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WINTER MORTALITY OF COMMON LOONS IN FLORIDA COASTAL WATERS

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ABSTRACT: Diagnostic findings are presented for 434 common loons (*Gavia immer*) found sick or dead on Florida beaches from 1970 through 1994, primarily during the months of December to April. The most commonly recognized problem was an emaciation syndrome (66%), followed by oiling (18%), aspergillosis (7%), trauma (5%) and miscellaneous disease entities (1%). The cause-of-death for 3% of the birds was not determined. Many of the carcasses examined (n = 173) were obtained during an epizootic which occurred from January to March of 1983 in which more than 13,000 loons were estimated to have died. An emaciation syndrome, characterized by severe atrophy of pectoral muscles, loss of body fat and hemorrhagic enteritis, was the primary finding in this epizootic. It was postulated to have a complex etiologic basis involving synergistic effects and energy costs of migration, molting and replacement of flight feathers, food resource changes, salt-loading, intestinal parasitism, environmental contaminants, and inclement weather.

Key words: Aspergillosis, common loons, emaciation, environmental contaminants, *Gavia immer*, oil spills, parasites, stress, trauma, winter mortality.

INTRODUCTION

Common loons (*Gavia immer*) overwinter in large numbers in the Gulf and Atlantic coastal waters of Florida, except for the southern part of the state and the Florida Keys where they occur only occasionally (Stevenson and Anderson, 1994). At least some of these loons are known to nest in Wisconsin, Minnesota, and Michigan during the summer months (Bird Banding Laboratory, 1995). Each winter, dead loons are found on both Atlantic and Gulf beaches of Florida, occasionally numbering in the hundreds or even thousands (Stevenson, 1970, 1971, 1972, 1974; Ogden, 1987; Simons, 1985; Alexander, 1991). Several individual mortality events have been linked to oil spills (Longstreet, 1953; Stevenson, 1970; White et al., 1976) and inclement weather (Simons, 1985).

In the winter of 1983 a large mortality event involving common loons occurred on the Gulf coast of Florida, particularly in the area from Pensacola (30°24′N, 87°13′W) to Naples (26°8′N, 81°47′W) (Hamel, 1983; Hoffman, 1983; Imhof, 1983; Kale, 1983). Estimates of the size of this epizootic varied from 1,000 (Imhof, 1983) to as high as 10,000 loons (Alexander, 1991). The latter author speculated that the die-off was due to high energy costs of feather replacement coincident with mercury contamination. Spitzer (1995) reviewed the available data and suggested that the 1983 die-off might have been caused by the coincident occurrence of wing feather molting and shortages of food.

Herein, we describe field and laboratory investigations which have been conducted over the past 25 yr in relation to this an-

nual loon mortality, with particular emphasis on the 1983 epizootic. Also, we suggest a complex multifactorial etiology to explain the severity of the epizootic in 1983.

MATERIALS AND METHODS

Necropsy and laboratory records of 434 common loons that were found moribund or dead on Florida beaches from 1970 through 1994 were reviewed. These records originated from three sources: The Department of Pathobiology (University of Florida, Gainesville, Florida, USA) (n = 247), The National Wildlife Health Center (Madison, Wisconsin, USA) (n = 97), and The Southeastern Cooperative Wildlife Disease Study (University of Georgia, Athens, Georgia, USA) (n = 90). Diagnostic findings on some of these loons have been presented elsewhere in a different context (White et al., 1976; Franson and Cliplef, 1993). Diagnostic procedures varied among cases since the purpose of the necropsies was to determine the cause-of-death. For each case the data reviewed included sex, total body weights, location, date, case history, and the primary diagnostic findings.

Carcasses came from the following general areas in Florida: the Gulf Coast (n = 257), the Atlantic Coast (n = 161), inland lakes in north central Florida (Alachua, Clay, and Marion counties) (n = 5), the Florida Keys (n = 1), and localities unknown (n = 10). The majority of these (91%) were obtained during December to April; the remainder were collected between May and August or in October and November. An additional 25 normal loons were collected by shooting and were used for comparative purposes. We defined "normal loons" as birds that were active, behaved as healthy birds, had ample amounts of body fat, robust pectoral muscles, and weighed more than 2.8 kg. These included 23 loons from the Gulf Coast (in the vicinity of Dog Island located off the coast of Franklin County; Fig. 1) in March and April, 1984, one from the Atlantic Coast (Palm Beach County in January of 1972), and one from an inland lake (Alachua County in May of 1973). Ages were not determined. The gender was determined for 327 of the loons; 157 were males and 170 were females. Except for the normal loons collected for comparative purposes and a small number of moribund loons, carcasses were in various states of decomposition and a complete evaluation was not possible on every bird. Twenty-one birds were received alive, the remainder were dead. Twenty-three loons were examined shortly after death; the remainder were frozen for later processing.

A subset of the main database was treated in more detail. These included the 286 loons that had no other primary diagnosis except emaciation. In addition, other studies were performed in order to understand the nature and cause of the emaciation condition including field and laboratory investigations into the 1983 epizootic. The procedures and materials utilized for these studies on emaciated loons follow.

Standard gross examinations were performed on all carcasses and where feasible, samples were obtained for histopathologic, microbiologic, parasitologic, and toxicologic studies. Blood samples were obtained from some emaciated birds and serum was separated and collected for use in testing for the presence of toxins of *Clostridium botulinum* Type C (n = 15) and Type E (n = 11) by the mouse-protection technique as described by Quortrup and Sudheimer (1943). Thin blood smears were made from 27 emaciated loons, air-dried, fixed in absolute methanol, stained by standard Giemsa technique, and examined microscopically in order to detect the presence of blood parasites.

Tissue samples from 17 emaciated birds were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 5 or 6 μm, and stained with hematoxylin and eosin (H & E) for examination by light microscopy. The tissues examined included lung (n = 13), air sacs (n = 1), liver (n = 15), kidney (n = 15), adrenal gland (n = 3), spleen (n = 4), heart (n = 9), bone marrow (n = 1), brain (n = 14), peripheral nerves (n = 2), ovary (n = 2), testis (n = 1), thyroid gland (n = 2), parathyroid gland (n = 1), esophagus (n = 1), proventriculus (n = 5), ventriculus (n = 4), pancreas (n = 3), intestines (n = 16), cloaca (n = 1), and salt gland (n = 1). When warranted, based on microscopic examinations of H & E stained tissues, Brown-Brenn bacterial stain and periodic acid-Schiff staining technique for fungi were utilized.

Aerobic and anaerobic cultures on blood agar and MacConkey agar were made from liver (n = 57), spleen (n = 5), intestinal contents (n = 65), heart blood (n = 5), pericardial fluid (n = 1), lungs (n = 10), kidney (n = 3), brain (n = 2), and abdominal fluid (n = 1) from 76 emaciated loons. Techniques for the isolation and identification of fungi and bacteria from tissues and intestinal contents have been described elsewhere (White et al., 1976; Franson and Pearson, 1995).

Tissues from eight emaciated loons (intestines, n = 6; spleen, n = 5; liver, n = 2 lung, n = 3; trachea, n = 1) were used for virus iso-

lation attempts in cell culture and embryonated chicken eggs as described by Docherty and Slota (1988) and Senne (1989).

Parasitologic examinations were conducted on 52 emaciated and 23 normal loons following the techniques given by Forrester et al. (1974). Total counts of intestinal trematodes were made by use of an aliquot system.

Samples of kidney, liver, and/or brain from 13 emaciated loons were wrapped separately in aluminum foil and then sealed in plastic bags to minimize dehydration and stored at −20 C until analyzed for environmental contaminants. The methods described by Haseltine et al. (1983) were used to detect residues in brain samples from seven loons for the following organochlorines: DDE, DDD, DDT, dieldrin, heptachlor epoxide, oxychlordane, *cis*-Chlordane, *trans*-Nonachlor, *cis*-Nonachlor, endrin, toxaphene, PCBs, several isomers of tetrachloro, pentachloro, hexachloro, and heptachloro diphenyl ethers, and several isomers of trichloro, tetrachloro, and pentachloro terphenyl. Lower limits of detection were 0.10 ppm for pesticides and 0.50 ppm for PCBs. Brain samples from six additional loons were tested using the methods of McMahon and Sawyer (1986) to detect residues of aldrin, BHC, carbophenolthion, chlordane, DDD, DDE, DDT, diazinon, dieldrin, endrin, ethion, heptachlor, heptachlor epoxide, lindane, malathion, methoxychlor, methyl parathion, mirex, PCBs, parathion, rabon, and toxaphene. Lower limits of detection were 0.01 ppm for aldrin, BHC, DDD, DDE, DDT, heptachlor, and lindane; 0.02 ppm for dieldrin, endrin, and heptachlor epoxide; 0.05 ppm for methoxychlor and parathion; and 0.10 ppm for carbophenolthion, chlordane, diazinon, ethion, malathion, methyl parathion, mirex, rabon, and toxaphene; and 0.20 ppm for PCBs. Tests for residues of mercury were conducted according to Halbrook et al. (1994) and Haseltine et al. (1983). Tests for selenium, cadmium, and lead followed those described by Haseltine et al. (1983) and Warren et al. (1990). Lower limits of detection were 0.02 or 0.20 ppm for mercury, 0.10 ppm for selenium, 0.03 ppm for cadmium, and 0.25 ppm for lead. Results are expressed on a wet weight basis.

Spleen extracts and intestinal contents from three emaciated loons from the 1983 epizootic were prepared for transmission experiments as described by Forrester et al. (1977). Four 1 to 2-wk-old Pekin ducklings and four 1 to 2-wk-old broad-breasted white domestic turkey poults each received 0.15 ml of spleen extract intramuscularly. Six other ducklings and six other poults received 0.5 to 1.5 ml of unfiltered intestinal contents and two ducklings and two poults each received 0.5 to 0.7 ml of filtered intestinal contents, all *per os*. All recipients were monitored three times each day for 7 days at which time one duckling from each of the experimental groups was killed and examined grossly and microscopically at necropsy. The remainder of the ducklings and all of the poults were killed and examined on day 18 post-exposure.

During 1983, an effort was made to determine the duration, geographic extent, and severity of loon mortality along the Gulf coast. Information on the duration of the event was obtained primarily by observations of loons in the vicinity of Dog Island from November 1982 through March 1983. An epizootic curve was constructed by partitioning the number of sick and dead loons per 2-wk interval for the period of 6 December 1982 through 13 March 1983. Two-wk intervals were used in order to insure adequate sample size. Data on the geographic extent and severity of the epizootic were compiled by surveying easily accessible beaches on foot or by the use of all-terrain vehicles from 18 to 25 March 1983. Records were kept on the linear distance of beach examined and the number of dead or sick loons observed. A total of 177.9 km of shoreline at 44 locations from Fairhope (Mobile Bay), Alabama to Boca Grande, Florida was searched by personnel with the Florida Game and Fresh Water Fish Commission, National Wildlife Health Center, and the Southeastern Cooperative Wildlife Disease Study. In addition, a telephone survey of state wildlife agencies of Louisiana, Mississippi, Alabama, Georgia, South Carolina, North Carolina, Virginia, and Maryland was conducted to ascertain whether loon mortality was occurring concurrently with the Gulf coast event.

The magnitude of mortality (M) for the 1983 epizootic was estimated as follows: $M = \Sigma(X_1 \cdot Y_1 + X_2 \cdot Y_2 + \ldots X_i \cdot Y_i)/0.49$; where X is the mean number of dead loons observed per km of shoreline for a given county, Y is the total number of km of shoreline for the county, and 0.49 is a factor accounting for the variable of carcass persistence.

The denominator was used to expand the estimated mortality beyond the product of loons per km multiplied by km of shoreline. This was done because carcasses of loons dying early during the epizootic should have decomposed and would not have been detectable later when the Gulf coast shoreline searches were conducted. An arbitrary carcass persistence time of 28 days was selected based on field observations of minimal carcass scavenging and our consensus that many carcasses were at least a few wk old. The denominator (0.49) represents the proportion of the Dog Island epizootic

TABLE 1. Primary diagnostic findings for 434 common loons from Florida, 1970–1994.

Primary diagnostic finding	Number of loons from each area					Total (%)
	Gulf Coast	Atlantic Coast	Inland Lakes[a]	Keys[b]	Unknown	
Emaciation syndrome	219	58	2	0	7	286 (66)
Oiling	5	71	0	0	0	76 (18)
Aspergillosis	14	13	1	0	3	31 (7)
Trauma	9	10	1	1	0	21 (5)
Miscellaneous	2	2	1	0	0	5 (1)
Undetermined	8	7	0	0	0	15 (3)
Total	257	161	5	1	10	434

[a] Includes freshwater lakes in Alachua, Clay, and Marion counties in north-central Florida.
[b] Big Pine Key in the Florida Keys (Monroe County).

curve for the 28 day period (14 February through 13 March 1983) preceding the Gulf shoreline searches. An additional variable not incorporated in the estimation procedure was expansion for less than 100% efficiency in carcass detection during searches; this was omitted because we wanted to be conservative in the mortality estimation and chose to not include a second speculative expansion factor.

Terminology (prevalence, intensity, and abundance) follows Margolis et al. (1982). The GLM procedure of the SAS system (SAS Institute, Inc., 1988) was used for all statistical computations. Analysis of variance (ANOVA) for unbalanced data was used to assess effects of the three factors of (1) mortality category, (2) location, and (3) gender on loon body weights and also on residues of mercury and selenium in livers. Interactions between these factors also were tested in the ANOVA. Means for factors with significantly different levels according to ANOVA were further compared using the least significance difference (LSD) criterion. Mercury and selenium residues were transformed through log 10 to normalized data and to linearize the relationship between mercury and selenium. Linear correlation was used to quantify strength of relationship between mercury and selenium. Statistical significance was accepted at $P \leq 0.05$.

RESULTS

The primary diagnoses for 434 common loons found moribund or dead in Florida from 1970 through 1994 are listed in Table 1. The most common finding was emaciation which occurred in 66% of the loons; it was characterized by muscle atrophy, abnormally low body weight, and depleted body fat. This condition was especially prevalent in birds overwintering in the northern Gulf Coast of Florida, but also occurred in loons from the Atlantic Coast and in a few inland freshwater lakes in northcentral Florida. Oiling was the second most important mortality factor and affected loons from both coasts. The majority ($n = 69$) of the oiled loons was found on beaches between St. Augustine and Flagler Beach (St. Johns and Flagler counties) in northeastern Florida, the consequence of an offshore oil spill from an unidentified source in January of 1974. Thirty-one loons died of pulmonary aspergillosis, whereas trauma, probably due to predation by sharks or other fish, was the cause-of-death of 20 loons. One loon died of gunshot wounds. Single cases of tuberculosis, intestinal coccidiosis, cardiac failure associated with bacterial infection, encephalitis, and pneumonia were recorded also.

The body weights (in kg) of loons in various mortality categories were compared with normal loons. Emaciated loons and loons with aspergillosis had the lowest weights ($\bar{x} \pm$ SE) of 2.00 ± 0.03 ($n = 155$) and 2.02 ± 0.09 ($n = 26$), respectively and did not differ significantly from each other. Weights of loons that died due to trauma (2.35 ± 0.19, $n = 18$) or oiling (2.46 ± 0.06, $n = 74$) were intermediate and were not significantly different from each other, but were heavier than emaciated birds and birds with aspergillosis. Normal loons (4.19 ± 0.20, $n = 12$) were significantly ($P = 0.05$) heavier than all other loons. Males

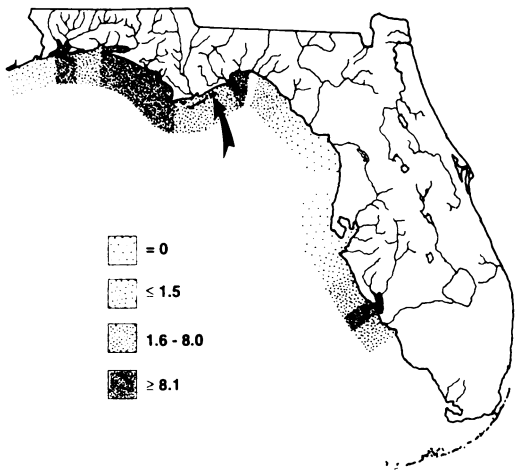

FIGURE 1. Distribution and concentrations (loons/km) of mortality of common loons on the Gulf Coast of Florida as determined by shoreline surveys from March 18 to 25, 1983. Arrow indicates location of Dog Island.

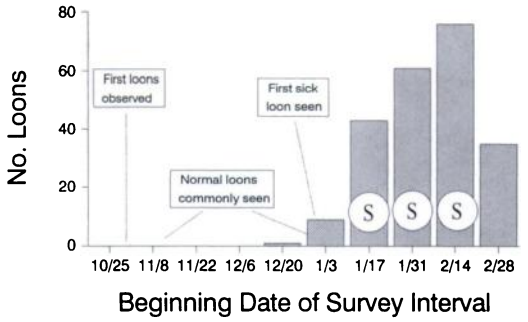

FIGURE 2. Chronology of observations and events including numbers of sick and dead common loons observed per 14-day interval from November 6, 1982 through March 13, 1983 in the vicinity of Dog Island off the northern Gulf Coast of Florida. Shaded bars indicate the numbers of dead loons found during a 2-wk time interval. An "S" within a circle on the bar indicates that there were records of storms during that time interval.

were significantly heavier (2.37 ± 0.05, n = 107) than females (2.14 ± 0.05, n = 106) (P = 0.0001). There were no significant differences between the weights of loons from the Atlantic Coast and the Gulf Coast and there were no interactions between gender and locality, gender and mortality category, locality and mortality category, or between gender, locality, and mortality category.

As stated previously in the Materials and Methods section we conducted further studies on the 286 emaciated loons which had no other obvious complicating disease factors. During the epizootic in 1983, 173 loons were studied; an additional 113 birds were obtained at different times throughout the 25 yr period. Along with field observations made during 1982–1983, these carcasses collectively formed the basis for the following results.

The ensuing field observations relate to the epizootic in 1983. The first record of common loons arriving in the vicinity of Dog Island was an observation of 25 to 50 loons on 6 November 1982. By 26 November, groups of apparently normal loons numbering 50 to 75 were observed commonly. The first observation of a sick loon was on 1 January 1983 when a single bird was observed in a tidal creek for 5 consecutive days. Such behavior is considered atypical. The first dead loon was noted on January 4, and from that date through 13 March, 225 dead and 28 sick loons were found on Dog Island. Grouping of the number of dead loons found on Dog Island by 14-day intervals produced a characteristic epizootic curve (Fig. 2). In total, 331 dead loons were found during searches of 177.9 km of shoreline at 44 locations along the Florida and extreme eastern Gulf coast of Alabama between March 18 and 25, 1983. The number of dead loons per km of shoreline averaged 1.9 and ranged from 0 for some areas of Baldwin County, Alabama and for Levy, Citrus, Pasco, and Pinellas counties in peninsular Florida to a maximum of 14 for a small section of St. Joe Beach in Gulf County, Florida. There was a major concentration of carcasses along the beaches in the central panhandle counties and fewer in the vicinity of Gasparilla Sound in Sarasota and Charlotte counties (Fig. 1). Very few (<20) carcasses of other species of birds were found during these searches and included several brown pelicans (*Pelecanus occidentalis*), double-crested cormorants (*Phalacocorax auritus*), unidentified gulls

and terns, and one great egret (*Casmerodius albus*). These carcasses were not examined at necropsy. Personnel of wildlife agencies in other southeastern states did not report loon mortality of a similar magnitude to that observed along the Florida Gulf coast. We calculated an estimated mortality of 13,610 loons for 1983 along the Florida Gulf coast.

Field necropsies were performed on 117 carcasses during the winter of 1983. Six of these were fresh and the others were in various stages of decomposition. The most common observations at necropsy (n = 114) were severe atrophy of muscles (especially pectoral muscles) and loss of body fat deposits. Seventy-three of these carcasses were in good enough condition so that the contents of their ventriculi could be evaluated. Food items were absent in 60 loons, seven contained pebbles, three had pieces of crab shells, two had snail shells, and one had fish scales. Blood or hemoglobin-stained fluid was present in the intestines of 59 of 62 loons for which this could be evaluated. This ranged from small dark flecks of clotted blood to extensive black tarry feces. Ninety-seven of the loons were in various stages of molt and 20 were fully feathered.

In addition to the carcasses mentioned above, 23 other comparatively fresh carcasses from the 1983 mortalities were examined in the laboratory and were found to be emaciated and anemic (pale tissues and watery blood). Most birds had molted and were in the process of replacing their primary and secondary wing feathers, which was considered normal for common loons at this time of year. Evidence of blood or dark tarry feces were found in the intestinal tract of each bird. Livers were generally pale and slightly swollen and spleens were shrunken. The kidneys frequently contained deposits of urates. Hearts were flaccid and contained small amounts of unclotted blood. Excess pericardial fluid and serous atrophy of coronary fat were observed consistently.

Tissues from 12 loons that died in 1983 were examined microscopically. Granulomatous inflammatory lesions in kidneys of 11 of 12 birds examined was the most consistent finding. The lesions were associated with the presence of either trematodes (*Renicola pollaris* and *Tanaisia fedtschenkoi*) or coccidia (*Eimeria gaviae*), or both. Various degrees of inflammation and necrosis of the collecting ducts were observed primarily in association with the coccidia or eggs of the trematodes. Little response was noted in association with the adult trematodes. The intestines of nine of the 12 loons had increased lymphocytic infiltrates in the lamina propria along with flattened or fused intestinal villi, and occasional dilated necrotic crypts associated with the presence of intestinal trematodes (primarily microphallids) and in one case cestodes. The trematodes did not appear to contain ingested blood even though evidence of intestinal bleeding was noted on gross examination of all of the loons. Hemosiderosis was present in livers and spleens of most birds. Subacute cholangitis was associated with trematodes (*Amphimerus arcticus*) in the liver tissue of at least two birds. Focal lymphocytic and histiocytic infiltration centered around bile ductules was noted and diffuse hepatocellular necrosis was observed in the livers of four birds. Bone marrow depletion was noted in the single bone marrow slide examined. Demyelinization or vacuolization of brain tissue were present in several loons. No significant lesions were found in other tissues examined. Five emaciated loon carcasses obtained in 1987 from a small die-off in the same area as the 1983 event had similar lesions as reported above. However, all five also had lesions in the brains consisting of diffuse leucoencephalopathy characterized by severe spongiosis and vacuolation of the white matter and to a lesser degree grey matter. Increased cellularity due to an inflammatory response was not documented in these birds.

No consistent bacterial or fungal isolates were obtained; however, the following

were found: *Aeromonas hydrophila* (from the liver of one loon), *Acinetobacter calcoaceticus* (from the lung of one loon), *Campylobacter* sp. (from the intestines of two loons and the liver of one), *Clostridium perfringens* and *Clostridium* sp. (from the intestines of one loon), *Escherichia coli* (from the intestines of 22 loons, the livers of four loons, and the lung, heart, pericardial fluid, and kidney of one loon each), *Enterobacter* sp. (from the intestines of two loons and the liver of one), *Pasteurella multocida* (from the intestines of one loon and the brain of another), *Peptostreptococcus* sp. (from the intestines of one loon), *Proteus vulgaris* (from the intestines of one loon), *Proteus* sp. (from the intestines of three loons, the liver of one loon, and the abdominal fluids of another), *Pseudomonas putrifaciens* (from the intestines of two loons), *Pseudomonas* sp. (from the lungs of one loon and the liver of another), *Salmonella agona* (from the intestines of three loons), *Salmonella blockley* (from the intestines of one loon), *Salmonella typhimurium* (from the intestines of one loon), *Serratia marcescens* (from the liver and kidney of one loon), *Streptococcus* sp. (from the abdominal fluid of one loon), *Vibrio alginolyticus* (from the intestines of two loons and the lungs of two loons), *Vibrio damsela* (from the liver and spleen of one loon), *Vibrio parahemolyticus* (from the intestines and lungs of one loon), *Weeksella virosa* (from the intestines of one loon), and *Aspergillus* sp. (from the air sacs and lungs of six loons). No viruses were isolated.

Blood smears were negative for protozoans. A large number of parasitic helminths (at least 49 species) were found in these loons, but because enteritis was present in many of them, the parasites considered to be of most significance were those that occurred in the small intestine. These were represented by 19 species of trematodes, 5 cestodes, 1 nematode, and 2 acanthocephalans. The most numerous of these were microphallid trematodes (three *Microphallus* spp. and two *Maritrema* spp.) which numbered in the tens of thousands in some loons. One undescribed *Microphallus* sp. comprised about 99% of the total numbers of microphallids. Trematodes were more prevalent and occurred in higher intensities in emaciated loons which died during the epizootic of 1983 compared to normal loons collected from the same area during 1984, the subsequent non-epizootic year. In 1983, 22 of 23 (96%) emaciated loons were infected with intestinal trematodes with a mean intensity of 7,665 (range = 1 to 82,625). In 1984, only 11 of 24 (46%) normal loons were infected with a mean intensity of 23 (range = 1 to 85). Abundances of 7,358 and 11 were recorded for loons in 1983 and 1984, respectively, a greater than 600-fold difference.

Serum samples from 15 emaciated loons were negative for toxins of *Clostridium botulinum* Type C. Eleven of these same loons were tested also for *C. botulinum* Type E and were negative.

Residues of DDE and PCBs (Aroclor 1260) were found in all brain samples of 13 loons that died in the epizootic of 1983. These varied from 3 to 20 (geometric mean = 7.8) and 11 to 51 (geometric mean = 23) ppm (wet weight), respectively. Low concentrations (<1 ppm) of dieldrin, heptachlor epoxide, oxychlordane, *trans*-Nonachlor, and *cis*-Chlordane were detected in 7, 6, 6, 6, and 1 loons respectively. Residues of other organochlorine compounds were not detected.

Kidney samples from 13 loons from the Gulf Coast during the 1983 epizootic all contained residues of cadmium (geometric $\bar{x}$ = 5.8; range = 3.1 to 12 ppm wet weight). Six of the 13 liver samples (46%) had detectable residues of lead ranging from 3.2 to 7.5 ppm, wet weight (geometric $\bar{x}$ = 4.2).

Residues of mercury and selenium in livers of 13 loons ranged from 7.9 to 90 ppm, wet weight (geometric $\bar{x}$ = 22) and 5.2 to 12 ppm, wet weight (geometric $\bar{x}$ = 10), respectively. These values for mercury and selenium are compared in Table 2

TABLE 2. Residues (ppm, wet wt.) of mercury and selenium in liver samples from common loons in Florida, 1973–1994.

Coast	Condition	Dates	Mercury			Selenium		
			n	Mean[a]	Range	*n*	Mean[a]	Range
Atlantic	Emaciated	1987	9	2.3	0.15–9.7	0	—	—
		1990–1991	2	1.4	1.3–1.4	0	—	—
		1993	2	4.8	4.1–5.6	0	—	—
		1994	5	4.3	1.8–17	0	—	—
	Oiled	1974	6	5.2	2.3–9.9	6	7.3	4.0–13
Gulf	Emaciated	1973–1974	5	17	9.2–29	5	12	8.5–29
		1974–1975	6	13	4.5–40	7	6.8	3.5–16
		1983	13	22	7.9–90	6	10	5.2–12
		1984	1	7.9	—	1	8.7	—
		1991	8	7.7	3.9–15	0	—	—
	Normal[b]	1984	23	4.5	1.1–56	23	5.8	2.3–29

[a] Geometric mean.
[b] Collected by shooting.

with residues from emaciated loons found in other years and localities, oiled loons, and normal loons. Residues of mercury ($P = 0.30$) and selenium ($P = 0.32$) in livers of male and female loons were not significantly different. Residues of mercury were significantly higher ($P = 0.0001$) in loons from the Gulf Coast than the Atlantic Coast; selenium could not be compared because no emaciated birds from the Atlantic Coast were tested for that metal. Residues of both mercury ($P = 0.0001$) and selenium ($P = 0.004$) were higher in emaciated loons than in normal birds. There was a linear correlation between residues of mercury and selenium ($r = 0.77$).

None of the ducklings or poults that had received spleen extracts or intestinal contents from loons exhibited abnormal behavior or signs of sickness during the period of observation. At necropsy all were normal grossly and histologically.

DISCUSSION

We conclude from our data and those from previous studies (Stevenson, 1970, 1971, 1972, 1974, 1977; Simons, 1985; Alexander, 1991) that episodes of loon mortality have occurred repeatedly on the wintering grounds in Florida since at least the early 1970's. In addition, we agree with Simons (1985), Alexander (1991), Franson and Cliplef (1993), and Spitzer (1995) that emaciation is the most common abnormal finding for loons found dead on their wintering grounds.

The magnitude of such losses either have not been estimated (Stevenson, 1970, 1971, 1972, 1974, 1977; Simons, 1985) or when estimates were made (Alexander, 1991; Franson and Cliplef, 1993), the parameters for those estimates were not well defined. Because we had more comprehensive information (e.g., duration, distribution, and carcass density) than previous studies, we were able to calculate a crude estimate of the magnitude of mortality for 1983. This calculation is based on three assumptions: (1) the epizootic curve constructed for mortality in the vicinity of Dog Island (Fig. 1) is representative of mortality within the Gulf region during that time interval, (2) the shoreline survey reasonably represents the relative magnitude of mortality in different regions of the Gulf coast, and (3) the mortality detected during the shoreline survey represents only a portion of that depicted within the epizootic curve for Dog Island. General support for the first assumption can be found in a temporally similar epizootic curve constructed for three sites in the northern Florida gulf over the period of

1983–88 (Alexander, 1991). With regard to the second assumption, we believe that sampling 44 locations covering 177.9 km of shoreline was adequate because the sampling was sensitive enough to detect differences in the spatial concentration of mortality (Fig. 1). Otherwise, mean carcass densities would be expected to have had a random geographic pattern. For the third assumption, we set the portion of the epizootic curve represented equal to the last 28 days in an effort to lessen the probability of an overestimate. The third assumption is the most troublesome because it implies specific knowledge of carcass persistence which is lacking. However, if our selection of the final 28-day portion of the epizootic curve was an insufficient time period for carcasses to disappear, thus producing an inflated estimate, it should be offset by the fact that our searches certainly did not detect all mortality.

Although the accuracy of our estimated mortality of 13,600 loons for 1983 along the Florida Gulf coast is debatable, it does at least demonstrate that loon mortality was considerable in the winter and spring of 1983 compared to other years. We believe that winter mortality is due predominately to an emaciation syndrome that is an important, recurring problem among common loons and possibly may be the major mortality factor for this species.

Three other data-sets on mortality of common loons are available for comparison with our observations in Florida, although it is important to remember that our data reflect winter mortality in contrast to other studies which resulted in information from loons on their breeding grounds in northern United States and Canada. One report contains information on 204 loons examined in Ontario (Canada) between 1971 and 1979 (Frank et al., 1983), one deals with 95 loons found dead in Minnesota (USA) between 1984 and 1990 (Ensor et al., 1992), and one is a nation-wide survey of 109 birds from 16 states in the USA. (excluding Florida and Minnesota) from 1976 to 1991 (Franson and Cliplef, 1993). An emaciation syndrome and trauma were found to be the leading causes of mortality in all three studies. The prevalence of emaciation was higher in our Florida sample (66%) than in the Ontario, Minnesota, and nationwide studies (12, 20, and 26%, respectively), whereas trauma was lower (5%) in Florida birds compared to the other studies (85, 39 and 17%, respectively).

Oiling was not reported in the Ontario and Minnesota birds (Frank et al., 1983; Ensor et al., 1992) and was identified in only three birds by Franson and Cliplef (1993), but accounted for 18% of the morbidity and mortality of loons wintering in Florida. Most (91%) of our sample of oiled loons came from a single incident that occurred along the Atlantic coastline of northeastern Florida during January of 1974 in which several hundred birds were involved (Stevenson, 1974; White et al., 1976). There have been a number of other reports of loons dying in Florida coastal waters and beaches due to oiling. The earliest record was found in Howell (1932, p. 73) who stated that "dozens were washed up on the beach near Daytona (Florida) during the winter and spring of 1925, their feathers clogged with oil." Longstreet (1953) observed oil-soaked loons on beaches in Volusia County (Florida) during the early 1940's. The largest reported oil spill involving common loons was in the winter of 1970 in Tampa Bay when "many hundreds" were affected (Stevenson, 1970). Lower than normal numbers of loons were observed in Tampa Bay in subsequent winters and this reduction was attributed to the oil-spill (Clapp et al., 1982). There is a higher probability that loons in Florida coastal waters will encounter oil than in freshwater habitats in northern states, mainly because of the activities of offshore oil production and numerous ships transporting oil and oil products in the marine environment. Clapp et al. (1982, p. 63) stated that common loons "are among the birds most vulnerable to oiling, which may cause considerable local

mortality." They stated that oiled loons do not often show up in large numbers on beached-bird surveys and suggested three reasons for this. These included: (1) loon populations are smaller and tend to be more dispersed than other seabirds, (2) loons may not seek shore as readily when oiled and are more likely to die in the water rather than on the beach, and (3) loons are less buoyant compared to other seabirds and sink more readily after dying. Thus, if the assertions of Clapp et al. (1982) are correct, it is possible that we may have underestimated the extent of loon mortality caused by oiling.

The prevalence of aspergillosis was the same in Florida loons (7%) as reported by Ensor et al. (1992) for Minnesota birds, but was lower (2%) in Ontario birds (Frank et al., 1983). This fungal disease has been observed most commonly as a sporadic infection of individual waterfowl, but also it is known to cause epizootics (Wobeser, 1981). It has been reported previously in common loons from Florida (Hartman, 1946; White et al., 1976) and has been observed in both oiled and non-oiled birds. Aspergillosis in waterfowl has been recognized as a sequel to oiling, malnutrition, other concurrent diseases, or captivity (Wobeser, 1981) and it is possible that the loons found with this as a primary diagnostic finding in Florida were representative of emaciated birds which became secondarily infected with *Aspergillus* sp. However, White et al. (1976) reported that aspergillosis occurred at a statistically significant higher prevalence in non-oiled loons than in oiled loons. Locke and Young (1967) reported on a case of aspergillosis in a captive common loon and, because of the rapidity of spread of hyphae along the capillary bed, suggested that this host "might be highly susceptible to this fungal infection."

The cause of the emaciation syndrome in loons appeared to be multifactorial. Infectious diseases were ruled out, since no viruses were isolated, various bacteria that were identified from these loons were not isolated consistently, and attempts to transmit an infectious agent to turkey poults and ducklings utilizing spleen extracts and intestinal contents from emaciated loons were unsuccessful. Other factors were judged to be contributory to the emaciation process in various ways and are discussed below.

The concentrations of DDE and PCBs in brain tissues were well below those reported to be lethal for birds (Stickel et al., 1970; Stickel, 1975); however, the sublethal effects of these organochlorines on loons are not known. The concentrations of DDE in loons from Florida were similar to those reported from common loons in Minnesota (n = 3; Ream, 1976), Michigan (n = 8; Fay and Youatt, 1967), and Wisconsin (n = 6; Belant and Anderson, 1990), but below that (130 ppm) found in the brain of a common loon that died of DDD poisoning in Mississippi (Prouty et al., 1975). PCB concentrations of Florida loons were also close to those given in previously published reports on common loons from Wisconsin (Belant and Anderson, 1990), Ontario (n = 157; Frank et al., 1983), and Mississippi (Prouty et al., 1975).

The significance of cadmium residues in loons is not known, although birds in general are thought to be relatively resistant to harmful effects of this metal (Eisler, 1985). In an experimental dosing study, White and Finley (1978) reported no mortality in mallards (*Anas platyrhynchos*) which received cadmium in their diets for up to 90 days. Residues in livers were over 100 ppm in some of their ducks.

Concentrations of lead in liver samples of two loons from the 1983 epizootic were 5.3 and 7.5 ppm and were similar to concentrations reported by Pokras and Chafel (1992) in common loons diagnosed with lead poisoning in New England. This might mean that these two Florida loons were somewhat compromised by ingested lead, although, other than emaciation, typical signs of lead poisoning were not observed at necropsy. Lead poisoning has

been reported also from common loons on their summer ranges in Minnesota, New Hampshire (USA), Wisconsin, and Maine (USA) (Ensor et al., 1992; Locke et al., 1981). In all of these cases reported from northern states, the cause of the lead toxicosis was linked to ingestion of fishing sinkers. We found no evidence of lead sinkers or other types of fishing tackle in loons from Florida waters.

Contamination of common loons by mercury has been studied in several northern locales including Ontario (Fimreite, 1974; Frank et al., 1983; Barr, 1986), Wisconsin (Belant and Anderson, 1990), and Minnesota (Ensor et al., 1992). Residues were found in all samples examined and ranged from 0.08 to 91 ppm (wet weight) and are comparable to the values found in loons from Florida. As mentioned previously, a high percentage of common loons overwintering in Florida are known from banding studies to nest in Wisconsin and Minnesota (Bird Banding Laboratory, 1995) and this may account for their contamination with mercury. Barr (1986) examined chicks (as well as adults) and showed that mercury was acquired by loons on their nesting grounds. Frank et al. (1983) found that residues were higher in emaciated loons (11–26 ppm) than in healthy loons (2–5 ppm), an observation that is similar to our findings in Florida. The significance of these mercury residues to the health of loons is not clear since there have been no experimental studies conducted on this species. Eisler (1987, p. 50) reviewed the literature on mercury contamination and concluded that "toxicity to birds varies with the form of the element, dose, route of administration, species, sex, age, and physiological condition. . ." The picture is further complicated by the possibility of interactions between mercury, selenium, cadmium, lead, and other contaminants such as DDE and PCBs (Mullins et al., 1977; Eisler, 1987; O'Brien et al., 1995). For example, a mutual protective effect of mercury and selenium has been found in experimental studies with Japanese quail (*Coturnix c. japonica*) (Stoewsand et al., 1974; El-Begearmi et al., 1977). In addition, G. H. Heinz and D. J. Hoffman (pers. comm.) found experimentally that there was a protective effect of selenium against mercury poisoning in adult male mallards (*Anas platyrhynchos*). This aspect needs to be investigated further in order to better understand the impact of environmental contaminants and their relationship to the health of common loons.

Although several factors such as inclement weather (Simons, 1985), oil spills (Stevenson, 1970; 1974; White et al., 1976), and high energy costs of feather replacement coincident with mercury contamination (Alexander, 1991) have been linked with winter mortality of loons, the emaciation syndrome appears to be the most significant. Information obtained over the past 25 yr, and especially during the 1983 epizootic, leads us to believe that the cause of the emaciation is complex and involves both biotic and abiotic factors. We suggest that the following stress factors may act synergistically: (1) energy depletion due to migration, molting, food shortage, salt-loading, and intestinal parasitism, (2) mobilization of environmental contaminants such as DDE, PCBs, selenium, mercury, lead, and cadmium which are stored in various tissues, and (3) inclement weather.

We postulate the following scenario. Common loons that nest in the Great Lakes region migrate to Florida in the fall, arriving over a several week period of time from the latter part of October through the middle of November (Alexander, 1991). Following their arrival in Florida coastal waters, they undergo a wing-feather molt and are flightless for a period of 3 to 4 wk (Woolfenden, 1967) which would limit their local movements. At this time they need to feed extensively to replenish their depleted energy reserves and to replace their molted feathers. Normally their diet consists predominantly of fish, but, to a lesser extent also crabs and shrimp (Howell, 1932; Sprunt, 1954). In the epi-

zootic of 1983, however, there was evidence that the loons fed more heavily on crabs than fish; this was concluded from our data on the abundance of intestinal trematodes, mainly microphallids. These trematodes were 600-fold more abundant in loons examined in 1983 than in those examined in 1984, a non-epizootic year. Since microphallids utilize arthropods such as crustaceans and shrimp as intermediate hosts (Schell, 1985), their presence in large numbers is an indication that the loons were feeding heavily on such prey items in 1983, but not in 1984. The differential heavy use of crustaceans and shrimp rather than fish during the epizootic year might have resulted in salt-loading since these animals are osmotic conformers (Bildstein et al., 1990) and have a greater concentration of salt in their tissues than fish. This would result in increased physiologic stress on the salt glands of the loons. This added stress would not occur in normal years when loons feed mainly on fish, which are not osmotic conformers and have low salt concentrations.

Livingston et al. (1997) conducted a long-term study of Apalachicola Bay (where Dog Island is located and where much of the 1983 loon mortality occurred) from February 1975 through July 1984 and found that freshwater input from rivers flowing into the area had a significant influence on trophic organization. They reported that a 2-yr drought in 1980–1981 caused sustained elevated salinities in the Bay which eventually led to major reductions of biomass at various trophic levels and progressive decreases in fish numbers. These biomass changes persisted for several years after the droughts occurred. In addition, although there was not a red tide episode occuring at the time of the 1983 epizootic, according to the records of the Florida Marine Research Institute, such an event did occur offshore several months earlier and resulted in extensive fish kills which may have had an effect on reducing fish populations inshore as well. Such events might have resulted in the loons feeding more commonly on crustaceans and less on fish in 1983 than is normal.

The final stress would be inclement weather. During the period of the 1983 epizootic (January to March), a series of cold fronts passed through Florida every few days, particularly in the northern Gulf area. These were characterized by above-normal amounts of rainfall (Kale, 1983; Hoffman, 1983). Another important aspect of these winter storms is that in addition to producing cold stress, they also increase turbidity in nearshore waters. Because loons are visual diurnal feeders (McIntyre, 1978), their foraging success may be diminished by storm-induced turbidity. The stresses placed upon loons by these various factors could result in excessive metabolism of fat reserves and catabolism of muscle and other tissues leading to mobilization of contaminants previously stored within body tissues. Some of the contaminants, for example mercury, can produce neurologic impairment (Eisler, 1987) which could further reduce the foraging ability of affected birds. Two of us (WRD and REL) made field observations of weakened loons with neurologic signs including tremors during the 1983 epizootic. The accumulative effects of the various stresses listed above could have led to higher than normal mortality of common loons which were overwintering in Florida waters in 1983.

Spitzer (1995) has suggested a scenario that is somewhat similar to ours to explain a die-off of several hundred common loons in emaciated condition on the Atlantic coast in the winter of 1993. He concluded that the stress of storms coupled with molting of the flight feathers and limitations of food were responsible for the mortality. He stated that the loons were emaciated, but gave no details on pathologic findings or other studies relating to toxicology, microbiology, or parasitology.

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