atomic emission spectroscopy at one of three laboratories specified by the US Fish and Wildlife Service (Hazleton Environmental Services, Inc., Madison, Wisconsin, USA, n=49; Research Triangle Institute, Research Triangle Park, North Carolina, n=21); and University of Pennsylvania, New Bolton Center, Laboratory of Pathology and Toxicology, Kennett Square, Pennsylvania, USA, n=189). Detection limits varied by laboratory between 0.05 and 1.0 part per million (ppm). Values below the detection limit were included in the calculations at the detection limit. Specific toxicologic analysis techniques are detailed in Pokras et al. (1991) and Dahlquist and Knoll (1978). If the carcass was in suitable condition, a standard set of tissues was taken for histopathology, including esophagus, proventriculus, ventriculus, intestine, cecum, pancreas, liver, gall bladder, spleen, heart, trachea, lung, kidney, gonad, adrenal, thyroid, pectoral muscle, sciatic nerve, and brain, including any significant gross lesions. These tissues were fixed in 10% neutral buffered formalin, dehydrated, and embedded in paraffin; 5 μm sections were stained with hematoxylin and eosin and examined by light microscopy at the Tufts University School of Veterinary Medicine Department of Pathology.

Determination of ultimate cause of death was based on history, clinical signs, and radiographic, gross, and microscopic findings. As described in Beyer et al. (1998) and Friend (1999), a diagnosis of lead toxicosis was made when a liver lead concentration >6 ppm wet weight was found in conjunction with either a lead object in the ventriculus or clinical, gross, or histologic signs typical for lead toxicosis, or both. A diagnosis of suspected lead toxicosis was made when a lead object was present in the ventriculus, with or without clinical or gross signs, but liver lead levels were not determined or were <6 ppm. This category also included a loon euthanized because of the radiographic presence of lead in its ventriculus, but which had liver lead <6 ppm. Presence of lead objects in the ventriculus was determined by radiographic and direct visualization of items of me-

tallic density. Metallic objects found in the ventriculus were tested by a commercial qualitative colorimetric swab method (Lead Check Swabs©, HybriVet Systems, Inc., Framingham, Massachusetts); an intense pink color on the swab confirmed the presence of lead in the object.

The category of trauma included birds observed struck by boats and those with injuries indicative of blunt trauma that was not consistent with loon aggression. A few trauma cases were loons attempting to land on wet asphalt surfaces that resemble open water. A diagnosis of intraspecific trauma was based on observations in the field or by lesions consistent with pecking or penetrating beak wounds. These wounds included punctures of pectoral muscles and sternum or abdomen, with hemorrhage and laceration of internal organs, or, in chicks, wounds and feather loss at the back of the head and neck. The category of fishing gear mortality included deaths caused by ingestion of fishing gear, with penetrating gastrointestinal wounds and peritonitis caused by hooks or gastric obstruction by other gear, but not including ingestion of lead sinkers. Entanglement with nets or monofilament line was also included in this category.

Fungal respiratory infections were considered a cause of death only when loons presented with massive involvement of multiple air sacs, lung parenchyma, and other tissues. These lesions were identified grossly by the presence of typical yellow, caseous, fungal plaques and nodules but were not cultured. When possible, the presence of *Aspergillus* sp. was confirmed histologically in these lesions by the presence of typical branching, thin-walled, and septate hyphae. Cases not confirmed were categorized as suspected fungal respiratory disease.

Bacterial infection was diagnosed by histologic examination of involved tissues or by diagnostic culture. Bacterial cultures were taken by culture swab from gross lesions suggestive of bacterial infection. Cultures were submitted to the Tufts Veterinary Diagnostic Laboratory (North Grafton, Massachusetts) for isolation and identification by standard microbiologic techniques.

Loons with gunshot wounds generally exhibited both an entrance and an exit wound or had bullet or pellet fragments lodged in tissues. Mortality associated with oil was diagnosed by the presence of significant amounts of oil on the feathers, with obvious disruption of waterproofing, with or without evidence of ingestion or internal lesions. Parasitic infections were determined to be the ultimate cause of death only when significant gross or microscopic lesions, or multi-systemic involvement was present. Parasites were identified by class as nematodes, cestodes, or trematodes but were not speciated for the purposes of this study; a subset of these helminths has been described previously (Chafel and Pokras, 1992). Predation was determined by the presence of puncture wounds or lacerations not consistent with intraspecific attacks. Loon chicks deemed victims of predation were typically recently hatched and found on or near the nest. Miscellaneous causes of mortality were identified based on their unique clinical, gross, and microscopic presentations. The category of unknown mortality included loons with no significant gross or histologic lesions and those found emaciated with minor fungal respiratory infections but no other obvious lesions, as well as carcasses too decomposed for thorough examination.

Body weight data for live loons were collected from 1995–97 as part of an on going capture and banding project in Maine and New Hampshire. Adult breeding loons were captured on freshwater lakes by a spotlighting technique, as previously described (Evers, 1992). Body weight was measured to the nearest 50 g using a 5 or 10 kg spring scale (Pesola Prazisionwaagen AG, Baar, Switzerland) and a mesh bag to contain the loon.

Annual common loon censuses were conducted by Loon Preservation Committee field biologists in New Hampshire to catalog individual loons and the breeding success of pairs. Lakes were prioritized for visitation according to a three-tiered system. Tier I lakes had a current history of loon pairs defending territories (minimum of 3 yr continuous occupancy or newly occupied) and required at least three site visits. Any lakes other than Tier I lakes that had a history of loon pairs were designated Tier II lakes. Tier II lakes support loon pairs on an irregular basis and at least one visit was required. Tier III lakes had few loon sightings on record. These lakes were visited as time allowed and prioritized by making observations on lakes not surveyed in the previous year. All observations were made from boat or by shoreline observations using 10× binoculars and/or 15–45× spotting scopes.

Annual mortality was estimated by use of the biologic effects submodel of the Natural Resource Damage Assessment Model for Coastal and Marine Environments (NRDAM/CME, National Technical Information Service, Springfield, Virginia, USA).

General statistical and data analyses were performed using commercially available software (SPSS version 6.1, SPSS Inc., Chicago, Illinois, USA). Differences in mean weight and liver lead values between groups were com-

TABLE 1. Population of common loons in New Hampshire with mortality estimates and carcass recoveries, 1996–99.

Year	Total adult loons in New Hampshire[a]	Estimated annual mortality[b]	Loon carcasses recovered	Percent recovered
1996	450	45	15	33%
1997	474	47	13	28%
1998	493	49	14	29%
1999	481	48	12	25%
Average	474.5	47	13.5	28%

[a] Data collected by New Hampshire Loon Preservation Committee.
[b] Assumes 10% annual mortality, as estimated by NRDAM model.

pared using independent-samples *t*-tests. Categorical variables were compared using a chi-square test. A Kolmogorov-Smirnov test was used to assess normal distribution of variables. The Fisher's exact test was used to compare distributions of causes of mortality between groups using the shareware program Exact2xK (Abramson and Gahlinger, 2001). All statements of statistical significance are based on $P<0.05$.

RESULTS

Population, estimated mortality, and recovery data of adult breeding loons from New Hampshire for 4 yr of the study are in Table 1. Mortality data for all loons included in the study are summarized in Table 2.

Cause of death varied significantly by age group ($P=0.001$) and habitat ($P=0.0008$). The majority of identifiable mortality in chicks ($n=103$) was from intraspecific aggression and other causes of trauma. Death in immature loons ($n=65$) was primarily from fungal respiratory disease and trauma. Causes of adult loon mortality differed significantly in breeding and wintering habitats. Wintering adults ($n=100$) primarily died of trauma and infection. In breeding adults ($n=254$) confirmed and suspected lead toxicosis from ingested fishing weights accounted for almost half of all mortalities. There was no significant difference ($P=0.34$) in causes of mortality between sexes.

Body weight and subjective body condition scores of adult loons varied significantly by gender, habitat, and cause of death. Weight was normally distributed within the population ($P=0.0001$). Male adult loons were significantly heavier than female adult loons ($P=0.001$), with weights of 4.2±1.2 kg (mean ± SD, $n=171$) and 3.3±1.0 kg, ($n=146$), respectively. Wintering adult loons had significantly ($P=0.001$) lower mean body weights

TABLE 2. Causes of common loon mortality in New England, 1987–2000, by age class and season.

	Chick		Immature		Breeding adult		Wintering adult		All loons	
Primary cause of death	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
Unknown	28	27	19	29	45	18	39	39	131	25
Lead toxicosis[a]	—[b]	—	3	5	111	44	4	4	118	23
Trauma	33	32	12	18	30	12	17	17	92	18
Intraspecific trauma	26	25	3	5	11	4	—	—	40	8
Fishing gear	2	2	4	6	24	9	9	9	39	7
Fungal respiratory disease[a]	—	—	13	20	18	7	6	6	37	7
Infection	10	10	5	8	4	2	11	11	30	6
Gunshot	—	—	—	—	8	3	5	5	13	2
Oil	—	—	3	5	1	<1	4	4	8	2
Miscellaneous	1	1	1	2	1	<1	5	5	8	2
Parasites	1	1	2	3	1	<1	—	—	4	1
Predation	2	2	—	—	—	—	—	—	2	<1
Total loons examined	103		65		254		100		522	

[a] Combined confirmed and suspected cases.
[b] None found.

TABLE 3. Average adult loon body weight for each category of mortality.

Primary cause of death	Average weight (kg)	Standard deviation (kg)	*n*
Intraspecific trauma	4.5	1.3	11
Lead toxicosis	4.3	1.0	106
Trauma	3.7	1.3	45
Fishing gear	3.6	1.4	30
Shot	3.5	1.3	12
Unknown	3.4	1.2	78
Infection	3.4	1.1	15
Fungal respiratory disease	3.4	0.9	22
Miscellaneous	3.3	1.1	6
Oil	2.6	0.3	5
Parasites	1.6	—	1
All adult loons[a]	3.8	1.2	331

[a] In cases where body weight was recorded.

of 2.9±0.9 kg (n=94) and significantly (P=0.001) poorer subjective body condition scores (91% versus 62% thin or emaciated body condition score) than breeding loons which had average weights of 4.1±1.2 kg (n=237). Table 3 lists mean body weights for adults in each category of mortality. Weights of live breeding adults from comparable populations, with average weights of 5.4±0.8 kg (n=83), were significantly higher than those of loons found dead (P=0.001). Comparisons of weights of chicks or immature loons were not performed because variability in age would confound results. Suspected and confirmed categories for both lead toxicosis and fungal respiratory infection were combined for these comparisons of body weight.

TABLE 4. Association of clinical signs and presence of gastrointestinal lead objects in confirmed and suspected lead toxicosis.

	Confirmed lead toxicosis	Suspected lead toxicosis
Liver lead (ppm, wet weight)	>6 ppm	Not tested or 2–6 ppm
Lead object alone	24	22
Clinical signs alone	3	0
Lead object and clinical signs	41	28
Total loons	68	50

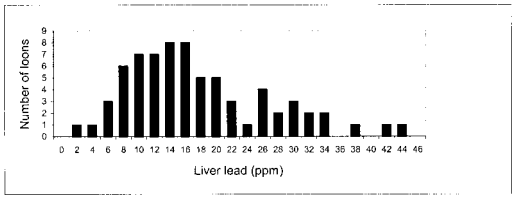

FIGURE 2. Liver lead histogram for common loons with confirmed and suspected lead toxicosis. n=71.

Table 4 delineates the assignment of loons to the categories of confirmed or suspected lead toxicosis. These categories do not include five loons that had liver lead levels >6 ppm but had no clinical signs or lead objects present in the gastrointestinal tract. Four of the 28 birds with clinical signs and lead objects, included in the suspected toxicosis column, were found to have slightly elevated liver lead levels between 2 and 6 ppm. Histograms of liver lead values for loons in the combined categories of confirmed and suspected lead toxicosis, or other causes of death are presented in Figures 2 and 3, respectively. Average liver lead value in loons dying from confirmed lead toxicosis was 17.6±8.8 ppm wet weight (n=68), compared to 1.0±2.5 ppm (n=194) in other loons, including suspected lead intoxication.

Typical clinical signs found with loons who had ingested lead included weakness and beaching, drooped wing posture, head tremors, and open-mouth breathing or dyspnea. Typical necropsy findings included green staining of feathers at the vent, impaction and dilatation of esophagus and

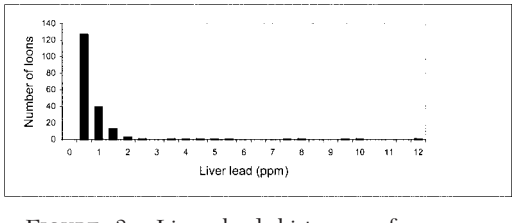

FIGURE 3. Liver lead histogram for common loons with non-lead associated mortality. n=194.

proventriculus with food items, dark green or black staining of ventriculus lining, a distended gall bladder, and dilation of intestine or cecum. Hepatic Kupffer cell hemosiderosis was often found on histologic examination of loons with elevated liver lead levels, which is suggestive of hemolytic anemia (Cullen and MacLachlan, 2001). Acid-fast renal inclusions were only rarely found.

Severe, systemic *Aspergillus* infections were confirmed in 14 loons and suspected in 23 others. Less severe lesions associated with fungal respiratory disease were found in 59 loons assigned to other categories of mortality. Causes of mortality for these 59 loons differed significantly ($P<0.008$) from those loons without fungal lesions ($n=329$). Loons in the categories of infection, trauma, and gunshot were most likely to be affected, with lesions found in seven (23%), 18 (20%), and three (23%) birds in these categories, respectively. Fungal growths in air sacs were found significantly more often in adult or immature birds from wintering populations ($P<0.003$). Adult loons with fungal lesions had significantly ($P<0.002$) reduced body weights (3.4 ± 1.0 kg, $n=56$) compared to those without (3.8 ± 1.2 kg, $n=275$). Frequency of fungal lesions differed significantly by age group ($P<0.00001$), with immature loons being most likely to be affected (46%, $n=30$), compared to adults (17%, $n=60$) and chicks (6%, $n=6$). No significant difference in prevalence of fungal lesions was found between male and female loons ($P<0.8$).

The miscellaneous category contained several diverse cases. Neoplastic lesions were the cause of death in two adult birds. These were a hepatic biliary ductular adenocarcinoma and a metastatic tumor of possible hepatic origin. Another loon died from complications of egg binding and peritonitis. One small adult loon asphyxiated after attempting to swallow a large fish. Another had gross changes in the kidney consistent with severe renal disease; histopathology was inconclusive because of autolytic changes in tissues. Two loons developed aspiration pneumonia after treatment and gavage at rehabilitation centers. Finally, after being observed separated from its parents during inclement weather, a loon chick became exhausted and drowned.

Direct anthropogenic causes of mortality, including confirmed and suspected lead toxicosis, fishing gear ingestion or entanglement, oil, gunshot, and most cases of trauma (assumed to result from boat collision) accounted for 270 of 522 (52%) cases of loon mortality among all age categories.

DISCUSSION

The loon population in New England is unusual in the degree of scrutiny it receives. The breeding loons in Massachusetts, Maine, and, especially, New Hampshire are monitored closely from the time they arrive in spring until the young fledge and migrate in the fall. This is possible in part because loon populations in this region, at the most southern extreme of their breeding range, are small as compared to more northern and less developed areas. Although every loon carcass was not found, our recovery was high, averaging an estimated 28% in recent 4 yr of the study in New Hampshire, as compared to the 6% recovery rate of wild waterfowl mortalities reported by Stutzenbaker et al. (1986). Given this recovery of carcasses, we believe this study to be a good indicator of overall mortality in this region with a few caveats described below.

This study emphasizes the importance of anthropogenic influences on this population of loons. Figure 1 shows that collection of loon carcasses was focused in a band along the New England coast and central/southern areas of New Hampshire, correlating with areas of high human population density and, therefore, more closely examined lakes. Our study highlights mortality occurring on the breeding grounds. Loons dying on the ocean are much less likely to wash ashore than those

dying on lakes and the amount of time a carcass lasts on the beach may be significantly limited by wind and weather conditions and scavenging (Forsell, 1999).

This study does not include an estimated 400 loons killed in a large oil spill off the Rhode Island coast in spring 1996 because full necropsy data were not available. Oil spills are a recognized danger for many seabirds and have a large impact on flightless, wintering loon populations. Several previous studies of loon mortality have also been associated with epizootics of varying etiologies (Brand et al., 1988; Forrester et al., 1997; Augspurger et al., 1998). The comparative effect of the mortalities reported here on regional loon populations is unknown.

Because this was a study of wild birds and the carcasses were often collected days after death, decomposition affected accurate determination of the cause of death. In cases where tissues were considerably decomposed, subtle metabolic or infectious diseases would be harder to detect, and obvious traumatic injuries may be given more weight. Additionally, because cultures were not routinely performed in this study, bacterial disease was probably under-diagnosed. Deaths due to predation of chicks were likely underrepresented in our sample because of the lack of physical evidence.

Habitat differences in body weights and mortality likely reflect the harsher environment and seasonal stresses of the coastal wintertime feeding grounds. Energy requirements for the winter molt would also add additional metabolic stress, resulting in higher rates of infectious and opportunistic disease. This is supported by our findings on fungal respiratory disease. Aspergillosis was more likely to be seen in both adult and immature birds found on saltwater in this study, and *Aspergillus* sp. has been reported as a common infectious agent in other populations of wintering loons (White et al., 1976; Alexander, 1991).

High human recreational pressure in the breeding territories on freshwater hypothetically leads to increased interactions between boats and loons, with resulting increases in traumatic injuries, line entanglement, and opportunity for exposure to lead fishing weights. Differences in mortality between age classes can similarly be explained by the differences in habitat and environmental pressures. Immature loon mortality appears to follow the patterns of both wintering adult and chick mortality, with trauma and *Aspergillus* infection as leading causes of death, reflective of these loons' presence in both the breeding and wintering habitats.

Differences in body weights and body condition were seen associated with several different factors. Female loons are about 25% smaller than males, despite their lack of dimorphism in plumage. Examination of the variations in body weight compared to cause of death gives an indication of the probable chronicity of the individual syndromes. For instance, loons with relatively chronic parasitic or fungal disease have low body weights; whereas loons dying of traumatic injuries have relatively higher body weights. Loons killed by other loons had the highest body weights, consistent with the acute, traumatic manifestations of territorial aggression observed in the field. Birds found with gunshot wounds typically did not die acutely but had chronic infections or wounds that would impair foraging ability, with intermediate body weights at death.

Lead intoxication in this population is associated with relatively high body weights compared to other causes of mortality. Apart from signs of lead toxicosis, few lesions were seen in these birds. We believe this indicates a relatively acute death, which is consistent with our clinical and field experience in this study. Other studies have found that loons with lead toxicosis are generally in poor body condition (Daoust, et al., 1998), and lesions similar to starvation are observed in other lead poisoned waterfowl (Beyer et al., 1998). However, Sanderson and Bellrose

(1986) indicate that weight loss is not always seen, especially in cases of acute toxicosis. Why this population of loons responds differently than that in the Canadian study is unclear, but may reflect the multifactorial nature of wildlife mortality and environmental stressors.

Liver lead levels of >6 ppm were considered indicative of toxicosis in this study, and levels between 2 and 6 ppm were considered suggestive. This is consistent with the literature in waterfowl and other birds (Franson, 1996; Pain, 1996). Although elevated liver lead levels have been reported in loons, no studies have attempted to determine threshold levels (Franson, 1996). Examining our data for lead levels found in unexposed and clinically affected birds in Figures 2 and 3, it can be seen that the majority of unexposed loons have lead levels at or below the threshold of detection (0.05–1.0 ppm), while the majority of loons found with ingested lead objects have levels much greater than 6 ppm. There were eleven loons in the population of loons with non-lead associated mortality that had elevated levels; we suspect these cases represented undetected exposure to lead objects in the environment. We suggest that in loons, conservative criteria for lead toxicosis are similar to those reported in other waterfowl, although any liver lead >2 ppm is suggestive of exposure. As has been previously proposed, in the face of gross and microscopic evidence of toxicosis, the presence, but not necessarily the degree, of elevations should be considered diagnostic (Franson, 1996).

The results of this study are similar to preliminary reviews of these data, which reported breeding loon mortality due to lead ingestion at 52% (Pokras et al., 1991; Pokras and Chafel, 1992). Lead toxicosis is still the largest cause of mortality in this population. Adult loon mortality due to lead toxicosis in New England was almost twice as common as in adjacent Canadian maritime populations (26%) (Daoust et al., 1998). This contrast may be due to the smaller sample size of loons in that study (n=31) or differences in monitoring of populations and collection of carcasses. It may also reflect differences in regional landscape, human population, or land and wetland usage. If a few lakes in southern New Hampshire, which are heavily used for recreation and have high adult lead-associated mortality approaching 100%, are removed from the analysis, the regional rates become comparable (data not shown).

Our results support the hypothesis that anthropogenic causes are the most significant determinants of loon mortality in New England, accounting for approximately half of all deaths in our study. These reasons for loon mortality are especially significant when considering sustainability of their populations in this region. Taken in conjunction with other indirect factors such as habitat encroachment and environmental contamination, these effects may be compounded (McIntyre, 1975; Yonge, 1981; Barr, 1986). Although wildlife mortality is dependent on a complex set of factors and stressors, it is clear from our data that future conservation and management of loon populations must consider human influences.

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