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Aflatoxin: A Dangerous Substance in Poultry Feed

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Introduction

Aflatoxicosis represents one of the serious diseases of poultry, livestock and other animals. The cause of this disease in poultry and other food-producing animals has been attributed to the ingestion of various feeds contaminated with *Aspergillus flavus*. This toxigenic fungus is known to produce a group of extremely toxic metabolites-aflatoxins. The 4 aflatoxins of concern are B1, B2, G1, and G2. However, B1 is the most predominant and toxic to poultry. Avian species especially chicks, goslings, ducklings and turkey poults are most susceptible to AFB1 toxicity. Even though prevention and avoidance are the best way to control aflatoxicosis, natural contamination of crops with *Aspergillus flavus*is sometimes unavoidable. Such aflatoxin-contaminated feeds can be decontaminated using various methods which mainly focus on physical removal or chemical inactivation of the toxins in the feeds. Moreover, dietary additives such as activated charcoal, phenobarbital, cysteine, glutathione, betacarotene, fisetin and selenium have also been reported to be effective in the reduction of aflatoxicosis in poultry.

Metabolism

Bioactivation is required for AFB_1 to be toxic and this processing predominantly occurs in hepatocytes. AFB_1 is initially absorbed in the small intestine, especially the duodenum. While bioactivating enzymes with low affinity for AFB_1 are present in the small intestine, the majority of the toxin is metabolized in the liver, where AFB_1 is converted by hepatic cytochromes P450 (*CYP*) enzymes into the reactive and electrophilic *exo*-AFB_1-8,9-epoxide (AFBO). An *endo* stereoisomer of the AFBO epoxide can also be produced, but is far less toxic and not relevant to AFB_1 toxicity.

Harmful effects of Aflatoxicosis:

Aflatoxicosis can develop in both acute and chronic form. The chronic form is the most common and is the result of continuous ingestion of contaminated feed over a period of time. The toxic effects of AFB1 are mainly localized in liver as manifested by hepatic necrosis, bile

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duct proliferation, icterus and hemorrhage. Chronic toxicity in those birds is characterized by loss of weight, decline in feed efficiency, drop in egg production and increased susceptibility to infections. Effects of aflatoxicosis are as follows:

- **Immunosuppression**: Increases susceptibility to secondary infection by opportunistic pathogens.
- **Reduced vaccine effectiveness**: Decreases resistance against viral pathogens, increasing the risk of acquiring the disease they are getting vaccinated against.
- **Gastrointestinal tract damage**: Causes erosion and roughening of the gizzard lining, resulting in decreased absorption of nutrients, stunted growth, nutrient-deficient related syndromes, and lowered feed conversion.
- **Liver damage**: The primary site of toxicity due to aflatoxins is the liver. Exposure causes hepatic lesions and enlargement of the liver as well as fatty liver.
- **Production losses**: Decreases egg production and egg quality in laying hens and hatchable eggs in breeding chickens.
- **Mutagenicity**: Binding of AFBO to DNA introduces G-T transversion mutations in hepatic DNA. AFBO is highly mutagenic in poultry.

Clinical sign of Aflatoxicosis:

- Loss in appetite
- Undigested feed droppings
- Stunted growth
- Weight loss
- Hudding
- Heat stress
- Paleness
- Deceased egg production
- Reduced egg size
- Increased feather pecking behaviour
- Lameness
- Convulsion
- Sudden death

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Susceptibility to aflatoxins

All animals are susceptible to aflatoxins, but the sensitivity varies between species. Poultry are sensitive to even low levels of AFB_1, and among species of agricultural importance, the order of sensitivity is ducks > turkeys > Japanese quail > chickens. Therefore, lower concentrations of AFB_1 are lethal to turkeys and ducks and more adversely affect production and health in these species.The incidences of hepatocellular tumors, particularly in ducklings, is considered to be one of the serious consequences of aflatoxicosis.In domestic turkey, efficient production of AFBO contributes to sensitivity. We have conducted considerable research to determine the P450 enzymes responsible for AFB_1 bioactivation and metabolism in turkey livers. This work revealed by turkey enzymes P450,encoded by *CYP1A5* and *CYP3A37*, predominantly responsible for converting AFB_1 into AFBO *in vitro* and *in vivo*. P450 1A5 has high-affinity (high V_{max}, K_{cat}; low K_m) and catalyzes the production of both *exo*-AFBO and the detoxified metabolite AFM_1 according to traditional Michaelis-Menten kinetics. P450 3A37 is the lower affinity catalyst, exhibiting apparent subunit allostery conforming to Hill enzyme kinetics and producing *exo*-AFBO and AFQ_1

Control of aflatoxins in poultry feed:

A practical aflatoxins control completed in two stages i.e. preventing the fungal growth as well as toxin production and secondly decontamination of existing aflatoxins in poultry feed. Several works as well strategies have been proposed by researchers such as plant breeding in such a way which can reduce the mould growth. Simultaneously improving harvesting and storage practices which can minimise contamination procedure hence reduce in aflatoxins production. For the toxin already present in the feed the most effective method for control is by addition of adsorbents which can prevent the toxin absorption from intestine. The addition of binder such as zeolite clays and aluminosilicates is very effective in reducing aflatoxins related toxicity. There are many clays base products marketed by various companies present in market having good efficacy against aflatoxins. One method of reducing moderate levels of aflatoxins contamination is by mixing good quality grain in aflatoxins contaminated feed, as this will help in reducing the overall level of aflatoxins in feed. This method is being applied by farmers at various level to control Mortality of birds as it helps in slowing down the metabolism of bird by lower density nutrition and secondly dilution of aflatoxins. Some Nutritional Modifications in feed are also very much useful for protection against aflatoxins

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which includes higher supplementation of vitamins, methionine and selenium. There are few chemicals such as sodium bisulphite, ammonia and peroxide acids tested for detoxification but found impractical especially due to safety and palatability issue

- Contaminated feed must be stopped (feed stock and feeds in feeding troughs) and replaced by another feed free from aflatoxin, birds recover fiorn most mycotoxins soon after replacing contaminated food by uncontaminated one.
- Treat the birds with some antitoxic substances that having the ability to bind with aflatoxin and prevent its absorption from the gastrointestinal tract, some of these substances can be used in the feed and the other in water as sodium calcium aluminosilicates or bentonites or zeolites or clinoptilolites fine particles size superactivated charcoal had marginal or no binding activity for aflatoxin or T -2 toxin.
- Because some vitamins E and C counteract the toxicity of T2 toxin or ochratoxin A. vitamins, trace elements especially selenium (protective by increasing the percentage of conjugated aflatoxin), protcins especially methionine and cysteine which counteract aflatoxin and lipids must be compensated by feed formulation.
- Birds vaccinated during the clinical signs of aflatoxicosis must be revaccinated
- Treat concurrent bacterial or parasitic infections.

Conclusion

Aflatoxins are a group of hepatotoxic compounds produced by the mold *Aspergillus flavus* when growing on feedstuffs. There are four naturally occurring aflatoxins: B1, B2, G1 and G2. B1 is the most concentrated and toxic of these**.** Aflatoxins adversely affect poultry resulting to liver damage, poor production and heavy economic loss. The numerous effective pre-harvest and post-harvest biocontrol method for aflatoxins mitigation have been applied. Nutritional management including use of binders and antioxidants helps in counteracting the deleterious impacts of AFB1 on poultry.