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Necropsy Findings and Environmental Contaminants in Common Loons from New York

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ABSTRACT: Diagnostic and analytical findings are presented for 105 common loons (*Gavia immer*) found dead or debilitated in New York (USA) from 1972–99. Aspergillosis (23% of cases) and ingestion of lead fishing weights (21%) were the most common pathologies encountered. Stranding on land, shooting, other trauma, gill nets, air sacculitis and peritonitis, and emaciation of uncertain etiology accounted for most of the remaining causes of disease or death. Analysis for total mercury in the liver of 83 loons yielded a geometric mean (gm) of 10.3 mg/kg (wet basis) and range of 0.07 to 371 mg/kg, with emaciated birds generally showing higher levels. Organochlorine contaminant levels in brain were generally low, principally consisting of PCB's (gm = 2.02 mg/kg) and DDE (0.47 mg/kg).

Key words: Aspergillosis, common loon, *Gavia immer*, lead poisoning, mercury, mortality, organochlorines.

Investigations into the mortality of common loons (*Gavia immer*) have identified several factors of regional and/or temporal importance. Drowning in fishing nets has at times claimed many loons on some large lakes in northern Canada (Vermeer, 1973). Type E botulism has historically killed many loons on Lake Michigan (USA, Canada) (Monheimer, 1968; Brand, 1988). Pokras and Chafel (1992) found ingestion of lead fishing weights to be the leading cause of death in adult loons on breeding lakes in New England (USA). Aspergillosis has frequently been identified as a cause of death in loons (McIntyre, 1988), although in most cases it is presumed secondary to other conditions. Common loons nesting on acidified lakes (Evers et al., 1998) or feeding downstream of anthropogenic sources of mercury (Barr, 1986) may accumulate high levels of mercury in their tissues with possible reproductive repercussions at minimum (Barr, 1986; Nocera and Taylor, 1998). An interaction of mortality factors has been proposed to account for die-offs that occasionally occur in common loons wintering along the Atlantic and Gulf coasts of North America (Spitzer, 1995; Forrester et al., 1997; Augsperger et al., 1998). Complex etiologies also were proposed for many of the thin loons in a mortality survey in Atlantic maritime Canada (Daoust et al., 1998). A better understanding of the mortality factors of this species will help identify present and future threats to this icon of the northern lakes and the ecosystems in which it lives. Here we report necropsy findings and contaminant data on common loons found dead or moribund in New York (USA) from 1972 through 1999.

Loons were submitted for diagnosis or examination as part of our routine activities as the wildlife pathology unit of the New York State Department of Environmental Conservation (NYSDEC; Delmar, New York, USA). Submitters included state fisheries and wildlife biologists, law enforcement personnel, wildlife rehabilitators, and the general public. Necropsies were performed on all specimens. Age was determined from plumage characteristics (McIntyre, 1988) or the development of reproductive organs and the bursa of Fabricius. Livers and brains were collected and frozen for contaminant analysis. Levels of lead and total mercury were determined in liver at the NYSDEC laboratory at Hale Creek (Gloversville, New York) or, more commonly, at contract laboratories,

particularly EnChem, Inc. (Madison, Wisconsin, USA) (and its predecessors Hazleton Laboratories (Madison, Wisconsin) and Raltech (Madison, Wisconsin)) and the Illinois Department of Agriculture Animal Disease Laboratory (Centralia, Illinois, USA). For lead determination, samples were prepared by acid digestion (with some variation in procedure between laboratories and over time), and analyzed by graphite furnace atomic absorption spectrophotometry according to EPA method 7421 (U.S. Environmental Protection Agency, 1988) or similar. Following identical or alternate acid digestion procedures, samples for mercury determination were analyzed by cold vapor atomic absorption spectrophotometry according to EPA method 7471A or similar (U.S. Environmental Protection Agency, 1995). Organochlorine pesticides and metabolites (including dichlorodiphenyltrichloroethane (DDT), dichlorodiphenyldichloroethane (DDD), and dichlorodiphenylethane (DDE), hexachlorocyclohexane isomers, hexachlorobenzene (HCB), dieldrin, endrin, heptachlor, heptachlor epoxide, alpha- and gamma-chlordane, oxychlordane, transnonachlor, toxaphene, and mirex), and total polychlorinated biphenyls (PCB's) were determined by gas chromatography of brain tissue by the same laboratories according to EPA method 8081 (United States Environmental Protection Agency, 1995), its forerunners, or similar. Both contract laboratories produced analytical results for mercury and organochlorines similar to those of the NYSDEC laboratory in a performance evaluation in 1999 (unrelated to the present study). The contaminant data were not normally distributed and were log-transformed prior to comparing subsets of interest by one-tailed or two-tailed *t*-tests (Snedcor and Cochran, 1980). Contaminant values below detection limits (DL) were assigned half the DL for purposes of calculation. All contaminant data are presented on a wet weight basis.

For diagnostic purposes, levels of organochlorine contaminants were compared with the following suggested lethal minimums based on experimental dosing of several passerine bird species: ≥300 mg/kg DDE for birds killed with DDE (Stickel et al., 1984a), 310 mg/kg total PCBs for birds killed with Aroclor 1254 (Stickel et al., 1984b), ≥177 mg/kg for mirex (Stickel et al., 1973), ≥4 mg/kg for dieldrin (Stickel et al., 1969), and ≥3.4 mg/kg heptachlor epoxide plus 1.1 mg/kg oxychlordane for birds killed with chlordane (Stickel et al., 1979). Lead levels in liver similarly were assessed relative to diagnostic minimums cited in reviews by Pain (1996) and Franson (1996). Mercury levels were compared with studies reviewed by Heinz (1996) and Thompson (1996).

During the 27 yr period, 105 common loons were examined; nine examined prior to 1980, 33 in the 1980's, and 63 in the 1990's. Surprisingly, only 16 loons originated from the Adirondack region which supported a breeding population of slightly in excess of 150 pairs in the mid-1980s (Parker, 1986) and perhaps more today. Of the remaining loons, 23 came from the Hudson River estuary or Long Island sites, 22 were from the Great Lakes (Lakes Erie and Ontario plus the Niagara and St. Lawrence Rivers), 17 were found on the Finger Lakes in the central-western part of the New York, and 27 were from other locations. Dead loons were found in all months of the year, although few were recovered in early fall and mid-to-late winter. Fifty-six percent of the loons bore plumage characteristic of breeding adults or exhibited anatomical evidence of having attained breeding age. Only three unfledged chicks were submitted. The remainder were immature birds (13%) or of uncertain age (29%).

The two most common maladies observed were ingestion of lead fishing weights and aspergillosis (Table 1). Lead fishing weights were found in the alimentary canals of 21 loons. The first case was recorded in 1982, and the occurrence of such cases has increased over time with

TABLE 1. Causes of morbidity and mortality observed in 105 common loons in New York, 1972–1999.

Diagnostic category	No. Cases (%)
Stranding[a]	11 (10)
Drowned in gill nets	7 (7)
Shot	7 (7)
Oiled	1 (1)
Other trauma	8 (8)
Ingestion of Pb fishing weights[b]	22 (21)
Other fishing tackle problems[c]	8 (8)
Aspergillosis[d]	24 (23)
Peritonitis and air sacculitis	9 (9)
Emaciation of unknown etiology	6 (6)
Open	2 (2)

[a] Inadvertent landing on wet pavement or other solid surfaces apparently mistaken for water. The proximate causes of death in these cases include impact trauma, complications of sublethal injuries, and/or dehydration and malnutrition subsequent to an inability to regain flight once grounded.
[b] Includes one case in which fishing weight ingestion was inferred from the lead level in the liver.
[c] Ingestion of hooks with injuries to the alimentary canal (5) or entanglement in monofilament line (3).
[d] Does not include nine additional cases where *Aspergillus* sp. lesions were found in loons compromised or killed by other identifiable primary causes.

increases in the number of loons examined overall (2 cases/13 examined (1980–84), 3/20 (1985–89), 6/26 (1990–94), 10/37 (1995–99)). Only one of these loons came from a marine site.

The level of lead in the livers of these loons exceeded the 5 to 6 mg/kg diagnostic thresholds for lethal lead poisoning determined for waterfowl or other birds in 18 cases ($\bar{x}$ = 19.3 mg/kg, range = 6.6–41.2 mg/kg). Lead poisoning also was considered a morbidity factor in the deaths of the other three loons with ingested fishing weights. One of the latter had been euthanized (liver lead = 3.24 mg/kg), another died in captivity two days after it was found debilitated on the shore of Lake Erie (USA, Canada) (4.18 mg/kg), and the third died of drowning under unknown circumstances in an Adirondack pond (1.57 mg/kg). Ingestion of a lead fishing weight may also have been involved in a loon that failed to migrate from an Adirondack Lake and was found dead in the ice in February. This loon showed emaciation, peritonitis, and had 4.9 mg/kg lead in the liver although no lead weight was found at necropsy. The geometric mean (gm) level of lead in the liver of loons that did not show ingested lead weights at necropsy (excluding this last case) was 0.07 mg/kg (n = 48, max = 1.1 mg/kg) including 21 cases where the levels of lead were below detection limits that ranged from 0.05 to 0.14 mg/kg.

Seven (33%) of the loons with ingested weights were in fair-to-good body condition or better. The remainder lacked fat and showed slight-to-severe atrophy of skeletal muscle. Food was noted in the upper alimentary canal in only three loons, each of which had ingested crayfish (53 individual crayfish in one case).

The lead fishing weights found in the stomach (19 cases), colon (one case), or cloaca (one case) were identified as sinkers (weights tied or clamped onto fishing line) in eleven loons, weighted lures (jigs) in nine, and one was not examined first-hand (removed by veterinarian prior to submission). One lure weight per bird was the rule except for one loon in which three sinkers were present. The heaviest sinker was a slightly worn swivel-type that weighed 9.5 grams, the lead portion of which measured 16.7 mm in length and 9.8 mm in diameter. The next heaviest was an elongated clamp-on type that weighed 5.1 g and measured 27.3 mm in length. The two largest jigs weighed 4.5 and 3.5 g despite considerable wear. The lure weights were accompanied by related fishing tackle (swivels, snap-swivels, hooks, line) in eight cases.

Fishing tackle also was implicated in the deaths of eight other loons. Five succumbed to complications of injuries to the alimentary canal caused by fish hooks. External entanglement in monofilament line claimed three others.

Chronic aspergillosis, as grossly described by O'Meara and Witter (1971), was the proximate cause of death in 24 loons, and *Aspergillus* sp. lesions were present in an additional nine cases, including

five of the lead poisonings. Half of the aspergillosis mortalities originated in marine or estuarine sites. With one exception, the body condition of these birds ranged from fair-to-poor to emaciated. Other lesions observed in these birds included minor foot injuries (four cases), healed puncture wounds to the breast, a healed fracture of the scapula, and abnormalities, damage or extreme wear to flight feathers (five cases). Whether some of these pathologies were related to factors that predisposed the loons to *Aspergillus* sp. infections, or were a consequence of the chronic fungal infection, was unclear.

Similarly in generally poor flesh were nine loons exhibiting chronic air sacculitis or peritonitis. At least five of these cases appeared linked to perforations of the alimentary canal: twice by fish bones, once by acanthocephalans, and two of unknown etiology. One loon that lacked any obvious alimentary canal injury had an extremely high level (371 mg/kg) of total mercury in its liver.

Inexplicable emaciation was observed in six loons, four from marine or estuarine sites. Two of the latter had extremely frayed and ragged plumage. Another, held briefly by a wildlife rehabilitator, appeared to have plumage that was not waterproof. One emaciated adult loon found at a pond in western New York in May had 130 mg/kg total mercury in its liver.

Mercury levels in the livers of 82 loons overall ranged from 0.07 mg/kg in a lead-poisoned unfledged chick to 371 mg/kg in the aforementioned adult female with air sacculitis. Although 29% of the levels were below 5 mg/kg and the geometric mean of the entire sample was 10.3 mg/kg, 23% of the levels were between 20 and 45 mg/kg, and 13% of the sample exceeded 65 mg/kg. Levels in loons lacking fat and showing notable muscle atrophy (gm = 12.4 mg/kg, $n = 53$) were generally higher ($P = 0.04$) than those in loons in good flesh (gm = 6.4 mg/kg, $n = 19$). No significant gender differences were noted.

Organochlorine contaminants were determined in the brains of 37 loons; 26 in poor flesh and the remainder in fair or better body condition. In general, the levels were not particularly elevated (Table 2) and, with one exception, never approached those associated with lethal intoxication. The exception was an emaciated adult male from Lake Ontario in 1983 which succumbed from aspergillosis and had elevated levels of DDE (9.18 mg/kg), PCB's (28.8 mg/kg), and a near lethal level of dieldrin (3.80 mg/kg). The highest PCB level (60 mg/kg) was recorded in an emaciated adult male found dead from aspergillosis along the Hudson River below Albany in 1981. Although experimental studies and field evidence suggest lethal brain levels begin around 300 mg/kg (Dahlgren, 1972; Sileo, 1977; Stickel et al., 1984b), dosing of great cormorants (*Phalacrocorax carbo*) with Clophen 60 yielded brain levels of 76–180 mg/kg (Koeman et al., 1973). The highest level of DDE (11.0 mg/kg) was recorded in a thin adult female succumbing to complications of ingested fish hooks in the Finger Lakes region in April. Another thin female from this highly ag-

TABLE 2. Organochlorine contaminants in brain tissue of 39 common loons from New York examined 1981–1999.[a]

Contaminants[b]	Levels in mg/kg, wet basis	
	% detection	Geometric mean (range)[c]
DDE	92	0.47 (0.004–9.7)
PCB's	92	2.02 (0.01–60)
Dieldrin	79	0.06 (<0.005–3.80)
Oxychlordane	64	0.02 (<0.005–0.42)
Transnonachlor	53	0.02 (<0.005–0.73)
Heptachlor epoxide	51	0.01 (<0.005–0.26)
Mirex	49	0.02 (0.001–1.30)

[a] Analyses by several laboratories (see text).

[b] Also detected less frequently were cis-nonachlor (43%, max = 0.18 mg/kg), HCB (38%, max = 0.07 mg/kg), DDT (24%, max = 0.15 mg/kg), DDD (19%, max = 0.02 mg/kg). Trace levels of alpha-chlordane were reported twice and endrin (0.026 mg/kg) was detected once. Photomirex (determined only by the New York State Laboratory at Hale Creek) was present in six of eight brain samples (range = 0.005–0.40 mg/kg).

[c] Minimums shown for DDE, PCB's and mirex were lower than the detection limits reported for some other samples.

ricultural region (dying from an air sacculitis) showed 193 mg/kg DDE in its liver (brain not analyzed).

Perhaps our most important finding was confirmation of ingested lead fishing weights as a major cause of common loon mortality. First described by Locke et al. (1982), this phenomenon was next reported by Pokras and Chafel (1992) as an important cause of death in New England loons, with an overall prevalence of 21% (n = 75), and a prevalence of 52% (n = 31) for breeding adults on freshwater lakes. Subsequently, Scheuhammer and Norris (1996) reported a prevalence of 30% (n = 127) for loons examined from angler-frequented loon habitat in eastern Canada, and a prevalence of 21% (n = 172) was recently calculated for loons from Michigan (T. Cooley, pers. comm.). A somewhat lower prevalence (7%, n = 95) was recorded in Minnesota (Ensor et al., 1992).

Pokras and Chafel (1992) noted, as we and others have, that lead fishing weights are frequently accompanied by other tackle (hooks, line, swivels) in the stomachs of loons, and proposed that loons in such cases were probably attracted to bait that accompanied these items. They also proposed that cases in which such associated tackle was not found may indicate that loons may be picking up lead fishing weights directly as pebble-like aids to digestion. It seems more likely, however, that these latter cases represent the loss of the associated tackle to abrasion and corrosion in the gizzard and/or loss by passage through the alimentary canal.

Although we recorded greatly elevated levels of mercury in some loons, the relationship of these levels to the cause of death is uncertain. Mercury levels in the livers of some specimens approached or exceeded those recorded in experimental poisonings of similarly piscivorus gray herons (*Ardea cinerea*) (Van Der Molen, 1982) and juvenile great egrets (*Ardea albus*) (Spalding et al., 2000). On the other hand, it has recently been shown that the mercury in the livers of common loons is principally in the less toxic inorganic form, and that the methylmercury fraction of the total declines as total mercury increases, generally remaining below 2.8 mg/kg (Pokras et al., 1998; Scheuhammer et al., 1998). Furthermore, mercury levels in the brains of three birds that we analyzed specifically to compare with elevated levels in the liver did not even approach the 15 mg/kg threshold for overt intoxication in non-piscivorus birds (Heinz, 1996). These three loons in our study with 371, 89, and 25.3 mg/kg mercury in liver respectively showed 4.5, 3.1, and 1.48 mg/kg in the brain, similar to the findings of Daoust et al. (1998) for ten loons from New Brunswick, Canada. In these respects, common loons may be similar to seabirds in tolerating high mercury concentrations (Thompson, 1996). Confirmation of incapacitation or lethal intoxication with mercury in common loons needs corroborative histopathological findings and/or additional clarifying data on the toxic levels of inorganic and organic mercury in loons or appropriate surrogate species.

For the majority of the aspergillosis cases, there were no obvious clues as to the identity of predisposing factors. Mercury contamination, at least, seems an unlikely factor as the geometric mean level of mercury in aspergillosis cases (8.9 mg/kg) was less than or no different from the means for other diagnostic categories for which emaciation was frequently recorded: lead poisoning (gm Hg = 8.51 mg/kg), unexplained emaciation (19.0 mg/kg), air sacculitis/peritonitis (30.5 mg/kg), and stranding (31.9 mg/kg).

As has been noted by Spitzer (1995), Forrester et al. (1997), Ausperger et al. (1998) and Daoust et al. (1998), mortalities of common loons may frequently be the result of a interplay of factors and/or events, and determining primary causes is often difficult. More straight-forward, however, is recognition of the substantial mortality of common loons at inland locations in eastern Canada, the northeastern United States, and probably elsewhere

from ingestion of lead sinkers and lure weights. Two states, New Hampshire and Maine, have enacted legislation to restrict the use of lead fishing weights based on the findings in New England. Restrictions have also been established at some national or provincial park lands in the United States and Canada. We encourage diagnosticians to publish or otherwise make available their findings on this topic to encourage additional restrictions elsewhere, preferably at a national or international level.

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