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Source: Journal of Wildlife Diseases, 34(3) : 524-531

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-34.3.524

Journal of Wildlife Diseases, 34(3), 1998, pp. 524–531

INTERACTIVE MORTALITY FACTORS IN COMMON LOONS FROM MARITIME CANADA

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ABSTRACT: Between August 1992 and November 1995, 31 moribund or dead common loons (*Gavia immer*) found in the three Maritime provinces of Canada (New Brunswick, Nova Scotia, Prince Edward Island) were necropsied. Eight of these birds were in good body condition and died acutely from drowning or trauma. The remaining 23 birds were in poor body condition and had either chronic lead poisoning, respiratory mycosis, or oil contamination of their plumage. Loons in poor body condition had significantly higher numbers of intestinal trematodes and significantly higher levels of total renal mercury than loons in good body condition. Therefore, poor body condition in many loons was associated with two or more concurrent potential disease processes, although we could not establish a cause-effect relationship among these processes in individual birds. These results suggest that mortality in chronically ill wild animals can result from synergism among several potentially debilitating agents present in their environment.

Key words: Common loon, *Gavia immer*, infectious disease, lead, mercury, synergistic interactions.

INTRODUCTION

Although the suitability of the common loon (*Gavia immer*) as a general indicator species for the quality of aquatic habitats is controversial (Strong, 1990), its long life span and high trophic level in the aquatic food chain make it a useful species for assessing the occurrence of contaminants in the environment. Studies on causes of mortality in this species may help to identify and address anthropogenic etiologies. Lead poisoning resulting from ingestion of lead fishing sinkers and mercury contamination resulting from bioaccumulation in the food web are two important toxicoses of common loons in eastern North America (Barr, 1986; Pokras and Chafel, 1992; Scheuhammer and Norris, 1996). Recently, adult common loons from the Canadian Maritimes were found to have the highest concentrations of total mercury in blood ($\bar{x}$ ± S.D. = 3.53 ± 1.86 μg/g) observed in any North American population of this species (Evers et al., 1998). These concentrations were strongly correlated between adults and chicks from the same family groups, indicating that concentrations in adult birds reflected exposure to mercury in their breeding lakes rather than in their marine wintering areas. Biomagnification of mercury in northeastern North American lakes probably is favored by the acidity of these lakes and by high levels of atmospheric deposition of mercury in this region (Evers et al., 1998).

Other causes of mortality reported in common loons include respiratory mycosis caused by *Aspergillus fumigatus*, drowning in commercial fishing nets, and trauma from motor-boats or gunshots (Frank et al., 1983; Pokras et al., 1991). The aim of the present study was to provide a detailed analysis of the health status of individual common loons found moribund or dead in the Maritime provinces of Canada and to correlate the results of toxicological analyses with other pathological findings. This complements similar studies elsewhere in North America (Frank et al., 1983; Pokras et al., 1991).

MATERIALS AND METHODS

Thirty-one common loons, collected in all months of the year between August 1992 and November 1995, were submitted for laboratory examination to the Atlantic Veterinary College (University of Prince Edward Island, Charlottetown, Prince Edward Island, Canada). These birds (23 from Nova Scotia, five from New Brunswick, three from Prince Edward Island)

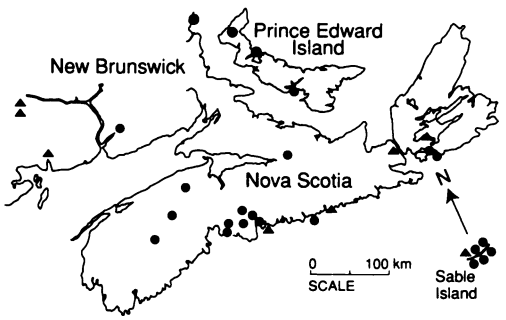

FIGURE 1. Collection sites of common loons in good (▲) and poor (●) body condition necropsied in the Canadian Maritime provinces between 1992 and 1995.

were found dead or moribund along shores of freshwater lakes, the Atlantic Ocean, or the Gulf of St. Lawrence (between 43°50′N, 59°40′W and 48°0′N, 67°50′W) (Fig. 1). Most carcasses were frozen for 1 to 15 mo before submission. A complete necropsy was performed on each bird. For each bird, the age groups of adult (at least 2-yr-old) versus immature were determined on the basis of degree of development of the gonad(s), presence or absence of a bursa of Fabricius, and plumage (for birds collected during spring and summer) (McIntyre, 1988). No unfledged birds were examined. Body condition was assessed at necropsy on the basis of the relative abundance of fat reserves, particularly in the subcutis and around the base of the heart, and the degree of pectoral muscle development. Birds with conspicuous fat reserves and well developed pectoral muscles were considered in good body condition. Birds with partial or complete absence of fat reserves and moderately to severely atrophic pectoral muscles were considered in poor body condition. One entire kidney from each bird was analyzed for lead and total mercury, and one half of the brain from 10 birds was analyzed for total mercury. Prior to analysis, these samples were stored in Whirl-pak plastic bags (Baxter Corporation, Canlab Division, Toronto, Ontario, Canada) at −20 C. The contents and mucosal lining of the entire intestinal tract from 26 birds were examined for helminths. The following tissues were collected for microscopic examination: brain, brachial and sciatic nerves, heart, lung, stomach, liver, kidney, spleen, thyroid, adrenal, and skeletal muscle. These tissues were fixed in 10% neutral buffered formalin, dehydrated in graded alcohol and xylene, and embedded in paraffin blocks; 5 μm-thick sections were stained with hematoxylin and eosin (Luna, 1968). Tissues from a single bird with gross lesions suggestive of a bacterial infection were examined bacteriologically. These tissues were cultured at 35 C in 5% CO_2 on 5% sheep blood and MacConkey agars (Oxoid Inc., Nepean, Ontario, Canada), and bacterial isolates were identified to genus (Quinn et al., 1994). Cases of respiratory mycosis compatible with aspergillosis were diagnosed on the basis of gross and microscopic lesions (Chute and Richard, 1991) but were not confirmed by cultural isolation and identification of the fungus.

For lead analysis of kidney samples, duplicate 1 g portions of fresh tissue weighed to the nearest 0.01 g were digested with deionized water and concentrated nitric acid (trace metal grade) for 30 min with a microwave sample preparation system (Model MDS-2000, CEM Corporation, Matthews, North Carolina, USA). Digested samples were reconstituted to 10 ml with deionized water. The lead concentration for each sample was determined by graphite furnace atomic absorption spectroscopy (Model Zeeman 5100, Perkin-Elmer Corporation, Ridgefield, Connecticut, USA). International Atomic Energy Agency (A-1400 Vienna, Austria) certified reference material (MA-B-3TM-fish tissue) was used as a quality control (90–110% recovery). The detection limit with this method was 0.25 μg/g (wet weight). For total mercury analysis of kidney samples, each tissue was weighed and homogenized with half its weight of 0.1N sulfuric acid (ACS grade). Samples of homogenates were weighed in PARR digestion bombs (Parr Instruments, Maline, Illinois, USA), mixed with 2.5% potassium dichromate in concentrated nitric acid (ACS grade), and heated at 140 C for 1.5 hr. The bombs were subsequently cooled, and digested tissues were diluted with deionized water to a final volume of 25 ml. Analysis was by cold vapor technique atomic absorption spectrophotometry (CVTAAS) (Model PE 305, Perkin-Elmer Corporation, Ridgefield, Connecticut, USA). National Research Council (NRC; Institute of Marine Biosciences, Halifax, Nova Scotia, Canada) DOLT-2 reference material (dogfish liver) was used as a quality control (110% recovery). The detection limit with this method was 0.1 μg/g (wet weight). For total mercury analysis of brain samples, homogenized tissues were weighed and freeze dried, and their dry weights recorded. Samples were digested in 0.5 ml 70% nitric acid (J. T. Baker, Instra-analyzed) per 0.1 g of dry tissue at 70 C in dri-baths (Multi-Blok, Lab-Line Instruments, Inc., Melrose Park, Illinois, USA). After digesting for 2 to 4 hr, 98% sulfuric acid (J. T. Baker, Instra-analyzed) and 36.5 to 38% hydrochloric acid (J. T. Baker, Instra-analyzed) were added. Samples were digested for 2 to 4 hr, then left to cool

overnight. Volumes were adjusted to 10 ml with 2 mM potassium dichromate (J. T. Baker) in 3% hydrochloric acid. Samples were further adjusted to 20 ml final volume with 1.5% hydrochloric acid and 100 μl Octanol (Fisher Scientific) as an antifoaming agent. Analysis was by CVTAAS (Model PE 3030B AAS, Perkin-Elmer Corporation, Norwalk, Connecticut, USA). NRC DOLT-2 (dogfish liver) and DORM-1 and -2 (dogfish muscle) were used as quality controls (110–115% recovery). The detection limit with this method was 0.02 μg/g (wet weight). Both lead and mercury levels in kidneys were reported on a wet weight basis. Mercury levels in brain were reported on a dry weight basis but, for comparison with renal values, were converted to wet weight levels based on the moisture content of the original samples. For lead, we considered renal levels of 6 μg/g and higher to indicate poisoning (Wobeser, 1981).

Intestinal contents were washed with tap water, passed through a sieve with a 150 μm pore size and fixed in hot (63 C) acetic acid-ethanol-formalin for collection of helminth parasites. A 10% aliquot was examined in a grid-marked petri dish with a dissecting microscope and the numbers of helminths were counted. The entire sample was examined for birds with <300 helminths, whereas a 1% aliquot was counted for birds with very high numbers of parasites (>10,000). Representative samples of trematodes, cestodes and acanthocephalans were stained with Semichons acetic-carmine and mounted on slides with Canada Balsam for identification. Duplicate voucher specimens were deposited in the Invertebrate Collection of the Canadian Museum of Nature (Ottawa, Canada; accession numbers: 1997-0077 for *Cryptocotyle lingua*, 1997-0064 for *C. concavum*, 1997-0068 for *Apophallus brevis*, 1997-0079 for *Stephanoprora pseudoechinata*, 1997-0080 for the microphallids, 1997-0069 for the echinostome) and in the United States National Parasite Collection (Beltsville, Maryland, USA; accession numbers: 87052 for *C. lingua*, 87038 for *C. concavum*, 87042 for *A. brevis*, 87054 for *S. pseudoechinata*, 87055 for the microphallids, 87043 for the echinostome). Trematodes were identified with standard keys (McDonald, 1981; Schell, 1985; Skrjabin, 1964).

Statistical comparisons were made between birds in poor body condition and those in good body condition with regard to total body weight, renal levels of mercury, and total numbers of intestinal helminths. Body weights were compared, using a two-sample t-test. Because the data on mercury levels and numbers of intestinal helminths did not follow a normal distribution, the nonparametric two-sample Wilcoxon rank sum (Mann-Whitney) test was used (Daniel, 1983). Mercury levels in brains and kidneys of 10 birds also were compared, using the nonparametric Wilcoxon signed rank test (SYSTAT, 1996).

RESULTS

Eight of the 31 loons examined (four adult female, two adult male, two immature male) were in good body condition. In three of these loons, the history indicated drowning from entanglement in fishing nets; no aspirated foreign material was seen grossly or microscopically in the respiratory tract of any of them. One bird was heavily oiled but otherwise had no significant gross or microscopic lesion; therefore, the cause of its death in relation to the exposure to oil was not determined. Three birds had died of acute trauma of undetermined cause characterized by severe internal hemorrhage and bone fractures. One bird had been shot through its mandible and had subsequently developed acute necrotizing and fibrinous focal stomatitis, focal tracheitis and left caudal thoracic air sacculitis, the tracheitis and air sacculitis possibly having resulted from aspiration of necrotic material from the mouth; *Pseudomonas* sp. and *Corynebacterium* sp. were isolated from a swab of the affected portion of the trachea.

The other 23 loons were in poor body condition. Their mean total body weight was significantly lower ($P < 0.001$) than that of birds in good body condition (Table 1). Of these 23 birds six (four adult female, two adult male, all found on lakes) died from lead poisoning (see below). Five birds (three adult male, one adult female, one adult of undetermined sex) had severe chronic gross and microscopic lesions of respiratory mycosis compatible with aspergillosis. In all five birds, these lesions involved large areas of the air sacs and, to a lesser extent, the lungs and/or major airways. Five birds (one adult male, two adult female, one immature male, one immature female; all found in a marine environment) had severe oil contamination of their plumage. One adult female had a locally

TABLE 1. Body weights (kg), concentrations of total mercury in kidneys (μg/g, wet weight), and numbers of intestinal trematodes in common loons from the Maritime provinces of Canada, according to body condition.

	Poor body condition			Good body condition		
	Body weight	Renal mercury	Intestinal trematodes	Body weight	Renal mercury	Intestinal trematodes
Number	15	23	19	8	8	7
Mean	3.1[a]	18.80[b]	4,560[c]	4.8[a]	3.07[b]	744[c]
(SD)	(0.8)	(17.82)	(6,975)	(1.0)	(2.40)	(1,790)
Range	2.1–5.1	0.16–61.0	0–30,080	3.8–6.6	0.45–6.45	0–4,800

[a] Significant difference ($P = 0.001$).
[b] Significant difference ($P = 0.019$).
[c] Significant difference ($P = 0.045$).

extensive chronic necrotizing esophagitis; a single lead pellet was found on the outer surface of the affected portion of esophagus. The amount of necrotic material associated with this lesion was considered sufficient to have interfered with the passage of ingesta. The primary cause of the poor body condition and death in the remaining six birds (two adult male, three immature female, one immature male) was not determined. On microscopy, of the four immature birds in this group, one had a mild nonsuppurative encephalitis of unknown cause (the amount of mercury in the brain of this bird was 0.85 μg/g). Another had chronic lesions of renal coccidiosis, which consisted of marked distention of the lumens of most collecting ducts by coccidial oocysts and an infiltration of their walls by epithelioid macrophages, multinucleated giant cells, some lymphocytes, and fibroblasts.

Six loons in poor body condition had renal concentrations of lead compatible with poisoning ($\bar{x} \pm$ S.D. = 91.6 ± 49.8 μg/g; range = 15.5–167.0 μg/g) (Wobeser, 1981). The gizzards of four of these birds contained remnants of lead sinkers (a single swivel in two cases, a swivel and a split shot in a third case, and two split shots in the fourth case); all lead masses were ≤8 mm in diameter. Two of these gizzards also had chronic traumatic lesions caused by penetration of a fishing hook and associated with a focal peritonitis. Renal concentrations of lead in the other birds in poor body condition and in the eight birds in good body condition were below the detection limit, except for one emaciated adult whose kidneys contained 0.6 μg/g of lead. The average amount of mercury in the kidneys of loons of all ages in poor body condition was significantly higher ($P = 0.019$) than in loons of all ages in good body condition (Table 1). This difference was greater ($P = 0.005$) when only adult birds were considered. The number of immature birds was too small for statistical comparison in this age group. In 10 birds in poor body condition for which data were available (eight adult, two immature), concentrations of mercury were significantly higher ($P = 0.0069$) in the kidneys ($\bar{x} = 28.80 \pm 19.17$ μ/g, range = 0.16–61 μg/g) than in the brains ($\bar{x} = 1.13 \pm 0.77$ μg/g, range = 0.198–2.58 μg/g).

Intestinal helminths collected from 26 loons included 11 species of trematodes, three species of cestodes, and 1 species of an acanthocephalan. In addition, one species of nematode was found in the proventriculus and two species of trematodes in the esophagus. The most numerous and prevalent helminths observed were the trematodes *C. lingua, C. concavum, A. brevis, S. pseudoechinata,* microphallids, and what appeared to be a single species of an echinostome (Table 2). A more specific identification was not possible for the echinostome and the microphallids because of the poor quality of the specimens. The mean number of intestinal trematodes

TABLE 2. Abundance and prevalence of the major intestinal trematodes collected in common loons from the Maritime provinces of Canada.

Trematode	Abundance			Prevalence		
	All hosts	Hosts in poor body condition	Hosts in good body condition	All hosts (n = 26)	Hosts in poor body condition (n = 19)	Hosts in good body condition (n = 7)
Cryptocotyle spp. (*lingua, concavum*)	1,269[a] (0–9,200)[b]	1,505 (0–9,200)	628 (0–4,240)	50.0[c] (13/26)[d]	47.4 (9/19)	57.1 (4/7)
Apophallus brevis	1,303 (0–28,080)	1,784 (0–28,080)	0 (0)	26.9 (7/26)	36.8 (7/19)	0.0 (0/7)
Stephanoprora pseudoechinata	127 (0–2,080)	156 (0–2,080)	49 (0–200)	38.5 (10/26)	42.1 (8/19)	28.6 (2/7)
Echinostomes	128 (0–1,978)	163 (0–1,978)	33 (0–200)	34.6 (9/26)	36.8 (7/19)	28.6 (2/7)
Microphallids	384 (0–9,900)	522 (0–9,900)	10 (0–70)	11.5 (3/26)	10.5 (2/19)	14.3 (1/7)

[a] Mean number of trematodes collected.
[b] Range of number of trematodes collected.
[c] Prevalence of infection in the loons expressed as a percentage.
[d] Number of infected loons/total number of loons examined.

in birds of all ages in poor body condition was significantly higher (P = 0.045) than in birds of all ages in good body condition (Table 1). However, there was no statistically significant difference (P = 0.071) in trematode numbers between adults in poor body condition and those in good body condition. As with mercury levels, the number of immature birds was too small for statistical comparison.

DISCUSSION

Differentiating proximate from ultimate causes of chronic debilitation and death in free-ranging wild animals can be difficult, since only the end results of chronic diseases are observed, with no benefit of prior history on the affected animals or on the environmental conditions under which the disease processes started. Moreover, as this and other studies (Spalding et al., 1994) suggest, a substantial proportion of chronically ill wild birds may be affected by two or more concurrent disease problems at the time of death, further obscuring the sequence of events leading to emaciation and death. Lead poisoning, external oil contamination, and, in this study, the case of chronic esophagitis which resulted from a gunshot wound could be interpreted as ultimate causes of poor body condition and death. This intrepretation assumes that the majority of wild birds that ingest fishing sinkers or jigs, that are exposed to oil slicks at sea, or that are injured by hunters are otherwise healthy. Chronic respiratory aspergillosis is a well recognized disease in many avian species. Spores of *A. fumigatus* are widespread in the environment and are thought to be inhaled commonly by birds. Whereas these spores can be eliminated from the lungs and air sacs of healthy birds (Beckman et al., 1994), they may be allowed to germinate and cause disease in birds whose defense mechanisms are depressed because of stress caused by concurrent disease or primary starvation (Wobeser, 1981). Therefore, caution must be used before interpreting chronic respiratory aspergillosis as an ultimate cause of death.

The proportion of adult loons with lead poisoning in our region (26% of adults in poor and good body condition) is comparable to that in other regions of Canada (30%) (Scheuhammer and Norris, 1996); it is lower than in the New England states of the U.S.A. (52%) (Pokras and Chafel,

1992), but the latter study included only birds found on lakes, where the likelihood of ingestion of fishing lures is higher. All six birds with lead poisoning in our study were in poor body condition, in contrast to most birds observed by Pokras and Chafel (1992), which were in good body condition. The reason for this difference is not known.

Fish-eating birds are exposed primarily to the methylated form of mercury, the main toxicological effects of which are reproductive and, at higher doses, neurological (Scheuhammer, 1991). Experimentally, the neurological effects generally occur at concentrations of total mercury of ≥15 μg/g in brain and at least 30 μg/g in liver or kidneys (Scheuhammer, 1991). However, a predatory bird, which needs full coordination to feed adequately, could be functionally affected by lower burdens of mercury, before overt clinical signs occur. Conversely, some species may be better able than others to convert methylmercury into the less toxic inorganic form. For example, albatrosses and petrels can accumulate high tissue levels of total mercury without showing adverse effects (Kim et al., 1996). Similarly, most mercury in liver and kidneys of common loons with high total mercury levels is in the inorganic form, although most of that in their pectoral muscles is methylated (Scheuhammer et al., 1998). The strong association found in our study between poor body condition and high renal levels of mercury suggests a somatic redistribution of this metal associated with loss of body condition. Similar observations were made by Spitzer (1995) and Forrester et al. (1997) in common loons and by Spalding et al. (1994) in great white herons (*Ardea herodias occidentalis*). Gradual muscle atrophy concurrent with emaciation/starvation may release back into the blood circulation a substantial amount of mercury stored in the muscular compartment, for redistribution to other more vital organs such as the kidneys and nervous system, or perhaps sufficient to adversely affect the animal's immune system (Bernier et al., 1995). In other words, a body burden of mercury which, in a healthy bird, would have little or no physiological effect might become significant following chronic debilitation. Nevertheless, the role played by sublethal mercury levels in initiating the decline into emaciation in these loons remains unclear. Cerebral and renal mercury levels available for 10 birds in poor body condition in this study did not suggest a preferential redistribution of this metal to the brain.

There was a statistically significant association between poor body condition and high numbers of intestinal trematodes in our loons, an observation also made by Forrester et al. (1997). We could not determine whether the high levels of intestinal parasitism were a cause or a consequence of the poor body condition or were an unrelated finding. The pathophysiological effects of these parasites in birds are poorly understood. The life cycles are known for *C. lingua*, *C. concavum* and *A. brevis*, and all involve fish as a second intermediate host (Margolis and Arthur, 1979; Sinclair, 1972; Stunkard and Willey, 1929; Wooten, 1957). The apparent paradox of birds showing evidence of both successful prey capture and poor body condition could be explained if high levels of parasitic exposure could occur through ingestion of relatively few fish. Reported prevalence and intensity of infection by *C. lingua* in cunner (*Tautogolabrus adspersus*) in Newfoundland (Canada) were 54 to 100% ($\bar{x}$ = 90%) and 1 to 14,030 ($\bar{x}$ = 965) metacercariae per fish, respectively (Sekhar and Threlfall, 1970). A prevalence of 100% and mean infection intensities of 25 to 432 metacercariae per fish were reported for *A. brevis* in yellow perch from various lakes in Ontario (Canada) (Sinclair, 1972). If similar levels of infection occur in fish that serve as the food source for common loons in the Maritime region of Canada, birds feeding on these fish could acquire the numbers of parasites observed in this study from relatively few prey. The

precise number of fish and the time frame needed to acquire these levels of parasitism are unknown for this region at this time. The presence of these same parasites in the intestines of loons in good body condition (except for *A. brevis*) indicates that they are part of the normal helminth fauna of this species in this region. Therefore, the very large numbers observed in some of the birds in poor body condition may not have resulted from the consumption of an excessive number of unusual prey species, as proposed by Forrester et al. (1997) in their investigation of loon mortality along coastal waters of Florida (USA). Instead, poor body condition may have depressed the immune function of these birds (Chandra, 1992), thus allowing maturation of an abnormally high proportion of the metacercariae encysted within prey items.

The results of this and other studies, identifying multiple concurrent disease problems in individual birds in poor body condition, suggest that gradual debilitation of a wild animal from a specific disease may predispose it to several other potentially debilitating agents existing within its environment. Therefore, determination of the clinical effects of various pathological processes under controlled laboratory conditions may underestimate the capacity of these processes to cause morbidity and death in the wild, when compounded by other diseases.

ACKNOWLEDGMENTS

We thank the many conservation officers from the Nova Scotia Department of Natural Resources, the New Brunswick Department of Natural Resources, and the Prince Edward Island Department of Fisheries and Environment, as well as Z. Lucas, who submitted common loons for necropsy over the years. R. Maloney did the quantitative analysis of the parasitology samples. E. Holmes performed the lead analyses and summarized the various methods of toxicological analysis. P. Longabardi did the mercury analyses on renal samples. D. Bond did the mercury analyses on brain samples. G. Clements prepared the illustration. I. Dohoo helped with the statistical analyses.

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Received for publication 13 March 1997.