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Aflatoxicosis in Layer and Breeder Hens

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1. Introduction

During the past few decades there has been a steady increase in global production of poultry meat and eggs. Although the high nutritive value of eggs and poultry meat has resulted in increasing demand, food quality and safety factors are becoming increasingly significant in determining market value of poultry products. Poultry production is one of the fastest growing sectors of Iranian agriculture. Egg production is increasing at the rate of 4-6 per cent per annum, while broiler production at the rate of 10-12 per cent.

At present, Iran is the largest producer of poultry meat in neighboring countries and ninth largest producer in the world, thanks to a 753% growth in meat production from 195 thousand ton in 1978 to 1468 1thousand ton in 2008-09. Broilers are the major source of meat supply in the country. About 270 million broilers are produced every year. Consequent to increased production, per capita consumption /availability has also increased from 7 eggs in 1961 to 42 eggs in 2008. The per capita consumption of poultry meat has increased from 5.4kg in 1978 to 21.8kg in 2008-09. This enormous growth and spurt in poultry production has put a tremendous pressure on proper feeding of poultry in order to sustain the poultry industry in Iran (Manafi, 2010).

As mycotoxins are one of the major factors suppressing poultry productivity and also product quality, control of their impact is critical (Oguz, 2011).

According to the United Nation's Food and Agriculture Organization (FAO), approximately 25% of world's grain supply is contaminated with mycotoxins. The greatest economic impact of mycotoxin contamination is felt by crop and poultry producers, as well as food and feed producers.

Contamination of poultry feeds with mycotoxins is one of the major problems associated with the feeding of poultry. Mycotoxins are the toxic metabolites synthesized by a certain naturally growing fungi on animal feed, feed ingredients and other agricultural crops. More than 350 mycotoxins have been identified so far in feedstuffs. Aflatoxin is the most commonly occurring mycotoxin in Iran. Aflatoxins are a group of secondary metabolites produced by a certain species of fungus of the genus *Aspergillus* (especially *A. flavus* and *A. parasiticus*). These fungi are capable of growing and contaminating the grains and cereals at any time before and after the harvest, during storage, transportation and processing of feed ingredients and the formulated feeds after processing. The spores of the fungi remain dormant but when the level of moisture is more than 12 per cent with a temperature of 25-35°C, with humidity of 80 per cent and adequate aeration initiate their growth. Mycotoxins have adverse effect on both health and productivity in almost all species of domestic animals including poultry. In general,

mycotoxicosis results in reduced feed intake, diminished feed conversion, decrease in production and subsequently increased susceptibility to various infections depending upon the type of toxins ingested (Xue et al., 2010).

Mycotoxins	Fungi
Aspergillus toxins	
• **Aflatoxins B_1, B_2, G_1 and G_2**	*Aspergillus flavus* and *Aspergillus parasiticus*
• **Cyclopiazonic acid**	*Aspergillus flavus*
• **Ochratoxins**	*Aspergillus ochraceus*
• **Sterigmatocystin**	*Aspergillus versicolor*
Pencillium Toxins:	
• **Ochratoxins**	*Penicillium viridicatum*
• **Citrinin**	*Pencillium citrinum*
Fusarium Toxins	
• **T-2 Toxin, HT-2 Toxin, Diacetoxyscirpenol (DAS), Monoacetoxyscirpenol (MAS)**	*Fusarium tricinctum,* *Fusarium solani*
• **Deoxynivalenol (DON, vomitoxin)**	*Fusarium graminearum*
• **Zearalenone**	*Fusarium graminearum, Fusarium roseum*
• **Fumonisins B_1, B_2**	*Fusarium moniliforme* *Fusarium proliferatum*
• **Moniliformin**	*Fusarium moniliforme*
Ergot toxins	
• **Ergopeptines**	*Claviceps purpurea*
• **Ergovaline**	*Acremonium coenophialum*

Table 1. Significant mycotoxins and fungi (molds) in foods and feeds

Aflatoxin contamination of feedstuffs has been reported to be of a wide range from 1 to 900µg/kg in commonly used ingredients as well as mixed feed samples in developing countries (Mohanamba et al., 2007). Poultry industry suffers greater economic losses due to the greater susceptibility of the species in comparison with other animals to the toxin apart from continuing intermittent occurrences in feeds (Fraga et al., 2007; Thapa, 2008).

2. Incidence of aflatoxin

It is imperative that food contaminated with aflatoxin is considered unsafe for human and animal health. Aflatoxins occur over a wide variety of substrates of practical importance to poultry feeding (maize, groundnut/meal, cottonseed meal, sunflower extractions, rice, soya bean meal and compounded feeds.) Because of increasing awareness of the risk of aflatoxin contamination of foods and feeds, this has opened a new vista to conduct survey of feed stuffs which are commonly contaminated with aflatoxin. The details of the survey are presented in Table 2.

Author and year	Ingredient	Level (ppb)
Shetty et al. (1987)	Mixed poultry feed	30-1610
Jelinek et al. (1989)	Corn and corn products Peanuts	0.1-1970 0.2-5000
Devegowda et al. (1990)	Groundnut Maize Bajra	48-900 32-1000 12-15
Hegazy et al. (1991)	Poultry feed	1-2000
Devegowda & Arvind (1993)	Maize Ground nut cake Others	25-1002 45-1500 10-80
Jindal et al. (1993)	Poultry feeds	> 300 30-160
Dhavan & Choudary (1995)	Feed ingredients and mixed feeds	High concentrations
Sala & Ueno (1997)	Maize	20-100
Chandrasekharan (2000)	Maize	21to 500
Pandey et al. (2001)	Maize Wheat Groundnut extraction	948 285 225
Chandrasekharan et al. (2002)	Different feed samples	0 - 50
Wang et al. (2003)	Different feed samples	8.27
Manafi, (2007)	Poultry feeds	500
Manafi et al. (2009)	Mash Poultry feeds	450
Manafi et al. (2010)	Pelleted Poultry feeds	470

Table 2. The results of surveys conducted by various investigators on natural occurrence of aflatoxins in various feeds and feed stuffs

Results of the contamination monitoring program for mycotoxins from 1976 till date showing that, much of the monitored grain contained aflatoxin above 20ppb, higher than the regulatory limits in feeds of most countries (Jelinek et al., 1989).

2.1 Safe/permissible level of mycotoxins in poultry feeds

What is a safe level? Can a contaminated grain source be safely fed to other animals if not poultry? What will be the economic impact of a given level of contamination? These are some of the questions commonly asked by people involved in poultry and livestock farming.

Strictly speaking **there is no *safe level.*** With reference to mycotoxins the risk directly depends on the level of the major mycotoxins in the feed and also on the co-occurrence and levels of other mycotoixns.

In order to reduce the toxic and economical impact of mycotoxins, several countries regulate the levels of certain mycotoxins in foods and feeds. Worldwide food and feed legislation safeguards the health of consumers and the economic interests of animal producers and traders. Virtually all countries with fully developed market economies have regulations with the exception of some African countries.

Since the main consumers of poultry products are humans, it becomes relevant to also view the problem of mycotoxins residues in poultry products from a human health standpoint.

Mycotoxin	**Poultry production**	**Carry over in Meat and Eggs**
Aflatoxin B_1	+	Liver
Ochratoxin A	+	Hatching eggs
Cyclopiazonic acid	+	Meat and eggs
Deoxynivalenol	+	Hatching Eggs
Zearalenone	+	Eggs
T-2 toxin	-	-
Diacetoxyscripenol	-	-
Fusarochromanone (*Fusarium* toxin)	+	Hatching Eggs
Aurofusarin (pigment)	+	Eggs

Table 3. Occurrence of mycotoxin residues in poultry products

3. Toxicity and mode of action

Aflatoxin B_1 is found to be highly toxic (6.1mg/kg body weight) to chicken as compared to other Aflatoxins. Chronic aflatoxicosis resulting from regular low level dietary intake of aflatoxin caused reduced weight gain, decrease in feed intake and poor feed efficiency. The important biochemical effects of aflatoxin B_1 are inhibition of DNA replication and RNA synthesis (Kichou & Walser, 1994). Hsieh (1985) reported inhibition of elongation and/or termination of the translational process of protein synthesis, interference in successive steps in mitochondrial respiratory chain, alteration in immune response and exert carcinogenic, teratogenic and mutagenic effects by reacting with nucleophillic sites in macromolecular components. Further, it was stated that aflatoxin is accumulated in liver and the high content of microsomal cytochrome P-450 enzymes of hepatic cells favors the formation of DNA- aflatoxin adducts. Hence, liver is the major target organ for the aflatoxin toxicity. Among avian species, the most susceptible are ducks and turkeys followed by pheasants, chickens and quails (Diaz et al., 1995).

4. Aflatoxicosis

Aflatoxicosis caused by consumption of aflatoxins represents one of the most serious diseases to man, as well as poultry, livestock and other animals.

Aflatoxicosis in poultry is characterized by hemorrhages, anorexia, mortality, decreased feed efficiency and production, pathological changes in the liver, kidney and bile duct. The economic loss in the poultry industry due to aflatoxicosis is estimated to run upto millions of dollars (Raju et al., 2005).

5. Aflatoxicosis in commercial layers

The most prominent manifestations of experimental aflatoxicosis in layers are reduced egg production and egg weight, increased liver fat and alterations in some serum biochemical parameters.

Sims et al. (1970) fed *ad libitum* aflatoxin -contaminated diet having levels of 2.00 to 8.00ppm aflatoxin B_1 for 17 days and observed a significant reduction in egg production. Egg weight was not affected and also they could not detect any fluorescent metabolites in the eggs or liver of hens fed dietary aflatoxin.

Hamilton & Garlich (1971) fed the Single Comb White Leghorn hens with 1.25-200ppm dietary aflatoxin for three weeks and reported a dose related decrease in egg production and egg size, but shell thickness was not affected. The lipid content of liver was significantly increased in aflatoxin fed hens (5.00ppm) when compared with the control group.

Garlich et al. (1973) reported that the White Leghorn hens receiving 20.00ppm of aflatoxin in their diet for seven days did not adversely affect egg production but plasma calcium, protein, cholesterol and triglycerides were all decreased. In this study, delayed adverse effect of aflatoxin on egg production was observed. Once the hens were returned to a control diet for recovery, egg production began to decline significantly from the first day of the recovery period. Egg production reached to a minimum of 35 per cent, seven days later and then returned to the level of the control group, 19 days after the withdrawal of the contaminated diet. This delayed effect on egg production emphasizes the severe epidemiological problem of mycotoxins. Under field conditions, the feed causing the problem can be totally consumed before its adverse effects are noticed to undertake any therapeutic measure to solve the problem.

Huff et al. (1975) investigated the effect of graded levels of dietary aflatoxin up to 10.00ppm on layers. After four weeks, liver size and liver lipid content were increased, while egg production and egg size were decreased. Dry weight and lipid content of the yolk were not affected but yolk and plasma carotenoid concentrations were elevated.

McDaniel et al. (1979) reported that feeding of 200ppb aflatoxin in the diets did not significantly alter shell thickness of eggs obtained from layers. They concluded a trend with the known phenomenon of inverse relationship between age of bird and egg shell thickness.

Boulton et al. (1981) recorded a significant reduction in HI titers in layer breeders at 500ppb levels of aflatoxin.

Iqbal et al. (1983) fed the White Leghorn layers up to 5.00ppm dietary aflatoxin for three periods each consisting of 28 days. They reported that feeding 1.00ppm level of aflatoxin resulted in a significant reduction in hen day egg production and 2.00ppm level onwards feed efficiency was adversely affected. Congested and haemorrhagic livers, enlarged spleens, and immature ova with congestion were commonly seen. However, none of the levels affected feed consumption, body weight, egg weight, shell percentage, Haugh unit scores and serum protein levels. According to Dalvi & McGowan (1984), chronic aflatoxin toxicity in birds was characterized by drop in egg production. Washburn et al. (1985) reported that dietary aflatoxin at 5.00ppm fed for three weeks had no detrimental effect on shell strength but egg weight was significantly reduced.

Johri & Sadagopan (1989) reported a significant reduction in hen day egg production of laying quails when fed with 0.50 or 0.75ppm aflatoxin. Johri et al. (1990) studied the effect of low levels of dietary aflatoxin (0.00-0.75ppm) in Japanese quail fed toxic diet for 100 d and reported that egg production, protein utilization and body weight were adversely affected by 0.50 and 0.75ppm, whereas feed consumption and hatchability of fertile eggs were adversely affected by 0.30ppm. At 0.75ppm level, fertility of eggs and serum total protein decreased and serum glutamic pyruvic transaminase (ALT) increased.

Aflatoxin when added at 0 and 10ppm, with tryptophan to a layer ration, showed significant reduction in egg production percentage (Rogers et al., 1991). Rao &Joshi (1993) included

1.25, 2.50, 5 and 10ppm aflatoxin B_1 in layer rations for four weeks and found decreased egg production in birds receiving 5 and 10ppm of aflatoxin B_1.
Fernandez et al. (1994) reported a significant reduction in egg production and oral lesions in layer chicken treated with 120ppb onwards for varying periods.
Azzam & Gabal, (1998) reported a significant reduction in egg production of commercial layers fed with high levels of aflatoxin for six weeks.
Kubena et al. (1999) studied the effect of diets containing 50 or 100mg/kg moniliformin fed to White Leghorn laying hens for 420 d and observed that egg production was reduced by approximately 50 per cent by the end of the second 28-d laying period. Egg weights were reduced by the 100mg/kg toxin. The hens in toxin-treated group also had significantly lower body weight than the other treatments. Mortality was minimal except in hens fed with 100mg toxin/kg diet.
Mukhopadhy et al. (2000) have also reported a significant reduction in egg production in commercial layers exposed to 500ppb aflatoxin given for 90 days.
Ginzberg et al. (2000) reported that the yolk color in the group fed on 5 per cent of *Spirulina* algae was 2.4 times darker compared to the control laying hens.
Nimruz (2002) found that yolk color index of layers was significantly improved by the addition of *Spirulina* in feed. He concluded that Zeaxanthin content in the yolk tended to increase significantly with the dosage of *Spirulina*.
Kim et al. (2003) found reduction in serum calcium, phosphorous and ALT and increase in gamma glutamyl transferase (GGT) levels in laying hens by dietary levels of 500ppb of aflatoxin given from week 67 in laying hens.
Chowdhury & Smith (2004) reported decrease in feed efficiency when layers fed *Fusarium* mycotoxins contaminated diets compared with control groups.
Ogido et al. (2004) reported an increase in feed consumption and decrease in egg weight in Japanese quails fed with combination of 50ppb of aflatoxin B_1 and 10ppm of fumonisin B_1 for 140d.
Verma et al. (2004) reported decrease in hen day egg production, egg weight, feed consumption, shape index, albumen index and Haugh unit due to feeding 1ppm of aflatoxinB_1 for 42 d to White Leghorn hens aged 42 weeks.
Svetlana Grigorova (2005) reported that when adding 2 per cent and 10 per cent of dry biomass from fresh water algae of Chlorella genus in the combined forages for laying hens, the yolk pigmentation became significantly more intensive by 2.5 units by the Roche's scale.
Ninety-six laying hens fed with 2.50ppm of aflatoxin B_1 for four weeks by Zaghini et al. (2005) showed decrease in egg weight, egg shell weight and increased protein percentage in albumen. They reported that aflatoxin influenced color parameters, which was attributed to interference of aflatoxin B_1 with lipid metabolism and pigmentary substances deposition in yolk. Further, no aflatoxin B_1 or aflatoxin M_1 residues were found in eggs of the experimental groups.
Pandey & Chauhan (2007) reported that feeding of aflatoxin B_1 at the dose rate of 2.50, 3.13, 3.91mg/kg to the White Leghorn layers from first week to 40 weeks of age did not affect the body weight but resulted in decreased feed consumption, reduction in both egg production and egg weight at 3.91mg/kg level and caused 11-47 per cent dose-dependent mortality. They also reported that feeding aflatoxin B_1 at the dose rate of 2.50, 3.19 and 3.91mg/kg to the White Leghorn layers resulted in paleness of breast muscles, discolored livers, enlarged and pale kidney. Enlarged hearts and lungs were noticed at 3.13 and 3.19mg/kg levels. However, there were no changes in the intestine and spleen at all levels, but the Bursa of

Fabricius was oedematous and enlarged at 3.91mg/kg level. Lymphoid depletion and lymphocytolysis and reticuloendothelial cell hyperplasia in the spleen were also observed in all the toxin fed groups.

Denli et al. (2008) reported a reduced daily feed consumption, egg mass, and serum triglyceride concentrations, while increase in the relative liver weight, the serum activity of alkaline phosphatase, and the serum concentration of uric acid in twenty-eight Hisex Brown laying hens of 47 weeks of age fed with ochratoxin A for 3 weeks when compared those fed with the control diet.

Thapa (2008) reported a significant reduction in egg production of layers fed with varying levels of aflatoxin for three periods.

6. Aflatoxicosis in breeders

When aflatoxin (20.00ppm) was incorporated into feed of mature broiler breeder males for four weeks, no alteration in spermatozoa counts, semen volume, or semen DNA, RNA or protein content was recorded (Briggs et al., 1974).

Howarth & Wyatt (1976) fed broiler breeder hens 5 and 10ppm of aflatoxin in their diet for four weeks and reported no reduction in fertility, whereas hatchability of fertile eggs declined significantly from 95.00 per cent in the control to 68.90 and 48.50 per cent, respectively in 5 and 10ppm aflatoxin fed groups. Egg production decreased significantly during weeks three and four after initiation of toxin feeding in hens fed with 10 and 5ppm aflatoxin, respectively. They also observed enlarged fatty and friable liver and enlarged spleens by feeding aflatoxin at the dose levels of 0.00, 5.00 and 10.00ppm. Further, they did not observe any latent effect of aflatoxin or its metabolites on the performance of the surviving chicks hatched from broiler breeder hens, fed with 0.00, 5.00 and 100µg/kg of aflatoxin for four weeks.

Sharlin et al. (1981) reported decreased semen volume and testes weight and disruption of the germinal epithelium in mature White Leghorn males fed with 20.00ppm aflatoxin for five weeks. They also noticed decrease in feed intake and body weight. However, there was no effect on per cent fertile eggs or per cent hatchability of fertile eggs from hens artificially inseminated with spermatozoa from the treated males.

When laying hens and mature cocks were fed diets containing 8.10ppm aflatoxin B_1 or 1.60ppm aflatoxin G_1 for three weeks, egg production ceased. Histological examination of the ovaries showed follicular atresia. On the contrary, no testicular lesions were seen in the males (Hafez et al., 1982).

Jayakumar et al. (1988) fed aflatoxin B1 at rate of 25µg/duck, daily for three months and noticed reduced fertility and hatchability. Khan et al. (1989) injected 26.00, 81.00 and 216.00 ng/egg of aflatoxin B_1 and reported that lethal dose was 216.00 ng/egg and it caused mortality of chick embryo by the fourth day of incubation.

Tiwari et al. (1989) compared the hatchability of chicks hatched from aflatoxin containing eggs and concluded that it was low in comparison to chicks hatched from aflatoxin free eggs. Further, they studied the post-hatch performance of chicks hatched from aflatoxin containing eggs and observed lower weight gains and impaired defense system in chicks fed on normal diet.

In a study by Abdelhamid & Dorra (1990) where the maternal diet contained 100.00ppb of aflatoxin, citrinin or patulin for six weeks, the chicks had significantly higher weight than the control.

Rao (1990) observed a drastic deterioration in semen quality of breeder cocks fed with 1.000ppm aflatoxin. The traits affected were semen volume, semen concentration, motility and abnormalities.

Stephen et al. (1991) reported a significant drop in egg production in layer chicken fed with 5.00 and 10.00ppm aflatoxin for three weeks.

Nelson-oritiz & Qureshi (1992) assessed single dose exposure of six day-old embryos to 0.100, 0.500 and 1.00µl of aflatoxin B_1 and concluded that rate of mortality of the embryos was dose related. Chick embryos, administered different levels of aflatoxin or ochratoxin on the chorio-allontoic membrane showed decreased weight and length. Further, abnormalities like everted viscera, exposed brain, crossed beak, underdeveloped eyes and head and twisted limbs were observed.

Bergsjo et al. (1993) reported chick developmental anomalies when laying hens were fed diets containing 4.90mg of DON/kg of feed for 10 weeks.

Diaz & Sugahara (1995) reported that birds fed aflatoxin at 0.66 or 3.00µg/kg diet did not show any adverse effect on chick performance.

Muthiah (1996) conducted an experiment to study the effect of graded dietary levels of aflatoxin B_1 (0.00, 0.50, 1.00 and 1.50ppm) on the reproductive performance of layer breeders. He reported that the sperm motility and concentration were not affected while the percentage of sperm abnormality increased when aflatoxin B_1 was included in the diet of breeder cocks. The feed consumption was significantly decreased and egg production declined in proportion to the level of aflatoxin B_1 incorporation in the diets. There was no effect on fertility but hatchability was affected. The chicks hatched from breeder hens, received graded levels of aflatoxin in their diets did not show any effects on body weight, weight gain, mortality and feed consumption during the 0-8 weeks post-hatch performance period.

Cotter & Weinner (1997) reported lowered hatchability in broiler breeder hens fed with four levels viz., 0.00, 308.00, 610.00 and 1834.00ppb of aflatoxin.

Brake et al. (1999) conducted an experiment by feeding diets with different levels of diacetoxyscirpenol (DAS) (ranging from 0.00 to 20.00mg/kg) to broiler breeders between 67 to 69 wk of age. They observed no effect on egg production, when DAS was fed upto the level of 5.00ppm. Furher, they have demonstrated that feeding diets contaminated with 10.00 and 20.00mg of DON per kg of feed decreased the fertility in broiler breeder males, though there was no difference in the volume of semen produced.

Brake et al. (2000) reported that there were dose-related decreases in body weight and feed consumption indicating feed refusal, as well as dose-related increases in the extent of mouth lesions of broiler breeders fed with 0.00, 5.00, 10.00, or 20.00mg DAS/kg diet from 24 to 25 wk of age.

Stanley et al. (2004) reported that feeding aflatoxin at the rate of 3mg/kg to 35 week's old Cobb broiler breeder hens for three weeks significantly reduced serum total protein, albumin, calcium and phosphorus levels.

Sypecka et al. (2004), reported that only trace amounts of *Fusarium* mycotoxins are transferred into the eggs of laying hens, which are unlikely to be of significance with respect to embryonic mortality.

Yegani et al. (2006) reported no effect in feed consumption, body weight, and egg production. However, increase in early embryonic mortality (1 to 7d) in eggs from birds fed contaminated grains with deoxynivalenol (12.60mg/kg of feed) was observed in broiler breeder hens. They also reported that the ratio of chick weight to egg weight was not affected. Weight gains of

chicks fed a standard broiler starter diet at 7, 14, and 21 d of age were also not significantly affected by previous dietary treatments for the dam. Feeding of contaminated diets did not affect semen volume, sperm concentration, viability, and motility. There was no effect of diet on the relative weights of liver, spleen, kidney and testes.

Histological evidence of adverse effects of aflatoxin on the germinal epithelium of the testes was reported in immature chickens dosed with 200µg of aflatoxin/day/chick for 35 days (Mohan et al., 2008).

Manafi et al. (2009) reported the aflatoxin fed at the levels of 300, 400 and 500ppb for three periods, each with duration of three weeks to broiler breeders from 28 to 36 weeks of age. Inclusion of 500ppb aflatoxin in the diet significantly ($P \leq 0.05$) affected feed consumption, feed efficiency, egg production, egg weight, fertility, hatchability, embryonic mortality, GGT and ALT levels in the serum, organ lesion scores (liver, kidney, proventriculus and gizzard), relative weights of heart as well as liver, antibody titers against Newcastle and Infectious bursal diseases when compared to that of control. The results indicated no significant ($P \geq 0.05$) effect of aflatoxin on body weight, shell thickness, Haugh unit, residue in eggs, sperm count, per cent live sperm, yolk color index and relative weight of spleen when compared to that of control.

Serum alkaline phosphatase levels were significantly higher, serum alkaline aminotransferase ($P=0.068$) and gamma-glutamyltransferase ($P=0.067$) levels tended to increase ($P<0.05$) in 58-wk-old Ross 308 broiler breeders fed with 100 µg aflatoxin-contaminated diet than those of hens fed the uncontaminated diet (Matur et al., 2010).

7. Aflatoxin residue in eggs

Although the concentration of mycotoxins and their metabolites are generally much lower in eggs than in animal feeds and are not likely to cause acute intoxications in humans. However, the residues of carcinogenic mycotoxins such as aflatoxin B_1 and M_1, (aflatoxin M_1 is a polar metabolite of aflatoxin B_1) and ochratoxin A, when present in animal products are a threat to human health and must be monitored. The limit for aflatoxin B_1 in complete feeds is 0.02mg/kg.

Trucsksess et al. (1983) were able to detect aflatoxin B_1 and M_1 residues in eggs of hens fed contaminated feed. After 7 days of withdrawal only trace amounts remained in eggs. According to Wolzak et al. (1985) clearance of aflatoxin occurs faster from the albumen than from the yolk.

Aflatoxin and some of their metabolites can be carried over from feed to eggs in ration ranging from 5,000:1 to 66,200:1 and even to 125,000:1, whereas in other trials no measurable residual aflatoxin B_1 or its metabolites were found in eggs (Oliveira et al., 2002).

Zaghini et al. (2005) reported that no traces of aflatoxin B_1 or aflatoxin M_1 residues were found in eggs of layer hens supplemented with diet containing 2.50ppm aflatoxin B_1.

In another study, Salwa & Anwer (2009) reported no traces of aflatoxin in the eggs of layers fed with 25.00, 50.00 and 100ppb of aflatoxin in their diet for 60 days.

8. Counteraction of aflatoxicosis

The infestation of agricultural products, intended for human and animal consumption with toxigenic fungi that are capable of producing highly toxic metabolites has been a worldwide problem. Increased efforts are being undertaken to develop cost effective and safe

procedures and products to effectively deal with the decontamination and remediation of mycotoxin contamination in feedstuffs. The available approaches were reviewed by Trenholm et al. (1996) and Devegowda et al. (2003).

The methods aimed at preventing or reducing the level of mycotoxin contamination were classified as preventive or curative. The following approaches were recommended:

1. Prevention of the initial growth of moulds and subsequent production of mycotoxin.
2. Detection of mycotoxin in feed and selective removal of contaminated portions.
3. Inactivation or destruction of the toxin by physical, chemical and biological means.
4. Utilization of mycotoxin resistant genetic resources.

8.1 Physical methods

According to Park & Liang (1993) Mycotoxins (aflatoxin, ochratoxin, T-2 toxin and citrinin) are highly soluble in organic solvents. Their extraction from the feed stuffs using several solvents or mixture of solvents has been proved to be highly effective.

Scott (1989) opined that thermal treatment appears to have little effect on the toxin content as mycotoxins are heat resistant. Irradiation of feed stuffs may reduce the toxin content considerably. Exposure of contaminated feed ingredients to sunlight may also prove to be effective. These methods have little practical applicability.

The utilization of mycotoxin-binding adsorbents, which do not get absorbed from the GIT and instead bind physically with mycotoxin, is the most applied physical method of protecting animals against the harmful effects of mycotoxin contaminated feed and has gained considerable attention in recent times. The efficiency of the adsorption depends on the chemical structure of both the adsorbent and the mycotoxin. Before applying this technique for routine use, it is essential to establish that the adsorbent does not remove essential nutrients from the diet (Manafi et al., 2009).

Clays like hydrated sodium calcium aluminosilicates, activated carbon, bentonite, clays and special polymers are made of two or more mineral-oxide layers. These layers are stacked parallel units of silica and alumina sheets. The silica form tetrahedral sheets and the alumina forms octahedral sheets. Some of these clay particles have the ability to absorb moisture and will expand while others do not. The difference is due to clay chemistry and the elements (cations) that are components of the layers.

8.2 Herbal methods

Application of some herbal extracts of plant origin like turmeric (*Curcuma longa*), garlic (*Allium sativum*) and asafetida (*Ferula asafetida*) have shown to counteract aflatoxicosis in animals and poultry through their antioxidant activity.

Several herbal products contain antioxidant substances capable of scavenging free radicals and enhancing antioxidant enzymes. Nyandieka et al. (1990) reported that use of ethanolic extract of *Cassia senna*, (herb) as laxative inhibited the mutagenic effects of aflatoxin B_1. Feeding of the extract of *Azadirachta indica* prevented metabolic activation of aflatoxin B_1 to its epoxide derivative. Hepatic antioxidant status of rats was enhanced by feeding of a phenolic-lignin enriched extract of the fruit *Schisandra chinensis* and provided hepato protection against aflatoxin B_1.

Oxidative changes (increased peroxides, reduced antioxidant enzyme activity) in liver and kidney due to aflatoxin B_1 were reversed in rats by feeding a root/rhyzome extract of *Picrorhiza kurroa* (Picroliv) and a seed extract of *Silybum marianum* (Silymarin) (Weiss, 2002). Similarly, Rosamarinic acid, a phenolic component of *Boragnaceae* species of plants (sage,

basil, and mint) reduced free radical oxygen formation, and inhibited protein/DNA synthesis as well as apoptosis of human hepatoma cells caused by aflatoxin B_1 and ochratoxin A (Manafi et al., 2009).
According to Gowda & Ledoux (2008) ellagic acid, a phenolic compound of strawberries and grapes showed anticarcinogenic activity and inhibited aflatoxin B_1 mutagenicity. S-methyl methane thiosulfonate present in cabbage and onion suppressed chromosomal aberrations due to aflatoxin B_1 in rat bone marrow cells. Diterpenes, cafestol and kahweol present in green and roasted coffee beans prevented the covalent binding of aflatoxin B_1 to DNA by modulation of the carcinogenesis enzyme system.
Iqbal et al. (1983) observed chemoprotective effect of piperine (1-piperoyl piperidine), an alkaloid of pepper against aflatoxin by inhibiting cytochrome P 450 bioactivation of aflatoxin B_1. The protective effect of chlorophylline (a derivative of the green pigment chlorophyll) against aflatoxin B_1 was also observed. The toxic effects of aflatoxin in chicken was reversed by the administration of an alcoholic extract of African nut meg. The carbonyl functional groups of the curcuminoids are thought to be responsible for their antimutagenic and anticarcinogenic action. Further, strong inhibitory effect of curcumin on superoxide anion generation was noticed.

8.3 Use of enzymes

The enzymes are believed to break the functional atomic group of the mycotoxin molecule and thereby render them nontoxic (Kumar et al., 1993). Enzymes viz., carboxyesterase present in the microsomal fraction of the liver, esterase and epoxidase are being tried for their practical applicability in the field conditions (Pasteiner, 1997).

8.4 Nutritional manipulations

Increasing the crude protein content and supplementation of additional levels of riboflavin, pyridoxine, folic acid and choline showed protective effect against aflatoxicosis (Ehrich et al., 1986). Anti-oxidants like BHT and β-napthoflavone, vitamin C and vitamin E offer protection against aflatoxin induced genotoxicity in *in vitro* studies (Johri et al., 1990).
Increase in dietary protein levels and supplementation of L-phenylalanine was revealed to be effective against aflatoxicosis and ochratoxicosis. Devegowda et al. (1998) reported that the supplementation of the diet with selenium and methionine partially alleviated the adverse effects of aflatoxin respectively.

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8.6 Biological methods

With the awareness of potential harmful effects of chemicals used for counteracting mycotoxins and the cost involvement with their usage has prompted the scientists to look

for alternative methods which are applicable and safe. A rapid explosion in the field of feed industry through the biotechnological methods has opened a new possibility of degradation of mycotoxins by microorganisms. Several yeasts, moulds and bacterial strains posses the ability either to destroy or transform mycotoxins successfully (Phillips et al., 1988).

8.6.1 Bacterial degradation

Acid producing bacteria's such as *Lactobacillus plantarum* and *Lactobacillus acidophilus* were found to detoxify aflatoxin in maize. Rumen bacteria were found to degrade ochratoxin A (OA), T-2 toxin and zearalenone (ZEA) (Linderfelser & Ceigler, 1970).

He et al. (1992) detoxified moldy maize diet containing 5.00ppm vomitoxin, microbially through incubation with the contents of large intestine of chickens having a detoxified vomitoxin of 2.10ppm.

8.6.2 Protozoan degradation

Tetrahymena pyriformis at a dose rate of $22x10^6$ cells detoxified aflatoxin B_1, by converting it into its hydroxyl products to an extent of 5 per cent in 24 hours and 67 per cent in 48 hours (Robertson et al., 1970). Intact rumen fluid containing various protozoa was reported to metabolize T-2 toxin and ochratoxin while no effect on aflatoxin was noted (Kiessling et al., 1984).

8.6.3 Fungal degradation

Some of the species of fungi have been found to detoxify aflatoxin. An intracellular substance was found to be responsible for *A. flavus* and *A. parasiticus* to degrade the formed toxins in a culture when their mycelium was subjected to fragmentation. The peroxidase enzymes produced by the fungal mycelium, which can catalyze hydrogen peroxide into free radicals, reacts with aflatoxin (Dvorak, 1989).

8.6.4 Degradation by yeast

Yeasts are being primarily used as growth promoters in poultry and animal feeds. Besides their beneficial effects on feed utilization and rich concentration of many vitamins, certain species and strains of yeasts have been observed to detoxify mycotoxins through its degradation (Cooney, 1980).

In the early 1990s, a commercially yeast culture preparation of *Saccharomyces cerevisiae*^{1026}, which was earlier noted as digestive aid and a growth promoter, was found to improve hatchability (McDaniel, 1991) and broiler body weights (Stanley et al., 1993). Further investigations lead to the establishment of yeast culture preparation's ability to adsorb aflatoxins in poultry feeds (Devegowda et al., 1995).

Supplementation of live cells of *Saccharmyces cerevisiae*^{1026} was found to be beneficial in counteracting the adverse effect of several mycotoxins (Stanley et al., 1993). It has also improved serum total protein and HI titer against Newcastle disease in aflatoxin fed broilers (Devegowda et al., 1996). In an another study, inactivated yeast at 0.2 per cent in the diet was found to alleviate the growth depression effects of aflatoxin up to 200ppb level (Devegowda et al., 1998).

Mycotoxin binding ability of Maannanoligosaccharides has been demonstrated in various *in vitro* trials (Devegowda et al., 1998) and *in vivo* trials (Raju & Devegowda, 2000; Swamy et al., 2004).

9. Conclusion

The addition of aflatoxin in layer and breeder hens could lead to aflatoxicosis which can adversely affect the performance and reproductive efficacy of the birds. Aflatoxicosis in poultry is characterized by hemorrhages, anorexia, mortality, decreased feed efficiency and production, pathological changes in the liver, kidney and bile duct. The economic loss in the poultry industry due to aflatoxicosis is estimated to run up to millions of dollars. In commercial Layers, the most prominent manifestations of experimental aflatoxicosis are reduced egg production and egg weight, increased liver fat and alterations in some serum biochemical parameters.In case of breeder hens, many of parameters like changes in body weight, feed consumption, feed conversion ratio, egg production, egg weight, shell thickness, Haugh unit score, yolk color index, fertility, hatchability and embryonic mortality and some of biochemical and immunological parameters like serum levels of GGT and ALT, visceral organ weight and organ lesions and serum antibody titers for ND and IBD could be altered due to aflatoxicosis. This chapter briefly reviews the impact of aflatoxicosis in commercial layer and breeder hens.

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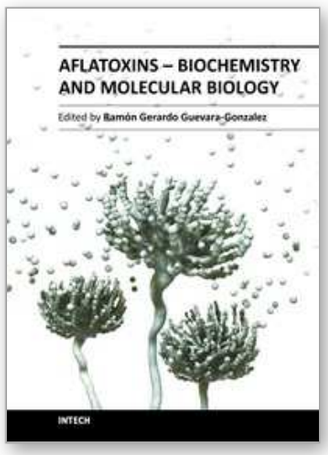
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Aflatoxins â€“ Biochemistry and Molecular Biology is a book that has been thought to present the most significant advances in these disciplines focused on the knowledge of such toxins. All authors, who supported the excellent work showed in every chapter of this book, are placed at the frontier of knowledge on this subject, thus, this book will be obligated reference to issue upon its publication. Finally, this book has been published in an attempt to present a written forum for researchers and teachers interested in the subject, having a current picture in this field of research about these interesting and intriguing toxins.

How to reference

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