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# Aflatoxicosis in Poultry

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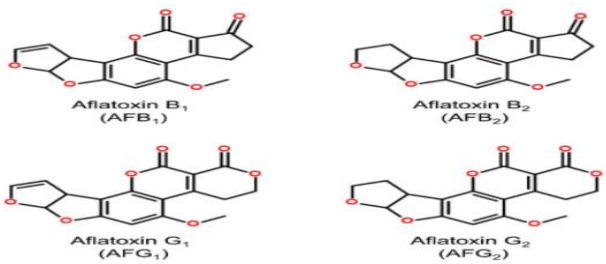
***Abstract:*** **Aflatoxicosis is among the major cause of economic losses in poultry production. Aflatoxins are a group of hepatotoxic compounds produced by the fungus of *Aspergillus* sps. when growing on feedstuffs. Aflatoxins are hepatotoxic, mutagenic and carcinogenic fungal toxin which is capable of producing diseases in farm animals as well as poultry. There are four primary aflatoxins: aflatoxin B1 (AFB1), aflatoxin B2 (AFB2), aflatoxin G1 (AFG1) and aflatoxin G2 (AFG2). Among these AFB1 is the most toxic aflatoxin. Aflatoxicosis in poultry is characterized by decreased growth rate, poor feed conversion, immunosuppression, passage of undigested food in the dropping, anemia, decrease egg production in layers quantitatively and qualitatively, decrease hatchability, embryonic mortality, reduced fertility due to decrease testicular weight, decrease semen volume and sometimes there may be lamness, ataxia, convulsions & death. In humans being acute aflatoxicosis is manifested by vomiting, abdominal pain, pulmonary edema, coma, convulsions, and death with cerebral edema and fatty involvement of the liver, kidney and heart.**

***Keywords:*** **Aflatoxin, Poultry, Hepatotoxic.**

## 1. INTRODUCTION

Mycotoxins are ubiquitous in nature and produced by several fungi. Many species of fungi like *Aspergillus, Fusarium, Penicillium, Claviceps,* and *Alternaria* etc particularly produces these mycotoxins. Mycotoxins are the secondary metabolites of fungi. Over 400 known mycotoxins have been identified with a potential 30,000 different metabolites. Most common mycotoxicosis affecting commercial poultry are Aflatoxicosis and ochratoxicosis (Pattison *et al*., 2008).

Aflatoxin is hepatotoxic, mutagenic and carcinogenic fungal toxin which is capable of producing diseases in farm animals as well as poultry and is known to be produced by specific moulds in feed commodities (Bennet and Klich, 2003). *Aspergillus* species produce most potent fungal toxin causing aflatoxicoses that have harmful effects on living beings. Aflatoxins belong to a heterologous group of fungal secondary metabolites called mycotoxins that adversely affect human and animal health. Aflatoxins are most commonly produced by strains of *Aspergillus flavus, A. parasiticus, and A. nominus,* although many other *Aspergilli* have aflatoxin producing capabilities. Of the over 180 species of *Aspergillus*, only a few are aflatoxigenic. Aflatoxins are named according to their blue or green fluorescence under UV light, there are four primary aflatoxins: aflatoxin B1 (AFB1), aflatoxin B2 (AFB2), aflatoxin G1 (AFG1) and aflatoxin G2 (AFG2) (Yiannikouris and Jouany, 2002). AFB1 is the most toxic and is classified as human carcinogen (Talebi *et al*., 2011).




**Molecular structures of the four primary aflatoxins**

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In humans, acute aflatoxicosis there is vomiting, abdominal pain, pulmonary edema, coma, convulsions, and death with cerebral edema and fatty involvement of the liver, kidney and heart (Strosnider *et al*., 2006).

**Sources:**

Poultry birds become exposed to AFB1 and other aflatoxins by consuming contaminated feed. Corn, cottonseed, peanuts, sorghum and other foods (figs,tree nuts and spices) are at especially high risk. Warm and humid conditions during maturation, harvest, transport or storage, promote *Aspergillus* growth and aflatoxin production. Aflatoxins can also be transfered to milk, meat and eggs of livestock and poultry fed the toxins. Therefore, AFB1 is a human food safety risk in both plant and animal products.

**Sensitivity:**

Ducks > Turkeys > Japanese quail > Chickens (Lozano and Diaz,2006; Rawal and Kim, 2010)

**AFB1 Metabolism:**

$AFB_1$ require bioactivation for to be toxic and bioactivation predominantly occurs in liver (Bedard and Massey, 2006 and Rawal and Kim, 2010). AFB1 is absorbed in the small intestine and majority of the toxin is metabolized in the liver. In the liver AFB1 is converted by hepatic cytochromes P450 enzymes into the reactive and electrophilic AFB1-8,9- exo-epoxide (AFBO) (Bedard and Massey, 2006; Eaton and Gallagher, 1994; Rawal and Kim, 2010; Klein *et. al*, 2000). An endo stereoisomer of AFBO epoxide can also be produced, but is less toxic and is not relevant to $AFB_1$ toxicity (Bedard and Massey, 2006; Eaton and Gallagher, 1994). AFBO is a highly unstable intermediate and quickly reacts to form adducts with DNA, RNA and proteins, responsible for AFB1 toxicity (Bedard and Massey, 2006; Eaton and Gallagher, 1994). AFBO is primarily detoxified by glutathione S-transferase (GST) enzymes that add glutathione (GSH) to form an 8,9-dihydro-8-(S-glutathionyl)-9-hydroxy-AFB1 (AFB1-SG) adduct.( Rawal and Kim, 2010; Eaton and Gallagher, 1994; Klein *et. al*, 2000). Other metabolites of AFB1 are aflatoxicol, aflatoxin M1, aflatoxin P1 and aflatoxin Q1.

**Clinical signs:**

Clinical signs of aflatoxicosis in poultry envolves decreased growth rate, poor feed conversion, immunosuppression, vaccination failure, decrease resistance to infectious disease, passage of undigested food in the dropping, anemia, decrease egg production in layers quantitatively and qualitatively, decrease hatchability, embryonic mortality, reduced fertility due to decrease testicular weight, decrease semen volume (sperm counts), and sometimes there may be lamness, ataxia, convulsions & death.

Symptoms depend on the type of toxin, amount, duration of the exposure, age, health status and synergistic effects of genetics, dietary status, and interactions with other toxic insults (Bennett and Klich, 2003).

**Post mortem findings:**

Liver becomes swollen, friable, pale or yellow due to precipitation of fat and hyperplasia of bile ducts epithelium, inflammation of these ducts , gallbladder enlarged and in the late stage fibrosis of the liver may occur. Enlargement of the kidneys, spleen and pancreas. Atrophy of bursa of Fabricius, thymus and testes. Hemorrhages present under the skin, on muscles and on the internal organs due to fragility of blood vessels (Biro *et al.*, 2002). In chronic stage there is ascites and hydropericardium.

## 2. HISTOPATHOLOGY

Liver reaveled vacuolation of hepatic cells and bile duct proliferation. Metabolic alterations caused by aflatoxins in chickens result in elevated lipid levels, disruptions in hepatic protein synthesis, which result in several blood coagulation disorders, immunosuppression and decreased plasma amino acid concentrations (Sumit *et al*., 2010). Kidneys revealed thickened basement membrane in the glomeruli and associated hyaline droplets in the renal tubules. It is not known if the glomerulus is damaged by toxins or by a leakage of unusual protein from a severely damaged liver. Liver lesions in chicken are characterized by retrogressive and regenerative parenchymal changes. (Herenda and Franco, 1996). R.E. cell hyperplasia along with depletion of lymphocyte observed in spleen. Bursa of Fabricus revealed degeneration and depletion of lymphoid cells in bursal follicle.

## 3. DIAGNOSIS

Diagnosis is based on history, clinical signs, gross and microscopic lesions. By examination of grains or feeds externally showing change in colour or growth of fungi. Definite diagnosis depends on identification of toxin.

Traditional methods: Bioassays using ducklings, chicken embryos and brine shrimp larvae were used for toxicity testing of feed ingredients (Watson and Lindsay, 1982; Celik *et al.*, 2000).

Now a days detection largely depends on chromatographic techniques using sophisticated equipments. HPLC using fluorescence detection become the most accepted method for the determination of aflatoxins (Blesa *et al.*, 2005). Rapid immunochemical screening methods like enzyme-linked immunosorbent assay (ELISA) and immunoaffinity column assay (ICA) have been developed and are commercially available (Schiefer, 1990). Rapid minicolumn method for aflatoxin detection in agricultural products (Arim *et al.*, 1999). El-Nagerabi and Elshafie (2001) developed a test known as the Bright greenish-yellow fluorescence (BGYF) test. Feed samples are inspected under UV light for a characteristic bright greenish yellow fluorescence in damaged kernels. ELISA has been routinely used for detection of aflatoxin B1 in cereals and animal tissues (Dutta and Das, 2001; Gathumbi *et al.*, 2003; Lee *et al.*, 2004). An antibody-colloidal gold probe conjugate has been developed recently for rapid and specific detection of AFB1. Mass spectroscopy(MS) in combination with liquid chromatography is becoming popular for identification of aflatoxins (Sulyok *et al.*,2006; Cavaliere *et al.*, 2007).

Recent and advanced techniques

- Biosensors: Biosensor assay works on the surface plasmon resonance (SPR) (Zheng and Binder, 2005; Logrieco *et al.*,2005).
- MicroSERS is a new biochip technology that uses surface-enhanced Raman scattering (SERS) microscopy for label-free transduction and detection of aflatoxins (Grow *et al.*, 2003).
- Polymerase chain reaction (PCR): Primers for aflatoxin regulatory (afl R), o-methyltransferase (omt) and oxidoreductase (ord) genes are used.

**Strategies to Reduce AFB1 Toxicity:**

- Use of preservatives and anti-fungal agents, such as propionic acid.
- Mould inhibitors like hydroxyquinoline effective in reducing aflatoxin formation (Rao, 1995).
- Chemical Detoxification*:* Ammonium hydroxide, calcium hydroxide, sodium hydroxide and sodium hypochlorite reduce AFB1 concentrations through hydrolysis.
- Probiotics: Many Gram-positive bacteria, including *Streptococcus, Enterococcus, Lactococcus, and Berevibacillus,* can bind AFB1. Interactions between AFB1 and *Lactobacillus rhamnosus GG L.rhamnosus LC-705* or mixtures of these strains have been shown to be especially effective.
- Selection for Resistance*:* Selection for AFB1 resistant lines of chicken and quail has been investigated.

## 4. PREVENTION

For prevention of aflatoxicosis shipments falling outside acceptable levels should be rejected. Antifungals as organic acids, copper sulphate, gentin violet must be added routinely to the feeds to prevent fungal growth in storage feeds.Ozone treatment of grain can be used. Feed must be stored in well ventilated place, and walls to avoid excess moisture. Feed should be prepared fresh as fresh (store time must not exceed than 14 days). Litter must stored in well ventilated places and in poultry house must preserved it dry. We should use the practice of regular inspection of feed and cleaning and disinfections of grain mills, feeding troughs with formalin 2-3 % or copper sulphate. Pelleted feed should be preffered because during their manufacturing process cause destruction of fungal spores.

## 5. CONCLUSIONS

Aflatoxins are considered as primary cause of abnormal health status and productive performances in birds. Because of increased susceptibility to infections and drastic reduction in production potential aflatoxins causes a significant loss to poultry industry. Aflatoxins are of much concern to humans also as it is present as residues in milk, meat, eggs and other animal products. Detoxifying agents and toxin binders have to be used in order to make sure that the animals are receiving safer feed materials. Future gene expression analyzes can provide insight into the mechanisms of aflatoxicosis and methods to reduce its effects. Education and awareness of farmers about aflatoxicosis is important for dealing with this problem. Organizations at National and International level are to constantly monitor the risk aflatoxins poses to animal health and need to draft legal limits for their effective control and prevention.

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