Mycotoxicosis

Definition

Mycotoxicosis is a disease caused by a toxic fungal metabolite. Mycotoxicoses may affect both humans and animals. Poultry mycotoxicoses are usually caused by fungi that colonize and invade grains and feeds, but other environmental aspects may be involved.

Occurrence

1. Grains and forages used as foodstuffs support the growth of certain fungi when environmental conditions of temperature and humidity are suitable. Some of these fungi produce metabolites that are toxic to humans and animals and cause disease (mycotoxicosis) by either ingestion or cutaneous exposure.

2. Mycotoxicoses occur throughout animal-rearing regions of the world. Although specific mycotoxins form more frequently in certain geographic locations, interstate and international shipment of grains may result in widespread distribution of a mycotoxin problem.

Diagnosis

1. A definitive diagnosis of mycotoxicosis should involve the isolation, identification, and quantitation of the specific toxin(s). This is usually difficult to accomplish in the modern poultry industry because of the rapid and voluminous use of feed and ingredients.

Control

1. Prevention of mycotoxicoses requires the detection and control of mycotoxin contamination in feed ingredients and the application of feed manufacturing and management practices that prevent mold growth and mycotoxin formation.

2. Feeds and grains can now be screened for several mycotoxins (aflatoxin, T-2 toxin, ochratoxin, zearalenone) using monoclonal antibody detection kits. Many poultry companies already routinely test grain for aflatoxin contamination by a chromatographic procedure (minicolumn technique).

3. Mycotoxins can form in decayed, crusted, built-up feed in feeders, feed mills, and storage bins. This can be prevented by inspection of bins between flocks to certify absence of feed residue and by cleaning bins and feeders when necessary. Use of tandem feed bins on farms allows cleaning between successive feed deliveries.

4. Antifungal agents added to feeds to prevent fungal growth have no effect on toxin already formed, but may be cost-effective management in conjunction with other feed management practices. Several commercial products, most of which contain propionic acid, should be applied according to manufacturers’ instructions.

5. Zeolytes, a class of silica-containing compounds used as anticaking agents in feed formulation, and as aids in the improvement of eggshell quality, show promise as a practical and economical method of reducing the effects of certain mycotoxins. Hydrated sodium calcium aluminosilicate has been shown to bind aflatoxin B1, possibly by sequestration in the digestive tract, and reduce its toxicity to chickens.

Treatment

1. Remove the toxic feed and replace it with unadulterated feed.

2. Treat concurrent diseases (parasitic, bacterial) identified in the diagnostic evaluation.
3. Substandard management practices should be immediately corrected as they have increased detrimental
effects in a flock stressed by mycotoxins.

4. Vitamins, trace minerals (selenium), and protein requirements are increased by some mycotoxins and can
be compensated for by feed formulation and water-based treatment.

I. AFLATOXICOSIS

HISTORICAL INFORMATION

1. During the 1950s, a disease in dogs called hepatitis X occurred in the southeastern United States and was
tied to the consumption of moldy dog food. It was later reasoned to have been caused by the same
mycotoxin responsible for high mortality in turkeys due to hepatic toxicity (turkey X disease) in England in
1960. Peanut meal imported to England from Brazil was highly contaminated with fungi of the Aspergillus
flavus-Aspergillus parasiticus group, which produced aflatoxins.

2. The aflatoxin story was historically important because unlike ergotism and alimentary toxic aleukia, which
were sporadic and relatively localized phenomena, aflatoxicosis attracted global attention concerning the
potential problems of mycotoxins in the food chain, and the ease by which these problems could be widely
distributed.

ETIOLOGY

1. Mycotoxins of the aflatoxin group (B1, B2, G1, G2) are the cause of aflatoxicosis. Aflatoxin B1 is the
most common in grains and is highly toxic. Aflatoxin forms in peanuts, corn, and cottonseed, and their
products, in other grains, and in poultry litter. A. flavus is the primary producer of aflatoxin in grains, but
not all strains of the fungus are toxigenic.

2. Like other mycotoxins, aflatoxin is produced only when substrate, temperature, and humidity are ideal.
Favorable conditions for toxin formation may be localized within a volume of stored or transported grain
creating toxic "hot spots". Once formed, the toxin is stable.

3. Grains damaged by insects and drought stress, and broken pieces of grain (screenings) are more likely to
support fungal growth and toxin formation.

4. Aflatoxin B1 is a potent, naturally occurring carcinogen and thus has special public health considerations.

CLINICAL SIGNS

Aflatoxicosis in poultry is primarily a disease of the liver [Fig. 1; Aflatoxicosis; Univ Missouri] with
important ramifications for other body systems, which may ultimately cause production problems and mortality.
Affected birds have reductions in growth, carcass pigmentation, egg production, and immune function, and have
increased nutrient requirements for protein, trace elements (selenium), and vitamins. The disease may be fatal.

LESIONS

At necropsy, lesions are minimal with either transient exposure or exposure to a low concentration of toxin.
Jaundice, generalized edema and hemorrhages, tan [Fig. 2; Aflatoxicosis; Univ Missouri] or yellow
discoloration of the liver, and swelling of the kidneys [Fig. 3; Aflatoxicosis; Univ Missouri] are seen with more
severe intoxication. Microscopic changes in the liver occur as necrosis of hepatocytes, lipid accumulation in
hepatocytes, bile duct proliferation, and fibrosis. These are common reactions of this organ to toxic insult and
although they may be suggestive of aflatoxicosis, are not pathognomonic.
II. CITRININ MYCOTOXICOSIS

ETIOLOGY

Citrinin is a mycotoxin that was first isolated from *Penicillium citrinum* but is also produced by other species of *Penicillium* and by a few species of *Aspergillus*. Citrinin may be a factor in renal disease in food animals in Denmark, but no other documented case studies involving poultry are known.

CLINICAL SIGNS

Experimental citrinin mycotoxicosis in the chicken, turkey, and duckling has shown that chickens are relatively resistant, but all develop clinical illness of marked watery fecal droppings related to increases in water consumption and urine output. Metabolic alterations of electrolytes and acid-base balance occur. Young birds have reduced weight gain.

LESIONS

Citrinin produces marked functional changes in kidneys, however, gross lesions may be slight or overlooked. Swelling of kidneys and microscopic lesions of nephrosis may occur following severe exposure. In these circumstances, lymphoid tissues may be depleted and necrosis occurs in the liver.

III. ERGOTISM

HISTORICAL INFORMATION

1. Ergotism was recognized in central Europe in the Middle Ages and is the oldest known mycotoxicosis. Humans with ergotism (St. Anthony's fire) experienced an initial cold sensation in the hands and feet followed by an intense burning sensation. Gangrene of the extremities developed in both humans and afflicted animals. The disease occurred where bread was made from rye and other grains parasitized by toxigenic strains of the fungus *Claviceps purpurea*. The mold colonizes and replaces kernels of grain to form a hard, dark purple or black mass called an ergot or sclerotium.

2. Although the pharmaceutical properties of the ergot were recognized in China 5,000 years ago, it was not until 1875 that alkaloids present in the sclerotium were recognized as the cause of ergotism.

ETIOLOGY

1. The ergot alkaloids are a large family of compounds, and may cause constriction of blood vessels (vasoconstriction) those results in their pharmacologic and toxicologic effects.

2. *Claviceps* spp. that colonizes wheat, rye, and triticeal are the most common causes of ergotism of humans and animals.

CLINICAL SIGNS

In chickens, ergotism causes reductions in growth and egg production, and nervous incoordination.

LESIONS

Lesions include abnormal feather development, necrosis of the beak, comb, and toes, and enteritis.
IV. OCHRATOXICOSIS

HISTORICAL INFORMATION

1. Ochratoxin has been detected in kidneys of chickens with renal lesions in a processing plant in Denmark.

2. Three disease outbreaks in the United States involving 360,000 turkeys were associated with ochratoxin concentrations of up to 16 ppm.

ETIOLOGY

Ochratoxins A, B, and C are usually produced by toxigenic strains of *P. viridicatum* but may be produced by other species of *Penicillium* and by *Aspergillus ochraceus*. Ochratoxin A is the most toxic and is the greatest threat to poultry production.

CLINICAL SIGNS

1. Reductions in feed intake and increases in mortality.
2. Weight loss.
3. Drops in egg production have been reported from Ochratoxin A.

LESIONS

1. Gross and microscopic lesions in the kidneys and liver.

2. Experimental ochratoxicosis in chickens causes a dose-related reduction in weight gain, and gross and microscopic lesions occur in the target organs, liver and kidney. Visceral gout and reductions in plasma carotenoids, immune function, and certain blood coagulation factors also occur.

V. OOSPOREIN MYCOTOXICOSIS

ETIOLOGY

Oosporein is a toxic pigment produced by *Chaetomium* sp. and other fungi and is a contaminant of cereal grains and feedstuff.

CLINICAL SIGNS

Oosporein mycotoxicosis, studied in chickens and turkeys, causes a dose-related decrease in growth and an increase in water consumption. Chickens are more susceptible than turkeys.

LESIONS

Visceral and articular gout as a result of nephrotoxicity.
VI. TRICHOThECENE MYCOTOXICOsis
(Fusariotoxicosis)

HISTORICAL INFORMATION

1. A disease called alimentary toxic aleukia occurred in the Russian people in the early 20th century, the 1930s, and especially during the Second World War. Labor shortages during the war necessitated the overwintering of grains (wheat, rye, and millet) in the fields and harvesting was delayed until spring. Bread made from the new grain caused acute gastroenteritis, followed by the formation of ulcers of the face and oral membranes, facial edema and lymph node enlargement, and in the later stages, bone marrow disorders, anemia, and uncontrolled hemorrhages. Morbidity and mortality greater than 50% occurred in some villages. Similar problems occurred in livestock and poultry in the region.

2. The disease is now recognized as a mycotoxicosis caused by colonization of grains by toxigenic species of *Fusarium*. These fungi produce mycotoxins of the trichothecene group, many of which cause caustic injury to mucous membranes and skin, the basis of the facial, oral, and gastrointestinal features of the disease. They also affect rapidly dividing cells (radionimetic effect) manifested by disorders of the bone marrow (anemia, hemorrhagic disorders) and by abortions.

ETIOLOGY

More than 40 trichothecene mycotoxins are known to exist. T-2 toxin is one of the most toxic to poultry.

CLINICAL SIGNS

1. Chickens with fusariotoxicosis (trichothecene mycotoxicosis) have had reduced growth, abnormal feathering [Fig. 1; Trichothecene; Auburn Univ], severe depression, and bloody diarrhea.

2. In chickens, pigeons, ducks, and geese, the caustic properties of the trichothecenes have been manifested as feed refusal, extensive necrosis of the oral mucosa and areas of the skin in contact with the mold, and symptoms of acute gastrointestinal disease.

3. Experimental fusariotoxicosis, reproduced in chickens with pure T-2 toxin closely resembled the spontaneous disease but lacked the extensive hemorrhages.

LESIONS

1. Trichothecene mycotoxicosis may cause necrosis of the oral mucosa [Fig. 2; Trichothecene; Auburn Univ], reddening of the mucosa of the remainder of the gastrointestinal tract, mottling of the liver, distention of the gallbladder, atrophy of the spleen, and visceral hemorrhages.

VII. ZEARALENONE MYCOTOXICOsis

HISTORICAL INFORMATION

In experimental studies, chickens have shown relative insensitivity to the effects of zearalenone. Zearalenone mycotoxicosis has been recognized since 1927 as the cause of a syndrome resembling estrogen stimulation in pigs and cattle in the United States and elsewhere.
MYCOTOXICOsis

ETIOLOGY

Zearalenone is a mycotoxin produced by *Fusarium roseum* (*Gibberella zeae*) and other *Fusarium* spp. A period of warm temperature and high humidity followed by low temperature is most conducive to toxin formation on grains.

CLINICAL SIGNS

1. Zearalenone-contaminated feed has been associated with high mortality (40%) in a flock of 24,000 broiler breeder chickens. Affected birds had cyanotic combs and wattles and had difficulty walking.
2. Turkeys may develop swelling of the cloaca and reduced fertility.
3. Male geese may have reductions in sperm quantity and viability.

LESIONS

1. Affected chickens have had ascites and cysts both inside and outside of the oviduct. The oviducts were swollen and inflamed, and were obstructed with fibrinous fluid. Some oviducts had ruptured.
<table>
<thead>
<tr>
<th>Names</th>
<th>Aflatoxins</th>
<th>Fumonisins</th>
<th>Oosporein</th>
<th>Tricothecene</th>
<th>Ochratoxins</th>
<th>Citrinin</th>
<th>Ergotism</th>
<th>Zearealenone</th>
</tr>
</thead>
<tbody>
<tr>
<td>B1, B2, G1 and G2 are natural contaminants</td>
<td>T2, DAS, DON more than 100 fungal metabolites</td>
<td>Ochratoxins A, B &amp; C</td>
<td>Ergot alkaloids (ergotamine, ergocristine)</td>
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<td>Major producers</td>
<td>Aspergillus mostly Aspergillus flavus and A. parasiticus</td>
<td>Fusarium moniliforme</td>
<td>Chaetomium trilaterale</td>
<td>Primarily isolated from Fusarium spp. Type A (more toxic to chickens) 14 PEB</td>
<td>Produced by several Aspergillus penicillium viridicatum and other species of penicillium Ochratoxin A – the most common toxic mycotoxin for poultry and the most toxic</td>
<td>Penicillium citrinum Claviceps purpurea or other claviceps species</td>
<td>Fusarium graminearum Fusarium roseum</td>
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<td>Toxic action</td>
<td>Target organ: Liver *potent hepatocarcinogen in humans</td>
<td>Disruption of sphingolipid synthesis Very low toxicity for poultry</td>
<td>Primary renal tubular damage</td>
<td>Primary inhibition of protein synthesis followed by secondary disruption of DNA &amp; RNA synthesis Affect rapidly dividing cells such as those lining the GI tract, the skin, and lymphoid and erythroid cells Extensive necrosis of mucous membranes and skin in contact with the toxin Acute effects on digestive tract and bone narrow Immunosuppressive</td>
<td>Target organ: the kidney Interferes with DNA, RNA &amp; protein synthesis Affects renal carbohydrate metabolism (gluconeogenesis) = damage to the epithelium of renal proximal convoluted tubules Decreased electrolyte absorption Increased water excretion</td>
<td>Reversible renal damage</td>
<td>Arterial and venous vasoconstriction Necrosis of peripheral tissues Decreased blood flow to extremities Possible endothelial damage</td>
<td>Potent estrogenic properties Low toxicity for poultry</td>
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<tr>
<td>Clinical signs</td>
<td>Decreased feed intake Decreased body weight poor skin Decreased egg production Decreased immunity</td>
<td>Decreased body weight</td>
<td>Dose-related decrease in growth Increased water consumption</td>
<td>Decreased feed intake Reduced growth Severe depression Abdominal feathering Bloody diarrhea</td>
<td>Decreased feed intake Weight loss Increased mortality Increased water consumption Humid litter Decreased egg production</td>
<td>Marked watery fecal droppings Increased water consumption Increased diuresis Reduced weight gain(young birds) Humid litter</td>
<td>Reduced growth Decreased egg production Nervous signs (incoordination)</td>
<td>Reduced fertility</td>
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<td>MYCOTOXICOSIS</td>
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<th><strong>Ergotism</strong></th>
<th><strong>Zearalenone</strong></th>
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<tbody>
<tr>
<td>Lesions</td>
<td>Jaundice</td>
<td>Increased liver weight</td>
<td>Dehydration</td>
<td>Pale and swollen kidneys</td>
<td>Swollen kidneys</td>
<td>Gangrenous-like lesions</td>
<td>Oviduct hypertrophy</td>
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<td></td>
<td>Generalized edema and hemorrhages tan or yellow discoloration of the liver</td>
<td>Increased kidney weight</td>
<td>Swollen and pale kidneys secondary visceral and articular gout</td>
<td>Secondary visceral gout</td>
<td>Degeneration and necrosis of tubular epithelial cells of both proximal and distal tubules.</td>
<td>Necrosis of the beak, comb, toes Enteritis</td>
<td>Cloacal swelling (turkeys)</td>
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<td>Liver: Periportal necrosis with bile duct proliferation and fibrosis</td>
<td>Liver hepatic necrosis with biliary hyperplasia</td>
<td>Circumscribed proliferative yellow caseous plaques in oral mucosa</td>
<td>Pale and enlarged liver</td>
<td>Pale and swollen kidneys</td>
<td>Reduction in sperm quantity and viability (geese)</td>
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<td></td>
<td>Depletion of lymphoid organs</td>
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<td>Reddening of GI mucosa</td>
<td>Regression and cellular depletion of lymphoid organs.</td>
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<td></td>
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<td></td>
<td>Mottling of the liver</td>
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<td>Gallbladder distention</td>
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<td></td>
<td></td>
<td></td>
<td>Splenic atrophy</td>
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<td></td>
<td></td>
<td></td>
<td>Visceral hemorrhages</td>
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<tr>
<td>Sources</td>
<td>Peanuts, corn, cottonseed, and their products, In other grains and in poultry litter</td>
<td>Corn and corn based feed</td>
<td>Fusarium spp. are important pathogens to plant producing cereal grains, (corn, wheat, barley, oats, rice, rye…)</td>
<td>Widespread natural contaminant of cereal grains (barley, oats, rye maize)</td>
<td>Often coexists in cereals with ochratoxin A (corn, wheat, barley, oats, rye and rice)</td>
<td>Open inflorescence of graminaceous plants (rye, wheat, triticale barley, oats, sorghum, corn, rice) and several grass species.</td>
<td>Corn, corn products, rice</td>
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<tr>
<td>Treatments</td>
<td>None</td>
<td>Vitamin C supplementation might reduce some adverse effects.</td>
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