What REALLY Poisons Birds:
Avian Toxicology
Teresa Lightfoot, DVM, DABVP
BluePearl Veterinary Partners
Tampa, FL

Birds have historically served as sentinels for human toxicity in the environment. Birds were used as sentinels for coal miners in the U.S. and England until the middle of the last century. Dangerous levels of carbon monoxide, methane and other poisonous gasses would affect sentinel canaries before they affected the miners, thus warning them of a dangerous environment. As our knowledge of avian toxicology increases, so likely will knowledge regarding the interrelationship of toxicity and its sequelae in birds and humans. As practitioners, we need to be aware of these potential links, share our findings with our colleagues, both veterinary and human, and inform owners of potential concerns with animal and human health.

On a larger environmental scale, our wild birds are objects of studies that detect dangerous levels of heavy metals, pesticides and other chemicals in bodies of water and sources of food for both animals and humans. Affects on their health and fecundity are of conservational import, and have great significance for the environment and human health. For purposes of this presentation, the emphasis will be on toxins that are most commonly encountered in pet birds and those that are specific to avian species.

Air-borne /inhalant toxins
Polytetrafluoroethylene (PTFE)
The avian respiratory tract is particularly sensitive to air-borne irritants and toxins due to specific anatomic and physiologic features of birds. Polytetrafluoroethylene (PTFE) is one of the most common causes of air-borne toxicity encountered in pet avian species. This product is found in non-stick cookware, irons, covers for ironing boards, heat lamps, among others. When PTFE is heated above 280 degrees Celsius it decomposes into particulates and fluorinated, acidic gasses, which are toxic when inhaled. Clinical signs may include dyspnea, incoordination, weakness, coma and death. Pulmonary lesions include severe edema, hemorrhage, and necrosis. Treatment consists of oxygen therapy, bronchodilators, anti-inflammatory drugs, diuretics, antimicrobials to prevent secondary infection, and analgesics. In clinically affected birds, the prognosis is generally poor. Smaller birds such as budgerigars seem to be most sensitive to the effects of PTFE toxicity.

Smoke
Smoke is another source of air-borne toxins. Smoke is the general term used for the solid and liquid matter released into the air by combustion (pyrolysis). Exposure to fires, malfunctioning furnaces, engine exhaust, burning food or cooking oil, self-cleaning ovens, or other sources of smoke may induce toxicity. Carbon monoxide, hydrogen cyanide, acidic fumes and particulate matter are components of smoke that cause similar clinical signs to those seen in PTFE toxicity. With smoke inhalation toxicity, dyspnea may not be immediately apparent. It may be several hours before exposed birds demonstrate clinical signs. Smoke inhalation may also lead to immunosuppression and increased susceptibility to infectious disease. In addition to oxygen and bronchodilator therapy, corticosteroids and diuretics are used to treat dyspnea; however, long-term use of corticosteroids may predispose affected individuals to secondary respiratory infections like Aspergillosis.

Nicotine
Nicotine in tobacco smoke may be toxic. Birds most likely to be affected are those chronically exposed, usually pets that live in smoking house-hold [12]. One study demonstrated that cotinine, a nicotine metabolite, was significantly higher in the plasma of birds housed in environments with chronic exposure to tobacco smoke than it was in controls. In humans this metabolite is linked to allergies, asthma, lower respiratory illnesses and heart disease. Clinical signs in avian patients may include conjunctivitis, rhinitis, other respiratory disease, and

Miscellaneous airborne toxins
Other air-borne toxins include air fresheners, hair products, nail polish, scented candles, aerosols, gasoline fumes, glues, paints, mothballs, fumigants, and cleaning products such as ammonia or bleach. Sodium hypochlorite (bleach) was shown to cause death within 6-12 days in seven birds housed in an aviary that was cleaned with this product. While all inhaled toxins have the potential to cause irritation and damage to the respiratory tract, they may also compromise the immune system.

Ingested toxins
Heavy metal toxicity
The definition of a heavy metal depends on its usage. In the strict chemical designation, heavy metals are defined as metals that do not normally occur in living organisms (i.e. mercury, lead, cadmium) and can cause illness. In a medical context, the term heavy metal generally refers to any metal that is potentially toxic. This discussion does not include the radioactive heavy metals, such as uranium and plutonium.
Heavy metal toxicosis is commonly seen in both pet and wild birds, with lead (Pb) and zinc (Zn) toxicity being the most frequently diagnosed in pet birds. Historically, lead was the most common metal toxicity seen in pet birds. However, in recent years as lead is used less frequently in home products and knowledge of its toxicity in children has expanded, the incidence of lead toxicity (also known as plumbism) in humans and other animals has decreased.

**Zinc**

Common sources of avian zinc toxicity include the coating on galvanized wire cages, galvanized toys, food and water dishes and hardware (note: the process of galvanization may include coating with a metal alloy that is > 98% zinc and can contain 1% lead). Larger birds may ingest pennies, and those minted after 1982 have a core containing a high percentage of zinc, [4, 21]. Clinical signs of zinc toxicity may include lethargy, weakness, polydipsia and polyuria, diarrhea, regurgitation (particularly passive regurgitation of water), and less commonly neurologic signs or hemoglobinurea. Radiographs may demonstrate metal density in the GI tract, usually in the ventriculus. However, it is possible in zinc toxicities to find no radiographic evidence of metal. Plasma zinc concentrations above 2-4 part per million are suggestive of toxicosis. It is important that blood samples be collected in royal blue top tubes (non-rubber) to prevent contamination of the sample from zinc found in other rubber stoppers. Post mortem diagnosis is best made through analysis of zinc levels in tissue samples of the pancreas, liver, and kidney.

**Lead**

Prior to 1955, house paints in the U.S. often contained up to 50% lead. The sweet taste of lead encourages ingestion in both children and birds. Lead is one of the most common sources of toxicity seen in water birds and raptors, however, it may be seen in pet psittacines as well. In pet birds, sources of lead include stained glass, lead solder, curtain weights, fishing sinker weights, and in older houses, Venetian blinds, linoleum and paint may contain lead. Bird ‘toys’ are sometimes, through oversight or ignorance, produced using lead solder. In acute cases, severe hemoglobinuria, PU/PD, depression, anorexia, and seizures often occur and death is common within 24 hours of ingestion.

Chronic or low level lead toxicity often mimics the clinical signs seen with Zn. For determination of blood lead concentration, whole blood may be submitted as described for zinc above. Non-toxic levels in whole blood are reported to be <0.2ppm, while >0.2ppm is suspect and >0.5ppm is diagnostic. Lead concentrations may also be measured in tissues at necropsy, most importantly the liver in bone where lead exposure has been chronic.

Chelation therapy is the principal means of treatment for lead and often for zinc toxicity, accompanied by rehydration and supportive care. Chelating agents work by binding the heavy metal, forming a non-toxic chelate that is excreted. Ca EDTA is the primary parenteral agent used for lead (and zinc) toxicity in humans and animals. Recommended dose in birds is 35-40mg/kg IM or IV every 12 hours for 5 days and repeated as needed. Fluid therapy should be included to prevent possible nephrotoxicity, although this has not been documented to occur in avian species. Meso-dimercaptosuccinic acid (DMSA) is another chelator that may be used. Administer at 30mg/kg BID PO for 7 days. D-penicillamine (cupramine) is another chelator that can be administered for heavy metal toxicity at 55mg/kg PO q12h for 7 to 14 days. In children, it is used for low to moderate levels of lead toxicity.

If large metal particles are identified within the GI tract, removal may be attempted via gastric lavage, endoscopically, retrieval with long biopsy forceps or a magnet, or by surgical removal. If particles are too small to be retrieved, cathartics such as lactulose or psyllium can be administered to accelerate excretion.

Rarely, avian metal toxicities may involve cadmium, copper, mercury or iron. Except for iron, these rarely occur in pet birds, as they are often a result of environmental contamination. Mercury is of increasing concern in wild life that feeds from aquatic species such as bivalves and fish, which accumulate mercury. Mercury toxicity may be treated with DMSA. (Note: mercury found in glass thermometers is not absorbed by the GI tract, and is therefore non-toxic). Human applications of DMSA for avian use have not been well documented. Clinical signs of mercury toxicity may include: lethargy, fluffed feathers, increased respiratory effort.

**Food**

Human foods can be a source of toxins for avian pets, as owners frequently share table food with their birds.

Chocolate Few cases of chocolate toxicosis have been reported in birds. Treatment includes removal of any remaining chocolate from the GI tract. This is accomplished by crop and proventricular lavage, followed by administration of activated charcoal at 1-3g/kg. Both of these procedures must be performed with care to prevent aspiration and generally require anesthesia and tracheal intubation. Emesis should generally not be induced in avian patients due to the likelihood of resulting aspiration.

Avocado (Persea americana) is known to be toxic to some avian species. All parts of the plant, including fruit, seeds, leaves, and bark can induce signs of toxicity. Not all species of birds are equally affected by the toxins, nor are all avocados equally toxic. Larger parrots are more likely to demonstrate ante-mortem clinical signs such as lethargy, fluffed feathers, and increased respiratory effort. The most consistent necropsy findings include pericardial effusion, subcutaneous edema and generalized congestion of organs, including the lungs and liver.

Onion and garlic (Allim sp.) have occasionally been cited as toxic to pet birds when excessive amounts are consumed.
Toxic plants
Exposure to toxic plants may also occur when a curious bird ingests house plants or landscaping. There are few reports of pet bird plant toxicosis; however, those listed include Crown vetch (Coronilla varia), Rhododendron (Ericaceae family), Kalanchoe species, Oleander (Nerium) Convallaria majalis (Lily of the Valley), Digitalis purpurea (Foxgloves). Thee cardiac glycoside containing plants may cause increased contractility, bradycardia, wide QRS complexes, ventricular arrhythmias, and death.

Oxalate containing plants produce severe oral irritation. Common houseplants that contain oxalates include Schefflera (Umbrella plant), Spathephyllum (Peace lily), Dieffenbachia (Dumb Cane), Epipremnum (Pothos), and Philodendron.

The listing of house plants that are potentially toxic is long and beyond the allotted length of these Proceedings. Please see the Chapter in Vet Clinics of North America for a more detailed list of plants of concern.

In any plant toxicity, after initial stabilization, removal of any remaining plant from the GI tract is indicated and is accomplished through crop and GI lavage. Activated charcoal is also effective in most plant intoxications, neutralizing toxic components remaining in the GI tract. Further treatment is directed at the organ systems affected and clinical signs.

Mycotoxins
Mycotoxins are toxins produced by fungi and commonly occur in fungal-contaminated grain products. Avian species are more susceptible than other affected species. Aflatoxin and fusariotoxin are often responsible for avian mycotoxicosis and are usually associated with cereal grains, corn, and peanuts that have been exposed to or kept in humid, moist conditions. Aspergillus flavus produces aflatoxins and Fusarium produces fusariotoxins. Mycotoxins are hepatotoxic. Clinical signs of chronic aflatoxicosis often include lethargy, weight loss, anorexia, regurgitation, and polydipsia. Testing for mycotoxins in food and in the patient can be difficult due to variation in toxic concentration and the inconsistent production of toxins. The best way to protect pet birds from exposure to mycotoxins is to feed only human-grade grain, corn, and peanut products, avoid spoiled foods, and store grain products in cool, dry places.

Pesticides
Pesticides are commonly encountered in free-ranging avian patient; however, pet birds may be exposed to pesticides used in a home setting. Organophosphate and carbamate toxicity is less common now than in the 1900's. When encountered diagnosis and treatment follow those for other species.

There are anecdotal reports of pyrethrin toxicosis in birds, but few if any confirmed cases in pet birds. Treatment follows dog and cat parameters.

Rodenticides
Anti-coagulent rodenticides work by inhibition of the extrinsic, vitamin K-dependant pathway, in particular factor VII. Since birds rely more on the extrinsic pathway, factor VII may play a less important role in avian species and thus explain their apparent decreased sensitivity to anticoagulant rodenticides. Treatment is similar to that in mammals and consists of vitamin K supplementation.

Hypercalcemic rodenticides have been reported to cause death in birds. These calciferol derivatives produce hypercalcemia, leading to increased ionized calcium levels, metastatic calcification, cardiac conduction disturbances, renal failure and death. Some rodenticides are a combination of anticoagulant and calciferol, with these two agents producing a synergistic effect. Clinical signs of exposure to hypercalcemic rodenticides vary widely. So in addition to a markedly elevated serum calcium, a history of potential exposure is needed for diagnosis. Treatment of rodenticide hypercalcemia includes intense diuresis, glucocorticoids and an antihypercalcemic. Pamidronate, acts to inhibit bone-resorption. This drug was designed for the hypercalcemia of malignancy in humans, and is preferred over calcitonin for treatment of rodenticide.

Iatrogenic toxicities
Any drug, medication, or supplement can be toxic at high enough doses. Avian patients may be more likely to suffer from iatrogenic toxicities due to their small size and resulting dosing mistakes. Furthermore, medications in aviary situations may be administered in food or water, which does not allow control over individual dosages. Anecdotal reports of toxicity in cockatiels following repeated fenbendazole administration have been frequently reported.

Conclusion
Birds may be exposed to toxins through a variety of sources in their everyday environment. Toxicity may occur through inhalation, oral or dermal exposures. It is the clinician’s responsibility to diagnose and treat these toxicities to the best of their ability in an effort to correct the disease of the individual patient. Recognition of toxicity in the avian patient has further significance as it relates to the patient’s environment, including the health of other animals, humans, and the ecosystem. Veterinarians diagnosing poisoning in animals have a responsibility to consider human health implications as well as treating their patient(s). While some toxicities, such as lead and zinc toxicosis, are well-documented in avian species, others are limited to anecdotal reports and extrapolation from other species. Continued research is needed in this area of avian medicine in order to expand our knowledge and improve our ability to diagnose and treat toxic conditions in birds.
References
Other references available upon request