

**Elevated Lead Levels in Farmed American Alligators
Alligator mississippiensis Consuming Nutria *Myocastor coypus*
Meat Contaminated by Lead Bullets**

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Compared to mammals and birds, reports of lead poisoning in reptiles are rare (Beresford et al. 1981; Cook et al. 1988, 1989; Frye 1991; Morgan 1994; Bennett 1996). Elevated blood and tissue lead levels were detected in diseased and apparently healthy farm raised American alligators *Alligator mississippiensis* consuming ground nutria *Myocastor coypus* carcasses found to contain fragments of lead bullets. This report details the clinical, toxicological and pathological findings associated with an unusual case of lead poisoning in commercially reared alligators.

Louisiana Department of Wildlife and Fisheries data indicate sales of alligator hides and meat represent an aquaculture industry in the state with a 1995 production valued at US \$11 million. The animals are typically raised indoors in buildings containing multiple pens with shallow pools provided with raised platforms for feeding and resting. At optimum temperatures of 30–32 C, market-sized alligators, averaging 120 cm, can be produced in approximately 2 yr. Diets on most farms consist of a combination of commercially available pellets, chicken parts, whole fish, and ground nutria carcasses. Relative proportions of the dif-

ferent components vary throughout the year based on price and availability.

Nutria are large South American rodents, introduced into Louisiana earlier this century, that once formed part of an economically important fur industry. In the past, nutria were harvested using steel leg traps and killed by a blow to the head. In recent years, declining fur prices have led to reduced harvests and problems of overpopulation and habitat degradation. Today, the .22 caliber rifle has largely replaced the leg trap, and nutria are often shot rather than trapped. Lead fragments enter diets when bullets are ground with the nutria carcasses and are subsequently fed to the alligators. Nutria meat is purchased during the winter trapping season and fed mainly during the spring and early summer months. The actual length of time it is fed is dependent on the freezer capacity of the individual producer.

In July 1997, seven dead and two moribund 1-yr-old alligators, ranging in length from 60–90 cm and weighing 1.75–2.5 kg, were presented for evaluation to the Aquatic Animal Disease Diagnostic Laboratory at the Louisiana State University, School of Veterinary Medicine. Low numbers of alligators had been dying in a single building (house 6) for a period of 3 mo and cumu-

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lative mortalities had reached 5% in an original population of 3,000 animals. Non-specific clinical signs, when present, included anorexia, weight loss, poor growth, lethargy and death. No additional problems or unexpected mortalities were occurring in other buildings on the farm.

The two moribund alligators were euthanized by overdose with pentobarbital. Bacterial cultures, tissue samples for histopathology and toxicological analysis were collected from these two animals and one dead alligator. Visceral cavities were opened aseptically via a ventral midline incision. Samples of liver and spleen were removed, cultured on 5% sheep blood agar, and incubated overnight at 29 C. Lungs were incised, swabbed with a sterile cotton tip applicator, and the material collected also cultured on blood agar.

Samples of liver, kidney, spleen, lung, heart, stomach, intestine, skeletal muscle, skin, brain and bone were collected from the three animals and fixed in 10% neutral buffered formalin for 24 h. Prior to processing, bone samples were decalcified in 5% formic acid for 2 wk, followed by neutralization in a saturated solution of sodium bicarbonate for 1 h. Tissue samples were processed routinely, embedded in paraffin, and sectioned at 4 μ m. Staining procedures included Gill's hematoxylin and eosin (H&E) and Ziehl-Neelsen acid-fast stain. Two additional reportedly healthy alligators were obtained from a second farm that also feeds nutria meat. Blood samples were collected, the animals euthanized, and tissue samples collected and processed as outlined above. Blood smears were prepared on these two individuals, air dried and stained with a modified Wright's stain.

Blood samples were later collected at random from 13 live alligators in multiple buildings on the original farm. Nutria meat was a major component of the diets of all animals sampled. Blood and tissue samples were also collected from three age-matched controls that had never been fed nutria meat. Blood was collected in heparinized

vacutainers via puncture of the occipital sinus. Lead determinations were performed on blood samples following dilution, and on tissues following digestion in concentrated nitric acid, using a Perkin-Elmer 5000 graphite furnace atomic absorption spectrophotometer at a wavelength of 283.3 nm.

Gross examination of the original nine alligators revealed them to be in poor physical condition, as evidenced by wasting of the tail musculature. Little fat was present in the abdomen or fascial planes of the tail. Gastrointestinal contents were scant. The stomachs of five animals contained variably sized 0.2–1.0 cm fragments of a soft gray metal suggestive of lead, one of which was a nearly intact .22 caliber bullet. In four of these alligators, the stomachs contained only metal fragments. Several fragments were collected and later confirmed as lead using atomic absorption spectrophotometry. The lungs of one alligator were partially filled by clear mucoid exudate. There were no additional significant gross necropsy findings.

An inspection of the farm was conducted several days after the necropsies were performed. The rectangular buildings were of concrete block construction with exposed wooden rafters and plywood roofing coated by a thick layer of foam insulation. A central walkway divided the building in half, with four approximately 4.6-m square pens on both sides. Individual pens had concrete floors covered by epoxy paint. Pens contained 15–20 cm of water and were flushed every other day with well water supplied by PVC plastic pipe. The alligators were being fed on platforms constructed of arsenate treated plywood. There was no history of pesticide or other chemical application in or near the buildings. Examination of nutria meat on the premises revealed metal fragments identical to those found in the stomachs of dead alligators. Entire carcasses were being ground in a large industrial meat grinder prior to feeding. The diet

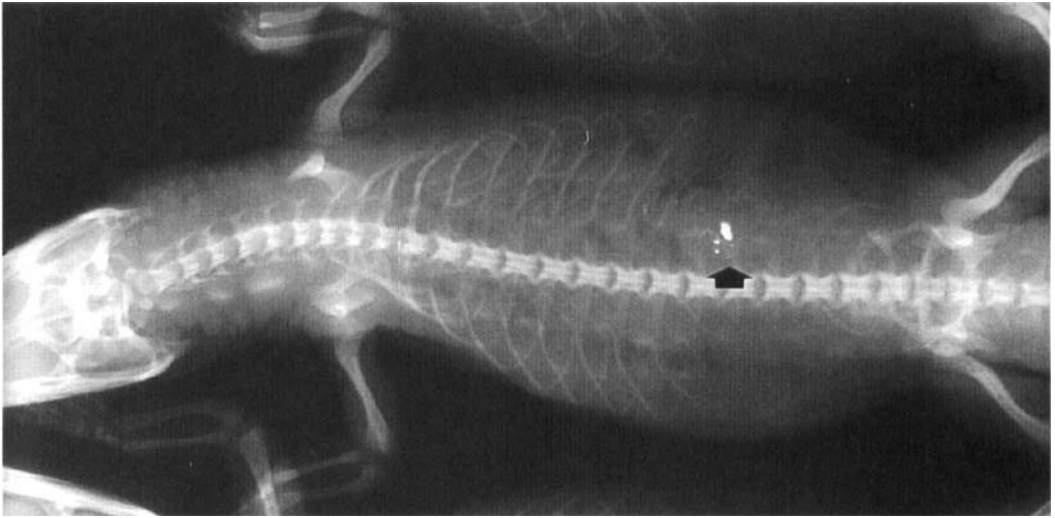


FIGURE 1. The arrowhead indicates radiopaque lead bullet fragments in the stomach of an alligator. The animal was anorectic and in poor physical condition prior to death, presumably from lead poisoning.

at this time consisted of three parts nutria meat to one part dry pellets.

Radiographs of four additional dead alligators revealed radiopaque material in the gastrointestinal tracts of three animals (Fig. 1). A composite radiograph of gastrointestinal tracts, collected at slaughter from an undetermined number of alligators, showed large numbers of radiopaque foreign bodies and nutria bones in the stomachs of many animals.

Bacterial cultures of liver and spleen were negative in the two live and one dead alligator examined. Lung cultures were negative from two animals, but a *Pseudomonas sp* was cultured in moderate numbers from the animal with mucoid exudate. Microscopic examination confirmed an acute mild heterophilic pneumonia in this individual. Renal changes consisted of random mild individual cell necrosis of proximal renal tubular epithelial cells, characterized by hypereosinophilia and nuclear pyknosis. Globular hyaline cytoplasmic inclusions, containing basophilic nuclear fragments, were infrequently present and interpreted as apoptotic bodies. Compared to negative controls, epithelial cell nuclei were often enlarged with marginated chro-

matin. Acid-fast intranuclear lead inclusion bodies were not present. There were no additional significant microscopic findings in any of the diseased or control animals examined. Blood smears prepared on two alligators with blood lead levels of 1.7 and 1.2 mg/L showed no evidence of basophilic stippling.

Results of toxicologic findings for lead are summarized in Table 1. In the absence of established criteria, blood lead levels greater than 0.2 mg/L were interpreted as suggestive of lead poisoning and levels greater than 0.5 mg/L were considered diagnostic. Blood lead levels ranged from 0.07 to 2.80 mg/L and were elevated in 11 of 15 (66%) randomly selected animals sampled from two farms. Blood levels in three control alligators ranged from 0.03 to 0.04 mg/L. Kidney and liver lead levels in control animals averaged 0.085 and 0.045 mg/kg, respectively. Kidney lead levels of 1.14, 1.20 and 1.30 mg/kg were found in three animals with blood lead levels of 1.50, 1.70 and 1.20 mg/L, respectively. Kidney levels in three frozen samples collected from alligators that had died were 0.40, 2.70 and 5.60 mg/kg. Liver lead concentrations of 0.46 and 0.37 mg/kg were

TABLE 1. Blood and tissue lead levels from alligators sampled on two farms feeding nutria carcasses as a major diet component. Blood lead levels of 0.2 mg/L are interpreted as suspicious of lead poisoning, levels of 0.5 mg/L are interpreted as diagnostic.

Location	Blood (mg/L)	Kidney (mg/kg)	Liver (mg/kg)	Muscle (mg/kg)
Control	0.03			
	0.03	0.04	0.03	<0.1
	0.04	0.13	0.06	<0.1
Farm A				
House 6	1.50	1.14		
	0.34			
	0.63			
	1.11			
	0.18			
	2.80			
	0.26			<0.1
				<0.1
		2.7		
		0.4		
		5.6		
House 2	0.10			
	0.88			
				<0.1
				<0.1
	0.91			
House 5	0.29			
	0.07			
	0.07			
Farm B				
	1.70	1.20	0.46	<0.1
	1.20	1.30	0.37	<0.1

found in two animals with blood lead levels of 1.70 and 1.20 mg/L. Muscle lead levels performed on six alligators consuming lead-contaminated nutria meat and two control animals were all less than 0.1 mg/kg and interpreted as insignificant. Arsenic determinations performed on the kidneys of three animals that had died revealed levels less than 0.1 ppm, indicating that arsenic was not leaching from the treated plywood onto the food in significant quantities. No additional metal analysis was performed.

Prior to the 1970s, lead poisoning or plumbism was the most common accidental poisoning seen in domestic animals. Poisoning resulted from ingestion of lead based paints, linoleum, used motor oil, storage batteries, greases, caulking materials, pipe dope, solder, forage contaminated by

emissions from lead smelters, soft water contaminated by lead pipes, and others (Aronson 1978; Hamir 1986). The incidence of lead poisoning in companion animals in the United States has declined since the enactment of laws to decrease the lead content of paints and gasoline (Morgan et al. 1991; Morgan 1994). Despite replacement of lead shot by steel for hunting purposes, lead poisoning from ingestion of spent lead shot remains a problem in waterfowl, due to its persistence in the environment (Degernes et al. 1989).

Lead poisoning in domestic animals has been reviewed by several authors (Osweiler et al. 1978; Lumeij 1985; Hamir 1986). The majority of ingested lead remains in the gastrointestinal tract and is excreted in the feces; absorbed lead is excreted mainly in

urine. Following absorption, lead is transported in erythrocytes, stored initially primarily in liver and kidney, then transferred slowly to bone. Lead is considered a protoplasmic toxin, capable of interrupting heme synthesis and erythrocyte maturation. Interference with the enzyme delta aminolevulinic acid dehydratase, involved in porphyrin synthesis, results in urinary excretion of delta aminolevulinic acid. Clinical signs are typically referable to the gastrointestinal and neuromuscular systems, but are often nonspecific and may be limited only to lethargy and emaciation. Common gastrointestinal signs include emesis, anorexia, diarrhea or constipation, and colic. Neuromuscular signs include seizures, hyperexcitability, hysteria, ataxia, muscle spasms, paresis, paralysis, blindness, and behavioral changes.

Gross lesions may be limited to weight loss or absent altogether, and when present are generally nondiagnostic; however, the presence of foreign material in the gastrointestinal tract should arouse suspicion if clinical signs are compatible (Zook 1972; Osweiler et al. 1978). On blood smears, basophilic stippling of erythrocyte cytoplasm accompanied by numerous immature and altered red blood cells is common (Hamir 1986). Microscopic lesions can be inconsistent, but most often involve the kidneys. Mild necrosis of proximal tubular epithelial cells, karyomegaly with margination of nuclear chromatin, and the presence of eosinophilic, acid-fast, intranuclear inclusion bodies are essentially pathognomonic. Lead inclusions occur less frequently in hepatocytes. The size and number of inclusions increase with increasing liver lead burdens. Less prevalent lesions include mild hepatic necrosis and hemosiderosis, transverse bands of metaphyseal sclerosis in the bones of young rapidly growing animals, and vascular and laminar necrosis in the central nervous system (Zook 1972).

Analysis of blood for lead content is considered the single most reliable test for confirmation of lead poisoning and levels of

0.4–0.6 mg/L are diagnostic in dogs (Zook et al. 1969). Detrimental effects have been associated with blood lead values in the 0.1–0.3 mg/L range, prompting some authors to advocate the use of additional markers, such as blood protoporphyrin or delta aminolevulinic acid levels, to demonstrate a biological effect in subclinical cases (Lumeij 1985; Berny et al. 1994). Blood lead levels in clinically normal and symptomatic birds can be much higher than in mammals, although considerable species variation exists (Lumeij 1985).

The metabolism of lead and its toxicity in alligators is unknown. Descriptions of lead poisoning in reptiles are limited, making it difficult to draw parallels between this case and the plethora of clinical, histopathologic and toxicologic data amassed in other animal species. The paucity of data on reptiles may be due to their more fastidious feeding habits and decreased likelihood to chew on foreign objects. Blood, kidney and liver lead levels, averaging 6.00, 8.45 and 21.6 mg/kg respectively, have been detected in apparently healthy wild box turtles *Terrapene carolina* collected in close proximity to a lead smelter (Beresford et al. 1981). Blood lead levels of 1.47, 1.78 and 2.47 mg/L were detected in two captive false gharials *Tomistoma schlegelii* and a Cuban crocodile *Crocodylus rhombifer*, respectively, at the New York Zoological Park. A portion of their diet consisted of feral urban pigeons, later found to contain high bone lead levels. None of the crocodilians exhibited signs referable to lead toxicosis. Blood lead levels in 23 other crocodilians at the zoo, not fed pigeons, were all less than 0.1 mg/L (Cook et al. 1988, 1989).

A case of plumbism was successfully treated in a tortoise following ingestion of lead based paint chips. The diagnosis was made on generalized neurologic signs and elevated blood lead levels (Bennett 1996). Blood lead levels of 2.21 mg/L (control 0.257 mg/L) and a blood protoporphyrin level of 2.20 mg/L (control <0.1 mg/L)

were found in another tortoise suffering from lead poisoning. Radiographs revealed radiopaque foreign material in the gastrointestinal tract, and paint chips were present in gastric contents evacuated by lavage. The animal recovered uneventfully following treatment with intravenous calcium ethylenediaminetetraacetic acid (CaEDTA) (Frye 1991). An iguana with radiopaque material in its gastrointestinal tract had signs of anorexia, lethargy and limb rigidity compatible with lead toxicosis. Blood lead determinations were not performed. Chelation therapy was attempted but the animal died (Morgan 1994).

Treatment of lead poisoning involves the administration of chelating agents, such as CaEDTA, penicillamine, or dimercaprol (BAL), which mobilize lead and allow it to be excreted in urine (Hamir 1986). Morgan (1994) advocates using a 1% solution of CaEDTA diluted in 5% dextrose, at a dose of 30–35 mg/kg body weight given intramuscularly (IM) three times daily for 5 d. Bennett (1996) recommends a dose of 10–40 mg/kg body weight given IM twice daily. Due to the expense and inherent difficulty of giving multiple injections to large numbers of alligators, therapy was not attempted in this case. Removal of gastrointestinal contents is also necessary prior to treatment, as CaEDTA enhances lead absorption and may exacerbate signs (Zook and Carpenter 1977).

It is difficult to interpret the significance of the elevated lead levels in the alligators examined, as they were found to be abnormally high in affected, dead and apparently healthy individuals; however, no other cause could be determined for the poor growth and mortalities. The marked variability in blood lead concentrations among individuals suggests ingestion of bullet fragments was a purely random occurrence. In dogs and birds, no correlation exists between blood lead concentrations, degree of hematologic changes, severity or nature of clinical signs (Zook et al. 1969; Lumeij 1985). The data collected here suggest that

alligators exhibit a relatively high degree of resistance to the toxic effects of lead. There were no hematologic or histopathologic lesions, particularly lead inclusions, to strongly support or dispute a diagnosis of lead poisoning. Karyomegaly with margination of chromatin was seen in renal tubular epithelial cells, but intranuclear lead inclusions were not. Nuclear enlargement with margination of chromatin appears to precede the development of inclusion bodies in dogs (Zook 1972). Inclusions may have developed with time and continued exposure in this case.

Clearly controlled studies are needed to establish lethal doses and to evaluate the impact sublethal levels of ingested lead may have on growth and performance of farm raised alligators. The feeding of nutria meat utilizes a nuisance animal as a readily available and economical food source for the alligator industry; however, its widespread usage and the common practice of taking the animals by gunshot suggest elevated lead levels may be widespread on alligator farms. Other farmers have indicated that they commonly find bullet fragments in their supply of nutria meat. Producers should be aware that feeding nutria carcasses contaminated with lead to alligators could represent a potential threat to production on commercial operations. In this limited study, muscle lead levels were very low and do not appear to pose a threat for human consumption of alligator meat, but further investigation should be performed to assure safety.

Acknowledgments

The authors would like to thank Dr. C. L. Shilling for providing control alligators used in this investigation.

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