Mycotoxins in Poultry Production

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Abstract: The occurrence of mycotoxins has become a problem to be discussed, due to its harmfulness to humans, animal's health and consider an obstacle to the poultry economy. Mycotoxins are toxic metabolites produced by certain species of fungi and may contaminate food. The fungi can grow in incorrectly stored feeds especially which stored under unfavourable environmental conditions (high relative humidity and elevated temperature). Aflatoxins are mainly produced by *Aspergillus flavus* and *Aspergillus parasiticus*, and B1, B2, G1 and G2 are its best known types. Fumonisin, with its B1, B2 or B3 types, are produced by Fusarium, while ochratoxin A is produced by Penicillium and Aspergillus. The main trichothecenes mycotoxins are T-2 toxin, deoxynivalenol and diacetoxyscirpenol. Zearalenone, produced by different species of Fusarium fungi affects chickens only when they are exposed to extremely high levels of contamination. Generally, immunosuppression, hepatotoxicity and nephrotoxicity as a decrease in performance and production gains are the most observed effects. There are several laboratory methods that can be used for the determination of mycotoxins. In order to control the contamination, it is necessary to adopt proper farming practices which prevent fungi growth.
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Introduction

Mycotoxins are toxic metabolites produced by some species of filamentous fungi and may contaminate food for human and animal consumption. About 400 different mycotoxins have been identified, which differ greatly in size and structural shapes **(Iamanaka et al. 2010)**.

In tropical and subtropical climates, the fungal development finds favorable conditions of humidity and temperature (Dilkin 2002). However, the occurrence of mycotoxins is not onlyaproblem from developing countries. Agribusiness in many countries is affected, and this may interfere with or even prohibit exportation, reducing animal and agricultural production **(Leung et al. 2006).**

The production of mycotoxins depends on fungal growth and may occur at any stage of the plant growth, harvest, or storage of food. However, fungal growth and mycotoxins are not synonymous, since not all fungi species produce toxins. The non-detection of signs of fungus presence does not indicate the absence of mycotoxins, since this may remain after elimination of the fungus or thermal treatment situations in which the fungus may have been deleted, but the toxin persists due to their thermotolerance **(Zhu et al. 2016).**

The contamination by a mycotoxin may occur indirectly and directly. Indirect contamination occurs when toxins in food remain even after the destruction of fungi. On the other hand, direct contamination occurs when the ingredient or the feed becomes contaminated by a toxigenic fungi, with subsequent formation of mycotoxins **(Frisvad & Samson 1991, Iamanaka et al. 2010).**

Analyses carried out in the 90s showed almost half of all commodities produced in the world, especially staple foods, was in some way contaminated by mycotoxins **(Bhat & Miller 1991).** In developing countries the problem is even more serious. Where as good quality products are usually exported, those commodities of inferior quality, with higher mycotoxins levels than those permitted in importing countries are sold and consumed internally, with obvious risks to human and animal health **(Dawson 1991).**

High levels of mycotoxins in feed contribute to acute mycotoxicoses and high mortality rate. Lower levels cause chronic mycotoxicoses with or without clinical symptoms, followed by considerable decrease in production performances, immunosuppressive effects and presence of residues in poultry meat and eggs. Primarily, the toxicity of mycotoxins depends on the type, quantity and duration of ingestion of mycotoxins, species, gender and age of the animal, general health, immune and nutritional status, as well as environmental factors. Aflatoxicosis and ochratoxicosis are the most common mycotoxicoses in commercial poultry **(Pattison et al., 2008).**

Interaction between mycotoxins and broiler immunity

With the consumption of contaminated food, cells of the intestinal mucosa, which possesses both components of innate as specific immunity may be exposed to large concentrations of these toxins **(Prelusky et al. 1996).** As described by **Bouhet & Oswald (2005),** the function performed by the physical barrier of the intestinal epithelium is achieved by trans-epithelial electrical resistance that exists in the cell monolayer.

Some toxins may affect this trans-epithelial electrical resistance in the intestinal mucosa. **Mclaughlin et al. (2004)** explained this can happen due to the decrease in the amount of proteins found in the cell junctions.

On the other hand, cells of the intestinal mucosa which make this innate physical protection.

Are comprised of a constantly renewed tissue to maintain the integrity of the epithelium, which occurs from the proliferation of differentiated cells from the crypt, which differentiate and move along, being eliminated by extrusion at the height of intestinal villi. It is also known that mucus production has an important function as a lubricant and protective.

barrier of this epithelium, and when the intestinal mucosa is challenged, there is an increase in the number of these cells in the intestine, with increased mucus production. The influence of toxic fungal metabolites in mucosal immunity can greatly affect the animal performance, as the induction of immunity is very important to ensure protection against various pathogens which typically invade these surfaces **(Streatfield 2006).**

Mycotoxins affecting broiler production

Aflatoxins

The term aflatoxin was created based on the name of its main producer (*Aflavus*). The main known aflatoxins are B1, B2, G1 and G2, with an established classification based on their fluorescence under ultraviolet light (B¼ blue, green G ¼) and mobility for thin layer chromatography. They are mainly produced by *Aspergillus flavus* and *Aspergillus parasiticus*. However, recently, the species *Aspergillus nomius*, *Aspergillus bombycis*, *Aspergillus pseudotamari* and *Aspergillus ochraceoroseus* have also proved to be aflatoxigenic **(Moss & Long 2002).** Aflatoxins are secondary metabolites associated with toxicity caused by food in animals and are reported to be hepatotoxic, mutagenic, immunosuppressive and neoplastic (**Ferreira et al. 2006).** They are responsible for major hazards in commercial poultry health and livestock production, mainly due to financial losses resulting from the decrease in animal weight gain **(Carão et al. 2014).** The high susceptibility of young birds to intoxication by aflatoxins compared to older birds has been demonstrated by **Arafa et al (2003)**. The response is directly related to the level of comfort of the birds, in other words, higher stress levels require fewer amount of toxins for changing the performance of animals **(Doerr et al. 1983).**The signs of intoxication by aflatoxins mainly depend on its concentration in the feed, the type of aflatoxin and the time of ingestion **(Ogido et al. 2004),** being characterized by immunosuppression and bone abnormalities, bleeding, depigmentation and changes in liver function **(Rosa et al. 2001, Miazzo et al. 2005, Tessari et al. 2010).** In research performed by **Tessari et al. (2005),** there was a reduction in total protein serum in broilers fed diets containing 200 µg of AFB1/kg for 20 days.

Fumonisin

Fumonisins (B1, B2 and B3) belong to a large group of mycotoxins produced by *Fusarium* fungi, natural contaminant of cereals, especially corn and its by-products. The occurrence of fumonisin B1 in food produced in Brazil have been described by several researchers, reaching nearly 90% of positivity, with levels up to 300 mg/kg feed. Fumonisins produced by *F. moniliforme* and *F. proliferatum*, are a family of mycotoxins can contaminate food, especially composed by corn **(Lino et al. 2004).**

In avian species, they are associated mainly with performance reduction and also severe immunosuppression, affecting cells and organs of the immune system **(Gertner et al. 2008),** besides affliction of liver function in broilers (Tessari et al. 2010). According to **Leung et al. (2003)**, the reduction in sphingolipid biosynthesis caused by fumonisins may alter the electrical regulation of epithelial cells. Fumonisins are also described as blocking of the mitotic cycle phases of epithelial cells, decreasing its proliferation **(Bouhet et al. 2004).**

Ochratoxins

Ochratoxin A (OTA) produced by fungi of the species *Penicillium* and *Aspergillus* naturally occurs throughout the world in various plant products, such as barley, coffee beans, cocoa beans, and nuts. It has been also detected in products made of cereals, wine, beer, grape juice and animal origin **(Nogueira & Oliveira 2006).**

In a study performed by **Gupta et al. (2008),** it was observed OTA produces nephrotoxic and hepatotoxic effects and may cause immunosuppression in broilers. It can also cause serious pathological changes in chicks, making the chickens fed foods contaminated with ochratoxin more susceptible to *Salmonella* infection. Even though this mycotoxin is extremely harmful to the birds, fortunately there is little contamination in poultry feed **(Santurio 2000).**

Garcia et al. (2003) concluded that broilers exposed to ochratoxin diet showed lower body weight, reduction in food intake, reduction in the levels of plasmatic proteins, albumins and globulins, with or

without the use of adsorbents. They also found an increase in blood uric acid level, necrosis of renal tubular cells and hepatocytes, bileduct hyperplasia and increase in the diameter of proventricular glands.

Trichothecenes

The main trichothecene mycotoxins are T-2 toxin, deoxynivalenol (DON) and diacetoxyscirpenol (DAS). All of them are produced by several species of *Fusarium* fungi, at temperatures below 15 °C **(Santurio 2000).**

T-2 toxin was shown to be, *in vitro*, toxic for chicken's macrophages, inhibiting their phagocytic capacity **(Kidd et al. 1995).** It may also form peroxides from lipids, resulting in a decrease of the concentration of vitamins in birds (**Hoehler & Marquardt 1996**). T-2 and DAS mycotoxins, at levels around 1 ppm in the feed, produced oral lesions in broilers, Low doses of Deoxynivalenol (DON) interfere with the differentiation of enterocytes **(Kasuga et al. 1998).**

Antonissen et al. (2014) found broilers experimentally infected with *Clostridium perfringens*, when fed diet containing 3,000 to 4,000 mg/kg DON, presented duodenum with lower electrical transepithelial resistance and lower villi height, suggesting disruption of the barrier and damage to the epithelium gut, which may lead to increased permeability and reduced absorption of proteins.

Burditt et al. (1983) observed its most significant toxic effects with the level of 4 ppm, where the birds showed low feed intake, delay in growth, changes in the blood parameters and neurotoxicity. Liver lesions were found in chickens that received T-2 in the diet **(Garcia et al. 2003). Dänicke et al. (2007)** noted lower feed intake in broilers when fed diets containing corn contaminated by DON.

Zearalenone

Zearalenone (ZEA) is a mycotoxin produced by various species of *Fusarium*, especially.

Fusarium graminearum and *Fusarium culmorum*, main agents involved in its formation **(Zinedine et al. 2007).** Except for extremely high levels of contamination, chickens are not affected by the ingestion of ZEA **(Lee et al. 1985).** Broilers were intoxicated with levels of 800 ppm of ZEA, and showed no changes in performance, however, a decrease in the number of white blood cells was observed, hypertrophy of some oviducts and comb shrinkage **(Chi et al. 1978).** The ZEA can also be hepatotoxic, and change some serum parameters (**Zinedine et al. 2007).**

Although the ZEA does not affect the performance of the chickens in natural contamination, it should be noted health authorities in some importing countries are on alert as the ZEA residues in chicken meat, as this mycotoxin, in certain concentrations, may induce an anabolic effect in humans and other mammals **(Santurio 2000)** and tumor development due to chronic exposure **(Yu et al. 2005).**

Prevention and control of mycotoxin formation

The best way to control aflatoxin and ochratoxin formation is to prevent the growth of fungi on harvested and stored grains and other susceptible commodities. Crops should be harvested at maturity and pre- or post-harvest mechanical damage should be avoided. Moisture contents of harvested crops should be reduced to a safe level.

Moisture build-up in the stored grain should be prevented by measures such as regular aeration. Aflatoxin production can be decreased by storing food in a low oxygen, high-CO2 environment. In areas of the southern United States, where the preferred conditions for aflatoxin production are common (25-30°C, humidity85%), refrigeration of food is often necessary to prevent aflatoxin production production**. (Ritchie, 1994)** In the United States, the Food and Drug Administration, has established a tolerance of 20 ppb of aflatoxin for foods other than milk, but European markets are striving for a lower Codex importation standard of 2 ppb. **(Abbas, 2005);** AFB1 contamination is practically unavoidable, chemoprevention strategies aimed to reduce AFB1 toxicity in poultry and in other animals have been the subject ofnumerous studies**. (Arafa et al., 1981; Leeson et al,1995).**

Several chemopreventives have been evaluated in poultry for reducing symptoms of aflatoxicosis. Because of their sensitivity, poultry have been used as models for discovering AFB1 chemopreventives.

Since 1990s, particular studies **(Maciorowski et al., 2007; Wyatt, 1991)** have shown the value of non-nutritive clays, such as aluminosilicates, zeolites, bentonites and clinoptilolites on aflatoxicosis prevention. They have high binding capacity against aflatoxin, reduce the absorption from the gastrointestinal tract and are generally inert, nontoxic and economical in use. Antifungal agents such as gentian violet and propionic acid have been evaluated and appear to be most promising substances in the control of aflatoxin-producing fungi. Similarly, benzoic acid has been found to be quite effective against *A. flavus*. Other feed additives including selenium and carotenes have also been reported to have the some value in reducing the toxicity of AFB1 in chickens and in turkeys. Also, high protein diet has been found to have protective effect against aflatoxins in chickens.

(Sumit et al., 2010).

The aflatoxin and ochratoxin content in food can be determined by analytical techniques such as: thin layer, gas or liquid chromatography, spectrofluorometry and spectrophotometry **(Talebi,**

2011). HPLC (high-performance liquid chromatography) still remains the technique of choice for aflatoxin and ochratoxinanalysis. HPLC methods include HPLC with fluorescence detection and HPLC with near-ultraviolet, laser- induced fluorescence detection (near- UV LIF) (Abbas, 2005). ELISA test for poultry are available for identification of totalaflatoxin and ochratoxin A. Detection of aflatoxin and ochratoxin residues in tissues requires 100 g of fresh or frozen liver or kidney. Samples for analysis should be placed in sealable plastic bags. Although not ideal, tissues from several dead birds can be pooled for analysis if necessary**. (Ritchie, 1994);** Chemical detoxification of aflatoxins (acid treatment, alkaline treatment with hydroxide, bisulfites, chlorinating compounds and oxidizing agents) in foods and feeds is important as a short-term postharvest solution to the problem. Although there are many chemical methods, ammoniation is still the most utilized and approved method for decontamination. New methods such as ozonation treatment do show promise, but require further testing for safety and scalability. With any chemical method, studies must be done to determine if new toxins are formed as a result of the treatment. It is also important to determine whether the treatment will alter the functional and nutritional characteristics of the products **(Abbas, 2005).**

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