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Famphur Toxicosis in a Bald Eagle

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On 24 November 1983, an adult female bald eagle (*Haliaeetus leucocephalus* L.) was found unable to fly near Lewes, Delaware. She was kept overnight by U.S. Fish and Wildlife Service personnel at Prime Hook National Wildlife Refuge and transported to the Patuxent Wildlife Research Center, Laurel, Maryland, the following afternoon.

On clinical examination the eagle was recumbent, unable to move except for slight rotation of the head, and exhibited extensor rigidity with legs directed backward and wings slightly extended. Pupils alternately dilated and contracted and were unresponsive to light stimulation; corneal reflex was absent. Palpation revealed a mass in the crop. Packed cell volume was 54% and blood protoporphyrin was 17 μg/dl. No abnormalities were evident radiographically, except for the presence of two shot which appeared to be in the stomach.

An endotracheal tube was inserted and a large mass of feathers and avian skeletal remains was manually removed from the crop with a long-handled hemostat. Stomach gavage with lactated Ringers solution yielded additional feathers as well as a dark fetid fluid. Supportive treatment included 70 mg prednisolone (Henry Schein, Inc., Port Washington, New York 11050, USA) and 20 ml lactated Ringers solution intravenously, and 35 mg gentamycin (Gentavet®, Burns-Biotec, Inc., Omaha, Nebraska 68103, USA) intramuscularly. An additional 40 ml lactated Ringers was administered subcutaneously. The eagle died several hr after treatment was initiated.

At necropsy, the carcass was in good flesh with abundant subcutaneous fat and weighed 4,162 g. No external lesions were noted and there was no indication of traumatic head injury. A regurgitated mass of dark feathers and mucous was present in the oral cavity. No lesions were observed in the viscera except for a slight thickening of the right abdominal air sac with some adherent yellow caseous material. Stomach contents consisted of about 100 cc of feathers and small bird bones; one lead shot was also recovered. Air sac, liver, and lung were submitted for routine bacteriologic culture to the Maryland Department of Agriculture, Animal Health Laboratory, College Park, Maryland, but

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no significant growth was observed. Feathers and bones in stomach contents were from brown-headed cowbirds (*Molothrus ater* Boddgert) and European starlings (*Sturnus vulgaris* L.).

Although lead toxicosis was considered in the differential, the elevated hematocrit and the low blood protoporphyrin concentration did not support this diagnosis. Blood protoporphyrin in four apparently healthy captive bald eagles at the Patuxent Center averaged 43 μg/dl (range = 37–55 μg/dl). Clinical signs, physical examination, and presence of a full crop suggested acute toxicity. Concentrations of organochlorines (including DDT derivatives, dieldrin, heptachlor epoxide, chlordane, endrin, toxaphene, and PCB) in the carcass and brain were not elevated. Stomach content was checked for strychnine, compound 1080, avitrol, and starlicide, but results were negative. Lead residues in blood and liver were determined according to Hinderberger et al. (1981, Atomic Spectroscopy 2: 1–7) and Haseltine et al. (1981, Pestic. Monit. J. 15: 90–97), respectively. Results were 0.06 ppm lead in blood and 0.23 ppm (wet wt) in liver, much lower than residues reported from experimentally poisoned bald eagles (Hoffman et al., 1981, J. Wildl. Dis. 17: 423–431; Pattee et al., 1981, J. Wildl. Manage. 45: 806–810).

Brain cholinesterase (ChE) activity was determined to be inhibited by 85% using the method of Ellman et al. (1961, Biochem. Pharmacol. 7: 88–95) as modified by Hill and Fleming (1982, Environ. Toxicol. Chem. 1: 27–38). Brain ChE activity in the poisoned eagle was 2.5 μmoles acetylthiocholine iodide hydrolyzed/min/g, wet weight, compared with a mean of 16.7 μmoles/min/g in brains from three eagles which had died previously from other causes. Stomach content was analyzed for anti-ChE compounds by gas chromatography. A 10 g sample was extracted with methylene chloride, filtered, and concentrated on an evaporator. Famphur (phosphorothioic acid *O*-[4-(dimethylamino) sulfonyl] *O,O*-dimethyl ester) was quantified (1.9 ppm, wet wt) with a flame photometric detector using a 3% OV-101 column at 205 C. After the extract was cleaned up with a high pressure liquid chromatograph equipped with a fraction collector and an RP-8 column, famphur was confirmed by gas chromatography–mass spectrometry.

Famphur is applied to the backs of cattle for treatment of warbles (*Hypoderma* sp.). Laboratory studies have shown famphur to be quite toxic to birds, with an LD_{50} of about 2–10 mg/kg (Tucker and Crabtree, 1970, U.S. Fish and Wildl. Serv. Resource Publ. 84, 131 pp.; Schafer, 1972, Toxicol. Appl. Pharmacol. 21: 315–330). Primary famphur toxicosis has been reported in black-billed magpies (*Pica pica* L.), European robins (*Erithacus rubecula* L.), and a dunnock (*Prunella modularis* L.) in the United Kingdom (Felton et al., 1981, Vet. Rec. 108: 440). In the United States, primary famphur poisoning has occurred in black-billed magpies, and secondary poisoning was observed in two red-tailed hawks (*Buteo jamaicensis* Gmelin); one had eaten a magpie and the other had eaten a European starling (Henny et al., 1985, J. Wildl. Manage. 49: In press). Hill and Mendenhall (1980, J. Wildl. Manage. 44: 676–681) demonstrated that barn owls (*Tyto alba* Scopoli) which consume prey poisoned by famphur could succumb to secondary poisoning. It is possible the cowbirds and starlings found in the eagle's stomach were poisoned after consuming famphur in or near a feedlot, and the eagle was secondarily poisoned after feeding on them as carrion.

Within the continental United States, the bald eagle is endangered in 43 states and threatened in five. The population has been making a gradual recovery since the ban of DDT in 1972, but effects of other environmental contaminants are poorly understood. Secondary poisoning by organophosphates such as famphur could be

a potential mortality factor for the bald eagle and other raptors.

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Thyroid Adenoma and Ovarian Luteinization in an Aged Fisher (*Martes pennanti*)

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Fishers are mustelid fur bearers of economic importance found only in North America (Powell, 1981, Mamm. Species 256: 1–6). Most current literature on fishers deals with biology, ecology, and management (Hamilton and Cook, 1955, N.Y. Fish Game J. 2: 13–35; Kelley, 1978, Fisher (*Martes pennanti*) biology in the White Mountain National Forest and adjacent areas, Ph.D. Thesis, University of Massachusetts, Amherst, 178 pp.; Powell, 1981, op. cit.; Strickland et al., 1982, *In* Wild Mammals of North America, Chapman and Feldhamer (eds.), University Press, Baltimore, pp. 586–598) or on parasites in specific geographic locations (Meyer and Chitwood, 1951, J. Parasitol. 37: 320–321; Hamilton and Cook, 1955, op. cit.; Dick and Leonard, 1979, J. Wildl. Dis. 15: 409–412). Strickland et al. (1982, op. cit.) stated that parasitism in fishers is frequent but does not appear to be a serious mortality factor. Very little information is available on naturally occurring diseases of fishers. In this report we describe the pathologic findings in a 11-yr-old female fisher that had been raised in captivity since shortly after birth.

Two female fisher kits were found in the spring of 1973 during a logging operation in the White Mountains of New Hampshire. Both were donated to the Science Center of New Hampshire on Squam Lake in 1974 as part of an education ex-

FIGURE 1. Large cyst (arrow) in caudal part of left thyroid of an 11-yr-old fisher.

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