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Source: Journal of Wildlife Diseases, 12(3) : 335-340

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-12.3.335

MERCURY POISONING IN A WILD MINK

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Abstract: Mercury poisoning was diagnosed in a clinically-ill wild mink (*Mustela vison*) on the basis of clinical signs, histopathologic lesions and tissue mercury concentrations. The probable source of mercury was through ingestion of fish from the nearby South Saskatchewan River which is known to be contaminated with mercury. This is believed to be the first documented case of mercury intoxication of a wild animal in North America.

INTRODUCTION

Although aquatic mercury contamination is widespread and high concentrations of mercury have been detected in a variety of piscivorous birds and mammals from several areas of the world, clinical mercury intoxication of such animals has been reported only from Japan[16] and Sweden.[6] The present case is the result of a fortuitous encounter by one of us (M.S.) with a clinically-sick wild mink (*Mustela vison*) near a river known to be contaminated with mercury.

HISTORY AND METHODS

In February, 1975, a wild female mink was found in a field approximately 0.5 km from the South Saskatchewan River, about 2 km downstream from the city of Saskatoon. When observed the animal was squealing, jumping in the air and attempting to burrow in the snow. It would lie on its back and then roll over, and fine, whole-body tremors were evident. The animal's eyes were open but it was oblivious to the observer. Tracks in the snow indicated that the animal had been moving in a circular manner within a 20 m diameter area for some time. The animal made no attempt to bite or escape when picked up; however, it became moribund and died within 2 hrs of capture.

The animal was submitted to the Department of Veterinary Pathology and a necropsy was performed approximately 1 hr after death.

At necropsy tissues were fixed in 10% buffered formalin for histopathology and portions of liver, kidney, skeletal muscle, fur and one-half of the brain were frozen in individual plastic containers for mercury analysis. A small portion of the brain was examined for Rabies antigen by fluorescent antibody technique.[2]

Total mercury content of tissue was determined using atomic absorption spectrophotometry as previously described.[19]

Tissues for histopathology were processed routinely, embedded in paraffin, sectioned at 6 μm and stained with Hematoxylin and Eosin.

RESULTS AND DISCUSSION

Pathology:

The mink was in good body condition and no gross lesions were visible. The histopathologic lesions found were very similar to those observed in mink experimentally poisoned with methyl mercury chloride.[20] The most severe lesions were found in the cerebral cortex, cerebellum, lymphoid tissues and myocardium. There was neuronal degeneration

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and necrosis in the cerebral cortex with astrocytosis and microgliosis, axonal degeneration and slight evidence of cavitation or formation of a "status spongiosus". (Fig. 1 and 2). These lesions were diffuse throughout the cortex but most severe in the occipital region. Occasional perivascular cuffs of mononuclear cells were encountered in the cortex in association with areas of neuronal necrosis. In the cerebellum, lesions were largely confined to the medial and basal areas in which there was a distinct pyknosis and loss of granular cells. (Fig. 3).

There was marked depletion of cells from the central areas of follicles in spleen and lymph nodes, and in some cases necrosis of cells was evident. There were multiple foci of histiocytic infiltration in the myocardium in association with foci of hyaline degeneration of myocardial fibres (Fig. 4). Myocardial lesions were not observed in the experimentally-poisoned mink[20] but have been reported in alkyl mercury poisoning in other species,[12,10,13,15] including the closely-related ferret (*Mustela putorius*).[11]

Mercury Analysis:

The concentration of mercury present in the tissues of this mink were similar to those in mink experimentally poisoned with methyl mercury chloride[20] and to concentrations reported in alkyl mercury intoxication of other carnivores (Table 1).

The concentration of mercury in the fur of the present case was much higher than that found in the experimentally poisoned mink; however, the latter animals received mercury after seasonal growth of fur had been completed so that little or no mercury accumulation would have been expected.[20]

Rabies antigen was not demonstrated in the brain of this animal.

From previous studies of experimental methyl mercury poisoning of mink we stated that a diagnosis of mercury poisoning could be made in this species on the basis of mercury concentrations of greater than 5 ppm in brain and muscle tissue, together with appropriate clinical and pathological findings.[20] The present case appears to fulfill these requirements.

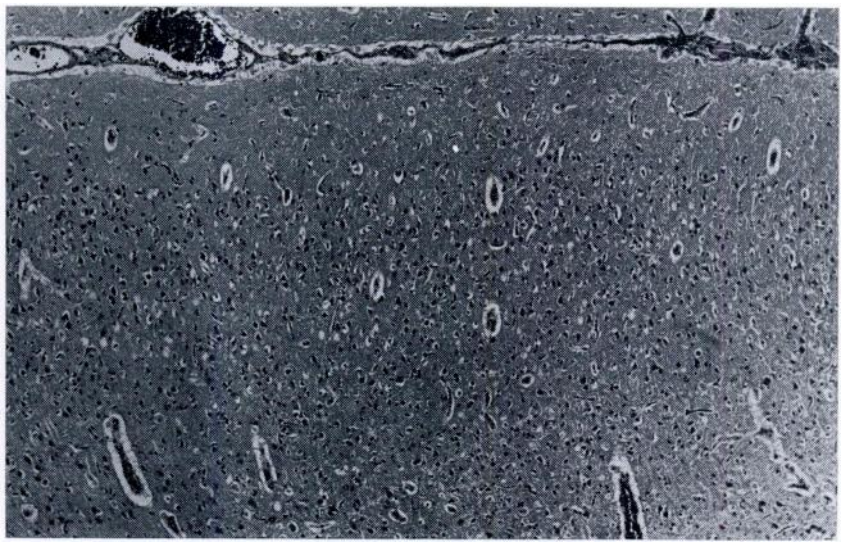

FIGURE 1. Cerebral cortex. Note increased cellularity and pyknosis of neurons in all laminae. H & E x 115

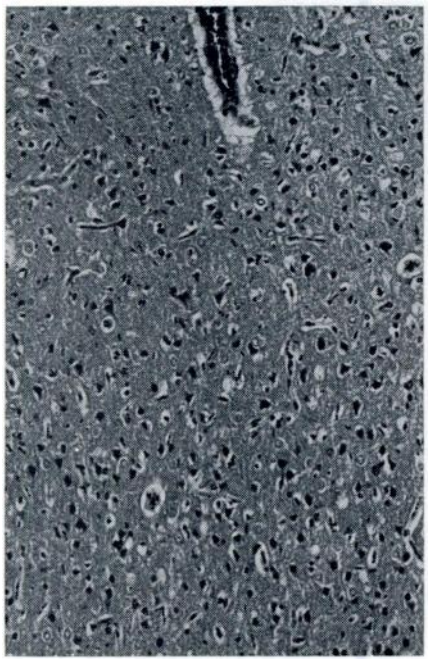

FIGURE 2. Central area of Fig. 1. Shrunken, angular necrotic neurons, with increased numbers of glial cells. H&E x 160.

FIGURE 3. Cerebellum. Pyknosis and loss of cells in the granular layer. H&E x 185

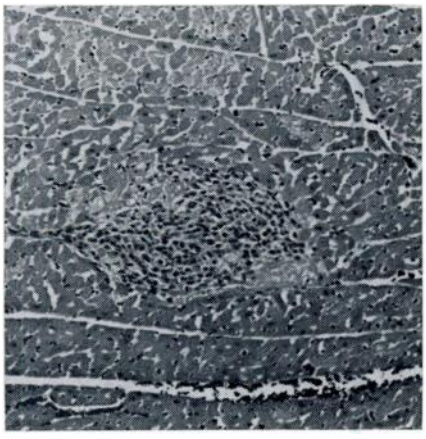

FIGURE 4. Myocardium—Focal areas of histiocytic infiltration in association with myocardial fibre degeneration. H&E x 60

The origin of the mercury in this case is unknown; however, the most likely source would appear to be contaminated fish from the nearby South Saskatchewan River. Under experimental conditions, the feeding of diets containing 1.8 ppm of mercury as methyl mercury chloride produced clinical intoxication and death of mink within as little as 59 days.[20] Fish from the immediate area of the river near where the mink was found may contain concentrations of mercury considerably greater than that level.[18,2]

High levels of mercury have been reported in tissues of Swedish otter (*Lutra lutra*) and mink[3,4,5,17] and the population of Swedish otter is reported to have decreased greatly during the past decade.[3,4] In some cases, Swedish mink appear to become contaminated with mercury through the consumption of seabirds rather than by direct consumption

TABLE 1. Total mercury concentration (ppm) in tissues of a wild mink, and mean concentrations reported in cases of alkyl mercury poisoning in carnivorous animals.

Species	Type of Poisoning	Hg (ppm)					Authority
		Liver	Kidney	Muscle	Brain	Fur	
Mink	Natural	58.2	31.9	15.2	13.4	34.9	present case
	Experimental*	24.3 ± 6.0	23.1 ± 4.1	16.0 ± 7.1	11.9 ± 3.9	1.5 ± 0.4	20
Ferret	Experimental*	53.8 ± 10.6	69.0 ± 10.8	34.3 ± 12.4	26.8 ± 14.3	————	11
Cat	Natural*	62.2 ± 21.6	19.8 ± 9.2	————	9.2 ± 1.6	45.9 ± 8.6	14
	Experimental*	74.3 ± 32.0	20.1 ± 10.8	————	12.8 ± 5.1	45.8 ± 34.3	14
	Experimental	39	31	27	18.0	————**	1
	Experimental	74.5 ± 7.3	37.1 ± 4.1	22.0 ± 3.5	18.5 ± 0.8	————	7
	Experimental	72.7 ± 12.0	27.0 ± 7.5	24.9 ± 6.1	17.4 ± 1.4	————	7

* mean and standard deviation calculated from published values

** fur contained fore than half of total body burden

of fish.[4] Fimreite[9] related that a trapper had discontinued trapping in an area of northern Ontario known to be contaminated with mercury, "because furbearers such as mink (*Mustela vison*) and otter (*Lutra canadensis*) had totally disappeared during the last years". High levels of mercury also have been reported in the hair of mink and otter from water systems in Georgia.[8] These reports together with the present case, suggest that mercury poisoning may be an unrecognized disease of wild mink and other piscivorous animals in areas where fish are contaminated with mercury. The lack of documented cases of poisoning may be due to difficulties in finding sick or dead wild animals or in diagnosing the disease, rather than due to a lack of cases of poisoning.

Acknowledgments

The aid of Professor Karl Borg who reviewed the manuscript and provided many helpful suggestions is gratefully acknowledged.

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Received for publication 24 October 1975