Chapter 37

Mycotoxins

Mycotoxins are toxins produced by molds (fungi) that, when they are ingested, can cause diseases called mycotoxicosis. These diseases are are not infectious. The effects on the animal are caused by fungal toxins in foods ingested, usually grains, and are not caused by infection with the fungus. Many different molds produce mycotoxins and many corresponding disease syndromes have been described for domestic animals. However, only two types of mycotoxin poisoning, aflatoxicosis and fusariotoxicosis, have been documented in free-ranging migratory birds.

Until recently, sickness or death caused by mycotoxins were rarely reported in migratory birds. Identification of mycotoxins as the cause of a mortality event can be difficult for a number of reasons. The effects may be subtle and difficult to detect or identify, or the effects may be delayed and the bird may have moved away from the contaminated food source before becoming sick or dying. Also, grain containing toxin-producing molds can be difficult or impossible to recognize because it may not appear overtly moldy.

Techniques to detect and quantify a variety of mycotoxins important to domestic animal and human health are available through many diagnostic laboratories that serve health needs for those species. These same techniques are applicable for wildlife. Further study and improved diagnostic technology is likely to result in identification of additional types of mycotoxins as causes of disease and death in waterfowl and other wildlife.

Aflatoxin Poisoning

Synonyms

Aflatoxicosis

Cause

Aflatoxins are a group of closely related toxic compounds produced by the fungi *Aspergillus flavus* or *A. parasiticus*. Four types of aflatoxins commonly are found in grains contaminated by these fungi: aflatoxin B1, aflatoxin B2, aflatoxin G1, and aflatoxin G2. These compounds become more toxic when they are metabolized after they are ingested. Aflatoxin B1 is the most commonly occurring and the most toxic. Aflatoxins present in very low concentrations, in parts per billion (ppb), can cause toxicosis.

Domestic ducklings are quite sensitive and the effects of aflatoxin exposure have been studied extensively in this species. In one study using 1-day-old ducklings, the LD_{50}, which is the dose of toxin required to produce death in 50 percent of the test animals via a single dose or single day's feeding, of aflatoxin B1 was 360 ppb aflatoxin.

Aflatoxins are often associated with groundnuts (peanuts) and corn, but they also have been found in other grains and nuts. *Aspergillus* sp. fungi can proliferate in improperly stored grain that has a moisture content of greater than 14 percent, relative humidity greater than 70 percent, and temperature greater than 70 °F. These fungi also can invade grains in the field, especially when there is drought stress, insect damage, or mechanical damage.

Species Affected

Aflatoxins can affect humans, many species of warm-blooded domestic and wild animals, and fish, most notably rainbow trout. Animals that consume grain are more likely to be affected than those that do not. Susceptibility depends on species, age, and diet. In general, birds are more susceptible than mammals, and young birds are more susceptible than adult birds. Mortality events caused by exposure to aflatoxins have been reported in free-ranging birds including a variety of duck species (mallard, black duck, lesser scaup, gadwall, and blue- and green-winged teal), Canada geese, snow geese, and sandhill cranes.

Distribution

Within the United States, the problem of aflatoxin-contaminated grain as a cause of disease in domestic animals and humans has been associated with the Southeastern and Gulf Coast States. Documented wildlife mortality events caused by aflatoxicosis are few; of those reported in wild birds, most occurred in Texas. However, the major fungi that produce aflatoxins, *A. flavus* and *A. parasiticus*, are widespread in temperate and tropical environments.

Seasonality

Most mortalities caused by exposure to acutely toxic levels of aflatoxins are reported in the fall and winter and coincide with times during migration and wintering when cranes and waterfowl are consuming waste grain in fields (Fig. 37.1). Mortality can occur at any time of the year when contaminated grain is provided at birdfeeding stations.

Field Signs

Field signs of aflatoxicosis reported in waterbirds vary from depression and lethargy to blindness, lack of awareness of surroundings, inability to fly, tremors, and wing flapping. Often the birds are simply found dead.

National Wildlife Health Center file photo

Figure 37.1 *Canada geese feeding on standing waste corn in a winter field.*

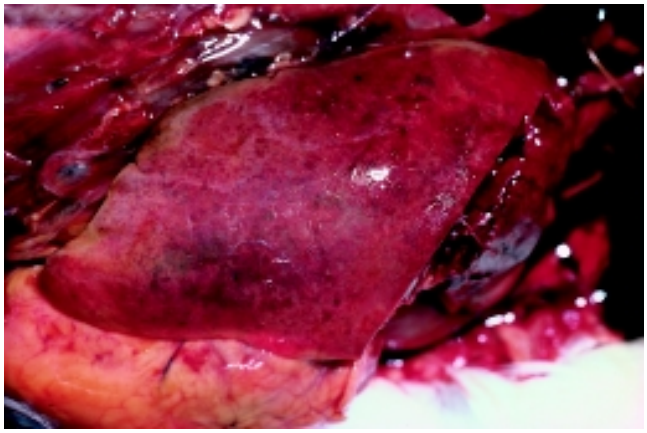

Photo by James Runningen

Figure 37.2 *Pale, enlarged liver with multiple, focal hemorrhages from a bird with acute aflatoxicosis.*

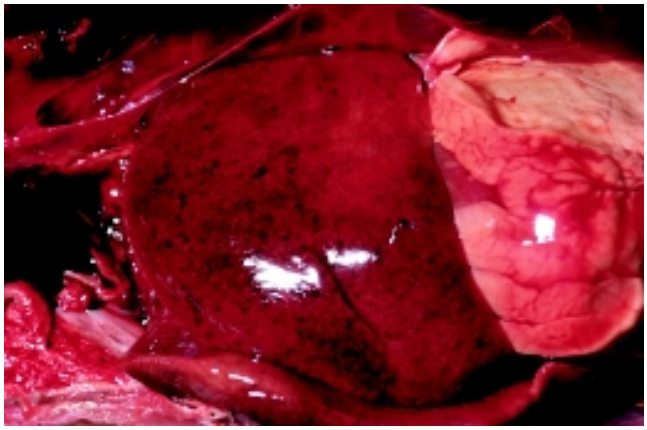

Photo by James Runningen

Figure 37.3 *Hemorrhagic liver from a bird with acute aflatoxicosis.*

Gross Lesions

Lesions of aflatoxicosis can be variable, depending on the amount of aflatoxin ingested and the length of time the animal is exposed. Birds exposed to high levels over a short period of time may have an enlarged, swollen pale liver (Fig. 37.2). Liver hemorrhages may be found in multiple focal areas, or may be diffuse and involve most of the liver tissue (Fig. 37.3). Hemorrhages and fluid may be observed in many organs in the chest and abdomen. Inflammation and bleeding of the gastrointestinal tract lining, which may cause the intestines to appear blackish-red throughout their entire length, may also be observed.

Birds that are exposed to small amounts of aflatoxin over a long period of time may not die suddenly, but rather may have chronic health problems. Chronic effects, which include appetite loss, weight loss, and general ill health, can be more insidious and difficult to definitively relate to aflatoxin exposure. Chronic exposure also may produce a shrunken, fibrous liver with regenerative nodules or tumors. In laboratory tests, aflatoxin B1 has been shown to cause genetic mutations, liver cancers, and, possibly, fetal defects. Chronic low level aflatoxin exposure also is known to suppress the immune system, which may predispose animals to infectious diseases.

Diagnosis

Diagnosis is made by examining tissues for gross lesions and typical microscopic lesions, which include liver tissue death or necrosis and proliferation of lesions in the bile duct. Measurement of aflatoxin levels in ingesta and tissues collected from affected birds and from the grain suspected of being contaminated is also crucial for confirming the diagnosis. The samples of choice include whole refrigerated carcasses for necropsy as well as grain that affected birds have been eating. Because mycotoxin occurrence can vary widely within an agricultural field, it is important to try to obtain a representative sample of the suspect grain. If possible, transport the grain frozen, and ensure that the sample remains frozen so that fungal growth and toxin production secondary to improper postcollection storage does not occur.

Control

Wildlife should not be fed grain that has levels of aflatoxins in excess of those allowed for use in human or domestic animal food (20 ppb for consumption by humans or young animals and dairy cattle; 100 ppb for mature poultry). In years when aflatoxins are a problem, grain from fields that are frequently used by wildlife should be checked for aflatoxin levels. If the fields are aflatoxin-contaminated, deep plowing of the contents can make the grain unavailable to wildlife. If the fields cannot be plowed, hazing wildlife from the area can lessen their exposure.

Human Health Considerations

Handling aflatoxin-poisoned sick or dead birds does not pose a human health risk. However, birds known to have died from acute aflatoxicosis should not be consumed.

Fusariotoxin Poisoning

Synonyms

Fusariomycotoxicosis, trichothecene mycotoxicosis, T-2 toxicosis, vomitoxicosis, zearalenone toxicosis

Cause

Fusariotoxin poisoning is caused by toxins produced by fungi of the genus *Fusarium*. There are two classes of toxins produced by these organisms: metabolites that have properties similar to the hormone estrogen such as zearalenone (F-2 toxin), and the trichothecenes. Zearalenone has been linked to hyperestrogenic or feminizing syndromes in domestic animals, but similar effects have not yet been observed in wild species. More than 50 trichothecene toxins have been identified from *Fusarium* cultures and field samples. However, the trichothecenes have rarely been documented as the cause of mortality in free-ranging birds.

The most frequently occurring trichothecene toxin in the United States is deoxynivalenol, commonly called vomitoxin. Others include T-2 toxin, diacetoxyscirpenol (DAS), neosolaniol and iso-neosolaniol. The feedstuffs involved in *Fusarium* sp. toxin production include corn, wheat, barley, oats, peanuts, and sometimes forages. *Fusarium* toxins differ from other mycotoxins in that they tend to be produced during the colder seasons of the year.

Species Affected

Poisoning caused by trichothecene toxins has been documented in domestic mammals, poultry, and waterfowl. However, reports of fusariotoxicosis in free-ranging waterfowl and other migratory birds are rare and poorly documented. Mortality attributed to trichothecenes has occurred on several occasions in free-ranging sandhill cranes. It is suspected that wild waterfowl could be affected by trichothecenes because these toxins tend to be produced in low temperature conditions when waterfowl make heavy use of waste grain as food.

Distribution

Fusarium sp. are widespread in the environment and they commonly occur as plant pathogens or contaminants in stored or waste grain and other plant parts. Toxin production from *Fusarium* sp. is most commonly a problem in the North-Central United States and Canada. However, the only documented locations of wild bird mortality caused by *Fusarium* toxins are in Texas and New Mexico, and these die-off events involved sandhill cranes that fed on *Fusarium*-contaminated peanuts.

Seasonality

Fusarium invasion often occurs during wet conditions in the summer and fall while crops are in the field. However, the organism also can grow in stored grain. The optimal temperatures for mold growth and toxin production differ. Temperatures that support growth of the vegetative form of the fungus are between 64 and 77 °F, but actual toxin production tends to occur at temperatures between 40 and 65 °F and, in some cases, has even been documented at near-freezing temperatures. Colder temperatures favoring toxin production coincide with times that cranes and waterfowl are using waste grain in fields during their fall and winter migration.

Field Signs

Different trichothecenes have different effects. In general, clinical signs in domestic poultry and geese include feed refusal, vomiting, and gastrointestinal bleeding. Some birds have been described as having neurologic abnormalities. Free-ranging sandhill cranes diagnosed with trichothecene toxicosis had difficulty keeping their balance and had flaccid paralysis or weakness of the neck and wing muscles. This created a stance characterized by a drooping head and wings (Fig. 37.4). The more toxic trichothecenes cause immune suppression and may predispose birds to secondary infections.

Gross Lesions

Inflammation and ulceration of the skin and mucosal surfaces of the oral cavity and upper gastrointestinal tract are the most commonly reported lesions in domestic animals and were observed in affected sandhill cranes (Fig. 37.5). Gross lesions described in sandhill cranes also included subcutaneous fluid over the head and neck (Fig. 37.6) and multiple hemorrhages and pale areas in skeletal muscle.

Photo by Ronald M. Windingstad

Figure 37.4 *A sandhill crane suffering from fusariotoxicosis. Notice the wing and head droop.*

Photo by James Runningen

Figure 37.5 *Inflammation and ulceration of the mucosal surface of the esophagus in a sandhill crane with fusariotoxicosis.*

Photo by J. Christian Franson

Figure 37.6 *Fluid beneath the skin of the head and neck of a sandhill crane with fusariotoxicosis.*

Photo by Ronald M. Windingstad

Figure 37.7 *Plowing of this field made these peanuts unavailable to the cranes and ended a fusariotoxicosis mortality event.*

Diagnosis

Diagnosis is made through observing the appropriate field signs, finding gross as well as microscopic tissue lesions, and detecting the suspected toxin in grains, forages, or the ingesta of affected animals. However, the tests required to detect these toxins are complex and few diagnostic laboratories offer tests for multiple trichothecenes. The samples of choice include both refrigerated and frozen carcasses for necropsy examination and a representative sample of the suspected contaminated grain source. Because the toxin is produced under cold conditions, the grain sample should be frozen rather than refrigerated for shipment to the diagnostic laboratory.

Control

Wildlife should not be fed grain with levels of fusariotoxins in excess of those recommended for use in domestic animal food. In years when fusariotoxins are a problem, grain from fields that are frequently used by wildlife should be checked for toxins. If the fields are significantly contaminated, deep plowing of the contents of the field can make the grain unavailable to wildlife (Fig. 37.7). If the fields cannot be plowed, hazing wildlife from the area can lessen their exposure.

Human Health Considerations

Handling fusariotoxin-poisoned sick or dead birds does not pose a human health risk, but birds known to have died from acute fusariotoxicosis should not be consumed. The more potent trichothecenes in grain or forages may present a health hazard when the fungal spores and contaminated plant parts are inhaled or when they contact the skin.

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Supplementary Reading

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