
WINTER MORTALITY OF BALD EAGLES ALONG THE LOWER WISCONSIN RIVER

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Abstract

Unusual morbidity and mortality of bald eagles (*Haliaeetus leucocephalus*) wintering in two counties along the lower Wisconsin River, Wisconsin, began in 1994-1995 with the deaths of at least 14 eagles. Nine eagles were found dead, five were collected alive but died within 2 days, and two additional birds were found sick, then rehabilitated and released after 2.5 mo. Bald eagles at roosts from 10-65 km upriver and 10-150 km downriver from the affected region and elsewhere in the state were not found sick or dead. Beginning in 2000-2001, after a hiatus of 4 yr, during which eagle populations in the region were carefully monitored, similar bald eagle morbidity and mortality has recurred each winter. The area of concern has expanded to eight counties, with mortality events occurring primarily in January and February, and infrequent cases found from late November to early April. Of 85 bald eagles that have died in the target area within the appropriate time frame, 63 have been necropsied; some evaluations are still in progress. Sick eagles present in good body condition, with weakness, incoordination, tremors, vomiting and seizures. Snow or litter around dead eagles is often disturbed, consistent with observations of terminal seizures. Eagles brought into veterinary and rehabilitation facilities frequently have repetitive seizures, refractory to medication, over hours or days before death or euthanasia. No other avian or mammalian species have been involved. At gross necropsy, no consistent abnormalities have been found. By light microscopy, a minimum of 40 affected eagles had mild to severe multifocal to diffuse hepatocellular cytoplasmic vacuolation. Special stains revealed the presence of lipid in the vacuoles of a subset of affected livers. Vasculitis and microhemorrhages in the brain have been noted; it is unclear if the hemorrhages are a consequence of the seizures. This suite of lesions has not been seen in more than 4000 bald eagles from throughout the United States. The characteristic lesions of avian vacuolar myelinopathy, initially noted in 1994-1995 in Arkansas, have not been seen in Wisconsin bald eagles.

Extensive laboratory investigations on dead and sick birds have been inconclusive. Investigations have focused on agrochemical and veterinary drug use, contaminants associated with the Badger Munitions Plant, livestock mortality events, fish kills, and forage fish species available to eagles. Toxicologic tests have ruled out heavy metals, organophosphorus and carbamate pesticides, organochlorines, 4-aminopyridine, white phosphorus, strychnine,

anticoagulants, and barbiturates as causative agents. Additional compounds, including sodium fluoroacetate and cyanide, have not been detected in a limited number of samples tested. Aerobic and anaerobic cultures, fungal cultures, viral cultures, assays for exposure to viruses and protozoal parasites, and tests for biotoxins have not found an etiology. Agricultural fields, where eagles feed on pig and duck carcasses, often with crows and turkey vultures, have been examined, farmers interviewed, and samples of pigs and ducks collected for evaluation. In 1995, a mortality event in the same area involving rock doves was investigated; the birds had severe, nonsuppurative encephalitis caused by pigeon paramyxovirus 1. There is no correlation with fish kills, and heavy metals and organochlorines in fish were below toxic concentrations. Forage fish have been tested for levels of thiaminase, with one species, gizzard shad, testing very high. The hypothesis that the syndrome is caused by a severe thiamine deficiency as a result of feeding largely on gizzard shad remains to be adequately tested. Evaluation of test results continues, and repeated multi-agency workshops allow for generation of new hypotheses.