Indian J. Anim. Nutr. 2022. 39 (3): 221-234
doi: 10.5958/2231-6744.2022.00028.7

Indian Journal of Animal Nutrition

Aflatoxins in Duck Production-A Review

S. Banerjee[1], R. Behera[2], S. Panda[1], G. R. Jena[1], D. Kumar[2]*, P. K. Naik[2], B. K. Swain[2], S. K. Mishra[2] and C. K. Beura[2]

College of Veterinary Science, Odisha University of Agriculture & Technology
Bhubaneswar, Odisha-751003, India
[1]CVSc&AH, OUAT, Bhubaneswar
[2]ICAR-DPR Regional Station, Bhubaneswar
*Correspondence: dhiruvet@gmail.com

ABSTRACT

Aflatoxins are fungal toxins produced by fungi Aspergillus, mainly *A. flavus*, *A. parasiticus* and *A. nomius*. The toxin includes B1, B2, G1, G2, M1, and M2 of which B1 (AFB1) is the most carcinogenic and hazardous to animals, birds and human beings having high potential to cause aflatoxicosis. The primary exposure to AFB1 occurs via contaminated feed. High temperature, humidity in fields, improper storage and presence of broken grain facilitates mycotoxins accumulation in feed. Liver is the prime site of metabolism where AFB1 is converted by hepatic cytochromes P450 (CYP) enzymes into the reactive and electrophilic exo-AFB1-8, 9 epoxide (AFBO). AFBO binds to guanine residues resulting in DNA mutations and hepatic cancer. Ducks are unable to metabolize aflatoxins efficiently and hence considered to be highly susceptible for aflatoxicosis. Median lethal dose for ducklings is 0.34 to 0.56 mg/kg body weight compared with 6.5 to 16.5 mg/kg body weight for chicks. Aflatoxins negatively affect growth and production in ducks. Many researchers have documented elevated serum AST, ALT, ALP, and GGT indicating hepatocyte damage in AFB1 intoxicated ducks. Aflatoxins causes granular degradation, cytoplasm vacuolation and fatty dystrophy of liver, negatively affects kidney function and suppresses the alternative complement activation pathway, suppressing immune system of ducks. Therefore, it is highly necessary to adopt strategies like proper storage of feed ingredients in cool and dry conditions to check fungal contamination, supplementation of antioxidants viz; vitamin E, selenium, beta-carotene, lycopene etc. in feed and use of mycotoxin binders for minimizing the toxin effect.

KEYWORDS: Aflatoxins, Duck, Mycotoxins

Article received : 04 August 2022; Article accepted: 10 September 2022

INTRODUCTION

Aflatoxins are classes of mycotoxins (fungal toxins), causing toxicity in human and animals. Two main groups of Mycotoxins, which affect public and animal health are Aspergillus and Fumonisins, mainly produced by *Fusarium verticillioides* and *Fusarium proliferatum*. Among all the mycotoxins, the most thoroughly studied is aflatoxins (Ramos et al., 1996). Aflatoxins are produced by fungi Aspergillus, mainly *A. flavus, A. parasiticus* and *A. nomius* (Moss et al., 1998). Aflatoxins are difuranocoumarin compounds and include B1, B2, G1, G2, M1, and M2 (Bilgrami and Chaudhary, 1998). Aflatoxin B1 (AFB1) is the most carcinogenic aflatoxin among the aflatoxin family and is hazardous to both animals and human beings having high potential to cause aflatoxicosis (Leeson et al., 1995; Limaye et al., 2018). International Agency for Research on Cancer (IARC) has classified it as class 1A carcinogen (IARC-WHO, 2002; Wu et al., 2021). Aflatoxin naturally contaminates cereal grains and animal feeds around the world (Monbaliu et al., 2010; Streit et al., 2012). Maize, wheat, soybean, rice, sorghum, cotton seed, sunflower seeds and animal feeds can naturally be contaminated by aflatoxin Â1, Â2, G1 and G2 (Cole and Richard, 1989; Robens and Richard, 1992; Martinez et al., 2010). Consumption of contaminated feed is the primary source of aflatoxicosis. High temperature

and humidity in fields, inappropriate storage and presence of broken grain facilitates mycotoxin accumulation in grains (Binder et al., 2007). Optimal conditions for growth of fungi temperature between 12–42 °Ñ and humidity 18% and 9–10%, respectively (WHO, 1979). Although, ducks are among the most sensitive species to mycotoxin contamination, the toxicity of aflatoxin in ducks is not much studied as chickens. On the other hand, the global production of duck meat and eggs is rising in the recent years, thus, it is necessary to invest more resources to investigate the harmful effects of aflatoxin in ducks, especially ducklings.

MATERIALS AND METHODS

AFB1 becomes toxic only after its bio-activation and this process mostly occurs in hepatocytes (Eaton et al., 1995; Bedard and Massey, 2006). AFB1 is initially absorbed in the duodenum (Gratz et al., 2005). Bio-activating enzymes for AFB1 are present in the small intestine (Guengerich et al., 1996). The majority of the toxin is metabolized in the liver, where AFB1 is converted by hepatic cytochromes P450 (CYP) enzymes into the reactive and electrophilic exo-AFB1-8, 9epoxide (AFBO) (Eaton et al., 1995; Bedard and Massey, 2006; Coulombe, 1993; Cullen et al., 1993). AFBO reacts with macromolecules of the cells including proteins and DNA, thus causing cyto-toxicity and genotoxicity (Doi et al., 2002). AFBO binds to guanine residues in nucleic acids leading to DNA mutations and liver cancer in humans, primates and ducks (Eaton and Gallagher, 1994; Verma, 2004; Do and Choi, 2007). However, there is limited evidence of carcinogenicity of aflatoxin B2 (AFB2). An endo stereoisomer of the AFBO epoxide which is far less toxic can also be produced but it is not relevant to AFB1 toxicity (Eaton et al., 1995).

Species susceptibility of Aflatoxins

The existence of specific CYP450 enzymes those able to bio transform AFB1 to AFBO coupled with poor conjugation ability with glutathione is regarded as an important factor in AFB1 sensitivity of a species (Guengerich et al., 1996; Gallagher et al., 1996). The highly susceptible species are rabbits and ducks (oral LD50 0.3 and 0.36 mg/kg body weight (BW), respectively while chickens (oral LD50 6.5 mg/kg BW) are relatively resistant to AFB1 (Pier et al., 1992). Ducks are considered as the most sensitive species of domestic fowls to acute aflatoxicosis. The median lethal dose for ducklings is 0.34 to 0.56 mg/kg body weight compared with 6.5 to 16.5 mg/kg body weight for chicks (Leeson et al., 1995). Ducks are unable to metabolize aflatoxins efficiently and hence considered to be highly susceptible (Dalvi, 1986). Ducks are also the only poultry species that develop hepatic tumors from chronic or sub-chronic aflatoxin exposure, an effect that is also seen in rats, primates and humans (Leeson et al., 1995). A study conducted by Diaz et al. (2010) showed cytochrome (CYP) P450 enzymes are responsible for the toxic effect of aflatoxin. The finding revealed that four CYP enzymes may be involved in AFB1 bioactivation in ducks and this explains the high sensitivity of this species to AFB1. The sensitivity of different fowl to AFB1from high to low order was ducklings > turkey > gosling > quail >chicks (Wu et al., 2021). It has been estimated that toxic levels of aflatoxin in domestic ducklings have been reported from 0.3 to 0.6 mg/kg (Edds, 1973). Ducks are about 200 times more sensitive than chickens because ducks have a different mycotoxin metabolism than other species. Ducks have a greater bioactivation activity of aflatoxin and less potential for detoxification and elimination of aflatoxin (Soriano, 2020) owing to CYPs producing excessive AFBO and AFL and inadequate detoxifying enzymes -glutathione S-transferases GSTs to metabolize detrimental AFBO. In contrast, chickens are capable of converting maximum AFB1 into AFL, thus minimizing the production of harmful AFBO, thus chickens are fairly resistant to AFB1 (Wu et al., 2021). Duck liver microsomes need only 50 seconds to process AFB1 LD50 concentration, in contrast chickens require 32 to100 minutes for the same (Patterson, 1973).

Toxic effects of Aflatoxins

Aflatoxin is an amino alcohol group of mycotoxins. It is hepatotoxic in nature and, thus, affects all liver functions viz; metabolism of nutrients

in the diet, as well as protein synthesis and immune effectors. Therefore, affected animals may suffer a decrease in productivity and immunosuppression (Soriano, 2020). Aflatoxin B1 (AFB1) is detrimental to both humans and animals because of its diverse toxicity that causes weight loss, immune-suppression, mutagenesis, reproductive alterations and carcinogenesis (Zychowski et al., 2013). In domestic fowls, aflatoxins induce numerous biological effects, such as liver diseases, alterations in growth rates, changes in the mechanism of immunogenesis, and carcinogenic and mutagenic effects (Pier, 1973). Aflatoxicosis in ducks caused delayed development, hyperkeratosis of cornea and oral mucosa, bone deformity and fragility, inflammatory edema of eyelids, paralysis of leg, dermatitis, poor feathering and fatty, pale, enlarged and friable liver (Singh et al., 2019). Wan et al. (2013) observed AFB1 contaminated diet increased bill decolourization (bill decolourization ratio % was 8.13 in control vs 40.10 in 25%, 50.80 in 50% and 76.52 in 100% AFB1 contaminated diet group). Mishra et al. (2021) reported that Pekin ducklings receiving 200 and 400 ppb AFB1 developed lameness across the hock joints. The lame ducklings continued to exhibit slower growth, lack of agility and deformities which remained irreversible even after therapeutic interventions.

Effect of Aflatoxins on growth and production

Presence of aflatoxin in the feed negatively affects growth in ducks. Diets containing 50 micrograms/kg aflatoxin B1 equivalent or more caused significant reduction in body weight gain and reduction in utilization of dietary protein in Alabio ducks as compared with White Leghorn chickens. With increasing level of aflatoxin content above 50 micrograms/kg, greater difference in performance between ducks and chicken were observed (Ostrowski-Meissner, 1983). Aflatoxin concentrations above 60 ppb reduced duck growth and at 120 ppb, weight gain was only 52.9% in comparison to an uncontaminated diet (Bintvihok, 2011). Wan et al. (2013) reported a linear decrease in the average daily gain with increased concentration of AFB1 in duck diet (66.61 g in control vs 65.85g in 25%, 64.72 g in 50% and 64.13 g in 100% aflatoxin contaminated diet).

Feeding of aflatoxin contaminated maize diet to ducks decreased day 7 body weights, average daily gain (ADG) and average daily feed intake (ADFI) by 22, 31 and 23%, respectively while there was 11% increase in FCR-feed conversion ratio. From day 8 to 14, birds fed AFB1 diets showed significantly ($p<0.05$) lower ADG, ADFI and FCR by 41%, 47% and 10%, respectively (Abbasi et al., 2018). There was a decrease in ADG (79.35g vs. 66.05g) and ADFI (164.92 g vs.107.56g) in ducks fed with maize naturally polluted with AFB1 (Feng et al., 2017). Liu et al. (2017) observed a reduction in FCR in the aflatoxin treated group than that of the control in male Cherry Valley ducklings at week 1 and 2. Valchev et al. (2013) documented 17.3% and 26.97% decrease in body weight of ducks fed with aflatoxin intoxicated diet @0.5mg AFB1/kg feed and 0.8 mg AFB1/kg feed, respectively and an increased FCR by 12.74%. Aflatoxins delayed growth of Cherry Valley male ducks and development of skeletal muscle (Chang et al., 2016). Ali et al. (2019a) documented linear decrease in mean body weight of ducks comparison to control group with rise in AFB1 dose (8th week BW in control 1908.33 ± 70.68 g vs 1810.83 ± 105.2, 1773.33 ±179.62, 1760 ±148.23 and 1631.67 ± 86.33 g in ducks receiving 6, 12, 24 and 48 ppb AFB1, respectively).

Similar trend of decreased feed intake, body weight gain, increased feed to gain ratio and selected organ weights (liver, kidney and pancreas) in AFB1-treated groups in day-old Cherry Valley ducks fed with diets containing different levels of AFB1contaminated rice for 6 weeks was reported by Han et al. (2008). Chen et al. (2014a) documented notable decrease in cumulative body weight gain (0-14 day) in white Pekin ducklings 728g/duck in control vs. 648 g, 499 g and 380g per duck in AFB1 intoxicated @0.11, 0.14 and 0.21g/kg feed, respectively. However, feed efficiency was not affected. Increasing concentrations of AFB1 reduced cumulative body weight gain and feed intake both linearly and quadratically. Regression equation

revealed that for every 0.1-mg/kg raise in AFB1 in diet, there were around 230 and 163g decrease per bird in cumulative feed intake and body weight gain, respectively. Grenier and Applegate, (2013) observed reduced digestibility of crude protein in ducks by 8-13% in aflatoxin contaminated feed at level of 20- 40 ppb.

Dietary treatment had no significant effect on egg production of laying duck (duck days average). The egg shell thickness (0.48±0.07 vs. 0.46±0.07) and the percentage of yolk weight (31.9±1.15 vs. 31.5±1.68) were slightly lower in duck received AFB1 diet@ 100ppb (Sumantri et al., 2019a). Aflatoxin contamination in the diets during early growth did not really affect characteristics of early laying of Mojosari and Tegal ducks, however, affected live weight of ducks at age at first laying (Prasetyo and Sushanti, 2010). AFB1 exposure at 70 ppb significantly ($P<0.05$) decreased the body weight of laying ducks by 0.87% while treatments had no significant effect on egg production and egg weight (Sumantri et al., 2019b).

Effect of Aflatoxins on haematology

In ducks, besides reduction in growth and utilization of protein, dietary aflatoxin caused liver damage and significantly affected most of the blood constituents, blood clotting time and De Riti's ratio in ducks fed with aflatoxin contaminated ground nut meal (Ostrowski-Meissner, 1983). Significant decrease in hematological values of hemoglobin, PCV and TLC was reported in Pekin duck groups fed with aflatoxin (12ppb, 24ppb and 48ppb) with significantly lower value in 48 ppb group. Haemoglobin (Hb g/dl) value of 13.316 vs. 9.717 and 9.633 was reported in control, AFB1 24 ppb and 48 ppb group, respectively. MCV, MCHC and MCH reduced significantly in all aflatoxin intoxicated ducks (MCV138.49 vs 90.99 and 89.77 fl in control, 24 ppb and 48 ppb group). Toxic level of AFB1 in white Pekin duck was confirmed at 48 ppb (Ali et al., 2019a). Effect of aflatoxin in duck feed articulated as fall in red blood cell counts, haemoglobin content, haematocrit value, platelet counts and rise in white blood cell counts. The percentages of the differential leukocyte counts expressed as increased percentage of heterophils and lower proportion of lymphocytes (Valchev et al., 2018). Decreased blood T3 and T4 value were noted in aflatoxin intoxicated duck groups (Valchev et al., 2014). Prolonged exposure to mild doses of aflatoxin in humans and livestock (rats, primate, and ducks) causes hepatocellular carcinoma, which is one of the most harmful type of cancer diseases (Benkerroum, 2020; Diaz and Murcia, 2011).

Effect of Aflatoxins on blood biochemical profile

Han et al. (2008) recorded significant increase in the activities of serum ALT and AST in AFB1-contamined groups. Chen et al. (2014a) found an elevated serum aspartate amino transferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP) and Gamma-glutamyl transferase (GGT) indicating hepatocyte damage in aflatoxin B1 intoxicated White Pekin ducklings. Apparent digestibility of crude protein (CP) was significantly lesser though activities of digestive enzyme like protease, trypsin, chymotrypsin and amylase were enhanced in AFB1-intoxicated group. Feeding AFB1, reduced serum glucose, creatinine, albumin, total protein, globulin, Ca, P and creatine phosphokinase linearly, while serum urea, Na, Cl, alkaline phosphatase, and aspartate amino transferase concentrations increased linearly with increasing AFB1 concentration in feed. AFB1 at very low concentrations can significantly impair liver function in Pekin ducklings. Enzymes related to liver function like ALT, AST and GGT and kidney function like blood urea nitrogen (BUN), creatinine exhibited rising trend with higher level of aflatoxin with drop in serum protein signifying liver and kidney dysfunction (El-Sheshtawy et al., 2021). There was a gradual fall of serum total protein from control (8.58 ±0.149) to ducks receiving AFB1@ 6ppb (7.06 ±0.312), 12ppb (7.277±0.107) followed by 24ppb (5.60±0.214) and 48ppb (5.51±0.532) group. Triglycerides (mg/dl) value showed a significant increase in group 24ppb (284.945 ±10.29) and 48ppb (338.625±17.983) than control group 196.44 ±8.56. Enzymes related to liver function like ALT (Control 23.77±0.289; 24 ppb 24.61±0.21; 48

ppb 26.01±0.285), AST (Control 65.2±1.559; 24 ppb 71.1875±3.154; 48 ppb 82.07±2.5124) and GGT (Control, 2.42±0.147; 24 ppb, 3.08±0.414; 48 ppb, 3.33±0.2102) and kidney function like blood urea nitrogen (Control, 2.6375±0.1261; 24 ppb, 3.735±0.554; 48 ppb, 4.82±0.339) creatinine (Control, 0.224±0.0132; 24 ppb, 0.316±0.0063; 48 ppb, 0.333±0.039) exhibited rising trend with higher level of aflatoxin (Ali et al.,2019a). Similar trend of reduced concentrations of biochemical parameters viz; urea, creatinine, uric acid, calcium, inorganic phosphate, sodium and potassium and higher creatine kinase activity was noted in aflatoxin intoxicated groups. (Grozeva et al., 2012). There was a linear decrease in serum albumin and globulin percent with increase in AFB1 in diet of ducks. Serum albumin % was 17.30 in control diet which decreased to 16.51% in 25% AFB1 contaminated diet; 16.10 and 15.97% in 50% and 100% AFB1 contaminated diet, respectively. Similarly, serum globulin was 25.17% in control group which decreased to 24.09, 22.45 and 21.70% in 25%, 50% and 100% AFB1 contaminated diet group, respectively (Wan et al., 2013).

Effect of Aflatoxins on duck mortality

Significant increase in mortality percent was reported by Wan et al., (2013) when Cherry Valley ducks were fed AFB1contaminated corn diet (0% mortality in control vs 1.88% in 50% and 3.75% in 100% contaminated diet. Abassi et al. (2018) reported an increased mortality rate in ducks fed with AFB1 contaminated diet (0% in control vs 5.80% in AFB1 treatment group). 31.25% increase in mortality was observed in ducks fed with naturally AFB1contaminated maize diet (Feng et al., 2017). Mishra et al. (2021) reported a mortality of 45% and 85% in ducklings when fed with a diet with 200 ppb and 400 ppb AFB1, respectively.

Effect of Aflatoxins on histopathology

While in chicken it is common to find lesions in gizzard and intestines due to mycotoxin intoxication, whereas in ducks such lesions are atypical. Liver, heart and immune system are the principal target organs for mycotoxins in ducks. Aflatoxin treated ducklings had increased relative weights of the liver, kidneys, and spleen, and decreased relative weight of bursa of fabricius after 2 wk of feeding (Liu et al., 2017). Han et al. (2008) reported that the pancreas sizes of ducks given diets containing 20 and 40 ppb aflatoxin were bigger than those fed a control diet. The increased relative weight of pancreases of ducks might be as a result of the increased mature crystalline granules in the cells of pancreas (Fouad et al., 2019). A study examining the effects of AFB1 on liver lesions in poultry reported that ducks generated hepatic lesions on alternate days when fed 15 µg AFB1 (Anbiah et al., 2004). Ducklings fed a ration containing AFB1 at 0.5 mg/kg feed exhibited an enlarged liver, brown-yellowish in colour, with striated and petechial haemorrhages while ducks fed with 0.8mg AFB1/ kg feed resulted in strong dilation of capillaries, pericapillary edema and Kupffer cells activation. Hepatocytes exhibited granular degradation and cytoplasm vacuolation, karyorrhexis, karyopyknosis and initial fatty dystrophy. Ducklings treated with 0.8 mg AFB1/kg feed exhibited more intense liver changes with predomination of fatty dystrophy. Morphological changes in kidney parenchyma observed as congestion, degeneration of renal tubules, desquamation and disintegration of tubular cells with pyknotic changes, necrobiotic changes and hemorrhages based upon the quantity of ingested toxin (Grozeva et al., 2012).

Wan et al. (2013) observed significant ($P<0.05$) decrease in relative weights of liver (3.525 in control vs 2.919 in 100% AFB1 contaminated diet), spleen (0.102 vs 0.087), thymus (0.408 vs 0.339), bursa fabricus (0.15 vs 0.132) and various organs (liver, spleen, thymus, bursa of Fabricius). However, Feng et al. (2017) recorded an increased relative weight of liver in ducks fed with maize diet naturally contaminated with AFB1 (3.19 control vs. 4.70). relative weights of liver, kidneys, heart, pancreas, gizzard and proventriculus were higher while the relative weight of the thymus, bursa of Fabricius and the spleen were considerably lower (Valchev et al., 2013).

Notable histopathological lesions in liver, kideney and heart were evident in Pekin ducks fed with 200 and 400 ppb AFB1. Liver was enlarged with congestion surrounded with pale-patches, loss of liver architecture with cloudy swelling and hepatocyte necrosis in 200 ppb AFB1 group while in case of 400 ppb fed ducks liver showed notable loss of architecture, necrosis with profuse RBC infiltration. Kidney showed enlargement across all the specimens with paleness evident in most samples. In ducks fed with 200 ppb AFB1, the spleen showed mild congestion with few nuclear debris and vacuoles; whereas in 400 ppb group spleen showed massive congestion of red pulp with more nuclear debris and vacuoles (Mishra et al., 2021). Mondal et al., (2018) observed pathological lesions in liver and kidney of ducks died due to aflatoxicosis owing to presence of 20-30 ppb aflatoxin in feed. The lesions included were- hemorrhages, fatty infiltration and necrotic patches, white calcified spots on liver in some cases, and yellowish, mottled liver. Kidney was enlarged with white calcified spots, pale and white spotted in colour. Liver of ducks died from aflatoxicosis fed with 48 ppbAFB1 expressed vacuolar degeneration of cells with mild sinusoidal congestion and focal necrotic patches. Tubular degeneration, interstitial congestion observed in kidney. Lymphoid depletion was apparent in bursa of Fabricius and thymus (Ali et al., 2019b).

Effect of Aflatoxins on immunology

The avian immune system comprises of bursa of fabricius, thymus and spleen to produce mature leukocytes. At low dietary concentrations, AFB1 can damage these immune tissues and suppress innate and adaptive immune responses at the cellular level (Coulombe, 1993; Leeson et al. 1995; Hoerr et al., 2010). Pier, (1992) stated that the immune system is sensitive to aflatoxin and observed lower complement production and reduction in the phagocytic activity. Aflatoxin is included in the amino alcohol group of mycotoxins. It is hepatotoxic and, therefore, affects all liver functions, such as the metabolism of nutrients in the diet, as well as protein synthesis and immune effectors. Therefore, affected animals may suffer a decrease in productivity and immunosuppression (Soriano, 2020). Prolonged exposure to mild doses of aflatoxin in primate, rats and ducks can cause hepatocellular carcinoma- one of the most dreaded cancer diseases (Benkerroum, 2020; Diaz and Murcia, 2011). Feeding aflatoxin to ducks via feeds adversely impacted the natural immunity by suppressing the alternative pathway of complement activation. Complement activity declined from 727.99 CH50 to 625.91 CH50 in ducks receiving 0.5 mg AFB1/kg feed and from 816.74 CH50 to 601.61 CH50 in ducks receiving 0.8 mg AFB1/kg feed (Valchev et al., 2015). Feeding of 0.11 to 0.21 mg of AFB1/kg impaired classical and alternative complement pathways in the duckling serum (Chen et al. 2014b). Number of viable cells picked up from the thymus was remarkably lower in aflatoxin-challenged birds. Birds challenged with aflatoxin also expressed remarkably lower E-coli O55 lipopolysaccharide induced mitogenic responses. In mallard ducklings, oral exposure to aflatoxin even in subacute dose lowered normal lymphocyte reactivity. Hence, waterfowl that consume even mild amount of mycotoxin-polluted waste grain are more prone for bacterial or viral diseases (Hurley et al., 1999). Guo et al. (2012) found a considerable decrease in the bursa of Fabricius, thymus indices and body weights in aflatoxin B1 treated group as compared to the control group. In addition, the spleen indexes reduced strikingly. AFB1 significantly affected growth and immune organs development of the ducklings.

Effect of Aflatoxins on oxidative stress

Oxidative stress is defined as a disturbance in the balance between antioxidants and prooxidants, with increased levels of prooxidants causing potential damage to body. This imbalance can be due to the decrease of endogenous antioxidants, low intake of dietary antioxidants or increased production of free radicals and other reactive species like reactive oxygen species (ROS). Metabolizing AFB1 increases the production of free radicals and lipid peroxides leading to cellular damage. Also, AFB1 intoxication led to a significant increase in superoxide dismutase (SOD) activity as observed

in Pekin ducks treated with AFB1(Barraud et al., 2001). The main way of AFB1 detoxification is through the AFBO conjugation with glutathione (GSH). AFB1 can cause an increase in ROS formation in target organs especially duck liver (Shen et al., 1996; Barraud et al., 2001; Guindon et al., 2007). Extremely high levels of AFB1-DNA adduct and AFB1-albumin adducts were detected in duck liver and serum, respectively as compared to other animal species exposed to a similar AFB1 dose (Barraud et al., 2001). The mechanism of AFB1-induced cytotoxicity may be due to the release of free radicals, which initiates lipid peroxidation and further damage the tissue (Shen et al., 1994). Towner et al. (2003) stated that there is direct evidence of the involvement of free radicals in AFB1 toxicity. It is proved that oxidative stress can play an important role in the initiation of carcinogenesis through DNA damage (Visioli et al., 2004). This may be one of the mechanisms responsible for AFB1-induced carcinogenesis. According to Bernabucci et al. (2011) mycotoxins have high potential cytotoxic effect and they act as secondary metabolites. The possible mechanism of cytotoxicity has been postulated by the induction of oxidative stress.

Mitigation strategies for Aflatoxicosis in ducks

As discussed above, aflatoxin can cause a huge loss to duck sector by lowering the production, immunity, overall health, increased mortality and cost of production. The presence of aflatoxin and other mycotoxins in the environment and commodities is inescapable therefore, it is important to formulate strategies to avoid their occurrence in the food chain to prevent contaminated commodities from entering food chain and feed processing units.

- The first step of prevention starts in the field where cereal is being harvested. Several strategies like use of mould resistant plants, crop rotation, maintaining the recommended row and intra-plant spacing to avoid overcrowding of the plants, weed control, proper use of insecticides and fungicides, use of scarecrow to keep birds away from field can be helpful (Singh et al., 2019)
- Use of clean and dry equipment for collecting and transporting the harvested grain.
- Soon after the harvest, grains should be dried to the recommended moisture level for safe storage of the concerned crop.
- Reduce the quantity of foreign materials and damaged kernels to the minimum
- Keep the storage area dry and protected from rain and ground water.
- Protect the stored crops from rodents and birds.
- Fungi are aerobic in nature. Thus, lower or nil concentrations of oxygen and high carbon dioxide will check aflatoxin production on stored grains.
- Judiciously use fungistats or mould inhibitors
- Control environmental factors that influence fungal growth:
    - Reduce moisture content of grain and feeds below13%
    - Reduce relative humidity of grain and feeds below70%
    - Reduce storage temperature of grain and feeds below20°C
    - Reduce oxygen availability during storage below 0.5% (Singh et al., 2019)

Minimizing harmful effects of Aflatoxins

Several researchers have documented different ways of minimizing the harmful effects of aflatoxin in ducks. Addition of glucomannan –yeast cell wall extract acts as aflatoxin adsorbent owing to its large porous surface to trap the toxins physically. When diet of ducks was intoxicated with 120 ppb aflatoxin, body weight gain reduced to 52.9% of the control. But, when glucomannan was included in the diet recovered growth rate was 67.4% of the control (Bintvihok, 2011). Curcumin supplementation in diet exhibited positive effects on the laying duck performance fed with AFB1-contaminated feed (Sumantri et al., 2019a). Feeding of aloe vera extract can be helpful in relieving the influence of the

aflatoxin on laying chicken (Mojaher et al., 2021). Several researchers have advocated that the detrimental effects of aflatoxin can be lessened by the inclusion of organic mycotoxin binders such as yeast, probiotics, antioxidant (Nalle et al., 2019; Fouad et al., 2019; Wade et al., 2018; Wade and Sapcota, 2017; Girish and Devegowda, 2006; Afzal and Zahid, 2004) and inorganic mycotoxin binder like sodium bentonite, clinoptilolite, and cyclopiazonic acid (Barati et al., 2017; Magnoli et al., 2011; Kumar and Balachandran, 2009; Ortatatli et al., 2005). Yet, the efficacy of each type of mycotoxin binder depends on the level of aflatoxin and species of fowl. The addition of 2 g/kg toxin binder mycotox NG to the feed had not significant protecting effect against the adverse effects of aflatoxin B1 on blood triiodothyronine and thyroxine concentrations (Valchev et al., 2014). Supplementation of Mycotox NG in diet decreased the severity of haematological changes and severity and frequency of histological lesions (Valchev, 2013). The addition of a Silicoglycidol-based mycotoxin binder allowed to get superior results in ducks fed a diet polluted with mycotoxins. The ducks those received the mycotoxin binder exhibited improved weight (+66 g/bird), superior feed conversion ratio (- 1.06%), and lesser relative weight of liver (- 4.72%) indicating a better status and efficiency of liver (Soriano, 2020).

Augmenting glutathione precursor amino acids like methionine and cystine levels in the diet can help in alleviating negative effects of aflatoxin. Glutathione produces conjugated complex with AFB1 in the liver and the complexes get eliminated via feces and urine. Boosting the amount of protein and calorie in the diet and vitamins mainly riboflavin and D3 can facilitate detoxification of AFB1. In contrast, deficiency of thiamin exhibit a protecting effect against aflatoxicosis as its deficiency mobilize the lipid reserves thus interfering the metabolism of aflatoxins in liver (Singh et al., 2019). Amongst the vitamins, vitamin E promptly reacts with peroxide radicals and produces stable radicals - tocopheroxyl capable to form á-tocopherol by reacting with ascorbate (Jacob, 1995). Incorporation of vitamins A and E ameliorated aflatoxin induced changes and checked aflatoxin provoked carcinogenesis by anti-mutagenic effect (Nyandieka and Wakhisi, 1993; Gradelet et al., 1997; Verma and Nair, 2001). Lycopene and silymarin reversed the damaging effects of aflatoxin on liver and kidney tissue owing to their antioxidant activity (El-Sheshtawy et al., 2021). Use of sodium selenite in duck diet could significantly improve the harmful effect caused by AFB1 (Shi et al., 2012). Inclusion of 0.1% clay adsorbent can protect ducks from the harmful effects of AFB1-intoxicated corn in diets (Wan et al., 2013).

The feeds can be decontaminated using various methods mainly focus on physical removal or chemical toxins in the feeds. Moreover, dietary additives such as activated charcoal, phenobarbital, cysteine, glutathione, beta-carotene, fisetin and selenium have also been reported to be effective in reduction of aflatoxicosis in poultry (Dalvi, 1986). Currently, use of adsorbents like zeolite, in the feed is widely popular to check aflatoxin absorption in the gastrointestinal tract. This approach is relatively economic, usually considered as safe and easy to mix in ration (Singh, 2019). Addition of 2% of zeolite in diet reduced the negative effects of aflatoxin-B1 on performance of laying duck (Sumantri et al., 2019b).

CONCLUSIONS

Ducks are unable to metabolize aflatoxins efficiently and hence considered to be highly susceptible for aflatoxicosis. Aflatoxin can cause a huge loss to duck sector by lowering the production, immunity and increased mortality and the cost of production. Therefore, it is highly necessary to adopt strategies like proper storage of feed ingredients to check fungal contamination, supplementation of antioxidants like vitamin E, selenium, beta-carotene, lycopene etc. in feed and use of mycotoxin binders for minimizing the toxin effects.

REFERENCES

Abbasi, F., Liu, J., Zhang, H., Shen, X. and Luo X. 2018. Effects of feeding corn naturally contaminated with aflatoxin on growth

performance, apparent ileal digestibility, serum hormones levels and gene expression of Na+, K+ -ATPase in ducklings. Asian-Australasian Journal of Animal Sciences. 31: 91-97.

Afzal, M. and Zahid, S. 2004. Effects of Addition of a Mycotoxin Detoxifier in Poultry Feed Containing Different Levels of Aflatoxins on the Performance of Broilers. Asian-Australasian Journal of Animal Sciences. Doi: 10.5713/ajas.2004.990.

Ali, I., Panda, S.K., Pati, S., Mishra, S.K., Mohanty, G.P., Jena, G.R., Acharya, A.P., Sahoo, P.R. Mohanty, L., Mishra, N., Kumar, D. and Sarkar, A. 2019a. Growth performance and hemato-biochemical alterations in induced aflatoxicosis in white pekin ducks (*Anas platyrhynchos domesticus*). Journal of Entomology and Zoology Studies. 7(3): 1291-1295.

Ali, I., Panda, S.K., Pati, S., Acharya, A.P., Mishra, S.K., Jena, G.R., Mohanty, G.P., Mohanty, L., Das, S. and Kumar, D. 2019b. Patho-morphological and Genotoxic Changes in Induced Aflatoxicosis in White Pekin Ducks (*Anas platyrhynchos* domesticus). International Journal of Current Microbiology and Applied Sciences. 8(5): 1225-1233.

Anbiah, S., Mohan, C., Manohar, B., Balachandram, C. 2004. Chronic aûatoxin B-1 induced hepatopathy in ducks. Indian Veteterinary Journal. 81: 1210–1212.

Barati, M., Chamani, M., Mousavi, S.N., Hoseini, S.A. and. Ebrahimi, M.T.A. 2017. Effect of commercial toxin binder, native probiotic strains, cell wall yeast and aluminosilicate in diets contaminated with aflatoxin, on the expression of GOT2, CYP450 1A5 genes and serum concentrations of liver enzymes in broiler chickens. Kafkas Üniversitesi Veteriner Fakültesi Dergisi. 23(6): 953-960. DOI: 10.9775/kvfd.2017.18022.

Barraud, L.T., Douki, S., Guerret, M., Chevallier, C., Jamard, C., Trepo, C.P., Wild, J.C. and Cova. L. 2001. The role of duck hepatitis B virus and aflatoxin B1 in the induction of oxidative stress in the liver. Cancer Detection and Prevention. 25:192-201.

Benkerroum, N. 2020. Chronic and acute toxicities of aflatox-ins: mechanisms of action. International Journal of Environmental Research and Public Health. 17: 423.

Bedard, L.L. and Massey, T.E. 2006. Aflatoxin B1-induced DNA damage and its repair. Cancer Letters. 28: 174–183.

Bernabucci, U., Colavecchia, L., Paolo, P., Loredana, D., Lacetera, B.N., Nardone, A. and Ronchi B. 2011. Aflatoxin B_1 and fumonisin B_1 affect the oxidative status of bovine peripheral blood mononuclear cells. Toxicology *in Vitro*. 25(3): 684-691.

Bilgrami, K.S. and Choudhary, A.K. 1998. Mycotoxins in preharvest contamination of agricultural crops, In Mycotoxins in Agriculture and Food Safety, Sinha KK, Bhatnagar D. Eds.; Marcel Dekker: New York, NY, USA, pp. 1–43.

Binder, E.M., Tan, L.M., Chin, L.J., Hand, J. and Richard, J. 2007. Worldwide occurrence of mycotoxins in commodities, feeds and feed ingredients. Animal Feed Science and Technology. 137: 265–282.

Bintvihok, A. 2011. Controlling aflatoxin danger to duck and duck meat. World's Poultry Science Journal.17:18–20.

Chang, W., Qing X., Zheng, A., Zhang, S., Chen, Z., Wang, J., Liu, G. and Cai, H. 2016. Effects of aflatoxins on growth performance and skeletal muscle of Cherry Valley meat male ducks. Animal Nutrition. 2(3):186-91. http://dx.doi.org/10.1016/j.aninu.2016.06.002

Chen, X., Horn, N., Cotter P.F. and Applegate, T.J. 2014a. Growth, serum biochemistry, complement activity, and liver gene expression responses of Pekin ducklings to graded levels of cultured aflatoxin B1. Poultry Science. 93: 2028–2036.

Chen, K., Fang, J., Peng, X., Cui, H., Chen, J.,

Wang, F., Chen, Z., Zuo, Z., Deng, J., Lai, W and Zhou, Y. 2014b. Effect of selenium supplementation on aflatoxin B1-induced histopathological lesions and apoptosis in bursa of fabricius in broilers. Food and Chemical Toxicology. 74: 91–97.

Cole, R. and Richard, J. 1989. Mycotoxins: economic and health risks. Council for Agricultural Science and Technology, ed K.A. Nisi, Ames, IA, pp. 1–91.

Coulombe, R.A. 1993. Biological action of mycotoxins. Journal of Dairy Science. 76(3): 880-91. doi: 10.3168/jds.S0022-0302(93)77414-7.

Cullen, J.M. and Newberne, P.M. 1993. Acute hepatotoxicity of Aflatoxins. In: The Toxicology of Aflatoxins: Human Health, Veterinary, and Agricultural Significance; Eaton DL, Groopman JD, Eds.; Academic Press: London, UK.

Dalvi, R.R. 1986. An overview of aflatoxicosis of poultry: Its characteristics, prevention and reduction. Veterinary Research Communication. 10: 429–443.

Diaz, G. J. and Murcia, H. W. 2011. Biotransformation of aflatoxin B1 and its relationship with the differential toxicological response to aflatoxin in commercial poultry species. Chapter 1 in Aflatoxins—Biochemistry and Molecular Biology. R. G. Guevara-Gonzalez, ed. In: Tech, Rijeka, Croatia. November 17th, 2010 Published: October 5th, 2011, DOI: 10.5772/22109.

Diaz, G.J, Murcia, H.W., Cepeda, S.M. and Boermans, H.J. 2010. The role of selected cytochrome P450 enzymes on the bioactivation of aflatoxin B1 by duck liver microsomes. Avian Pathology. 39(4): 279-285.

Do, J.H. and Choi, D. 2007. Aflatoxins: detection, toxicity, and biosynthesis. Biotechnology and Bioprocess Engineering. 12: 585-593.

Doi, A.M., Patterson, P.E. and Gallagher, E.P. 2002. Variability in aflatoxin B1 macromolecular binding and relationship to biotransformation enzyme expression in human prenatal and adult liver. Toxicology and Applied Pharmacology. 181: 48 59.

Eaton, D.L. and Gallagher, E.P. 1994. Mechanisms of aflatoxin carcinogenesis. Annual Review of Pharmacology and Toxicology. 34: 135–172.

Eaton, L.D., Evan, P.G., Theo, K.B. and Kent, L.K. 1995. Role of cytochrome P4501A2 in chemical carcinogenesis: Implications for human variability in expression and en-zyme activity. Pharmacogenetics. 5(5):259-74. doi: 10.1097/00008571-199510000-00001.

Edds, G.T., Nair, K.P. and Simpson, C.F. 1973. Effect of aflatoxin B1 on resistance in poultry against Newcastle disease, infectious bronchitis and infectious bursal disease. Avian Pathology. 27: 290–295.

El-Sheshtawy, S.M., Amal, F., E-Zoghby, Nesreen, A., Shawky, and Dalia, H. S. 2021. Aflatoxicosis in Pekin duckling and the effects of treatments with lycopene and silymarin. Veterinary World. 14(3): 788–793.

El-Sheshtawy, S.M., El-Zoghby, A.F., Shawky, N.A. and Samak, D.H. 2021. Aflatoxicosis in Pekin duckling and the effects of treatments with lycopene and silymarin. Veterinary World. 14(3): 788-793. doi: 10.14202/vetworld.2021.

Feng, G. D., He, J., Ao, X. and Chen, D. W. 2017. Effects of maize naturally contaminated with aflatoxin B1 on growth per-formance, intestinal morphology, and digestive physi-ology in ducks. Poultry Science. 96: 1948-1955.

Fouad, A. M., Ruan, D., El-Senonseuj, H. K., Chen, W., Jiang, S. and Zheng, C. 2019. Harmful effects and control strategies of aflatoxin B1 produced by *Aspergillus flavus* and *Aspergillus parasiticus* strains on poultry: review. Toxins. 11:176.

Gallagher, E.P., Kunze, K.L., Stapleton, P.L. and Eaton, D.L. 1996. The kinetics of aflatoxin

B1 oxidation by human cDNA-expressed and human liver microsomal cytochromes P450 1A2 and 3A4. Toxicology and Applied Pharmacology. 141(2):595-606. doi: 10.1006/taap.1996.0326.

Girish, C. K. and Devegowda, G. 2006. Efficacy of modified glucomannan(Mycosorb) and HSCAS to alleviate the individual and combined toxicity of aflatoxin and T-2 toxin in broiler chickens. XXII'nd World Poultry Congress, 8-10 September, Istanbul, Turkey.

Gradelet, S., Astrong, P., le Bon, A.M., Berges, R. and Suschetet, M. 1997. Modulation of Aflatoxin B1 carcinogenicity, genotoxicity and metabolism in rat liver by dietary carotenoids: evidence for a protective effect of CYP1A inducers. Cancer Letters, 19:114(1-2):221-223.

Gratz, S., Mykkänen, H. and el-Nezami, H. 2005. Aflatoxin B1 binding by a mixture of Lactobacillus and Propionibacterium. *In vitro* versus ex vivo. Journal of Food Protection 68: 2470–2474.

Grenier, B. and Applegate, T. J. 2013. Modulation of Intestinal Functions Following Mycotoxin Ingestion: Meta-Analysis of Published Experiments in Animals. Toxins. 5: 396-430.

Grozeva, N., Valchev, I., Kanakov, D. Hristov, Ts., Lazarov L., Binev, R. and Nikolov, Y. 2012. Investigations on liver function in mulards with experimentally induced aflatoxicosis. Agricultural Science and Technology. 4(4): 371 – 377.

Guengerich, F.P., Johnson, W.W, Ueng, Y.F., Yamazaki, H. and Shimada T. 1996. Involvement of cytochrome P450, glutathione S-transferase, and epoxide hydrolase in the metabolism of aflatoxin B1 and relevance to risk of human liver cancer. Environmental Health Perspectives. 104: 557–562.

Guindon, K.A., Bedard, L.L. and Massey, T.E. 2007. Elevation of 8-hydroxydeoxyguanosine in DNA from isolated mouse lung cells following in vivo treatment with aflatoxin B1. Toxicological Sciences. 98(1): 57-62. doi: 10.1093/toxsci/kfm073. Epub 2007.

Guo, S., Shi, D., Liao, S., Su, R., Lin, Y., Pan, J. and Tang, Z. 2012. Influence of selenium on body weights and immune organ indexes in ducklings intoxicated with aflatoxin B. Biological Trace Element Research. 146(2):167-70.

Han, X.Y, Huang, Q.C., Li, W.F., Jiang, .J.F. and Xu, Z.R. 2008. Changes in growth performance, digestive enzyme activities and nutrient digestibility of cherry valley ducks in response to aflatoxin B_1 levels. Livestock Science. 119(1–3): 216-220.

Hoerr, F.J. 2010. Clinical aspects of immunosuppression in poultry. Avian Diseases. 54: 2–15.

Hurley, D.J., Neiger, R.D., Higgins, K.F., Rottinghaus, G.E. and Stahr, H. 1999. Short-term exposure to subacute doses of aflatoxin-induced depressed mitogen responses in young mallard ducks. Avian Disease. 43(4): 649-55.

IARC-WHO, 2002. Some Mycotoxins. Lyon (France): IARC. Report No. 82. Monographson the evaluation of the carcinogenic risk of chemicals to humans: Some natural occurring substances: Food Items and Constituents, Heterocyclic Aromatic Amines and Mycotoxins.

Jacob, R.A.1995.The integrated antioxidant system. Nutrition Research.15:755–766.

Knutsen, H.K., Alexander, J., Barregård, L., Bignami, M., Brüschweiler, B., Ceccatelli, S., Cottrill, B. and Dinovi, M. 2018. Scientific Opinion on the risk to human and animal health related to the presence of 4,15-diacetoxyscirpenol in food and feed. Eurpean Food Safety Authority Journal. 16: 5367.

Kumar, R. and Balachandran, C. 2009. Histopathological changes in broiler chickens fed aflatoxin and cyclopiazonic acid. Veterinarski Arhiv. 79: 31-40.

Leeson, S., Diaz, G.J. and Summers, J.D. 1995. Poultry Metabolic Disorders and Mycotoxins; University Books: Guelph, Canada.

Limaye, A., Yu, R.C., Chou, C.C., Liu, J.R. and Cheng, K.C. 2018. Protective and detoxifying effects conferred by dietary selenium and curcumin against AFB1-Mediated Toxicity in Livestock: A Review. Toxins. 10(25): 1-18.

Liu, J., Song, W.J., Zhang, N.Y., Tan, J., Krumm, C.S., Sun, L.H. and Qi, D.S. 2017. Biodetoxification of aflatoxin B1 in cottonseed meal by fermentation of Cellulosimicrobiumfunkei in duckling diet. Poultry Science. 96: 923–93.

Magnoli, A.P., Monge, M.P., Miazzo, R.D., Cavaglieri, L.R., Magnoli, C.E., Merkis, C.I., Cristofolini, A.L., Dalcero, A.M. and Chiacchiera, S.M. 2011. Effect of low levels of aflatoxin B1 on performance, biochemical parameters, and aflatoxin B1 in broiler liver tissues in the presence of monensin and sodium bentonite. Poultry Science. 90: 48–58.

Martínez-de-Anda, A., Valdivia, A. G., F.Jaramillo-Juárez,. Reyes, J. L., Ortiz, R., Quezada, T., de Luna, M. C. and Rodríguez, M. L. 2010. Effects of aflatoxin chronic intoxication in renal function of laying hens. Poultry Science. 89: 622–628.

Mishra, S.K., Jena, C.R., Naik, P.K., Kumar, D., Swain, B.K., Panda, S.K., Pamia , J. and Jena, G.R. 2021. Impact of dietary Aflatoxin-B1 on juvenile growth in White Pekin Ducks. Indian Journal of Animal Research. DOI: 10.18805/IJAR.B- 4556.

Mohajer, A., Seifi , S., Rezaie , S., Khaniki , G.J., Samarghandian , S., Farkhondeh , T. and Sadighara, P. 2021. Effect of aloe vera extract on reducing aflatoxin B1 in eggs of laying hen and egg yolk oxidative stability. Biointerface Research in Applied Chemistry. 11(5): 12680–12688. https://doi.org/10.33263/BRIAC115.1268012688.

Monbaliu, S., Van Poucke, C., Detavernier, C., Dumoulin, F., Van De Velde, M., Schoeters, E., Van Dyck, S., Averkieva, O., Van Peteghem, C. and De Saeger, S. 2010. Occurrence of mycotoxins in feed as analyzed by a multi-mycotoxin LC-MS/MS method. Journal of Agricultural and Food Chemistry. 58, 66–71.

Moss, M.O.1998. Recent studies of mycotoxins. Journal of Applied Microbiology. Symp. 84: 62–76.

Nalle, C., Angi, A., Supit, M., Ambarwati, S. and Yuliani, N. 2019. Response of broiler chickens fed on diets containing low level of Aflatoxin (B1 and B2) and supplemented with mycotoxin binder. Journal Kajian Veteriner. 98-102. https://doi.org/10.35508/jkv.v0i0.1592.

Nyandieka, H.S. and Wakhisi, J. 1993. The impact of vitamins A,C,E, and selenium compound on prevention of liver cancer in rats. East Africa Medical Journal. 70(3): 151-3.

Ortatatli, M., Oðuz, H., Hatipoðlu, F. and Karaman, M. 2005. Evaluation of pathological changes in broilers during chronic aflatoxin (50 and 100 ppb) and clinoptilolite exposure. Research in Veterinary Science. 78: 61–68.

Ostrowski-Meissner, H. T. 1983. Effect of contamination of diets with aflatoxins on growing ducks and chickens. Tropical Animal Health and Production. 15:161–168.

Patent-Duckling feed capable of relieving aflatoxin poisoning of duckling retrieved from URL thttps://patents.google.com/patent/CN102178118A/en.

Patterson, D. S. P. 1973. Metabolism as a factor in determining the toxic action of the aflatoxins in different animal species. Food and Cosmetics toxicology. 11(2): 287-294.

Pier, A.C. 1973. An overview of the mycotoxicosis of domestic animals. Journal of The Medical Association of Thailand. 163: 1259–1261.

Pier, A.C. 1992. Major biological consequences of aflatoxicosis in animal production. Journal

of Animal Science. 70: 3964–3967.

Prasetyo, H.L. and Sushanti, T. 2010. Effect of genotypes and aflatoxin levels in the diets on early laying characteristics of local ducks. International System for Agricultural Science and Technology. 15(3): 215-219.

Ramos, A. J., Fink-Gremmels J. and Hernandez. E. 1996. Prevention of toxic effects of mycotoxins by means of nonnutritive absorbent compounds. Journal of Food Protection. 59: 631-641.

Robens, J. F. and Richard, J. L. 1992. Aflatoxinsin animal and human health. Reviews of Environmental Contamination and Toxicology. 127: 69–94.

Shen, H.M., Shi, C.Y., Lee, H.P. and Ong, C.N.1994. Aflatoxin B1-induced lipid peroxidation in rat liver. Toxicology and Applied Pharmacology. 127: 145–150

Shen, H.M., Shi, C.Y., Shen, Y. and Ong, C.N. 1996. Detection of elevated reactive oxygen species level in cultured rat hepatocytes treated with aflatoxin B1. Free Radical Biology and Medicine, 21: 139–146.

Shi, D., Guo, S., Liao, S., Su, R., Pan, J., Lin, Y. and Tang, Z.2012. Influence of selenium on hepatic mitochondrial antioxidant capacity in ducklings intoxicated with Aflatoxin B1. Biological Trace Element Research. 145: 325–329.

Singh, R., Mandal, A.B. and Biswas, A. 2019. Mycotoxicosis and its Management in Poultry. Technical Bulletin. Avian Nutrition and Feed Technology Division, Central Avian Research Institute, Izatnagar – 243 122 (UP), India.

Soriano, D.M. 2020. Aflatoxins in ducks and use of mycotoxin binders, Veterinary Digital retrieved from URL https:// www.veterinariadigital.com.

Streit, E., Schatzmayr, G., Tassis, P., Tzika, E., Marin, D., Taranu, I., Tabuc, C., Nicolau, A., Aprodu, I., Puel, O. and Oswald, I.P. 2012. Current situation of mycotoxin contamination and co-occurrence in animal feed–focus on Europe. Toxins. 4: 788–809.

Sumantri, I., Sulaiman, A., Gulo, N. and Wahyuni, P. 2019a. Effects of curcumin supplementation in aflatoxin B1-contaminated diet on the performance and egg quality of laying duck. IOP Conference Series Earth and Environmental Sciences. 387(1): 012102. DOI: 10.1088/1755-1315/ 387/1/012102.

Sumantri, I., Herliani, H., Rajibi A.N. and Edriantina, R. 2019b. Effects of zeolite inclusion in aflatoxin B1-contaminated diet onthe performance of laying duck. Journal of the Indonesian Tropical Animal Agriculture. 44(3): 277-285.

Valchev, I., Grozeva, N., Kanakov, D., Hristov, T.S., Lazarov, L. and Binev, R. 2018. Effect of experimentally induced aflatoxicosis on hematological parameters and bone marrow morphology in mulard ducks. Agricultural Science and Technology. 10(3): 208–214.

Valchev, I., Grozeva, N., Lazarov, L., Kanakov, D., Hristov, T.S., Binev, R. and Nikolov, Y. 2013. Investigations on kidney function in mulard ducklings with experimental aflatoxicosis. Agricultural Science and Technology. 5(3): 282–289.

Valchev, I., Lazarov, L., Hristov, T.S. Kanakov, D., Binev, R. and Nikolov, Y. 2014. Blood triiodothyronine, thyroxine and thyroid stimulating hormone concentrations in Mulard ducks with experimental aflatoxicosis. Bulgarian Journal of Veterinary Medicine. 17(3): 191-198.

Valchev, I., Koynarski, T., Sotirov, L., Nikolov, Y. and Petkov, P. 2015. Effect of aflatoxin B1 on Moulard duck's natural immunity. Pakistan Veterinary Journal. 35(1): 67-70.

Verma, R.J. 2004. Aflatoxin causes DNA damage. International Journal of Human Genetics. 4: 231-236.

Verma, R.J. and Nair, A. 2001. Vitamin E ameliorates aflatoxin-induced biochemical changes in the testis of mice. Asian Journal of

Andrology. 3(4): 305-9.

Visioli, F., Grande, S., Bogani, P. and Galli, C. 2004. The role of antioxidants in the mediterranean diets: focus on cancer. European Journal of Cancer Prevention. 13: 337–343.

Wade, M.R. and Sapcota, D. 2017. Effect of dietary esterified glucomannan on the performance of broiler chickens during experimental aflatoxicosis. Animal Nutrition and Feed Technology. 17: 107-116. DOI: 10.5958/0974-181X.2017.00011.7.

Wade, M.R., Sapcota, D. and Verma, U. 2018. Ameliorating aflatoxicosis in commercial broiler chickens by dietary mycosorb: Heamato-biochemical studies. 2018. Indian Journal of Animal Research. 52(1): 46-50. DOI : 10.18805/ijar.B-3182

Wan, X.L., Yang, Z.B., Yang, W.R., Jiang, S.Z., Zhang, G.G., Johnston, S.L. and Chi, F. 2013. Toxicity of increasing aflatoxin B1 concentrations from contaminated corn with or without clay adsorbent supplementation in ducklings. Poultry Science. 92:1244–1253.

World Health Organization. 1979. Environmental health criteria, safety evaluation of certain food additives: 1-127.

Wu, H.C. and Santella, R. 2012. The role of aflatoxins in he-patocellular carcinoma. Hepatitis Monthly. 12(10 HCC):e7238. doi: 10.5812/hepatmon.7238.

Wu, K., Jia, S., Zhang, J., Hang C., Wang S., Rajput, S.A. and Sun, L. 2021. Transcriptomics and flow cytometry reveals the cytotoxicity of aflatoxin B1 and aflatoxin M1 in bovine mammary epithelial cells. Ecotoxicology and Environmental Safety. 209: 111823.

Wu, K., Liu, M., Wang, H., Hang., Rajput, S.A. Shan Y and Qi D. 2021. The mechanism underlying the extreme sensitivity of duck to aflatoxin B1. Oxidative Medicine and Cellular Longevity. 2021;1-8. https://doi.org/10.1155/2021/9996503.

Zychowski, K.E., Pohlenz, C., Mays, T., Romoser, A., Hume M, Buentello A, and Phillips, T.D. 2013. The effect of Nova Sil dietary supplementation on the growth and health performance of Nile tilapia (*Oreochromis niloticus*) fed aflatoxin-B1 contaminated feed. Aquaculture A. 376-379: 117-123.