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Aflatoxicosis in Poultry: A Review

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Abstract

Aflatoxins are secondary metabolites produced by toxigenic strains of *Aspergillus flavus* and *Aspergillus parasiticus* on peanuts, soybeans, corn (maize) and other cereals either in the field or during storage when moisture content and temperatures are sufficiently high for mold growth. The toxic response and disease in mammals and poultry varies in relation to species, sex, age, nutritional status and the duration of intake and level of aflatoxins in the ration. Earlier recognized disease outbreaks were called "moldy corn toxicosis," "poultry hemorrhagic syndrome," and "Aspergillus toxicosis" may have been caused by aflatoxins.

Key words: Aflatoxicosis, Aflatoxins, Poultry

Introduction

Aflatoxicosis occurs in many parts of the world and affects growing poultry (especially ducklings and turkey poults), young pigs, pregnant sows, calves, and dogs. Experimentally, all species of animals tested have shown some degree of susceptibility. Dietary levels of aflatoxin (in ppb) generally tolerated are ≤ 50 in young poultry, ≤ 100 in adult poultry. Approximately two times the tolerable levels stated is likely to cause clinical disease, including some mortality.^[1]

Pathogenesis

Aflatoxins are metabolized in the liver to an epoxide that binds to macromolecules, especially nucleic acids and nucleoproteins. Their toxic effects include mutagenesis due to alkylation of nuclear DNA, carcinogenesis, teratogenesis, reduced protein synthesis, and immunosuppression. Reduced protein synthesis results in reduced production of essential metabolic enzymes and structural proteins for growth. The liver is the principal organ affected. High dosages of aflatoxins result in hepatocellular necrosis; prolonged low dosages result in reduced growth rate, immunosuppression, and liver enlargement.^[2]

Clinical findings

In acute outbreaks, deaths occur after a short period of inappetence; other acute signs include vomiting, depression, haemorrhage, and icterus. Subacute outbreaks are more usual, with unthriftiness, weakness, anorexia, reduced growth, feed efficiency, and occasional sudden deaths. Laboratory changes in most species are related to liver damage, coagulopathy, and impaired protein synthesis. Specific laboratory changes include increased AST, ALT, and alkaline phosphatase; hypothermia, prolonged prothrombin and activated partial thromboplastin times, hyperbilirubinemia, hypocholesterolemia, hypoalbuminemia, and variable thrombocytopenia. Generally, aflatoxin concentrations in feed twice the tolerable levels given above are associated with acute aflatoxicosis. Liver damage can lead to reduced clotting factor synthesis with acute to chronic haemorrhage. Subclinical effects include reduced growth rate and feed efficiency, hypoproteinemia, and reduced resistance to some infectious diseases despite vaccination.^[3]

Lesions

In acute cases, there are widespread haemorrhages and icterus. The liver is the major target organ. Microscopically, the liver is enlarged and shows marked fatty accumulations and massive centrilobular

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necrosis and haemorrhage. In sub acute cases, the hepatic changes are not so pronounced, but the liver is somewhat enlarged and firmer than usual. There may be edema of the gallbladder. Microscopically, the liver shows periportal inflammatory response and proliferation and fibrosis of the bile ductules; the hepatocytes and their nuclei (megalocytosis) are enlarged. The GI mucosa may show glandular atrophy and associated inflammation. Rarely, there may be tubular degeneration and regeneration in the kidneys. Prolonged feeding of low concentrations of aflatoxins may result in diffuse liver fibrosis (cirrhosis) and rarely, carcinoma of the bile ducts or liver may be seen.^[4,7]

Diagnosis

Disease history, laboratory data, necropsy findings, and microscopic examination of the liver should indicate the nature of the hepatotoxin, but hepatic changes are somewhat similar in *Senecio* poisoning. The presence and levels of aflatoxins in the feed should be determined. Acutely affected animals have increased liver enzymes (alkaline phosphatase, AST or ALT), bilirubin, serum bile acids and prothrombin time. Chronic exposure can cause hypoproteinemia (including decrease in both albumin and globulin). Aflatoxin M₁ (principal metabolite of aflatoxin B₁) can be detected in urine, liver, kidney.^[5]

Prevention and control^[6,7]

- Contaminated feeds can be avoided by monitoring batches for aflatoxin content. Local crop conditions (drought, insect infestation) should be monitored as predictors of aflatoxin formation.
- Young, newly weaned, pregnant, and lactating animals require special protection from suspected toxic feeds. Dilution with non-contaminated feedstuffs is one possibility, but this may not be acceptable on a regulatory basis.
- Cleaning to remove lightweight or broken grains will often substantially reduce mycotoxins concentration in remaining grain. Ammoniation reduces aflatoxin contamination in grain but is not currently approved by the FDA for use in food animals in the USA because of uncertainty about by-products produced.
- Numerous products are marketed as anticaking agents to sequester or "bind" aflatoxins and reduce

absorption from the GI tract. One effective binder for aflatoxins is hydrated sodium calcium aluminosilicates (HSCAS) which reduce the effects of aflatoxin when fed to pigs or poultry at 10 lb/ton (5 kg/tonne). They also provide substantial protection against dietary aflatoxin. HSCAS reduce aflatoxin M₁ in milk by ~50% but do not eliminate residues of aflatoxin M₁ in milk from dairy cows fed aflatoxin B₁. Other adsorbents (sodium bentonites, polymeric glucomannans) have shown variable but partial efficacy in reducing low-level aflatoxin residues in poultry and dairy cattle.

- The use of clay-based adsorbents has proved effective at reducing the toxic effects of aflatoxin-contamination in animal feeds

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