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## Heavy Metal Poisoning in Birds

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### Key Points

- Heavy metal poisonings in birds most commonly occur from ingestion of substances containing lead or zinc.
- Lead that is ingested can be absorbed through the gastrointestinal tract and then taken up by soft tissues and eventually by bone.
- Lead affects all major organs and can cause damage to the gastrointestinal tract, red blood cells, kidneys and liver.
- Clinical signs of heavy metal toxicosis may include non-specific signs of illness, gastrointestinal signs, urinary tract problems and neurological deficits.
- Antemortem diagnosis of heavy metal toxicity relies on blood lead or zinc levels. In some BUT NOT ALL cases, ingested lead will also be visible on survey radiographs.
- Remove heavy metal from the bird's tissues with a chelation agent such as calcium EDTA.
- Pieces of heavy metal within the digestive tract may also be removed endoscopically, surgically or via gastric lavage.

### Introduction

Heavy metal poisoning in birds most commonly occurs from ingestion of substances containing lead, or less commonly zinc. Acute heavy metal toxicity is occasionally seen in companion parrots that ingest or chew on objects containing metal because of their curious nature and innate desire to forage. Chronic lead poisoning most frequently affects free-ranging wildlife such as ducks, geese, swans and loons. Lead toxicity in wild birds is most commonly seen during migration in the late fall and early spring. In heavily contaminated areas, toxicity may be observed at any time of the year. Lead toxicity also occasionally occurs in upland game birds such as mourning doves (*Zenaida macroura*), wild turkey (*Meleagris gallopavo*), pheasants and quail. Lead poisoning has also been reported in small mammals, such as raccoons (*Procyon lotor*), and raptors, presumably from the ingestion of lead contaminated prey.

[↑ top](#) <sup>[3]</sup>

### Lead

Lead is relatively insoluble. After ingestion, small amounts are absorbed from the gastrointestinal tract and released into the bloodstream. The presence of grit in the ventriculus or gizzard increases the absorption of lead, which is first retained in soft tissues and eventually bone.

Lead causes endothelial damage while also inhibiting enzymes needed for cellular metabolism. Pathologic changes may include:

- Epithelial necrosis in the gastrointestinal system
- Increased erythrocyte fragility

- Bone marrow suppression leading to inhibition of erythrocyte production and function
- Damage to capillaries within the brain resulting in cerebral edema

Lead may be found in many household items (see Box 1):

#### Box 1. Sources of lead

- Leaded paint (particularly in older homes)
- Antique or imported metal cages
- Plaster and caulking compounds, roofing materials
- Batteries
- Solder
- Curtain weights
- Fishing weights, jig heads
- Shotgun pellets
- Stained glass
- Chandeliers
- Foil from champagne bottles
- Linoleum
- Improperly glazed ceramic
- Golf balls

Although lead shot has been banned for hunting waterfowl in the United States, spent lead shot is still present in waterways. Ingestion of one to three lead shotgun pellets has been reported to be lethal in waterfowl (Beasley 1999).

### Clinical signs of lead toxicosis

Birds can present with a variety of clinical signs depending on the rate of toxin exposure. In cases of acute exposure, birds typically show non-specific signs of illness as well as problems associated with the gastrointestinal tract, urinary tract and/or nervous system (Box 2).

#### Box 2. Signs of acute lead toxicosis in birds

Non-specific signs of illness	Weakness or depression, pallor and anorexia
Gastrointestinal signs	Anorexia, <a href="#">crop stasis</a> <sup>[4]</sup> , vomiting or regurgitation, <a href="#">biliverdinuria</a> <sup>[5]</sup> and <a href="#">diarrhea</a> <sup>[6]</sup> (i.e. loose, dark or black stool)
Urinary signs	Hematuria or <a href="#">hemoqlobinuria</a> <sup>[7]</sup> , particularly in Amazon parrots ( <i>Amazona</i> spp.)
Neurologic signs	Twitching, circling, convulsions and/or blindness

Chronic toxin exposure may be associated with all of the aforementioned signs as well as gradual weight loss.

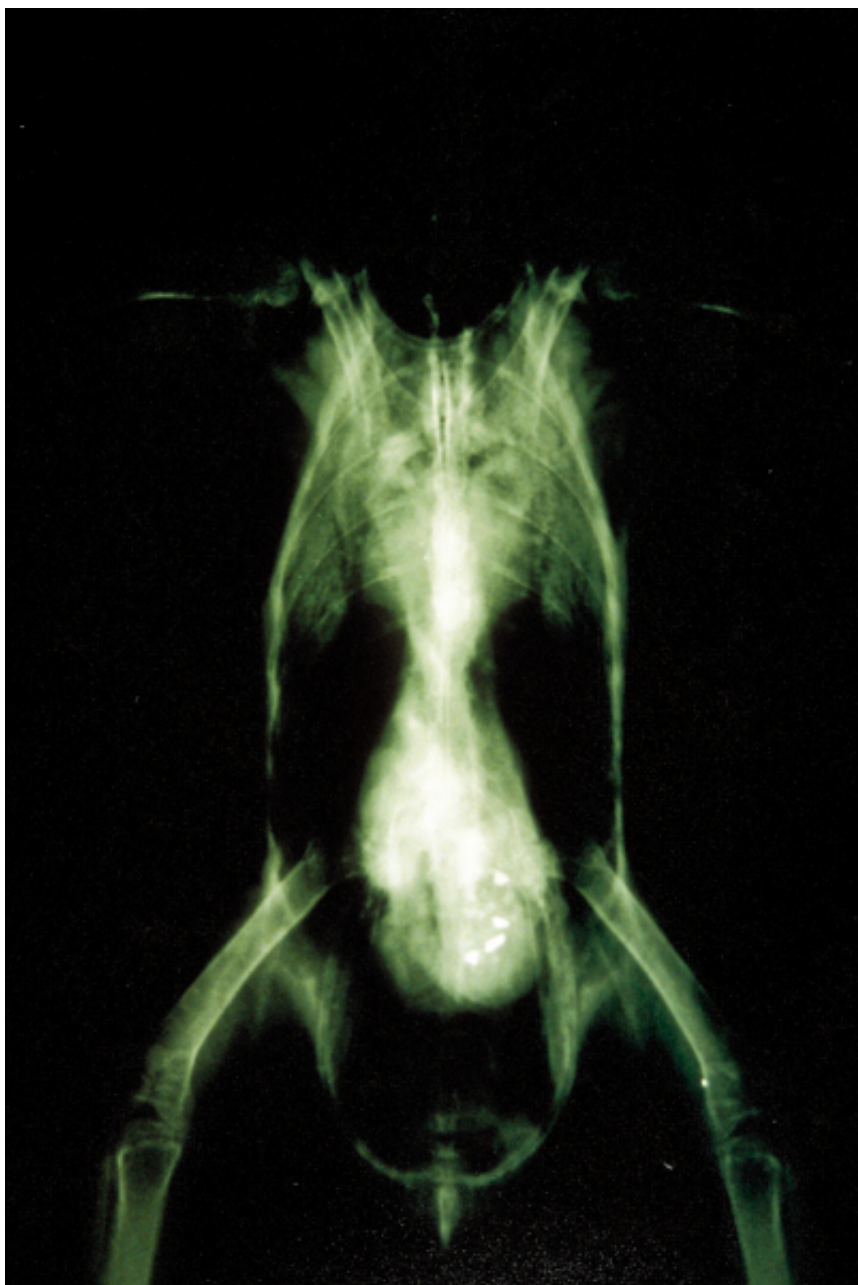
### Diagnosis of lead toxicosis

- A history of exposure to lead should significantly increase the index of suspicion.
- If lead toxicosis is suspected, perform a [complete blood cell count](#) <sup>[8]</sup> or at minimum a packed cell volume. Mild to moderate anemia may be documented; however basophilic stippling is extremely rare in birds (Box 3).

#### Box 3. Diagnostic testing for lead toxicity

History of exposure	Mild to moderate anemia
Hematology	Mild to moderate anemia (basophilic stippling rare)
Survey radiographs	Metallic density
Whole blood levels	Lead > 0.2 ppm

- [Survey radiographs](#) <sup>[9]</sup> are also recommended since some, BUT NOT ALL cases of lead toxicosis will reveal a discrete, metallic density within the gastrointestinal tract (Fig 1). In a survey of mallards (*Anas platyrhynchos*) dosed with lead shot, only 1.2% had a pellet in their ventriculus at necropsy (Rodriguez 2010).



[10]

**Figure 1.** Metal density in the ventriculus. Image provided by Dr. Greg Rich.

- Collect heparinized whole blood to measure lead levels. In the presence of clinical signs, blood lead levels above 0.2 ppm are suggestive for lead toxicity. Although portable testing equipment is commercially available, the most direct route for laboratory heavy metal testing is to send a sample to:
  - [Louisiana Animal Disease Diagnostic Laboratory](#) [11]
  - [Michigan State University Diagnostic Center for Population and Animal Health](#) [12]
  - [University of Minnesota Veterinary Diagnostic Laboratory](#) [13]
  - [Utah State University Veterinary Diagnostic Laboratory](#) [14]
- Inhibition of the enzyme,  $\delta$ -aminolevulinic acid dehydratase (ALAD) also occurs with lead toxicity and ALAD levels may be measured. Aminolevulinic acid dehydratase levels have been described primarily in wildlife studies; ALAD is typically not measured in companion bird care.
- Necropsy findings most consistently reported in wild birds include an enlarged gallbladder, impacted proventriculus, and a cracked, peeling ventricular lining, with or without lead shot present. Collect liver and kidney tissue to measure tissue lead levels.

## Treatment of lead toxicosis

There are three goals for treatment of heavy metal toxicity:

- Stabilize the patient by providing **supportive care** <sup>[15]</sup> such as **supplemental heat** <sup>[16]</sup>, **fluids** <sup>[17]</sup> to prevent dehydration and medication to stop **tremors or seizures** <sup>[18]</sup>.
- Remove heavy metal from bodily tissues with a binding or **chelating agent**:
  - Calcium EDTA (Calcium disodium versenate, 3M Pharmaceuticals) 30-35 mg/kg IM BID x 5 days
  - Dimercaptosuccinic acid (DMSA or Succimer) 25 mg/kg PO SID x 10 days. In a study evaluating chelating agents in cockatiels. Although DMSA is administered orally, it may be easier than other chelating agents for bird owners to administer at home, however, DMSA has a narrow margin of safety so this drug should always be used with caution (Denver 2000).
  - D-Penicillamine (Cuprimine, Merck) 30 mg/kg PO BID x 7 days minimum

Chelation therapy may not be necessary in cases where prompt removal of the lead source is accomplished. When chelation is started, monitor renal parameters during therapy (Richardson 2006).

- If a source of heavy metal is seen on radiographs, **removal of lead** from the gastrointestinal tract via endoscopy, surgery, or gastric lavage may also be indicated. Lubricants such as mineral oil or corn oil, cathartics (i.e. magnesium sulfate), or bulk agents such as peanut butter, psyllium or oral cellulose products may also be used to remove heavy metal from the digestive tract.

Removal of particles with an iron base using a feeding catheter loaded with neodymium-ferro-borium alloy magnets has also been described.

## Prevention of lead toxicosis

Since companion parrots are curious by nature, it can be challenging to prevent chewing and ingestion of undesirable objects. Pet birds should always be supervised during their time outside of the cage, and owners should also remove all known sources of heavy metal or limit exposure to areas with heavy metals (Box 1).

Control of problem areas for wildlife consists of plowing to lessen the availability of spent shot to birds. The use of non-toxic steel or bismuth shot for waterfowl hunting is also now required in the United States. This switch from lead to non-toxic shot has reduced the number of birds dying from lead poisoning in America. The United States and Canada are also considering a ban on lead fishing sinkers.

[↑ top](#) <sup>[3]</sup>

## Zinc

Zinc is a trace metal or mineral essential for health. Zinc is involved in cell replication and in development of cartilage and bone (McDonald 2006). The primary target organs in zinc toxicity are the kidneys and pancreas.

### Sources of zinc

Zinc toxicity usually arises from the ingestion of zinc-coated wire or metallic foreign bodies such as pennies minted after 1983 (Box 4). One penny contains approximately 2440 mg of elemental zinc (Richardson 2006).

Box 4. Sources of zinc

- Galvanized clips and wires
- Some powder coated cages
- Washers, nuts and bolts

- Snap fasteners, padlocks, some toy hangers
- Pennies made in 1983 or later
- Zinc oxide cream or ointment

Galvanized wire is another common source of zinc intoxication in parrots, especially cheap, imported wire. Aviary birds are often housed in galvanized steel wire cages. Galvanized coatings can contain up to 99.9% zinc, however galvanized wire can also contain lead (Platt 2006). Galvanized dishes should never be used since zinc can leach into the water.

Powder coating is a protective coating for the cage. While formulas differ by manufacturers, most contain no zinc, however some imported powder-coated cages use zinc to expedite setting of powder coating (Richardson 2006).

Zinc can also leach out of zinc-coated iron shot into the environment. Waterfowl can then ingest vegetation and sediments contaminated by zinc (Platt 2006).

A number of websites report that the adhesive in paper towel and toilet paper rolls contains significant amounts of zinc, however this appears to be more urban legend than fact.

*Once, when we had a look at the actual zinc content in the glue in those paper rolls, and then "borrowed" the nutritional requirements for zinc in chickens; presuming that the requirements in a parrot would be similar, we were able to show that if a parrot ate toilet paper exclusively with that zinc containing glue, there would still be a need for zinc supplementation to meet the nutritional requirements for the bird. (Speer 2010, Veterinary Information Network)*

## Clinical signs of zinc toxicosis

As with lead toxicity, signs of zinc toxicity can be vague and non-specific, but clinical signs are often related to disease of the gastrointestinal tract, pancreas, kidneys and/or central nervous system (Box 5).

### Box 5. Clinical signs of zinc toxicity

- Lethargy
- Crop stasis, regurgitation
- Green or yellow diarrhea
- Weight loss
- Seizure activity
- Paresis
- Polyuria/polydipsia
- Feather loss? Feather damaging behavior?

Although this is a bit controversial and reports are anecdotal, zinc toxicity has also been associated with an "extreme" loss of plumage and feather damaging behavior.

## Diagnosis of zinc toxicosis

The minimum database in zinc toxicosis may be relatively unremarkable. Mild to moderate regenerative anemia due to erythrocyte loss has been described with zinc toxicity (Box 6). Since zinc toxicosis typically results from chronic exposure to fine metal powder, a metallic density is rarely observed on survey radiographs.

### Box 6. Diagnostic testing for zinc toxicity

Hematology           Mild to moderate anemia  
Whole blood levels Zinc > 2 ppm

The normal blood zinc ranges are only weakly understood for a variety of species. Additionally, in a trial following induced zinc toxicity in cockatiels, blood zinc levels were extremely inconsistent as a diagnostic predictor (Howard 1992). Part of the confusion stems from the fact that zinc is an essential nutrient. Normal homeostasis regulates zinc levels based on variations in gastrointestinal content, bioavailability and individual nutritional needs. Significant diurnal variations in zinc values have also been documented in 15 adult

psittacine birds (Rosenthal 2005).

Nevertheless in the presence of clinical signs, blood zinc levels exceeding 200 µg/dl (or 2 ppm) are suggestive of toxicity. Collect heparinized whole blood to measure zinc levels. Take care in how the blood sample is drawn and stored to avoid contamination.

- Although microtainers are typically used for companion birds, remember that the rubber stoppers in red-topped tubes contain zinc. The presence of rubber can cause an artifactual elevation in zinc levels.
- Royal blue-topped tubes are an alternative collection tube to test for zinc and other metals.

The pancreas is the tissue of choice for postmortem zinc analysis (Box 7). Liver and kidney samples may also be collected to measure tissue zinc levels.

Box 7. Pancreatic tissue zinc levels in cockatiels (Dumonceaux 1994)

Normal 26.11 µg/gram (dry weight basis)

Toxic 312.4-2418 µg/gram

Important pathologic lesions seen with zinc toxicosis include ileus and focal mononuclear degeneration of the liver, kidneys and pancreas (LaBonde 1995). Necrotizing pancreatitis and erosive ventriculitis are other common manifestations. Histologically, the koilin layer is disrupted and there is ulceration of the underlying mucosa and dysplasia of the ventricular glands.

## Treatment of zinc toxicosis

The goals for treatment are the same as with lead toxicity. The most important difference is that zinc is not stored in bone, and therefore blood and tissue levels equilibrate faster. This means that the response to chelation therapy is also faster (LaBonde 1995).

## Prevention of zinc toxicosis

Scrubbing all new galvanized cage wiring with a mildly acidic solution such as vinegar, then drying carefully, may reduce zinc levels.

[↑ top](#) <sup>[3]</sup>

## Copper

Copper toxicosis is rare in birds. Sources of copper include wire, pennies minted before 1982, copper sulfate, anti-fouling paints and possibly copper ammunition (Christian Franson 2011). An important source of acute copper toxicosis in free-ranging waterfowl and other aquatic birds are acid metalliferous water bodies.

Clinical signs of copper toxicosis may include depression, weakness, anemia, convulsions and coma. Black discoloration of the parenchyma is an important gross finding. Common histopathological lesions include proventricular and ventricular necrosis, ventricular hemorrhage and/or congestion, erosion and ulceration of the koilin layer and duodenal hemorrhage (Isanhart 2011).

Like zinc, significant diurnal variations in blood copper levels have been documented (Rosenthal 2005).

## Iron

Iron toxicosis in companion parrots can result from exposure to cast-iron feeding bowls with chipped enamel. Non-specific signs of illness predominate such as lethargy, emaciation and anorexia. Deferoxamine is the treatment of choice in mammals, but calcium EDTA also works well (LaBonde 1995).



For more information on iron overload, see [Iron Storage Disease in Birds](#) [19].

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[↑ top](#) [3]

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