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Lead Poisoning of Sandhill Cranes (*Grus canadensis*).¹

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ABSTRACT

Two wild and two captive sandhill cranes (*Grus canadensis*) were diagnosed by National Wildlife Health Laboratory personnel as having died from lead toxicity. Ingestion of lead fishing weights by the wild cranes and of unspent .22 caliber shell cartridges by the captive cranes were responsible for these deaths. One crane force-fed lead pellets showed an increase of blood lead levels from 0.77 ppm to 23.8 ppm (wet weight) just before its death 15 days following exposure. Liver lead concentrations of sandhill cranes dying of causes other than lead toxicity are presented.

INTRODUCTION

Lead poisoning has been well described in many species of waterfowl but reports of lead toxicity in cranes are rare. Captive sandhill cranes (*Grus canadensis*) have been known to die from ingesting lead-based paint (Kennedy et al. 1977). The feeding habits of cranes in the wild may also place them in danger of ingesting lead shot. In addition, the tendency of cranes to pick up shiny objects such as shiny lead sinkers or unspent ammunition cartridges also places them in danger of ingesting objects containing lead.

The present paper reports the loss of two wild and two captive sandhill cranes to lead poisoning. In addition, we present information on a sandhill crane experimentally dosed with lead.

MATERIALS AND METHODS

Sandhill cranes found dead in the wild and in captivity were submitted to the National Wildlife Health Laboratory (NWHL) for necropsy. The birds had been chilled when shipments could be made immediately, or frozen for later analysis if shipment was delayed. Upon arrival at the NWHL, we examined carcasses using standard necropsy procedures. Selected tissues were examined for pathogenic bacteria, viruses, and parasites. Tissues for microscopic examination

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were fixed in 10% formalin, stained with either the hematoxylin and eosin or Ziehl-Neelsen carbol fuchsin stains. Livers, spleens, or kidneys were analyzed for lead levels by atomic absorption spectrophotometry. All lead values given are expressed in ppm, wet weight, unless otherwise indicated. Liver lead concentrations greater than 6 ppm, wet weight, or about 24 ppm, dry weight, were used as indicative of lead toxicity when accompanied by pathological findings.

For an experimental dosing study, an adult sandhill crane was given per os an initial dosage of 613 mg of lead (three No. 4 lead shot). Three weeks later an additional dose consisting of a larger lead pellet (59 gms) was given per os, and another three No. 4 lead shot were provided with its grit. Corn, game bird ration, and water were provided ad libitum. Blood samples were taken periodically throughout the study to monitor the blood lead concentrations. At necropsy, portions of the liver, spleen, kidney, brain, and bone were saved and analyzed for lead. Feces collected during the trial were cultured for *Salmonella* sp. and examined for parasitic ova by standard fecal flotation techniques.

RESULTS

Field Cases.

Case One: A female sandhill crane was found moribund in March 1976 near the Platte River in south central Nebraska and died immediately after capture. This crane was severely emaciated with marked pectoral atrophy, had very little visceral fat, and no subcutaneous fat. A portion of a lead fishing sinker was found in the gizzard contents. Analysis of tissues revealed high levels of lead to be present in the liver (23.0 ppm), kidney (29.8 ppm), and spleen (24.4 ppm).

Virological examinations were negative for Newcastle disease and avian influenza viruses. No pathogenic bacteria were found. The cause of the morbidity and subsequent death was determined to be lead poisoning.

Case Two: An adult female sandhill crane was found in December 1979 on its wintering grounds at the Aransas National Wildlife Refuge, Texas, in a weakened condition and unable to fly. It was euthanized and submitted to our laboratory for necropsy. The bird was extremely thin, weighing only 2.3 kg. Pectoral muscles were atrophied, no subcutaneous fat was present, and the coronary fat was undergoing serous atrophy. Intestines were discolored by bile. The gizzard contained gravel, plant remains, and a portion of a lead fishing weight (0.7 gm). The lead concentration (dry weight) in the kidney was 113.4 ppm and in the liver 258.8 ppm. Lead poisoning was diagnosed as cause of the moribund condition of this crane.

Of 17 sandhill cranes found in spring 1977 in Nebraska and necropsied in the field, three livers had lethal lead levels present (greater than 6 ppm). Unfortunately, cause of death of these birds was undetermined since accompanying lesions were not noted. Six of the birds had one or more lead shot present in the gizzard.

The livers of 31 other sandhill cranes necropsied at the NWHL were examined for lead concentrations. The following lead levels (dry weight) were found. One, an adult female captured at the Jasper-Pulaski Wildlife Area, Indiana, in October 1977, died from mycotic pneumonia and had an elevated liver lead concentration of 12.6 ppm. A crane that died from unknown causes at the Merced

National Wildlife Refuge in California had a liver lead level of less than 0.3 ppm. An immature female sandhill illegally shot in Wisconsin in October 1979 had a lead residue of 1.9 ppm in its liver. Nineteen lesser sandhill cranes found dead near a power line on March 31, 1981, on the Platte River near Kearney, Nebraska, had liver lead levels below 1.0 ppm as did three others found on the Platte in the spring of three other years (1977, 1979, and 1980). Two sandhill cranes that died from lightning strikes in Nebraska in March 1978 had liver lead levels of 1.6 ppm and 1.2 ppm and another crane found dead from an unknown cause on the Platte River had a lead level of 4.3 ppm detected in its liver. Three crane carcasses found on the Platte River in March 1982 had liver lead levels of 5.6 ppm, 4.3 ppm, and 2.6 ppm. Trauma was determined as cause of death for the first two cases and myositis diagnosed in the third crane.

Captive Cases.

Two of three captive-reared, juvenile sandhill cranes kept for public display at a county park near Stevens Point, Wisconsin, died within five days of each other six months after their arrival at the park.

At necropsy, both birds were in excellent body condition with good deposits of subcutaneous and mesenteric fat. One weighed 4.2 kg and the other 4.6 kg.

Gizzard pads of both birds were leathery. One crane's gizzard contained two macerated, unspent .22 caliber rifle cartridges. The gallbladder of this bird was engorged with bile. Chemical analysis of the liver for lead was 30.0 ppm and copper was 6.7 ppm (wet weight).

The second crane had obvious bile staining of the vent area and the gizzard pads were bile-stained. A well-worn, copper-coated penny was found in the gizzard contents. Lead concentration of the liver was 24.0 ppm and the copper level was 9.5 ppm (wet weight). Tests conducted on spleens, intestines, livers, and kidneys were negative for pathogenic bacteria. Histological examination showed a mild nephrosis and hepatic hemosiderosis. The final diagnoses for both captive cranes was lead poisoning.

Experimental Case.

A crippled adult female greater sandhill crane, found in the wild with a portion of its left wing missing was submitted by the International Crane Foundation to the NWHL on July 2, 1976. The wing stub had healed, but weight of the bird (3.2 kg) was less than normal. On July 5, three No. 4 lead shot (613 mg) were given per os (crane weight 3.4 kg). Blood lead levels remained low (< 1.0 ppm) for 18 days and the bird continued to gain weight. On July 23, the crane weighed 3.8 kg and an additional dosage of lead, a 59 gm pellet and three No. 4 lead shot, was administered. Within 6 days blood lead levels increased from 0.77 ppm to 9.7 ppm and by 10 days to 15.0 ppm. By August 7, the body weight was down to 3.6 kg and blood lead levels had risen to 23.8 ppm. Respiratory difficulty was observed at this time and the bird had difficulty holding its head erect. The right wing was drooping noticeably. The following day the crane was found dead and a necropsy was performed.

Examination of the carcass showed severe pectoral muscle atrophy. The gizzard was one-half its normal size. The gizzard pads were hardened and easily

pulled from the mucosa. The gallbladder was slightly enlarged and liver stained from bile retention. No significant bacteria, viruses, or parasites were found upon laboratory assays.

Lead concentrations in tissues were liver 26.3 ppm, spleen 10.6 ppm, kidney 13.2 ppm, brain 12.3 ppm, and bone 37.2 ppm. No acid fast intranuclear inclusion bodies similar to those often found in lead poisoned mallards were found upon microscopic examination of the liver and kidney (Locke et al. 1966).

DISCUSSION

Lead concentrations in the livers of sandhill cranes we diagnosed as dying from lead poisoning are compatible with that reported by Kennedy et al. (1977) from a crane that died following ingestion of lead-based paint. Similarly, the level of lead concentrations from these sandhill cranes has been reported for other avian species dying of lead toxicity (Bagley et al. 1967, Clausen and Wolstrup 1979, Cook and Trainer 1966, Del Bono and Bracha 1973). Common loons (*Gavia immer*) that had accidentally ingested fishing sinkers had liver lead concentrations similar to those we detected in the cranes (Locke et al. 1982). The sandhill crane found in the wild and subsequently euthanized (Case Two) indicates that some individuals may develop high levels of lead in the tissues before finally dying. This crane was obviously impaired as was evidenced by its severely emaciated state.

Diagnoses of lead poisoning in two captive cranes on display in a park emphasizes the importance of informing the public of the potential hazard of throwing shiny objects containing lead into pens where such birds are retained.

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