

Review Article

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A review on Poultry Mycotoxins: Aflatoxins and Ochratoxins

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Abstract

In many developing countries, especially countries with tropical climatic conditions are favorable for a fungal growth during different stages of crop harvest such as pre-harvest, post-harvest and storage times. Most common mycotoxins producing fungi are *Aspergillus species*, *Penicillium species*, *Fusarium species* and *Claviceps species*. The mycotoxigenic fungal growth may eventually result in mycotoxins production as secondary metabolites which are more chemically stable than fungi themselves and may cause mycotoxicoses in different animals including poultry and human beings. Mycotoxicosis is the term used to define a disease condition in man and animals due to the exposure to mycotoxins. Among those mycotoxins; aflatoxins and ochratoxins are most common and pathogenic in poultry. Poultry acquire mycotoxins from toxin contaminated cereal grains, litter and soil through ingestion or skin contact. Young poultry are more sensitive to aflatoxin and ochratoxin than adults. Diffuse syndromes characterize mycotoxicosis, but lesions predominate in certain organs, as liver, kidneys, epithelial tissue and central nervous system, depending on the type of toxin. Economic significance of poultry mycotoxicoses is greatly considerable due to mortality, decreased egg production, reduced egg size, loss of appetite and decreased feed conversion efficiency. Condemnations can occur during processing, due to bruising and haemorrhages, low hatchability and growth inhibition in commercial poultry. Diagnosis of poultry mycotoxicoses depends on the observation of manifested clinical signs, qualitative diagnosis and ELISA. Though treatment of poultry mycotoxicoses is not much effective, removal of contaminated feedstuffs and treating other disease complications would give good result. Regarding public health concern mycotoxins are able to transmit through meat and eggs to humans causing toxic effects. Prevention and control of poultry mycotoxicoses depend on the selection of uncontaminated poultry feeds, proper harvesting of grains, using appropriate drying techniques, maintaining proper storage facilities and whenever necessary using chemicals to inhibit fungal growth and toxin production.

Keywords

Aflatoxin,
Mycotoxin,
Ochratoxin,
Poultry.

1. Introduction

Mycotoxins are a diverse group of secondary metabolites produced by certain moulds when they grow on agricultural products. They do not belong to a single class of chemical compounds, and they differ in their toxicological effects. It was the outbreak of turkey X disease in United Kingdom (UK) during 1960, and the subsequent discovery of the aflatoxins and their toxic effects by scientists at Tropical Development and Research Institute (TDRI) and other institutions, which promoted the study of mycotoxins as a specialized field of scientific activity [1]. Mold occurrence and growth on poultry feeds is one of the major threats to poultry economy and health. Besides their negative impacts on nutritional and organoleptic properties, molds can also synthesize different mycotoxins [2].

More than 100,000 fungal species are considered as natural contaminants of agricultural and food products. However, due to genetically and ecological factors, relatively few can actually generate mycotoxins [2]. The most important mycotoxins in agriculture are the aflatoxins, ochratoxins, 12, 13-deoxytrichothecenes and the fumonisins; which are produced by species of fungi belonging to the genera *Aspergillus*, *Penicillium* and *Fusarium* [3]. Other toxigenic fungi include *Stachybotris atra* and the species of *Alternaria*, *Paecilomyces*, *Trichoderma* and *Trichothecium*, all of these fungi occur commonly in soil, agricultural products, grain dust and house dust [3].

Mycotoxin contamination of feedstuffs can impose health hazards to human as mycotoxins appear in animal's tissue and body fluids. Monogastric farm animals such as poultry and swine are more susceptible to AF due to the following two reasons: first, cereals are a large part of their diet and second they lack the ruminal reservoir of a multitude of microorganisms. Ruminants seem to be less susceptible to mycotoxins rather than other animals as their rumen flora has the ability to convert some mycotoxins into less carcinogen metabolites or

biologically inactive compounds (for example, AFB1 is converted to its less carcinogen metabolite AFM1 in ruminants body [4].

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 type, quantity and duration of ingestion of mycotoxins, species, sex and age of the animal, general health, immune and nutritional status, as well as environmental factors. Aflatoxicoses and ochratoxicoses are the most common mycotoxicoses in commercial poultry [5].

Aflatoxin producing fungi and aflatoxin-contaminated animal feed stuffs are recognized worldwide, usually with adverse implications for poultry production [6]. The direct and indirect effects of aflatoxicoses include increased mortality from heat stress (broiler breeders); loss of egg production (leg-horns); anemia, hemorrhages, liver condemnation, paralysis, lameness, and impaired performance of broilers [7].

The Food and Agricultural Organization estimate that as much as 25% of the world's agricultural commodities are contaminated with mycotoxins, leading to significant economic losses [8]. The world market poultry meat and egg market has been influenced by the production and managerial style from the developing countries. Avian mycotoxicosis is a great constraint in poultry industry, because the disease is characterized by immunosuppression, hepatotoxicity, nephrotoxicity, loss of egg production, mutagenicity and teratogenicity [9].

Mycotoxins are currently considered as a serious threat to the poultry farming in terms of diseases leading to synergistic interactions with other infections. Exposure to micro fungi-laden dust in poultry housings can result in a variety of disease

conditions. Mycopathology is a collective term used for diseases caused by fungi, either living or dead or their metabolic products (toxins, allergens or enzymes). This complex term comprises disease manifestations e.g. mycosis, mycotic colonization, myco-allergosis, mycetism and mycotoxicoses [10].

Therefore, the objectives of this paper are:

To review major poultry mycotoxins and their effect on poultry production particularly on aflatoxins and ochratoxins.

To review about potential transmission of poultry mycotoxicoses into the poultry products and associated public health problems.

2. Major poultry mycotoxicoses

Mycotoxicosis is the term used to define a disease condition in man and animals due to the exposure to mycotoxins. The severity is determined by the type of mycotoxin involved, the animal species and the effect are dose-dependent and there is an individual variation even within the same species. Mycotoxicosis is characterized by being related to food ingestion; it is neither contagious nor infectious and is always caused by the toxins produced by fungi [11]. Diffuse syndromes characterize mycotoxicosis, but lesions

predominate in certain organs, as liver, kidneys, epithelial tissue and central nervous system, depending on the type of toxin. However, two or more mycotoxins can be present at the same time, which can potentiate their toxic effects on a susceptible individual. Several mycotoxins have been identified in foods consumed by humans and animals. Among many mycotoxins; aflatoxicoses and ochratoxicoses are the most common mycotoxicoses in commercial poultry [5].

Mycotoxicosis has been a major but unrecognized food safety issue for several centuries. They are naturally occurring contaminants that cause health related problems when it gets into the body through natural route of ingestion, inhalation or may be absorbed through the skin [10]. Mycotoxin production takes place in the mycelium after active fungal growth, but may accumulate in specialized structures such as sclerotia, canidia, or in surrounding area [12]. Based on the moisture requirements, mycotoxin producers can be divided into three groups: field fungi, storage fungi, and advanced decay fungi. The field fungi mycotoxin producers require a grain moisture content of 22% to 25%, while storage mycotoxin producers usually grow in grain with a moisture content of 13% to 18% (equal to 70% to 90% relative humidity), and advanced decay fungi require over 18% moisture [13].

Table 1: Classification of mycotoxins according to the main organ they affect

No.	Affected organ systems	Mycotoxins
1.	Hepatotoxins	Aflatoxin Sporidesmin Luteoskyrin Cyclochlorotine Rubratoxin Sterigmatocystin
2.	Nephrotoxins	Ochratoxin Citrinin
3.	Neurotoxins	Penitrema Patulin Citroviridin

4.	Cytotoxins (Alimentary tract toxins)	Trichothecenes T-2 toxin Diacetoxyscripenol Neosolaniol Diacetylivalenol Deoxynivalenol (DON, vomitoxin) HT-2 toxin Fusarenon
5.	Estrogenic mycotoxins	F-2 toxin (zearalenone)
6.	Others	Ergot Fescue Lupinosis

Source: <http://www.fao.org/docrep/x5036e/x5036eob.htm>

2.1. Avian Aflatoxicosis

2.1.1. Etiology

Avian aflatoxicosis is a disease of poultry caused by aflatoxin. Aflatoxins (AF) are widely distributed toxins produced by *Aspergillus*. Of the over 180 species of *Aspergillus*, only a few are aflatoxigenic. After the discovery of AF in the 1960s, *Aspergillus flavus* and *Aspergillus parasiticus* of the section *Flavi* were the only known aflatoxin producers producing the B and B/G types of AF, respectively [14]. Other aflatoxigenic species that subsequently emerged are *Aspergillus nomius* (B and G types), *Aspergillus bombycis* (B and G aflatoxin), *Aspergillus ochraceoroseus*, and *Aspergillus pseudotamarii* (B type), but they occur less frequently [15].

The unexpected new comer is *Aspergillus niger*, an ochratoxin (OT) producer which was discovered over four decades ago but was never associated with AF synthesis. However, in a search for aflatoxigenic fungi in Romanian medicinal herbs, showed the capacity of some

strains of *Aspergillus niger* to produce AFB₁ [16]. AFB₁ is a member of aflatoxin group is one of the most carcinogenic natural product formed in nature. AF has been detected in most countries of the world. Four toxins soon identified: Aflatoxin B₁, B₂, G₁, G₂-blue or green florescence under UV light (ultra violet) [17].

2.1.2. Host sensibility

Young poultry are more sensitive to aflatoxin than adults. There are also large species differences: ducks being 10 times more sensitive than chickens. Furthermore, certain poultry breeds are more sensitive than others. Chickens and quails are considered relatively resistant, so acute intoxication occurs relatively rare. Chronic intoxication with aflatoxin is the results of aflatoxin ingestion for several weeks (one week minimum). Aflatoxin in concentration of 0.7ppm reduces the growth rate of turkey but without any effect in quails and chickens [18]. A diet containing 400ppm of AFB₁ severely affects body and liver weights in turkeys, with no effect in chickens and showed that the lethal dose is different between poultry species [19].

Table-2: Oral lethal dose of aflatoxins in different poultry species.

No.	Species	Oral lethal dose (ppm)
1.	Chick embryo	0.025
2.	Duckling	0.3
3.	Turkey poultry	0.5
4.	Chicken, New Hampshire	2
5.	Chicken, Rhode Island	6.3

Source (Dalvi, 1986) [20].

2.1.3. Pathogenesis

After absorption, the highest concentration of the toxin is found in the liver (target organ). At liver level, aflatoxin B₁ is metabolized by microsomal enzymes into different metabolites. In one day-old chicks, AFB₁ reduces the activity of liver uridine 5'-diphospho (UDP) glucose glycogen transglucosylase resulting in depletion of hepatic glycogen stores [21]. Also, lipid accumulation occurs in the liver of chickens and ducklings exposed to aflatoxin. Aflatoxin is rapidly excreted in the bile (eliminated in faeces) and urine, and does not accumulate or persist in body tissues. This perhaps explains the rapid recovery of egg production and hatchability after cessation of toxin ingestion [22]. Aflatoxin reduces resistance of poultry to infection with *Pasteurella multocida*, *salmonella species*, marek's disease virus, infectious bursal virus, *coccidia*, and *Candida albicans* [23].

2.1.4. Factors enhancing production of aflatoxin in poultry feed

There are several factors enhancing the prevalence of aflatoxigenic fungi and aflatoxin production in developing countries. The factors include the following: the food materials must be infected by aflatoxigenic fungi which deposit the toxins on feed ingredients and concentrated feeds. Among cereals, the size and integrity of the seed coat also affects the susceptibility of fungal infections of chickens and mycotoxin formation [24]. The substrate which may be feed ingredients and concentrated feeds possesses a source of energy in the form of carbohydrates, organic and inorganic source of nitrogen, trace

elements and moisture for growth of mould and toxin production [8].

The environmental condition favoring mould growth and aflatoxin production are hot and humid conditions, the optimal temperature varies between 24⁰C and 28⁰C and seed moisture content of at least 17.5% [25]. Soil types also affect the level of AF contamination of crops, for example, light sandy soil support rapid growth of fungi. Presence of other microorganism either bacteria for example presence of *Streptococcus lactis* and *Lactobacillus casei* causes reduction in AF production by *Aspergillus parasiticus* [26]. Agricultural practices also affect AF contamination of feed ingredient. Off season harvesting and harvesting system that enhances seed breakage would also increase the degree of AF production [27].

2.1.5. Distribution of Aflatoxin

AF has been found as contaminants in animal feed ingredient worldwide. The occurrence in developing countries is more because there is no strict food and feed quality control programmes to reduce the burden of AF. Also their environmental condition presented as hot and humid climate makes most developing country vulnerable to AF in poultry feed [27].

AFB₁ was detected mostly from animal and feed ingredients from developing countries. Among the feeds ingredients; sorghum, wheat, and maize were the most investigated, however, the data on groundnut cake, cotton seed meal and fish meal showed high level of AF contamination [27]. Higher level of contamination of AF were found

in the animal feed than the feed ingredient possibly because of the storage condition which allowed growth and proliferation of mycotoxigenic fungi [28].

2.1.7. Metabolism and Residues of Aflatoxin

In broilers, metabolites of aflatoxins B₁ and B₂ concentrated in kidney and liver but cleared within 4 days. Then metabolism of aflatoxin B₁ into conjugated aflatoxins B_{2a} and M₁ occurred in the liver, which will be metabolized to aflatoxicol [29]. The half-life of aflatoxin B₁ in laying hens is about 67 hours, though feed: egg transmission is about 5000:1. Most aflatoxin excreted through the bile and intestine, but aflatoxin B₁ and aflatoxicol were detected in ova and eggs for 7 days or longer [30].

Aflatoxin B₁ accumulated in reproductive organs and its subsequent transmission to eggs and hatched progeny (yolk sac and liver) in poultry. It is well established that AFB₁ is both carcinogenic and cytotoxic [31]. The activated AFB₁ metabolite (i.e. AFB₁-8, 9-epoxide) forms a covalent bond with the N7 of guanine and forms AFB₁-N₇-guanine adducts in the target cells. The results are G T transversions, DNA repair, lesions, mutations and subsequently tumor formation [32]. The reactive epoxide can also be hydrolyzed to AFB₁-8, 9-dihydrodiol which ionizes to form a Schiff's base with primary amine groups in the proteins [30]. The short-lived epoxies AFB₁ has also been associated with coagulopathy due to reduced synthesis of vitamin K and other clotting factors as a result of sub-lethal intoxication of animals [33].

The pharmacological interactions of aflatoxicosis has effect on plasma half-life, thus it affects drug effect in the body. Observed that chlortetracycline plasma concentrations were lowered due to decreased drug binding to plasma protein [34]. Though opinion differed considerably on sparing or aggravating effect caused by the addition of chlortetracycline to feed contaminated with aflatoxin [35].

2.1.8. Clinical signs

Aflatoxicosis does not induce mortality directly, although high levels (>10ppm) may be lethal. Acute aflatoxin poisoning leads to impaired pinhead sized lesions and paleness in liver. Histopathological, liver damage is manifested as vacuolation of hepatic cells and bile duct proliferation [36]. In aflatoxicosis, a thickened basement membrane in the glomeruli and associated hyaline droplets in the renal tubules are noted. It000 is not known if the glomerulus is damaged by toxins or by a leakage of unusual protein from a severely damaged liver [37].

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 [36]. Liver lesions in chicken are characterized by coordination of movement, vertigo, and paresis, followed by diarrhea with admixtures of blood, haemorrhages, tumescence, jaundice (icterus), coma and death [37].

The levels of serum proteins are reduced by aflatoxins. The synthesis of albumin and of the most of the globulins takes place in liver and in chronic hepatic diseases, hypoalbuminaemia occurs. The level of globulins decreases, but not as much as the albumin because not all the globulins are formed in the liver, such as gamma-globulins [38]. Intoxicated adult hens, with concentration above 2ppm have decreased egg production and the hatchability of their eggs is reduced. In adult male breeder, testicular weights and sperm counts are reduced. Dietary exposure of broiler hens to AF (10ppm) resulted in embryonic mortality and reduced the immunity in the progeny chicks. Embryonic exposure with AFs resulted in long-term depression of the immune function in chickens [37].

2.1.9. Lesions

Lesions depend on the age of the host and the dose of toxin ingested and can include: hepatosteatorrhea or fatty liver, kidney hypertrophy,

splenomegaly, atrophy of the thymus, testes and bursa of fabricius. Bruising associated with an increase of capillary fragility and haemorrhagic points (petechial haemorrhages) on the surface of muscles of the leg and breast have been described in aflatoxicosis of broiler chickens [39]. Acute toxicity of aflatoxins in chickens may be characterized by liver necrosis with icterus, distended gallbladder and bile ducts, white pin head sized lesions and paleness in liver. Histopathological liver damage is manifested as vacuolation of hepatic cells and bile duct proliferation [36].

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Acute and chronic toxic effects of aflatoxins, particularly AFB1, in turkeys are not very different from those in chickens: diffuse necrosis of liver parenchyma, proliferation of the bile duct epithelial cells and small haemorrhages. The prominent cellular changes in liver included swelling and vacuolation of the parenchyma cells and enlargement of the nucleus. Prolonged exposure of the duck to low levels of aflatoxins leads to marked nodular hyperplasia of the liver, bile duct proliferation and fibrosis and hepatocellular carcinoma. Chickens and turkeys have not been shown to develop liver neoplasm after ingestion of aflatoxins. Since metabolites of aflatoxins are implicated in carcinogenesis and ducks are known to metabolize aflatoxins rapidly, they appear to be prone to aflatoxin-induced carcinoma [37].

2.2. Avian ochratoxicosis

2.2.1. Etiology

The name “ochratoxin” derives from *Aspergillus ochraceus*, the first fungus discovered to produce this toxin. Ochratoxins are mostly produced by *Penicillium verrucosum*, but five other species of *Aspergillus* and six other species of *Penicillium* produce it as well. Ochratoxin-A (OTA) is the most commonly detected and most toxic member of the family. OTA is a common contaminant of cereals (corn, wheat, barley, oats, rye, sorghum) and peanuts, as well as soya, coffee and cocoa beans. Environmental conditions for ochratoxin production are similar to those for aflatoxin and simultaneous contamination with both is common [5].

Ochratoxins are isocoumarin compounds linked to L-b-phenylalanine and are designated A, B, C, and D, because of their methyl and ethyl esters. Ochratoxin A (OTA) is the most common and most toxic, and is relatively stable [41]. OTA production is dependent on different factors such as temperature, water activity and medium composition, which affect the physiology of fungal producers. In cool and temperate regions, OTA is mainly produced by *Penicillium verrucosum* or *Penicillium nordicum*. *Penicillium verrucosum* mainly contaminates plants such as cereal crops, whereas *Penicillium nordicum* has been mainly detected in meat products and cheese [42].

In tropical and semitropical regions, OTA is mainly produced by *Aspergillus ochraceus* (*A. ochraceus*). *Aspergillus ochraceus* have been reported in a large variety of matters like nuts, dried peanuts, beans, spices, green, coffee, beans and dried fruits; but also in processed meat and smoked and salted fish [43].

2.2.2. Occurrence of ochratoxicoses

Ochratoxicosis occurs less frequently in poultry than aflatoxicosis but is more lethal because of its acute toxicity. Ochratoxins are a family of toxic compounds consisting of three members, A, B

and C, which are structurally related and are produced as secondary metabolites of several species of fungus. Ochratoxin A contaminates agricultural products and due to its accumulation in food, represents a serious threat to human and animal health worldwide [5]. Ochratoxins are among the most toxic mycotoxins to poultry. They are nephrotoxins found in grains and feeds worldwide [44].

2.2.3. Host sensibility

Young poultry are more sensitive to ochratoxin ingestion than adults and ducks are seven times more sensitive than chickens. Quail and turkeys are also more sensitive to ochratoxicosis than chickens. Variations in sensitivity towards OTA exists among avian species, as LD₅₀ ranges from 0.5 to 16.5ppm body weight for ducks and Japanese quail, respectively, chickens 2-4ppm [5].

2.2.4. Pathogenesis

After resorption, the highest quantity of ochratoxin can be found inside kidneys and liver, and a considerably smaller extent in muscle. It is characteristic of poultry to have a more efficient and faster excretion of ochratoxins than other animals, approximately 48 hours. One of the profound effects of OTA is the its ability to alter the function of the immune system in avian species, causing severe leucocytopenia, impaired complement activity, reduction in immunoglobulin and several functional properties of macrophages and heterophils and finally it causes atrophy of the lymphoid organs along with depletion of lymphocytes [45].

OTA causes enlargement of the kidney and subsequently impairing its function, therefore, considered as a nephrotoxic mycotoxin in birds. Ochratoxin A inhibits protein synthesis, produces acute proximal tubular epithelial necrosis in the kidneys and inhibits normal renal uric acid secretion. A decrease in the concentration of proteins, triglycerides, cholesterol, calcium, phosphorus and potassium is followed by an increase in the level of uric acid and creatinine and a decrease in glomerular filtration [46].

Some authors explain how OTA inhibits respiration in mitochondria, where it acts as a competitive inhibitor of the carrier's proteins, localized on the inner membrane of mitochondria. Furthermore, it is considered that OTA represents a teratogenic agent for chickens, but not for other domestic animals [47].

2.2.5. Clinical signs

The unspecific clinical image of chronic ochratoxicosis in poultry is followed by a decrease in egg production of laying hens, whereas, as far as broilers are concerned, their growth is hindered and conversion of food is higher levels of dietary OTA (4 and 8ppm), resulting in yellow enlarged liver. Signs of liver diseases were further supported by the significant decrease in total protein, albumin and globulin. There is also some mild decrease in bursal and thymic size consistent with immunosuppression [40].

The egg shell often becomes thin and fragile, with different discoloration appearing on the surface. Growth inhibition is linked with malabsorption syndrome, as confirmed by the presence of hypocarotenoidemia [40]. The minimum amount of ochratoxin also causes reduced bone firmness and poor pigmentation. Acutely intoxicated birds are depressed, dehydrated and often polyuric and die in acute renal failure. Survivors will be; poorly feathered, have delayed sexual maturity, increased clotting times, anemia and immunosuppression [37].

2.2.6. Lesions

The enlargement of the liver and kidney in OTA intoxications is caused by the involvement of these organs in detoxification and elimination. Affected kidneys are white to tan, swollen, hard and may have white pinpoint urate crystals. If damage is extensive enough to cause renal failure, dehydration, hyperuricaemia and visceral urate deposition appears at kidney level. Pasty white urates are deposited on pericardial, perihepatic, peritoneal and articular surfaces. These deposits may be mistaken with inflammatory exudates but

their true nature can be determined by microscopic examination. More commonly, birds survive in compensated renal failure and kidneys appear enlarged, fibrotic and pale [39].

Other lesions include a thickened basement membrane in the glomeruli and lymphoid depletion from the lymphoid organs. In addition to the renal lesions there is mild to moderate glycogen deposition in hepatocytes, mainly at the periphery of the liver lobes at higher levels of dietary OTA (4 and 8ppm), resulting in yellow enlarged livers (Herenda and Franco, 1996). Tubular dilatation and hypertrophy, swelling of tubular epithelial cells, localized necrosis, and desquamation of the tubular basement membrane as signs of tubulonephrosis were reported [48].

Glomerulonephrosis, tubulonephrosis, focal tubular epithelial cell proliferation and the multiplex adenoma-like proliferation of renal parenchyma are considered to be primarily related to the toxin, while focal intertubular infiltration of lymphocytes and histiocytes can also occur either primarily or secondly as reparation of tubulonephrosis or as a consequence of immune stimulation [46].

The reduction seen in the number of lymphocytes in the pulpar region of the spleen has also been reported and detected a marked degree of lymphocytic depletion and obscure distinction between red and white pulp in some areas of the spleen [49]. The lack of visible damage in heart and muscles indicates a low sensitivity of these tissues to OTA toxicity [50].

2.3. Economic significance of mycotoxicoses on the poultry production

The most obvious effect of mycotoxins on poultry production is mortality, as this can be readily diagnosed and quantified. The outbreak of Turkey X disease resulted in the death of 100,000 turkey poultry and many ducks, chickens and pheasants. The total annihilation of 15,000 turkeys in North Carolina by aflatoxin, and the death of 59% of a 16,000-flock of turkeys from ochratoxicosis have also been reported [51].

Egg production can also be affected by mycotoxins in the diet. Aflatoxin apparently inhibits egg production at the point of commitment of an ovum to maturation; ova already committed are not influenced apart from less yolk than normal being deposited [51]. Decreased egg production, with thin, rubbery shells which break more readily than normal have been observed during field outbreaks of ochratoxin. Aflatoxin has also been implicated in hatchability, impaired semen quality in White Leghorn strains, under pigmentation, altering the response of chickens to environmental extremes and increased sensitivity to sodium chloride. In addition to affecting pigmentation, ochratoxin decreases the tensile strength of the large intestine by decreasing its collagen content and thus causes the fragile large intestine to rupture during processing [52].

Besides reducing egg production, aflatoxicosis is also responsible for the reduction in egg size and the proportional decrease in yolk size, as a result of the interference with protein and lipid synthesis [53]. According to Hamilton & Jones (1971), [52] the yolk size reduction is due to damages to the liver where layers fed diets with 10ppm aflatoxin during three weeks had 48% fat in the liver while layers fed an aflatoxin-free diet had 28%, an evidence of fat accumulation in the liver. As a consequence, there is a decrease in fat deposition in other structures, as in yolk.

Aflatoxins can enter the egg at any stage of its development, and it takes 7 to 8 days for an oocyte to mature and another 24 hours to become an egg. As the embryo absorbs the yolk during the third week of incubation, high levels of embryonic deaths are expected during this stage. The levels of aflatoxin in the eggs reach a plateau after 4-5 days and the same time to disappear if the intoxication is interrupted. According to the reports of Qureshi *et al.* (1998) [54], high rates of late embryonic mortality in birds that ingested 5 and 10 ppm AF, and concluded that the effects were more marked with a longer exposure time to the toxin. This effect is due to the transfer of AF metabolites or the toxin itself to the egg, causing immune alterations in the embryo.

However, it is the chronic effects of mycotoxins which could pose the most problems to poultry producers. Feed refusal which is a rapid, direct response to the presence of mycotoxin in the feed can be a problem with some animals. In poultry, aflatoxin has been found to be associated with the "bruising" syndrome of broiler chickens. The development of bruises and hemorrhages on the processing lines can result in a downgrading and condemnation of birds and customer dissatisfaction due to infernal bruising not visible to the inspectors [51].

Growth inhibition is another important toxic effect of mycotoxins in poultry feed. A diet high in protein largely prevents the inhibitory growth caused by aflatoxin, but at a cost. The concentration of bile required for lipid absorption and digestion from the intestinal tract is decreased during aflatoxicosis. The practical consequence of the malabsorption is to decrease the efficiency of feed conversion into animal products and hence increase the cost of production [51].

2.4. Public health significance

Most grains used as feed stuffs for poultry and livestock are also ingredients for human food and carry the same risk for fungal growth contamination. Coffee, wheat, cereal grains, flours, spices and rices are typical foods from which ochratoxins and other fusarial toxins can be detected [55]. Poultry products present low risk for human toxin exposure because mycotoxins have minimal distribution to skeletal muscle, the major form poultry consumption. Mycotoxin distribution to liver, kidney and eggs is of greater concern. Mycotoxins contaminated poultry feed can lead to the transfer of toxins through meat and egg to human beings [12]. A number of epidemiological studies have linked aflatoxin with human liver cancer and there is also potent synergy between the hepatitis B virus infection and aflatoxin in the induction of liver cancer [56].

In United States, the Food and Drug Administration (FDA) has an action level of 20 ppb for aflatoxin in foods (which would include poultry and eggs) and 0.5 ppb for aflatoxinM₁ in

milk [57]. Because of the global nature of food production and international movement of grains and food commodities, international standards and surveillance are important. Broadening the safety net to prevent mycotoxin exposure in the food chain requires international cooperation for safety, risk assessment, action levels and advisories [58].

2.5. Diagnosis for poultry mycotoxicoses

Diagnosis is made through observing the appropriate field signs, finding gross as well as microscopic tissue lesions, and detecting the suspected toxin in grains, forages, or the ingesta of affected animals [59]. The samples of choice include both refrigerated and frozen carcasses for necropsy examination and a representative sample of the suspected contaminated grain source. Because the toxin is produced under cold conditions, the grain sample should be frozen rather than refrigerated for shipment to the diagnostic laboratory [60].

The aflatoxin and ochratoxin content in food can be determined by analytical techniques such as: thin layer, gas or liquid chromatography, spectrofluorometry and spectrophotometry [61]. HPLC (high-performance liquid chromatography) still remains the technique of choice for aflatoxin and ochratoxin analysis. HPLC methods include HPLC with fluorescence detection and HPLC with near-ultraviolet, laser- induced fluorescence detection (near- UV LIF) [50]. ELISA test for poultry are available for identification of total aflatoxin and ochratoxin A. Detection of aflatoxin and ochratoxin residues in tissues requires 100 g of fresh or frozen liver or kidney [62].

2.6. Treatment against poultry mycotoxicoses

Toxic feeds should be removed and replaced with unadulterated feeds and poultry generally recover from mycotoxicoses soon after an uncontaminated diet are available [63]. Treatment of ongoing parasitic or bacterial diseases will alleviate additive or synergistic interactions. Substandard management practices are especially detrimental

to poultry stressed by mycotoxins and should be corrected [64].

Vitamins, trace minerals (especially selenium), protein and lipid requirements are increased by some mycotoxins and can be compensated by feed formulation and water based treatment. Vitamin A and C partially counteracted the toxicity of ochratoxin A in chicks. Increasing crude protein, dietary energy and vitamin supplementation can counteract the effects of aflatoxin [52]. Another strategy for treatment of mycotoxins in the presence of contaminated feeds involves specific nutrients that metabolize to detoxicants. Supplemental methionine and N-acetyl cysteine counteracted aflatoxin likely through the enlargement of glutathione formation [64].

2.7. Prevention and control of poultry mycotoxicoses

Prevention and control of mould in feed is key to achieving control of avian mycotoxicosis. Following good agricultural practices during both pre-harvest and post-harvest conditions would, minimize the problem of contamination by mycotoxins such as aflatoxins, ochratoxin and trichothecene mycotoxins. These include appropriate drying techniques, maintaining proper storage facilities and taking care not to expose grains [50]. 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. 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 low oxygen, high carbon dioxide environment [62].

AFB1 contamination is practically unavoidable, chemoprevention strategies aimed to reduce AFB1 toxicity in poultry [19, 65]. Since 1990s, particular studies 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 [66,67]. 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 *Aspergillus flavus* [36].

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 [36]. 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 [50].

Decontamination of mycotoxin from cereal crops used in the production of poultry feed can be classified as physical decontamination, biological decontamination and chemical decontamination [68]. The physical method of mycotoxin decontamination include density segregation and floatation, cleaning and washing, sieving, dehulling, hand picking, irradiation, milling, thermal degeneration [69]. Feed additive like mycotoxin inactivates trichothecenes by enzymatic decontamination of the 12-13 epoxy ring, and zealenone by the enzymatic opening of the lactone ring [70]. Moist ozone and dry ozone were able to reduce mycotoxin concentration in contaminated corn up to 90% and 70% respectively [71].

3. Conclusion

We should always keep in mind that mycotoxicoses can occur even in poultry consuming balanced feeds. and result in significant economic losses. In fact, tropical climatic conditions support fungal growth being more susceptible for subsequent mycotoxins productions which are chemically stable causing

mycotoxicoses. Monogastric animals such as poultry, swine, human beings, etc., are more susceptible to mycotoxicoses because much of their feedstuffs comprise cereal grains; which are greatly damaged by fungi, and also they lack intestinal microflora. Mycotoxins may be encountered in poultry products that can pose hazards to public health even in very small doses having synergistic effect with hepatitis-B virus and causing liver cancer. Generally, the source for mycotoxins in poultry farm is not only mould growth on grains, but also litter and soil can also be potential sources. Treatment of poultry mycotoxicoses is not much effective and more important approach is prevention and control. As a result, effective poultry production starts with a strict selection of raw material, a correct feed storage and monitoring and, when necessary, ends with the correct choice of additives that can control the mycotoxins that contaminate the feed, in an efficient and safe way.

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