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Mercury Poisoning in a Free-Living Northern River Otter (*Lontra canadensis*)

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ABSTRACT: A moribund 5-year-old female northern river otter (*Lontra canadensis*) was found on the bank of a river known to be extensively contaminated with mercury. It exhibited severe ataxia and scleral injection, made no attempt to flee, and died shortly thereafter of drowning. Tissue mercury levels were among the highest ever reported for a free-living terrestrial mammal: kidney, 353 μg/g; liver, 221 μg/g; muscle, 121 μg/g; brain (three replicates from cerebellum), 142, 151, 151 μg/g (all dry weights); and fur, 183 ug/g (fresh weight). Histopathologic findings including severe, diffuse, chronic glomerulosclerosis and moderate interstitial fibrosis were the presumptive cause of clinical signs and death. This is one of a few reports to document the death of a free-living mammal from presumed mercury poisoning.

Key words: *Lontra canadensis*, mercury poisoning, northern river otter, Virginia.

The accumulation of mercury in northern river otters (*Lontra canadensis*) is well documented (Mierle et al., 2000; Ben-David et al., 2001; Yates et al., 2005), and has been suggested as a cause of population declines (e.g., Hyvarinen et al., 2003) or reduced survivorship (Mierle et al., 2000). Mercury intoxication can cause behavioral aberrations in both American mink (*Neovison vison*) and otters, including circling and attempts to burrow into the ground (Wobeser et al., 1976; Wren, 1985). Experimental studies have been used to determine the possible level at which mercury results in toxic effects in these species by dosing animals with methylmercury (e.g., O'Connor and Nielsen, 1980). However, documented cases of fatal mercury accumulation in free-ranging wildlife are rare. The use of otters and mink as biomonitors for ecosystems is increasing (Basu et al., 2007; Wolfe et al., 2007). Background levels of mercury in otters in ecosystems contaminated primarily through atmospheric deposition are under 9.0 μg/g (wet weight [ww]) in the liver (Yates et al., 2005) and lower in the kidney (Kucera, 1983). Levels of mercury in ecosystems contaminated with point sources can result in mortalities, as reported for a dead otter from Ontario with hepatic mercury levels of 96.0 μg/g, ww (Wren, 1985). The fortuitous discovery of a moribund animal in the South River, Shenandoah Valley, Virginia, a site where invertebrates, fish, and birds are known to be extensively contaminated with industrial mercury (Cristol et al., 2008), provided a rare opportunity to associate specific tissue levels with mortality in a wild otter. Analyses revealed the highest levels of mercury reported in otters.

In April 2006, a moribund northern river otter was observed on the western bank of the South River (38°12.531′W, 78°50.477′N), Augusta County, Virginia. Visual examination of the animal from close range revealed tremors, bilateral scleral injection, and marked ataxia. The animal made no attempt to flee when approached and eventually returned to the water and drowned within minutes. The carcass was collected and refrigerated for 18 hr until a gross necropsy could be performed. Samples of ovary (with follicle), liver, lung, kidney, and heart were placed in 10% buffered formalin. These tissues as well as muscle (biceps femoralis), bile, blood, and fur from the hind leg were collected and frozen at −30 C. The

rest of the carcass, including the head, was also stored in a secure freezer at −30 C. The head was taken to the Virginia Department of Agriculture and Consumer Services Animal Health Laboratory, Harrisonburg, Rockingham County, Virginia, where the brain was removed for rabies testing at the Division of Consolidated Laboratory Services, Richmond, Virginia, and three samples of cerebellum were retained frozen for mercury analysis. A tooth was removed for cementum age analysis (Matson's Laboratory LLC, 8140 Flagler Road, Milltown, Montana, USA).

Tissues in 10% buffered formalin were submitted to Southeastern Cooperative Wildlife Disease Study, College of Veterinary Medicine, University of Georgia, Athens, Georgia, USA for histopathologic evaluation. Tissue sections were embedded in paraffin, sectioned at 3 μm, and stained with hematoxylin and eosin and Ziehl-Neelsen acid-fast for light microscopy.

Analysis for total mercury was completed with cold vapor atomic absorbance spectroscopy with the use of a direct mercury analyzer (DMA-80 Milestone, Inc., Shelton, Connecticut, USA) at the Trace Element Research Laboratory (Texas A&M University, College Station, Texas, USA). The factory-calculated instrument detection limit for the direct mercury analyzers used was 0.005 ng and was calculated to be between 0.013 and 0.026 ($n=9$) during this analysis. Mean percent recovery of standard reference materials (DORM-2, DOLT-3; National Research Council Canada, Ottawa, Ontario, Canada) was 100.75±4.79% ($n=4$) during the running of the samples reported while spike recovery was 100.70±1.37 ($n=13$) for the week of analysis.

No gross lesions were observed on necropsy, except for lack of body fat. Histopathologically, the mesangium of the glomeruli was thickened by fibrous connective tissue. Fibrous connective tissue also expanded the cortical interstitium and to a lesser extent the medullary interstitium (Fig. 1). The liver contained a few to moderate numbers of lymphocytes and plasma cells and rare neutrophils that surrounded periportal areas. Similar infiltrates surrounded bronchioles in the lung. Microscopic lesions were not apparent in the heart or reproductive tract. The brain was immunonegative for rabies by fluorescent antibody and the lung and liver were immunonegative for canine distemper virus (CDV). The otter was determined to be 5 yr of age and the cementum annuli were clear and distinct.

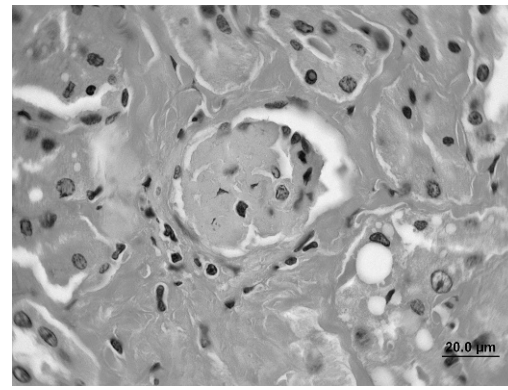

FIGURE 1. Histopathologic section of a kidney of a river otter (*Lontra canadensis*) with presumed mercury poisoning. Note the thickening of the mesangium of the glomeruli by fibrous connective tissue with expansion into the cortical interstitium, and to a lesser extent the medullary interstitium. H&E stain. Bar=20 μm.

Mercury concentration of tissues was (dry weight, except for fur): fur, 183 μg/g (fresh weight); kidney, 353 μg/g (75.1% moisture); liver, 221 μg/g (74.1% moisture); muscle, 121 μg/g (74.6% moisture), brain (3 replicates from cerebellum), 142 (78% moisture), 151 (78.2% moisture), 151 μg/g (74.1% moisture).

The clinical, pathologic, and toxicologic findings are consistent with the few previously published case reports of mercury poisoning in otters (Heinz, 1996). The lesions seen in the kidneys were severe and chronic and most likely led to the clinical signs in this otter; however, as brain tissue was not available for histopathology, other causes of the central

TABLE 1. Mercury levels reported in tissues from various mammal species.

Species	Location	Level (μg/g) (dry weight)	Tissue	Reference
Otter	Clay Lake, Ontario, Canada	96.0	Liver	Wren (1985)
		58.0	Kidney	
		36.0	Muscle	
		30.0	Brain	
Otter	Experimentally killed	33.4	Liver	O'Connor and Nielsen
		39.2	Kidney	(1980)
		15.7	Muscle	
		18.9	Brain	
Mink	South Saskatchewan River,	58.2	Liver	Wobeser and Swift
	Saskatoon, Canada	31.9	Kidney	(1976)
		15.2	Muscle	
		13.4	Brain	
		34.9	Fur	
Feral cats	Minamata Bay, Japan	37–145	Liver	Takeuchi et al. (1977)
Striped dolphin	French Mediterranean Coast	68–2,272	Liver	Augier et al. (1993)
		14–34	Kidney	
		7–155	Muscle	
		4–81	Brain	
Florida panther	Everglades, Florida, USA	330	Liver	Dunbar (1994)[a]

[a] Originally reported as wet weight. For the purposes of comparison we assumed that wet weight value was equal to dry weight value/3 (Puls, 1994).

nervous system signs such as encephalitis due to toxoplasmosis cannot be ruled out (Fernandez-Moran, 2003). However, the mercury concentration in the brain of this animal was markedly higher than previously reported levels in naturally and experimentally exposed otters (142–151 μg/g versus 30.0 and 18.9 μg/g, respectively) (O'Connor and Nielsen, 1980; Wren, 1985), suggesting mercury poisoning as the proximate cause of death.

Although the most consistent effects of methylmercury are on the central nervous system, renal necrosis and fibrosis have been reported in several experimentally dosed species, including guinea pigs, mink, and harp seals (*Phoca groenlandicus*), as well as in a naturally exposed horse (Heinz, 1996). The etiology of the renal pathologic changes is thought to be autoimmune glomerulopathy due to chronic inorganic mercury filtration (Eto et al., 1997). The remarkable finding in this case was the extremely high body burdens of mercury. With the possible exception of a Florida panther (*Felis concolor coryi*), these levels are higher than previously reported values from other nonmarine mammals (Table 1), and set an upper boundary for documented lethal levels of mercury in otters. To the authors' knowledge this case report is the first to describe histopathologic changes associated with markedly elevated tissue mercury levels in a naturally exposed river otter.

The value of the river otter as an established indicator species for sublethal effects of mercury is recognized by national mercury monitoring programs (Wolfe et al., 2007); however, it has not been well documented that otters or other animals might suffer mortality from direct exposure to mercury pollution. Although sublethal and reproductive-effect thresholds are increasingly being identified in free-living piscivorous birds and mammals (Scheuhammer et al., 2007; Burgess and Meyer, 2008; Evers et al., 2008; Scheuhammer et al., 2008; Basu and Head, 2010), evidence of mortality related to mercury contamination in ecosystems, such as this account, is rare.

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