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Assessment of Aflatoxins production and its various Control strategies: A Review

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ABSTRACT: *Mycotoxins specifically the aflatoxins contaminate about 25% of the agricultural products in the world. They effect greatly the growth rate, feed intake, and feed conversion ratio. There is a great economical loss to farmers due to loss of food values and effecting human as well as animal health. The different preventive measures are in use to control fungal contamination such as chemicals (acids, bases, and salts), HSCAS ,zeolite polymers, activated charcoal and yeast with variable findings. Fraction of yeast sludge has proved to minimize the adverse effects of aflatoxicosis due to the presence of manno-oligosaccharides in its cell wall. The present review highlights the remedial measures particularly yeast sludge to control aflatoxicosis.*

Keyword: *Aflatoxin, Contamination, Yeast sludge, Glucomannan*

The Historic Background of Mycotoxins

Mycotoxin's effects on humans and animals health has been reported since the 14th century. In 1952, the ingestion of mold-contaminated maize feed from swines in South USA resulted in an epidemic of "mouldy corn toxicosis" (Sean, 2002). AFB1 found to be the main cause for turkey deaths (Agag, 2004).

In 1962, aflatoxins were named due to the presence of *Aspergillus flavus*. This Aflatoxin have shown green and blue UV fluorescence. It can cause toxicity, mutagenicity and teratogenicity and are mainly produced by certain toxigenic fungal strains like *A. paraciticus* and *A. flavus.* Aflatoxins can be further divided into AFB and AFG (Agag, 2004). In 2004, hundred Kenyans became critically ill and nearly 125 victims were registered in 2004

because of acute aflatoxicosis (Wu et al., 2011).

Mycotoxins

Mycotoxins are secondary metabolites derived from mycelial filament frameworks (Dewegowda et al., 1998; Hussein and Brasel, 2001). The most common mycotoxins are the types of *Aspergillus, Penicillium, Fusarium, Zearalenones* and *Ochratoxins* (Binder, 2007).

Grains are more infected by mycotoxins as pathogensare present in fields.Secondary metabolites from moulds enforce harmful effects (Hussein and Brasel, 2001). Different routes including ingestion, absorption through the skin, and ingestion can lead to mycotoxins entering the body. This leads to various lethal effects which can be carcinogenic, immunotoxic, neurotoxic and teratogenic (Casteel and Rottinghouse, 2000; Frisvad et al., 2007). Species, sex, age, nutritional health, etc. are the magnitude of the results recently been reported to be carcinogenic (Yiannikouris and Jouany, 2002).

Cereal products are widely used for incursions into fungi, leading to production of aflatoxins (Zaki et al., 2012). Some 25 to 40% of the total global production of cereals is polluted with mycotoxins, particularly aflatoxins, directly and indirectly. Aflatoxins are manufactured in storage by *Aspergillus flavus* and *Aspergillus parasiticus.* They can be located in hot and humid tropical countries in contrast with moderate regions (Yiannikouris and Jouany, 2002; Maurice, 2002).

Aspergillus flavus is typically present in rice, peanuts and cotton. *Aspergillus parasiticus* is uncommon in South-East Asia, but it does not have *Aspergillus flavus*, which is only threatened by *Aspergillus parasiticus* (Pitt et al., 1997). AFB1 and AFB2 are generally formed by the *Aspergillus flavus*, while AFB1, AFB2, AFG1, and AFG2 are created by *Aspergillus parasiticus* (Agag, 2004).

The study of aflatoxins indicates that AFB1 is up to 77% the largest grain contaminant (Wilson and Payne, 1994). The embryo part of the grains is the key location for the development of *Aspergillus flavus*, which results in further aflatoxin bloom (Ghahri et al., 2010). In regions between 40 °N and 40 °S equator in latitude at temperatures 24-35 °C and humidity content exceeding 10% (Williams et al., 2004).

About 4.5 billion people in developed countries are exposed to aflatoxins, and high levels of aflatoxins in tropical and sub-tropical regions are in deteriorated areas (Verma, 2004).

There can be no place in the world free of aflatoxins due to the transport of agricultural supplies (Maurice, 2002).

Throughout Pakistan, aflatoxins are found to be 91.1% of the most prevalent mycotoxins in survey of poultry diets in 2 decades. The aflatoxin B2 were found to be present in ten commercial feed mills in Punjab. AFB2 was found to be more than 20 µg/kg in layer as well as broiler starter poultry (Shareef, 2010).

AFB1 was most often found to be 26.1 percent of the samples are aflatoxins, in particular meat, cereal as well as oil seeds (Bokhari, 2002). The overall level of exposure of aflatoxins to shell walnuts is 40%, with no shell at 70%, and peanuts with shell at 40 percent, is observed in KPK and northern parts of Pakistan (Luttfullah and Hussain, 2011).

The highest incidence of aflatoxin is in 60% maize, 40% sorghum and 25% in various trade shops (Lutfullah and Arshad, 2012). *Aspergillus flavus* in Pakistan had the highest incidence (Shah et al., 2010).Borutova et al., (2012) reported the occurrence in 2010 in the Asian area of various mycotoxins on various feeds including rice, wheat, soybeans, maize gluten, dried distillery, etc.

Chemical Nature and Structural illustration

300-400 secondary fungal metabolites and mycotoxins have been found in different commodities (Binder, 2007). Aflatoxins are molecular derivatives of bifuranocoumarin. The chemical composition contains a furan ring and the coumarin nucleus with such a pentenone ring (AFB and AFM) and a lactone ring with six members (AFG) (Brase et al., 2009).

Fluorescence under ultraviolet light can distinguish the four most common compounds such as B1, B2, G1, G2, M1 and M2 (Fig. 1) (Agag, 2004). Aflatoxins are soluble in methanol, chloroform and acetone. They are reactive and these contaminants decline at their boiling points, which vary from 237°C to 299°C but do not damage under standard cooking conditions (Jallow, 2015).

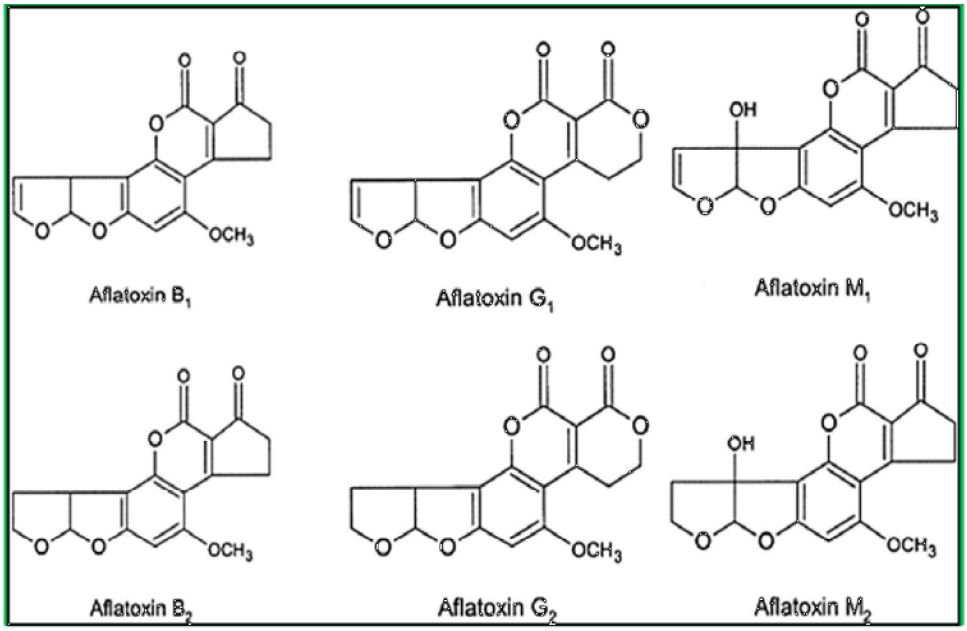

Fig. 1. Chemical structures of aflatoxin B1, B2, G1, G2, M1 and M2 (Musleh et al., 2017)

Factors that affect Toxicity

Physical, biological and chemical factors that influence toxin development are specific. Moisture, heat, humidity as well as mechanical damage are physical influences. Relative oxygen, carbon dioxide, medium structure, contaminants, and fungicides are the chemical influences included. Biological factors include plant variety, pressure, various insects and aggregation of spores that can influence the level of toxicity in development (Wayne, 2012). For the development of fungal mycotoxins, water, oxygen, temperature and pH (3-8) play a key role. Water activity can differ from 0.61 to 0.91, as the majority of fungi are grown at 0.75. Limited temperature varies from 12-41 °C in the processing of aflatoxins by *Aspegillus flavus* and *Asprgillus parasiticus* with maximum productivity at 25-32 °C. However, the synthesis of the aflatoxins increases by upwards of 27 °C and moisture by over 62 per cent (Agag, 2004).

The synthesis of AFB1 is higher as compared to AFG1. AFB1 is best produced at 24-28°C while 23°C is best temperature for the production of AFG1. The output of equal volumes of AFB and AFG is reassured by a low temperature of 8 to 10 °C. However, the production of total aflatoxins is limited and longer is required (Agag, 2004). Fungi are active in food as food spoilers

at higher levels of moisture (Zaki et al., 2012). In comparison, *Aspergillus* species may be at lower moisture levels than *Fusarium* species (Wayne, 2012). Oxygen is a crucial growth driver as it influences the production of mushrooms. Its development is controlled to below 1% oxygen (Zaki et al., 2012).

Aflatoxins pollution is positively impacted and potentially overlapped by high temperature and drought by insect damage to plants (Wayne, 2012). These conditions allow "hot spots" to expand in stored grains. Specific maize grains can be occupied with aflatoxins of 400,000 μg/kg (Richard, 2007). Before and after harvesting the accumulation of mycotoxins meditates primarily on climate factors, such as *Fusarium* species developed cereal toxins under high moisture conditions during harvest periods, and before harvesting crop pollution with aflatoxins, such as peanuts and maize, combined with high temperatures, insect damage and extreme drought conditions (Wayne, 2012). Genetic fungal genes and pathways have been dislocated for the manufacturing of mycotoxins and parameters (Yu and Keller, 2005; Bhatnagar et al., 2008). These genes may aid the development of plants which are difficult to aggregation of toxins (Wu et al., 2004).

Effects of Aflatoxins

Toxicity ofdifferent types of aflatoxins such as B1, G1, B2 and G2, depends on feed exposure period, species, sex, health status, and age of animal (Richard,2007; Denli et al., 2009). The disturbance of well-developed hepatic enzyme processes and break down of toxins potentially makes children less immune than adults (Quist et al., 2000).

The most infected species by aflatoxins are turkey chickens, broilers and layers (Huff et al., 1986). Goslings, pheasants and quails have an intermediate role for susceptibility, whereas chickens seem to be extremely immune. Ducklings are 5-15 times more sensitive than laying hens; but within layers, few varieties can be three generally more concentrated than other strains of layers (Azizollah et al., 2009; Suksombat et al., 2011).

Any mycotoxin after intakehas ability to affect body functions and contributes in diseases. Clinical signs and disease can differ even between species as the Aflatoxins which did not seem to affect the wellbeing of the broiler yet had a detrimental effect on the human community development

(Haschek et al., 2002; Grenier et al., 2011; Wayne, 2012).

Chronic aflatoxicosis, overt or indirect signs take in to account growth depression, lower feed volume and lowered nutritional intake (Humphrey and Klasing, 2004; Klasing, 2007). The decline in growth following the ingestion of aflatoxins is responsible for the low production of pancreatic digestive enzymes.The ingestion of aflatoxins has not been shown to affect apparent metabolizable energy (AME) in foods, but substantially decreases weight gain energy efficiency, AME energy intake and higher heat performance in broilers tissue gain (Wayne, 2012). Aflatoxin in feed may trigger an increase of 5% depression of broiler (Dersjant Li et al., 2003) and the poultry industry to cause a big difference between benefit and loss at each aflatoxin dose rate as mg/kg of feed (Kaki et al., 2012).

Aflatoxins Detoxification

Universally, the feed contaminated by aflatoxins B1 is almost unavoidable (Rawal et al., 2010). Decontamination of mycotoxins is referred to different methods by which their toxicity is removed. They may be physical and chemical methods (Diaz and Smith, 2005).

Physical Methods

Different methods have proven effective in reducing moderate mycotoxins concentrations as cleaning the kernel surface (Huwig et al., 2001; Wayne, 2012). Conversely, this seems relatively lengthy to remove highly contaminated feedstuffs.

Chemical Methods

A group of acidic compounds, alkaline compounds, salts, oxidants, reducing agents etc., are being utilized for degradation of mycotoxins in feedstuff (Jalili et al., 2011). These available methods are ineffective and relatively pricey. To reduce aflatoxins levels, ammoniation has been established but it is not acknowledged in the United States (Park and Price, 2001). Ammoniation abolishes the mycelia and lethal spores of fungi. Moreover, elevated quantities of doses of acetic acid, isobutyric acid, propionic acid, methyl bromide, ethylene oxide, ethylene dibromide, propane or propene, sulfur dioxide and phosphine show fungicidal activity. Although, these chemical compounds decrease dietetic quality and are corrosive to humans and animals.

Any of them will confiscate aflatoxins during the digestive phase as they are added to foods infected with

aflatoxins, causing mycotoxins to travel harmlessly through livestock gastro intestinal regions (Phillips et al., 1990). The more successive and realistic approaches to solving the issue of aflatoxins are the application of adsorbents and the degree of measured adsorption will range from 0% to 87% from a number of different resources of clay minerals (Schiedeler in 1993). Analysis has shown that aflatoxins can be adsorbed by sodium aluminosilicates and determines the type (Mahesh and Devegowda, 1996).

Mineral adsorbents such as zeolites, silica and phylosilicates demonstrate a range of binding potential for aflatoxin and are active, at basal levels, in channels of interlaying, on the surface, pores as well as at particle edges (Daković et al., 2000). It can be done the bentonite adsorption potential varies from 17% to 36%. An additional significant advantages of such adsorbents are the relatively cheap, healthy and easy to apply to animal feedingstuffs (Magnoli et al., 2008).

Soybean bentonites are white, light in weight and formed from volcanic centigrade, which primarily contain montmorillonite and are made up of salt from Na, K, Ca of hydrated aluminosilicates sometimes containing Fe, Mg, Zn and Ni etc (Diaz and Smith, 2005). The layered microphase of these adsorbents makes it possible for aflatoxins to react for adsorption at locations and boundaries surfaces in the interlayer region (Ramos et al., 1996). Solid colloidal compounds of zeolites have the ability to swell and raise water easily, which produces a gelatinous, thixotropic material (Pasha et al., 2008; Safaeikatouli et al., 2011).

The interlayer of montmorillonite that convinces the adsorption of multiple organic molecules including mycotoxins, zeolite and montomorillonite particles. The theory of atmosphere creating the hydration of exchangeable cations. Zeolites surfaces attract polar feature community of aflatoxins (Kolossova and Stroka, 2009).

In comparison to hydro-calcium aluminosilicate (HSCAS), the influence of aflatoxins on baby animals is decreased by 1 percent (Kolossova and Stroka, 2009). The strong negative charges of HSCAS are balanced with cations such as magnesium, potassium and sodium which are present in the cavities and which, by neutral pH or alkaline origin, are not reacted with feedstuffs and behave as an inert matter (Khanedar et al., 2012).

Aluminosilicates are being used as "anti-caking" agents at a frequency of

up to 2% but there are many drawbacks to the decrease in the use of minerals and a limited range in binding ability (Kolossova and Stroka, 2009). Bentonite minerals can affect ca-metabolism, nitrogen cations like NH_4^+ and can be used for the adsorption of AFB1. The development of tibial mineral values in chick feeds that have nutritional deficiencies is not shown to harmful effects (Southern et al. 1994).

There was a competition, at lower levels of toxins between AFB1 and montmorillonite clay supplemented feed of broiler chicken for adsorption sites (Liu et al., 2011). Bio-transformation of mycotoxins into less toxic metabolites, is another alternative by the application of microorganisms like *Corynebacteriumrubrum* (Yiannikouris and Jouany, 2002). This takes action in the intestinal area of animals earlier to absorption of mycotoxins but toxicity of products by enzymes, undesired sound effects of fermentation and with microorganisms on food quality is leftover.

The efficacy of Hydrated Sodium Calcium AluminoSilicate (HSCAS), with a blend of clays and yeast cell wall against Aflatoxins were determined on one day old chicks in 11 treatments with 5 replicates pens and observed mortality rate for control with FCR was not affected by dietetic treatments. So two adsorbents comparison indicated that chicks gained more with HSCAS supplemented feed than yeast cell wall feed (Zhao et al., 2010).

Control and Management

Strategies to control mycotoxins ought to be economically consistent and meet up the standards of FAO, UNEP or WHO on toxicity of mycotoxins. According to international criteria, control strategy should inactivate the toxin with no carcinogenic products, nodestructionof nutritive value of products and no change in quality of products (Liu et al., 2011).

Anokwuru et al. (2011) found the effect of factors like proper irrigation, genetically resistant crops and bio pesticide therapy to remove the toxicity produced by using *Aspergillus* species and determined the ability of ethanolic aqueous extract of *Acacia indicia* bark to lessen hepatotoxicity. Aqueous extracts of a variety of medicinal plants like *Punicagranatum, Cassia alata, Daturastramonium, Polyanthia longifolia* and *Annonasquamosa* were analyzed against *Aspergillus parasiticus* to inhibit production of aflatoxins (Rajani et al., 2012) .

Fruit extracts of *Libidibiaferrea* and *Paulliniacupana*showed significant

antifungal activities (Breda et al., 2016). Production of aflatoxins can be restricted by maintaining cereals without moisture and use of propionic acid to inhibit growth of molds by decreasing pH. Irradiating of UltraViolet rays, X-rays or microwave is also one of the convenient method to detoxify aflatoxins (Yiannikouris and Jouany, 2002).

Yeast Sludge

In Asia and African countries, yeast groups, chiefly *Saccharomycese cerevisiae*, contribute the principal character in fermentation of food, along with lactic acid bacteria (LAB) for food processing as well as preservation (Sari et al., 2008). In Pakistan, yeast sludge can be collected from sugar mills and distilleries (Mumtaz et al., 2000). Hassan et al. (2012) evaluated that 100 ml. of yeast sludge contains 8.96 gm of yeast cells; 0.26 % of mannan oligosaccharide and this is the prime compound that binds the aflatoxins and Ochratoxin A and improves the practical yield and financial side of profitable in poultry fabrication.

Saccharomyces cerevisiae and lactic acid bacteria (LAB) like *propionibacteria*, *bifidobacteria* and *lactobacillus rhamnosus* bind themselves by cell wall components against mycotoxins powerfully without toxic effects on animal health (YiannikourisandJouany, 2002; Kolossova and Stroka, 2009). In early 1990s, *Saccharomyces cerevisiae* (yeast), was used as a growth promoter and found to have valuable effects on weight gain and immune system in broilers exposed to aflatoxins (Rawal et al., 2010).

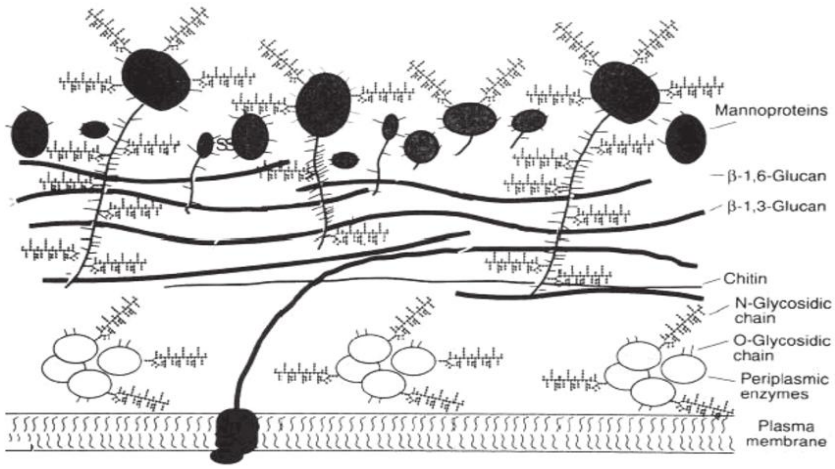

Fig. 2. Composition and structure of the yeast cell wall

CONCLUSION

Aflatoxins are a major source of disease outbreaks due to lack of knowledge and consumption of contaminated food andfeed worldwide. Extreme levels of aflatoxins in food inundeveloped countries are of major concern. Several effective physical, chemical, biological, and genetic engineeringtechniques have been employed for the mitigation, effective control and management of aflatoxins in food. But, developing fungal resistant and insect resistant hybrids/crops to combat pre-harvest infections and their outcome is a major issue of concern. Post-harvest treatments to remove aflatoxins such as alkalization, ammonization, and heat or gammaradiation are not generally used by farmers. However, some of plants have the ability to degrade and reduce the aflatoxin contamination in different types of agricultural products. Therefore, methods of using yeast sludge to reduce aflatoxin are currently being focused. Moreover, application of genetic recombination in *A. flavus* and otherspecies is being investigated for its potential to mitigateaflatoxins to ensure the safety and quality of food.

REFERENCES

1. Agag BI (2004). Mycotoxins in foods and feeds: 1-aflatoxins. Assiut Univ. Bull. Environ. Res. 7(1): 173-206.
2. Anokwuru CP, Anyasor GN, Ajibaye O, Fakoya O, Okebugwu P (2011). Effect of Extraction Solvents on Phenolic, Flavonoid and Antioxidant activities of Three Nigerian Medicinal Plants. Nat. Sci. 9(7): 53-61.
3. Azizollah K, Abdoullah H, Sudabeh M (2009). Effect of yeast glucomannan on performance of broiler chicken. Int. J. Agric. Biol. 11(1): 49-53.
4. Bhatnagar D, Rajesekaran K, Payne GA, Brown RI, Ye J, Cleveland TE (2008). The‘omics’ tools: genomics, proteomics, metabolomics and their potential for solving the aflatoxin contamination problem. World Mycotoxin J. 1(1): 1–10.
5. Binder EM (2007). Managing the risk of mycotoxins in modern feed production. Anim. Feed Sci. Technol. 133(1): 149–166.
6. Bokhari FM (2002). Aflatoxin production by *Aspergillus flavus*, Isolated from different food stuffs commonly used in Jeddah region, Saudi Arabia. Pak. J. Biol. Sci. 5(1): 69-74.

7. Borutova R, Aragon YA, Nährer K, Berthiller F (2012). Co-occurrence and statistical correlations between mycotoxins in feedstuffs collected in the Asia–Oceania in 2010. Anim. Feed Sci. Technol. 178(3–4): 190-197.
8. Brase S, Encinas A, Keck J, Nising CF (2009). Chemistry and biology of mycotoxins and related fungal metabolites. Chemical Rev. 109(1): 3903–4399.
9. Breda CA, Gasperini AM, Garcia VL, Monteiro KM, Bataglion GA, Eberlin MN, Duarte MC (2016) Phytochemical Analysis and Antifungal Activity of Extracts from Leaves and Fruit Residues of Brazilian Savanna Plants Aiming Its Use as Safe Fungicides. Nat. Prod. Bioprospect. 6(1): 195–204.
10. Casteel SW, Rottinghouse GE (2000). Mycotoxicoses. Enyclopedia Mircobiol. 3(1): 337–348.
11. Denli M, Blandon JC, Guynot ME, Salado S, Perez JF (2009). Effects of dietary AflaDetox on performance, serum biochemistry, histopathological changes, and aflatoxin residues in broilers exposed to aflatoxin B1. Poult. Sci. 88(1): 144-1451.
12. Dersjant-Li Y, Verstegen MWA, Gerrits WJJ (2003). The impact of low concentrations of aflatoxin, deoxynivalenol or fumonisins in diets on growing pigs and poultry. Nutr. Res. Rev. 16: 223–239.
13. Dewegowda G, Raju MVLN, Swamy HVLN (1998). Mycotoxins: novel solutions for their conteraction. Feed stuffs. 7(12): 15.
14. Diaz DE, Smith TK (2005). Mycotoxins sequestering agents: practical tools for neutralization of mycotoxins. The Mycotoxin Blue Book. Nottingham University Press, England. 323-339.
15. Frisvad JC, Smedsgaard J, Samson RA, Larsen TO, Thrane U (2007). Fumonisin B2 production by *Aspergillus niger*. J. Agri. Food Chem. 55(23): 9727-9732.
16. Ghahri H, Habibian R, Fam MA (2010). Effect of sodium bentonites, mannan oligosaccharide and humate on performance and serum biochemical parameters during aflatoxicosis in broiler chickens. Glob. Vet. 5(2): 129-134.
17. Grenier B, Loureiro-Bracarense AP, Lucioli J, Pacheco GD, Cossalter AM, Moll WD, Schatzmayr G, Oswald IP (2011).

Individual and combined effects of subclinical doses of deoxynivalenol and fumonisins in piglets. Mol. Nutr. Food Res. 55: 761–771.

18. Haschek WM, Voss KA, Beasley VR (2002). Selected mycotoxins affecting animal and human health. 2 ed. New York: Academic Press. 1(1): 645–699.
19. Hassan MA, Howayda MES, Noha HO, Maha MEK, Ragaa AS (2012). Efficiencyof use of yeast extract and acetic acid in detoxifications of aflatoxin contaminated chicken feeds. New York Sci. J .5(6): 66-72.
20. Huff WE, Kubena LF, Harvey RB, Corrier DE, Mollenhauer HH (1986). Progression of aflatoxicosis in broiler chickens. Poult. Sci. 65: 1891-1899.
21. Humphrey BD, Klasing KC (2004). Modulation of nutrient metabolism and homeostasis by the immune system. World Poul. Sci. J. 60(1): 90–100.
22. Hussein HS, Brasel JM (2001). Toxicity, metabolism, and impact of mycotoxins on humans and animals. Toxicol. 167(2): 101-134.
23. Huwig A, Freimund S, Käppeli O, Dutler H (2001). Mycotoxin detoxication of animal feed by different adsorbents. Toxicol. Lett.122: 179-188.
24. Jalili M, Jinap S, Son R (2011). The effect of chemical treatment on reduction of aflatoxins and ochratoxin A in black and white pepper during washing. Food Addit. Contam. 28(4): 485- 493.
25. Kaki S, Moeini MM, Cheraghi J (2012). Effects of zeolite and mycosorb on serum biochemical and hematological parameters of broilers chicken aflatoxicosis. J. Blood Lymph. 2(2): 1-4.
26. Khanedar F, Vakili R, Zakizadeh S (2012). Effects of two kinds of bentonite on the performance, blood biochemical parameters, carcass characteristics and tibia ash of broiler chicks. Glob. Vet. 9(6): 720-725.
27. Klasing KC(2007). Nutrition and the immune system. Br. Poult. Sci. 48(1): 525–537.
28. Kolossova A, Stroka J (2009). Evaluation of the effect of mycotoxin binders in animal feed on the analytical performance of standardised methods for the determination of mycotoxins in feed. 29(12): 1959-1971.
29. Liu YL, Meng GQ, Wang HR, Zhu HL, Hou YQ, Wang WJ, Ding BY (2011). Effect of three mycotoxin

adsorbents on growth performance, nutrient retention and meat quality in broilers fed on mould-contaminated feed. Bri. Poult. Sci. 52. 255-63.

30. Lutfullah G, Arshad H (2012). Studies on contamination level of aflatoxins in some cereals and beans of Pakistan. Food Control. 23(1): 32-36.
31. Magnoli AP, Tallone L, Rosa RCA, Dalcero AM, Chiacchiera SM, Sanchez RMT (2008). Structural characteristic of commercial bentonites used as detoxifier of broiler feed contamination with aflatoxin. Appl. Clay Sci. 40: 63-71.
32. Mahesh BK, Devegowda G (1996). Ability of aflatoxin binders to bind aflatoxin in contaminated poultry feeds- an in vitro study. In: XX World's Poultry Congress. 4: 296.
33. Maurice OM (2002). Risk assessment for afatoxins in foodstuffs. Int. Biodeterior. Biodegrad. 50: 137-142.
34. Mumtaz S, Sheikh MA, Iqbal T, Rehman KU, Rashid (2000). Bioconversion of distillery sludge (treated) to lysin and its biological evaluation. Int. J. Agric. Biol. 2(2): 274-277.
35. Musleh MH, Al-Ouqaili MT, Al-Kubaisi SM (2017). A Molecular Study of Aflatoxin and its Toxic Effect on Hematopoietic Stem Cells (Doctoral dissertation, M. Sc. Thesis, Department of Biology, College of Education for Pure Sciences, University of Anbar, doi: 10.13140/RG. 2.2. 33673.54889/1).
36. Park DL, Price WD (2001). Reduction of aflatoxin hazards using ammoniation. In: Rev. Environ. Contam. Toxicol. Afla. Red. 171(1): 139-175.
37. Pasha TN, Mahmood A, Khattak FM, Jabbar MA, Khan AD (2008). The effect of feed supplemented with different sodium bentonite treatments on broiler performance. Turkish J. Vet. Anim. Sci. 32(4): 245-248.
38. Phillips TD, Sarr AB, Clement BA, Kubena LF, Harvey RB (1990). Prevention of aflatoxicosis in farm animals via selective chemiosorption of aflatoxin. 1(1): 223-237.
39. Pitt JI, Hocking AD, Diane A (1997). Fungi and food spoilage. New South Wales, Australia: Springer.
40. Quist CF, Bounous DI, Kilburn JV, Nettles VF, Wyatt RD (2000). The

effects of dietary aflatoxin on wild turkey poults. J. Wildl. Dis. 36(3): 436-444.

41. Rajani, P,Sridevi V, Lakshmi MVVC (2012). A review on biological control of aflatoxin crop contamination. Int. J. Chem. Environ. Pharm Res. 3(1): 83-86.
42. Ramos AJ, Hernández E (1996). In vitro aflatoxin adsorption by means of a montmorillonited silicate. A study of adsorption of isotherms. Anim. Feed Sci. Technol. 62(2): 263-269.
43. Rawal S, Ji EK, Roger C (2010). Aflatoxin B1in poultry: Toxicology, metabolism and prevention. Res. Vet. Sci. 89(3): 325-331.
44. Richard JL (2007). Some major mycotoxins and their mycotoxicoses—An overview. Int. J. Food. Mircobiol. 119(1-2): 3-10.
45. Safaeikatouli M, Jafariahangari Y, Baharlouei A (2011). An evaluation on the effects of dietary kaolin and zeolite on broilers blood parameters, T4, TSH and growth hormones. Pak. J. Nutr. 10: 233-237.
46. Sari L, Marija MS, Marijana BS, Dragana VP, Abarkapa IS (2008). Effect of the baker′s yeast on the production of aflatoxin B1 by *Aspergillus flavus*. Food Process. Qual.Saf.35(4): 165-168.
47. Schiedeler SE (1993). Effects of various types of aluminosilicates and aflatoxin B1 on aflatoxin toxicity, chick performance, and mineral status. Poult. Sci. 72(2): 282-288.
48. Sean PA (2002). Mycotoxins and in-door molds. Indoor Envir. Connect. 3(4): 14-24.
49. Shah HU, Simpson TJ, Alam S, Khattak KF, Perveen S (2010). Mould incidence and mycotoxin contamination in maize kernels from Swat valley, North West Frontier province of Pakistan. Food Chem. Toxicol. 48(4): 1111–1116.
50. Shareef AM(2010). Molds and mycotoxins in poultry feeds from farms of potential mycotoxicosis. Iraqi J. Vet. Sci. 24(1): 17-25.
51. Southern LL, Ward TL, Bidner TD, Hebert LG (1994). Effects of sodium bentonite or hydrated sodium calcium aluminosilicate on growth performance and tibia mineral concentrations in broiler chicks fed nutrient deficient diets. Poult. Sci. 73(6): 848-854.
52. Verma RJ(2004). Aflatoxin cause DNA damage. Int. J. Hum. Genet. 4(4): 231-236.

53. Wayne LB (2012). Mycotoxin contamination of the feed supply chain: Implications for animal productivity and feed security. Anim. Feed Sci. Technol. 173(1–2): 134-158.
54. Williams JH, Phillips TD, Jolly PE, Stiles JK, Jolly CM, Aggarwal D (2004). Human aflatoxicosis in developing countries: a review of toxicology, exposure, potential health consequences, and interventions. Am. J. Clin. Nutr. 80(5): 1106-1122.
55. Wilson DM, Payne GA (1994): Factors affecting Aspergillus flavus group infection and aflatoxin contamination of crops. Academic Press, San Diego.
56. Wu F, Clare M, Marites T, Yan L (2011). The health economics of aflatoxin: Global burden of disease.
57. Yiannikouris A, Jouany JP (2002). Mycotoxins in feeds and their fate in animals: a review. Anim. Res. 51(2): 81-99.
58. Yu JH, Keller N(2005). Regulation of secondary metabolism in filamentous fungi. Annu. Rev. Phytopathol.43: 437–458.
59. Zaki MM, El-Midany SA, Shaheen HM, Rizzi L (2012). Mycotoxins in animals: Occurrence, effects, prevention and management. J. Toxicol. Environ. Health Sci. 4(1): 13-28.
60. Zhao J, Shirley RB, Dibner JD, Uraizee F, Officer M, Kitchell M, Vazquez AM, Knight CD (2010). Comparison of hydrated sodium calcium aluminosilicate and yeast cell wall on counter acting aflatoxicosis in broiler chicks. Poult. Sci. 89(10): 2147-2156.