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Pancreatitis in wild zinc-poisoned waterfowl

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Four waterfowl were collected in the Tri-State Mining District (Oklahoma, Kansas and Missouri, USA), an area known to be contaminated with lead, cadmium and zinc (Zn). They were part of a larger group of 20 waterfowl collected to determine the exposure of birds to metal contamination at the site. The four waterfowl (three *Branta canadensis*, one *Anas platyrhynchos*) had mild to severe degenerative abnormalities of the exocrine pancreas, as well as tissue (pancreas, liver) concentrations of Zn that were considered toxic. The mildest condition was characterized by generalized atrophy of exocrine cells that exhibited cytoplasmic vacuoles and a relative lack of zymogen. The most severe condition was characterized by acini with distended lumens and hyperplastic exocrine tissue that completely lacked zymogen; these acini were widely separated by immature fibrous tissue. Because the lesions were nearly identical to the lesions reported in chickens and captive waterfowl that had been poisoned with ingested Zn, and because the concentrations of Zn in the pancreas and liver of the four birds were consistent with the concentrations measured in Zn-poisoned birds, we concluded that these waterfowl were poisoned by Zn. This may be the first reported case of zinc poisoning in free-ranging wild birds poisoned by environmental Zn.

Introduction

Although zinc (Zn) poisoning has been reported in captive wild birds that have ingested bits of Zn as galvanized hardware or coins (Droual *et al.*, 1991; Zdziarski *et al.*, 1994), it has not been described convincingly in free-ranging wild birds. Gasaway & Buss (1972) suggested that waterfowl in the Coeur d'Alene River Basin might be dying from exposure to Zn; although this remains a possibility, later studies at that site (Sileo *et al.*, 2001) identified lead (Pb) as the cause of death in metal-poisoned waterfowl. Three Canada geese (*Branta canadensis*) and a mallard (*Anas platyrhynchos*) recently collected from the Tri-State Mining District had pancreatic lesions similar to those reported in captive waterfowl experimentally or accidentally poisoned with Zn (Dewar *et al.*, 1983; Kazacos & Van Vleet, 1989; Zdziarski *et al.*, 1994). These four wild waterfowl are the subject of this report. Dr James Carpenter of the College of Veterinary Medicine at Kansas State University has recently diagnosed zinc poisoning in a trumpeter swan (*Cygnus buccinator*) from the Tri-State Mining District (James Carpenter, personal communication, 2003).

The Tri-State Mining District covers about 3000 km^2 and includes parts of Ottawa County in Oklahoma, Cherokee County in Kansas, and Jasper and Newton Counties in Missouri (Gibson, 1972). The District extends from the northwestern edge of the Ozark Uplift across rolling prairie west to the Neosho River. The area has been mined from about the 1850s to the 1970s with the peak of activity in the first half of the twentieth century. The sulfide forms of lead (galena) and zinc (sphalerite) and to a lesser extent zinc carbonate (smithsonite), lead carbonate, lead phosphate (pyromorphite) and other less abundant ores were mined (Gibson, 1972; Weidman, 1932). Phillips & Lincoln (1930) reported the deaths of many

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mallards, pintails and teal in February 1923, on Spring River, near Riverton, KS. These authors concluded that the well-filled crops and other signs observed in the waterfowl were consistent with Pb poisoning. The suggested source of the Pb was mine refuse that had been dumped into the river. Two samples of sediment from Spring River, taken in 1987 near the mouth of Short Creek, contained an average of 23 000 mg/kg Zn and 1600 mg/kg Pb (Ferrington, 1989), suggesting that waterfowl might have been exposed to toxic concentrations of Zn near Riverton.

Although Zn poisoning has not yet been reported in free-ranging wild birds, it has been studied in other birds. Dewar *et al.* (1983) experimentally poisoned chickens and reported lesions of the exocrine pancreas including dilation of acinar lumina, cytoplasmic vacuolation, cytoplasmic globule formation, necrosis, numerous mitotic figures and interparenchymal fibrosis. In an ultrastructure study of Zn toxicity in Pekin ducklings, Kazacos & Van Vleet (1989) reported apoptosis; attenuated, cuboidal, atrophic acinar cells; interstitial fibrosis; and the formation of duct-like structures embedded in fibrous connective tissue. They found only minimal inflammatory response and the islets were normal. Similar lesions were reported in four species of captive diving ducks that had ingested pennies (Zdziarski *et al.*, 1994).

Methods

The birds were collected under the authority of permits from the US Fish and Wildlife Service and from the states of Oklahoma and Kansas. The geese were collected on 8 February 2001 and the mallard on 9 February 2001.

The four waterfowl collected were part of a larger study to determine whether birds from the site were exposed to toxic concentrations of Zn, Pb, and cadmium. The primary target organs (liver, kidney, spleen, and pancreas) of these metals were collected for histopathology examination and metal analyses. This study was not designed to determine other potential causes of morbidity in wild birds and did not include ancillary microbiological, virological, and parasitological laboratory tests.

One of the geese was weak and was captured alive by a bird dog. The two other geese and the mallard were active and were collected by shotgun and steel pellets. The impaired goose (number 1) was captured near an area of surface subsidence about 3 km west of Baxter Springs, Cherokee County, KS. It was examined clinically at the collection site and then was euthanized with carbon dioxide. Geese 2 and 3 were collected at Doubthat Bridge in Ottawa County, OK. The mallard was collected near the Atlas Chat Pile, also in Ottawa County, OK. The term 'chat' refers to the crushed rock remaining after the recoverable Zn and Pb have been removed.

Immediately after death, about 1 ml blood was taken by cardiac puncture with a needle and a vacutainer (both treated with lithium heparin) from each of the four birds. The haematocrit was measured in microhaematocrit tubes after centrifugation and the remaining blood was divided into two portions. One portion was frozen in liquid nitrogen and stored in an ultralow freezer until it was analyzed for activity of delta aminolevulinic dehydratase (ALAD), as in Pain (1989) and Henny *et al.* (2000). ALAD activity is a sensitive indicator of exposure to Pb. A unit of ALAD activity was defined as a 0.001 increase in absorbance at 555 nm with a 1.0 cm light path per milliliter of erythrocytes per hour at 38°C.

The second portion of blood was frozen and saved for metal analysis. Portions of the liver, pancreas, kidney and spleen of each bird were preserved in formalin and the remainders of the liver, pancreas and kidney were saved for chemical analysis. The gizzard contents were examined for ingested shot and other metallic artifacts but none were found. Concentrations of metals were determined by inductively coupled plasma-atomic emission spectrometry (Zn, cadmium and copper (Cu)) or by inductively coupled plasma-mass spectrometry (Pb) at the Research Triangle Institute (Research Triangle Park, NC, USA). Concentrations measured in reference materials, spiked samples and replicates were found to be within acceptable limits by the Patuxent Analytical Control Facility (US Fish and Wildlife Service, Laurel, MD). The formalinized tissues were submitted to the College of Veterinary Medicine, University of Wisconsin for histological processing. Sections stained by the haematoxylin and eosin method were examined by light microscope.

Results and Discussion

The impaired goose (Goose 1) was collected in Kansas within a few kilometers of Spring River, close to Riverton, where the waterfowl die-off was reported in 1923 (Phillips & Lincoln, 1930). This goose was a hatch-year male with a bursa of Fabricius. Its legs had reduced motor function; a foot prick did not produce a response. Its wings drooped and it had reduced wing proprioception. Gasaway & Buss (1972) and Zdziarski *et al.* (1994) reported paralysis of the legs as one of the few clinical signs of Zn poisoning in mallards. Grandy *et al.* (1968) reported that mallards poisoned with Zn shot showed reduced wing proprioception as well as a loss of control of their leg muscles. The goose's breastbone was prominent and it had very little subcutaneous or intra-abdominal fat, which is consistent with the weight loss observed by Levengood *et al.* (1999) and Zdziarski *et al.* (1994). The

goose's mouth, tongue and phonations were normal. An enlarged spleen was the only internal lesion. Its ALAD activity was 208 u and its haematocrit was 54.

Goose 2 was a female in normal body condition with no bursa of Fabricius. It had ALAD activity of 89, and a haematocrit of 39. There was a healed wound in its intestines, probably a penetrating gunshot wound, but no other gross lesions. Goose 3 was a male in normal body condition with no bursa of Fabricius, an ALAD activity of 38 and a haematocrit of 45. The only gross lesion noted at necropsy was a firm and fibrotic pancreas with a haemorrhagic surface. The mallard was a female with ALAD activity of 5 and a haematocrit of 51. It had a granuloma in the proventricular adventitia, but no other gross lesions.

Although erosion of the lining of the gizzard of captive birds experimentally or accidentally poisoned with Zn has been reported (Dewar *et al*, 1983; Droual *et al.*, 1991; Zdziarski *et al.*, 1994), the linings of the gizzards of these four waterfowl were unremarkable.

Henny *et al.* (2000) reported ALAD activity as 156 ± 68 (mean $\pm$ standard deviation) in adult reference mallards and 183 ± 54 in adult reference Canada geese. These reference values suggest that the mallard and geese 2 and 3 had had elevated exposure to Pb. Both Zn poisoning and Pb poisoning have been reported to depress mean haematocrits of waterfowl slightly, but responses of individual waterfowl are variable. Haematocrits of adult reference Canada geese are 43 ± 3.3 and those of adult reference mallards are 45 ± 2.5 (Henny *et al.*, 2000). Judged by these values, Canada goose 2 had a slightly lower than normal haematocrit.

Microscopic examination revealed mild to severe degenerative abnormalities of the exocrine pancreas in each of the four birds. The mildest condition, which occurred in Goose 1, was characterized by generalized atrophy of exocrine cells, a mild lack of zymogen, and empty cytoplasmic vacuoles approximately 5 μm in diameter; also present were a few scattered, prominent, individual cytomegalic exocrine cells with larger (approximately 10 to 15 μm diameter) but otherwise similar cytoplasmic vacuoles. The most severe condition occurred in the mallard and was characterized by widely separated acini of hyperplastic exocrine tissue that completely lacked zymogen and had distended lumens. Separating these acini were prominent bands of immature fibrous tissue. Mitotic figures were apparent (Figure 1).

The severity of the lesions in the other two geese (geese 2 and 3) was intermediate, with subcapsular and interacinar fibrosis being a prominent change; also present were a mild lack of zymogen, increasingly prevalent karymegaly, and cytoplasmic vacuolation (Figures 2 and 3). Inflammation was limited to a few interstitial granulocytes and lymphocytes. Apoptosis was not a prominent feature and islets were not affected. Although the lesions in the geese were less severe than those in the mallard, they may have been more chronic. In a study with ducklings of the relation between

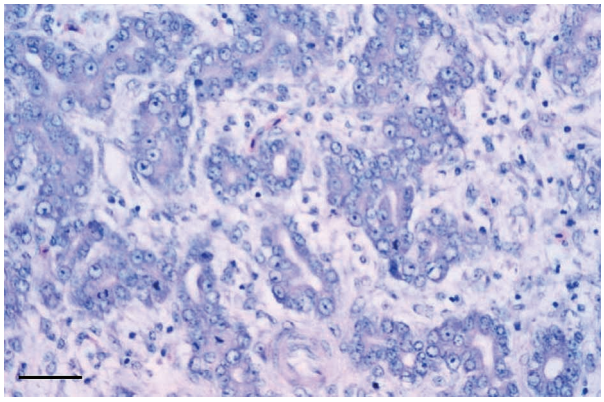

Figure 1. *Mallard pancreas that had a concentration of 440 mg/kg dry weight of Zn. Note widely separated acini with disorganized, misshapen cells that lack zymogen. Mitotic figures, marked variation in nuclei size, and prominent acinar lumens are evident. Scale bar = 40 μm.*

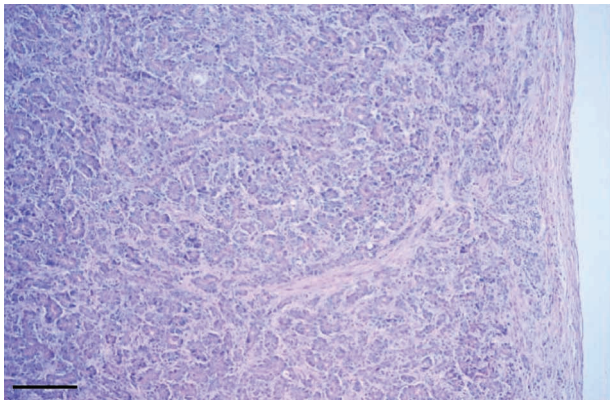

Figure 2. *Canada goose pancreas that had a concentration of 2400 mg/kg dry weight of Zn. Note the disorganized acini and prominent subcapsular and interacinar fibrosis. Scale bar = 150 μm.*

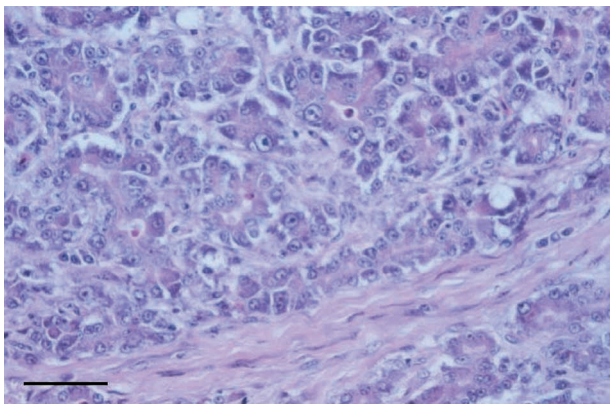

Figure 3. *Close up of Figure 2. Note disorganized, misshapen cells, marked variation in nuclei size, cytoplasmic vacuoles, and interacinar fibrosis. Scale bar = 40 μm.*

dietary selenium and vitamin E and silver, Cu, cobalt, tellurium cadmium and Zn, Van Vleet *et al.* (1981) reported that excessive Zn caused pancreatic necrosis and fibrosis, but the other metals did not. Several other microscopic lesions of minimal to mild severity occurred in the geese and mallard. The cause of these was unknown; they might have been a consequence of Zn intoxication, or unrelated. Goose 2 had moderately severe multifocal hepatic necrosis, minimal nephrosis, mild renal haemosiderosis, and fibrinoid necrosis of the media of a splenic artery; although these changes are not specific to any particular etiology, they are sometimes associated with Pb poisoning in waterfowl (Sileo *et al.*, 2001). However, the concentrations of Pb in the tissues of this goose were not remarkable (Table 1). The mallard had marked hepatocellular vacuolar degeneration, also of undetermined cause.

Zn is bound to metallothioneins found in the liver, kidney, intestinal mucosa and especially in the pancreas (Oh *et al.*, 1979). Animals usually regulate Zn effectively, and consequently hepatic concentrations of Zn do not vary proportionately with dietary exposure. For example, the whole body concentrations of Zn in songbirds from a site severely contaminated with Zn from smelting were less than 20% greater than those of songbirds from a reference site, although the A1 soil horizon of that contaminated site had more than 10 times the Zn concentration of the reference site (Beyer *et al.*, 1985). In experimental studies on chickens, hepatic Zn concentrations (wet weight) remained constant as the dietary concentration was increased from 37 to about 110 mg/kg (Stahl *et al.*, 1989).

Table 1. *Concentrations of Zn, Pb, Cd and Cu detected in tissues of Canada geese and a mallard from the Tri-State Mining District of Kansas, Oklahoma and Missouri*

Metal and bird	Concentration in tissue (mg/kg dry weight)			
	Liver	Kidney	Pancreas	Blood
Zn				
Goose 1	2900	970	260	160
Goose 2	1000	510	2400	39
Goose 3	1100	560	2300	53
Mallard	280	220	440	32
Pb				
Goose 1	2.1	4.5	0.31	1.7
Goose 2	2.9	11	0.68	2.2
Goose 3	2.2	6.5	1.1	2.0
Mallard	3.8	51	2.1	5.9
Cadmium				
Goose 1	3.7	12	0.24	<0.1
Goose 2	12	126	4.1	<0.1
Goose 3	8.2	41	2.3	<0.1
Mallard	0.42	3.4	0.14	<0.1
Cu				
Goose 1	19	21	<0.1	1.9
Goose 2	16	104	14	2.2
Goose 3	50	23	9.8	2.1
Mallard	21	150	4.9	1.6

Homeostatic mechanisms fail, however, at extremely high Zn concentrations. Hepatic Zn increased more than 10-fold in that study, when the dietary concentration was increased to about 2200 mg/kg (Stahl *et al.*, 1989). In mallards, liver concentrations (wet weight) increased from 54 to 401 mg/kg Zn as the dietary concentration increased from control concentrations to 3000 mg/kg (Gasaway & Buss, 1972). The proportional increase in Zn concentrations in kidneys and pancreases was greater than that in livers of dosed birds (Gasaway & Buss, 1972; Levengood *et al.*, 1999). Zn concentrations were higher and more variable in the pancreas than in the liver of Zn-poisoned birds and, when dietary concentrations of Zn were reduced, Zn concentrations remained longer in the pancreas compared with other tissues examined (Williams *et al.*, 1989). Tissue Zn concentrations in chickens drop rapidly after exposure returns to normal (Oh *et al.*, 1979). The half-lives of Zn in metallothionein were estimated as 2.3 days in the pancreas, 1.5 days in the liver and 0.9 days in the kidney (Oh *et al.*, 1979).

Although tissue concentrations of metals reported in wild waterfowl are quite variable (Di Giulio & Scanlon, 1984) we can draw some conclusions about the concentration detected in the four waterfowl in our study. The concentrations of Zn in the tissues of these geese were many times the values for control birds in toxicological studies (Table 2) and the hepatic concentrations were comparable with those in waterfowl killed by Zn in laboratory studies or accidentally killed by ingesting zinc pennies in zoos (Table 2). The concentrations of Pb (Pain, 1996) and cadmium (Furness, 1996) in the geese tissues were well below those associated with histological lesions. The concentrations of Zn in the tissues of the mallard were elevated, but were not in the range associated with death. Concentrations of Pb in the kidney and blood of the mallard also were elevated.

Zn exerts its toxicity partially by interfering with Cu metabolism (National Research Council, 1980). Geese fed a diet adequate in Cu were reported to have hepatic Cu concentrations of 6.0 to 26 mg/kg wet weight (about 20 to 97 mg/kg dry weight) and renal Cu concentrations 3.0 to 9.0 mg/kg wet weight (about 10 to 30 mg/kg dry weight) (Puls, 1994). Stahl *et al.* (1989) found that exposure to high Zn concentrations in chickens reduced hepatic Cu, and Levengood *et al.* (1999) found that exposure to high Zn concentrations in mallards reduced hepatic Cu concentrations and increased renal Cu concentrations. Both an increase (Levengood *et al.*, 1999) and a decrease (Stahl *et al.*, 1989) in Cu concentrations in the pancreas have been reported in response to exposure to high Zn concentrations. Goose 3 had normal Cu concentrations in the liver and kidney, based on the criteria of Puls (1994). Goose 1, however, had an

Table 2. *Concentrations of Zn in tissues of reference waterfowl and waterfowl experimentally or accidentally poisoned by Zn*

Waterfowl	*n*	Concentration in tissue (mg/kg dry weight[a]) Liver	Kidney	Pancreas
Reference values				
Control Canada geese (Hoffman *et al.*, 2000), minimum and maximum	3	110 to 180	50 to 59	
Control mallards (Levengood *et al.*, 1999), mean	20	210	90	290
Control mallards (Gasaway & Buss, 1972), mean (standard deviation)	6	180 (57)	90 (17)	300 (120)
Lethally dosed waterfowl				
Zn-poisoned mallards (Levengood *et al.*, 1999), mean	20	1300	1000	7300
Zn-poisoned mallards (Gasaway & Buss, 1972), minimum and maximum	24	1100 to 1600	1000 to 1700	4200 to 8900
Accidentally poisoned waterfowl (Zdziarski *et al.*, 1994), minimum and maximum	6	707 to 1827		

[a] Published concentrations expressed as wet weights were converted to dry weight assuming 70% moisture.

abnormally low hepatic Cu concentration of 19 mg/kg and the Cu concentration in its pancreas was below detection limits. Goose 2 had an abnormally low hepatic Cu concentration of 16 mg/kg and an abnormally high renal Cu concentration of 104 mg/kg. The hepatic Cu concentration in the mallard was low or normal (21 mg/kg) and its renal Cu concentration was much higher (150 mg/kg) than normal. These abnormal Cu concentrations in tissues of Goose 1, Goose 2, and the mallard are consistent with a diagnosis of Zn poisoning.

Vacuolar change, atrophy, and fibrosis of an exocrine pancreas are common in poultry and considered idiopathic or associated with selenium deficiency or zinc toxicity (Goodwin, 1996). However, in our experience, atrophy and fibrosis of exocrine pancreas is uncommon in free-ranging waterfowl. Because the lesions were virtually identical to the lesions reported in chickens (Dewar *et al.*, 1983) and waterfowl (Grandy *et al.*, 1968: Van Vleet *et al.*, 1981; Kazacos & Van Vleet, 1989; Levengood *et al.*, 1999; Zdziarski *et al.*, 1994) that had been experimentally or accidentally poisoned with ingested Zn, and because the concentrations of Zn in the pancreases and livers of the four birds were significantly elevated (Table 1), we conclude that Zn was responsible for the pancreatic lesions and that these waterfowl were poisoned by Zn.

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RÉSUMÉ

Pancréatite chez des palmipèdes empoisonnés par le zinc

Quatre palmipèdes ont été ramassés dans le district Tri-State Mining (Oklahoma, Kansas, Missouri, USA), une zone bien connue pour être contaminée par le plomb (Pb), le cadmium (Cd) et le zinc (Zn). Ils faisaient partie d'un groupe de 20 palmipèdes capturés pour déterminer l'exposition des oiseaux de ce site, à la contamination par les métaux. Les quatre palmipèdes (*3 Branta canadensis, 1 Anas platyrhynchos*) ont présenté des anomalies dégénératives faibles à importantes du pancréas exocrine, ainsi que des concentrations en Zn des tissus (pancréas et foie) considérés comme toxiques. Le cas le moins sévère a été caractérisé par une atrophie généralisée des cellules exocrines qui présentaient des vacuoles cytoplasmiques et un manque relatif de zymogène. Le cas le plus sévère a été caractérisé par des acini avec des ouvertures distendues et un tissu exocrine en hyperplasie qui était totalement dépourvu de zymogène ; ces acini étaient largement séparés par du tissu fibreux immature. Du fait que les lésions étaient presque identiques à celles décrites chez le poulet et les palmipèdes en captivité qui avaient été empoisonnés par ingestion de Zn, et du fait que les concentrations en Zn au niveau du pancréas et du foie des quatre palmipèdes étaient compatibles avec les concentrations en Zn mesurées chez les oiseaux empoisonnés, il a été conclu que ces palmipèdes avaient été empoisonnés par le Zn. Ce doit être le premier cas rapporté d'empoisonnement par le zinc chez des oiseaux sauvages en liberté dû aux conditions environnementales.

ZUSAMMENFASSUNG

Pankreatitis in wild-lebendem, durch Zink vergiftetem Wassergeflügel

Vier Wasservögel wurden in dem Drei-Staaten-Minen-Distrikt (Oklahoma, Kansas, Missouri, USA), einem Gebiet, von dem bekannt ist, dass es mit Blei (Pb), Cadmium (Cd) und Zink (Zn) kontaminiert ist, eingesammelt. Sie waren Teil einer größeren Gruppe von 20 Wasservögeln, die entnommen worden waren, um die Exposition der Vögel mit Metallkontaminationen in diesem Gebiet zu bestimmen. Die vier Wasservögel (3 *Branta canadensis, 1 Anas platyrhynchos)* hatten gering- bis hochgradige degenerative Veränderungen des exokrinen Pankreasgewebes sowie Zn-Konzentrationen in den Organen (Pankreas, Leber), die als toxisch zu bezeichnen waren. Die mildesten Veränderungen waren durch eine generalisierte Atrophie der exokrinen Zellen, die zytoplasmatische Vakuolen und einen relativen Mangel an Zymogen aufwiesen, charakterisiert. Die schwersten Alterationen waren gekennzeichnet durch Azini mit ausgeweitetem Lumen und hyperplastisches exokrines Gewebe, dem Zymogen vollständig fehlte; diese Azini waren durch unreifes fibröses Gewebe deutlich voneinander getrennt. Da die Läsionen nahezu identisch waren mit Veränderungen, die bei durch Zn-Aufnahme vergifteten Hühnern und in Gefangenschaft gehaltenem Wassergeflügel beschrieben worden waren und da die Zn-Konzentrationen in Pankreas und Leber der vier Vögel mit den Konzentrationen übereinstimmten, die bei mit Zn vergifteten Vögel festgestellt worden waren, folgerten wir, dass diese Wasservögel eine Zn-Vergiftung hatten. Dies ist die erste Fallbeschreibung einer Zn-Vergiftung in freilebenden Wildvögeln, die durch in der Umwelt vorkommendes Zink vergiftet wurden.

RESUMEN

Pancreatitis en aves acuáticas salvajes intoxicadas por zinc

Se recogieron cuatro aves acuáticas en el Tri-State Mining District (Oklahoma, Kansas, Missouri, USA), una área que se sabe que está contaminada con plomo (Pb), cadmio (Cd) y zinc (Zn). Éstas formaban parte de un grupo mayor de 20 aves acuáticas recogidas para determinar la exposición de las aves a la contaminación por metales del lugar. Las cuatro aves acuáticas (3 *Branta canadensis*, 1 *Anas platyrhynchos*) sufrían anormalidades degenerativas de moderadas a intensas en el páncreas exocrino, así como concentraciones tisulares (páncreas, hígado) de Zn que fueron consideradas tóxicas. El estadío más leve se caracterizaba por una atrofia generalizada de las células exocrinas que mostraban vacuolas citoplasmáticas y una pérdida relativa de zimógeno. El estadío más grave se caracterizaba por la presencia de acinos con la luz distendida y tejido exocrino hiperplásico que había perdido por completo el zimógeno; estos acinos estaban ampliamente separados por tejido fibroso inmaduro. Debido a que las lesiones eran casi idénticas a las lesiones descritas en pollos y aves acuáticas cautivas intoxicadas por la ingestión de Zn, y a que las concentraciones de Zn en el páncreas e hígado de las cuatro aves eran comparables a las concentraciones medidas en las aves intoxicadas por Zn, concluimos que estas aves acuáticas se intoxicaron con Zn. Este sería el primer caso de intoxicación por Zn en aves salvajes libres intoxicadas con Zn ambiental.