Pathological Studies on the Effect of Prebiotic and Probiotic on Aflatoxicosis in Broiler Chickens

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Abstract

Chicken is a cheap source of animal protein to overcome the world over increasing population problem, attention must focus on poultry industry. Broilers have many advantages including fast growth, high acceptability to consumers, high density of stocking. Moreover, it allows poultry farmer to have regular contact with their chicks permitting early detection of management trouble and diseases problem. The co-contamination of feed samples with mycotoxins/metabolites of varying concentrations suggests possible health risk to broiler industry. Various analytical methods were applied for detection of mycotoxins in commercial poultry rations, HPLC was more useful for detection of aflatoxin fragments. Broilers birds feed on higher mycotoxins ration showed depression, ruffled feather, friable liver with extended bile duct, enlarged kidney with urates and asities in addition to very bad performance and histopathological changes. While the addition of some feed additive (HASCS, yeast, probiotic and zinc) to the mycotoxin’s rations explain highly protective effect against these mycotoxins. Mycotoxins very difficult to remove from contaminated commodities, so continuous monitoring is essential and efficient protective products strategies are needed to deal with such outbreaks.

Key Words: Broiler, aflatoxin, Histopathology, Probiotic, Probiotic, HASCS, feed additives.

Introduction

The poultry business has emerged as a key sector of the global economy in recent years (Griggs and Jacob, 2005). Poultry at large-scale rearing facilities face stressful conditions and disease-related issues (Nava et al., 2005). High
temperatures and high humidity are ideal conditions for the growth of mould and yeast in improperly stored food and agricultural by-products. The presence of these fungi, in addition to spoiling the products, reduces its quality and facilitates the excrete mycotoxins, which are fungal secondary metabolites (Watts et al., 2003). Fungi produce mycotoxins as secondary metabolites, and these poisons can contaminate grain crops all over the world (Smith et al., 1995). Fungi (Asp. flavus, Asp. parasiticus, and Penic. puberulum) create aflatoxins, which are poisonous compounds (Peterson, et al., 2001). There are four distinct subtypes. According to research by Cheeeke and Shull (1985), aflatoxin B1 has the highest level of biological activity among the aflatoxin family. Animals' susceptibility to aflatoxins varies with factors like gender, age, species, and diet (Eaton & Groopman, 1994). Feedstuffs can develop aflatoxins under field or storage circumstances with the right amount of moisture and heat (Chao, 1991). Hepatitis, icterus, haemorrhage, and mortality are all symptoms of aflatoxicosis, a unique clinical condition (Abd El Hamid et al., 2002). Using feed additives to promote growth efficiency and feed utilisation is a key strategy for a more productive chicken industry (Pirgozliev et al., 2019). In order to aid in the clearance of mycotoxins through the gastrointestinal system, certain salts and mycotoxin binders are commonly utilised in poultry breeding (Murugesan et al., 2015). Activated, broad-spectrum, hydrated sodium/calcium aluminosilicate (HSCAS) (Huwig et al., 2001) is a common example of a material having a high adsorptive capacity. Probiotics are live non-pathogenic microorganism’s bacteria (Lactobacillus plantarum, and Pediococcus), yeast, and fungi (Pourakbari et al. 2016). Probiotics are live microbial which enhance intestinal microbial balance (Sethiya, 2016). Probiotics contain much type of Microorganisms as various species of lactobacilli or bifido bacteria (Qiao, et al., 2019). Microorganisms preent in probiotic produce many metabolites inhibit or kill pathogenic bacteria (Kizerwter and Binek, 2009). Probiotics is lowering intestinal pH leading to inhibit growth of pathogenic bacteria (Ashayerizadeh et al., 2009), probiotics maintaining normal intestinal flora by competitive exclusion and antagonism (Bengmar, 1998). Prebiotics are non-digestible food ingredients used as feed additives to improve health and protect poultry against pathogens (Pirgozliev et al. 2013). Prebiotics is reducing colonization of foodborne pathogens (Micciche et al., 2018). The present study was designed to evaluate prophylactic effect of probiotic and prebiotic to modulate
the toxic effect of aflatoxin in pathological changes in chickens.

**Material and Methods**

**HPLC analysis for two ration samples**

Reagents and chemicals Fisher Scientific (UK) supplied the acetonitrile, methanol water (HPLC grade), and n-hexane, while Sigma-Aldrich (USA) supplied the aflatoxin standards and trifluoroacetic acid (TFA). The aflatoxin concentration was determined utilising a pump and quaternary gradient system in an HPLC apparatus (Thermoscientific suryor Hpyc). Quantitative analysis was performed using a fluorescence detector with excitation and emission wavelengths of 360 and 440 nm, respectively. Analytical column dimensions were 150 mm in length and 4.6 mm in inner diameter, and the particle size was 5 m for a Supelco Discovery® C18. The mobile phase consisted of acetonitrile, methanol, and water (in the ratio of 8:27:65), and the flow rate was set at 1.0 mL min for the HPLC analysis. After degassing in an ultrasonic bath for 25 minutes, the mixture was filtered through a membrane filter and ready for use. The sample loop size was 20 l.

**Broilers and experimental design**

One hundred and fifty chicks aged one-day old were divided into 5 groups, group A, B, C, D fed on a diet with higher aflatoxins diet (20.88 ppb); while group E was fed on a diet with a normal limit of mycotoxins. Group B received HASCS as feed additives, while group C received (avi bac, pro powder and byg35), and group D received HASCS, avi bac, pro powder, and byg35.

**Feed additives used as anti-mycotoxins.**

**A- AVI5 Bac Probiotic** is an American product directed for use in poultry feed produced by ProByn, Inc. USA as powder form, composed of: Lactic acid bacteria, $1.6 \times 10^9$ CFU/gm (**L.acidophilus, L.Planterum, L.bervis**), Amylase, 224 AU/gm and β-glucans 144 B GU/gm.

**B Flow gard "HASCS" 100%**: It contains Hydrated Aluminum sodium calcium silicate. Lot Nu 02, Reg-Nu 1/315 Origin USA

**C- Bgy 35** It contains Inactive dried brewer's yeast (Saccharomyces cerevisiae) 100% [vitamins, minerals, Amino Acids]. Manufacture date (1/2021), Expiry date (1/2023), Lot Number 05, Reg-Number in Egypt 2/54, USA origin.

**D- Prebiotic (Pro-power)** It contains Yeast cell wall (Saccharomyces cerevisiae) 300 gm, Mannan oligosaccha-ride 170 gm, Beta-glucans 130gm, Dried breweris yeast 300gm, Diatomaccous earth 400gm (Humic acid) Manufactured date (1/2021), Expiry date (1/2023), number 090, Reg-Number 8490, origin USA.

**E- Zinc powder**: Used as a carrier for mixing all feed additives.

**Clinical observations:**
a) Clinical signs: observed the signs appear on the birds daily from the 1st day of experiment till the end.
b) Postmortem lesions: observed lesions appear in the different organs of dead birds during the experiment.
c) Morbidity, Mortalities: the mortality and morbidity observed daily in all groups from the first day till end of experiment.
D) Lesions score: it calculated according to clinical observations - No lesion=0 - diarrhea=1 - Ruffled feather + diarrhea=2 - R + D + bone strength=3 - R + D + b + Inhomogenous growth=4 - Mortality=5
E) Score lesion index: calculated by divides total score lesion / total number of life birds3.2)

Growth performance:
a) Body weight gain: Fifteen birds from each group were weighted weekly to observe the treatment effect on body weight and compare between groups (Jindal et al., 1994).
b) Feed intake: It was calculated weekly according to the feed consumption of ration by birds.
c) Feed conversion ratio: it is the conventional measure of livestock production efficiency. It calculated by division of the weight of feed intake / the body weight gained by the bird.

Histopathological examination
Specimens from liver, kidney, spleen, intestine, and bursa of Fabricius were excised immediately after death or slaughter after careful PM examination and fixed in 10% neutral buffered formalin. Five-micron thick paraffin sections, stained with hematoxylin and eosin and examined microscopically (Bancroft & Gamble, 2002).

Results
HPLC-1
two ration samples from different companies were assayed for the aflatoxins (B1,B2,G1,G2) concentrations using HPLC, the results are showed in fig-1-4/ tab-1.

Clinical observations
Clinical signs and postmortum lesions: At frist week group A showed clinically depression, diarrhea, ruffled feather, emaciation. At 2nd week group A showing Liver and Kidney enlargement Hydropericardium, Regression of the bursa of fabricus, distended gallbladder and bile duct, Liver damaged, swelling, friable, Dehydration, pasty white urate are deposited on pericardial, prehepatic, peritoneal articular surfaces. At 3rd weeks group A showed CRD lesions, Marked enlargement of liver, Urates in the ureters, Seroperitonites, Enlargement of the kidneys, Atrophy of bursa fabricus, Enlargement of spleen, pancreas, Ascites, hydropericardium.

Morbidity, Mortalities:
Morbidity, Mortalities were higher in group A than other groups, it followed by group B and C, while group D and E don’t show any morbidities or mortalities all over the experimental period.

Lesion score and Lesion score index: Group A showed higher
lesion score and lesion score index in comparison with other groups all over the experimental period. But other groups (B, C, D and E) showed non significant difference between each others.

**Growth performance (BW, FI, FCR):**

**Body weight (BW):** One-Way ANOVA results revealed that there was highly statistically (significantly) different among groups in body weight (p-value <0.01). Group A showed significant decreased in the body weight gain compared to other groups B, C, D and E; It followed by group B which showed significat decrease than groups C, D, E all over the 4th week of the experiment (p-value <0.01). while Both group D, E showed significant increase than other groups with non significant difference between each others. Group C showed non significant difference with groups D, E at 1st, 2nd, and 3rd weeks of the experiment but significant decrease at 4th week of the experiment.

**Feed intake (FI):** It was caculated weekly according to ration consumption by the birds. At frist week was 160 gm ration/bird, at 2nd week was 600 gm ration/bird, at 3rd week was 1000 gm ration/bird and at 4th week was 2800 gm ration/bird.

**Feed conversion ratio (FCR):** It was calculated according to the feed consumption in related to body weight, and it depend mainly on the changes on body weight weekly among groups. Group A in all weeks of the experiment showed significant increase in FCR compared to other groups. Group B showed significat increase compared with groups C, D, E in 1st week of experiment. While in 2nd, 3rd and 4th week of the experiment it showed non significant different with group C. while other groups C, D, E showed non significant different between each other all over the experiment.

**Histopathology**

**Liver:** The hepatic parenchyma suffred from various degenerative changes varied from acute cell swelling to micro steatosis or minate areas of coagulative necrosis scattered within the liver tissues (Fig-10.1). The majority of portal areas had prolifervative bile duct epithelium extravasted erythrocyties and lymphocytic aggrefation (Fig-10.2) Necrotic hepatic cells replaced by monocular cells.

**Kidneys:** Intense nephrotic changes of the tubalor epithelia represented manily by necrotic tubules and heamorrhags were common (Fig-10.3) sometimes hemorrhages blood casts and leukocytic aggregation mainly lymphocytic beside hyperitophied and vacuolated vascular media of renal arterioles were also notioced (Fig-10.4). Other tabules showed cellular casts or urates deposites inside their lumina-intersitil mononuclear cells aggregation and hyper cellutits of some glomeruli were observed.

**Spleen:** Pronuced depletion and cuagulative necrosis inved the
majority of splenic white pulps were detected (Fig-10.5). The necrotic lymphoid tissue had numerous reticulo endothelsal cells (REC) beside endotheliosis of some central arterioles (Fig-10.6) hyper trophied splenic septae capsule caulde be seen.

**Bursa of Fabricues:** The majority of lymphoid follicles were inactive and deplated with thickened interfollicular tissue by fibrous tissue (Fibroplasia) (Fig-10.7) necrotic follicles contained numerous minutes cystic spaces and few lymphocytes in both cortex and medullary zones (Fig-10.8).

**Intestine:** All intestinal villi in different segments of intestine appeared blunted denaded (Loss of villus enterocytes) and fused with reduction of their lengths (Fig-10.9). Submacosa showed displaced lymphoid follicles with degenerated and necrotic intestinal crypts (Fig-10.10) A few glands had hyperplasia of (goblet cells) or partial destruction partial hyaline degeneration or necrosis of muscular coat coulde be seen.

**Treated group:**

**Liver:** The hepatic cells were apparently normal with interstitial and portal lymphocytic aggregation (Fig-10.11), hyperplasia of kuffer cells could be also noticed.

**Kidneys:** Mild reversible nephrotic changes or apparently normal tubular epithelia with or without regenerative attampts were common (Fig-10.12) a few extravasted erythroytes inter renal capillaries and normal renal blood vasels were encontensed. All the tabular Lumina were empty and free from casts and urates.

**Spleen:** All splenic elements manily (white pulps) were activated and hyperplastic with over populativa of spleenic tissue by Lymphocytes (Fig-10.13, 14).

**Bursa of fabricas:** The Bulsal lymphoid follicles of treated group was activayed and hyperplastic (Fig. 15) the latter represented by less demarcted corico medullary junction and neumerous regenerated lymphoid elemeats scattered in both scortex and medulla (Fig. 16) the covering epithelium was normal or showed mild hyperplasia.

**Intestine:** Elongated and broad intestienal villi with agreat absorptive surface covering intestinal villi of different intestinal segments were common (Fig-10.17). Mild mucosal lymphocytic infiltration could be seen. Submucasal activated of lymphoid follicls with pronuced proliferative intestinal crypts were seen (Fig-10.18).
**Fig (1):** Liquid chromatogram of aflatoxins standard 50 ppb
Liquid chromatogram of aflatoxins standard 50 ppb (AFB1 at RT. 11.6 min., AFB2 at RT. 14.1 min., AFG1 at RT. 15.6 min., AFG2 at RT. 19.2)

**Fig (2):** Liquid chromatogram of aflatoxins standard 20 ppb
Liquid chromatogram of aflatoxins standard 20 ppb (AFB1 at RT. 11.6 min., AFB2 at RT. 14.1 min., AFG1 at RT. 15.6 min., AFG2 at RT. 19.2)
**Fig (3):** Liquid chromatogram of Aflatoxins in Feed Sample No. 5 (AFB1 at RT. 11.8 min., AFB2 at RT. 14.08 min., AFG1 at RT. 15.6 min.)

**Fig (4):** Liquid chromatogram of Aflatoxins in Feed sample No. 2 (AFB1 at RT. 11.6 min., AFG1 at RT. 15.4 min.)

**Table (1):** Liquid chromatogram of Aflatoxins in Feed Sample No. 5 and 7
<table>
<thead>
<tr>
<th>Sample No.</th>
<th>AFB1 (ppb)</th>
<th>AFB2 (ppb)</th>
<th>AFG1 (ppb)</th>
<th>AFG2 (ppb)</th>
<th>Total (ppb)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>19.5 (88%)</td>
<td>2.4 (11%)</td>
<td>0.25 (1.1%)</td>
<td>---</td>
<td>22.15</td>
</tr>
<tr>
<td>2</td>
<td>7.3 (85%)</td>
<td>---</td>
<td>1.3 (15%)</td>
<td>---</td>
<td>8.6</td>
</tr>
</tbody>
</table>

Figure (5): Chick of group A at 7 days showing sever congestion of liver gallbladder enlarged, filled with bile.
Figure (6): Chick of group A showing enlargement of kidney and ascites after three weeks, sever accumulation if urates in ureters.
Figure (7): Chick of group A showing sever congestion, enlargement of the liver, hydropericardium, Seroperitonitis.

Figure (8): Chick of group A showing sever congestion and enlargement of the heart. In compere with normal
Figure (9): Chick of group A showing sever congestion and enlargement of spleen in compare with normal.

Table (2): lesions score in different organs.

<table>
<thead>
<tr>
<th>parameter</th>
<th>1&lt;sup&gt;st&lt;/sup&gt; week</th>
<th>2&lt;sup&gt;nd&lt;/sup&gt; week</th>
<th>3&lt;sup&gt;rd&lt;/sup&gt; week</th>
<th>4&lt;sup&gt;th&lt;/sup&gt; week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TLS</td>
<td>LSI</td>
<td>TLS</td>
<td>LSI</td>
</tr>
<tr>
<td>Gp (A)</td>
<td>23/30</td>
<td>0.77</td>
<td>47/28</td>
<td>1.5</td>
</tr>
<tr>
<td>Gp (B)</td>
<td>7/30</td>
<td>0.2</td>
<td>3/29</td>
<td>0.1</td>
</tr>
<tr>
<td>Gp (C)</td>
<td>7/30</td>
<td>0.2</td>
<td>3/29</td>
<td>0.1</td>
</tr>
<tr>
<td>Gp (D)</td>
<td>2/30</td>
<td>0.1</td>
<td>4/30</td>
<td>0.13</td>
</tr>
<tr>
<td>Gp (E)</td>
<td>2/30</td>
<td>0.1</td>
<td>3/30</td>
<td>0.13</td>
</tr>
</tbody>
</table>

lesion score in the experimental groups: Total Lesion score = TLS. Lesion score index  = LSI. Score lesion: (No lesion=0 , Diarrhea(D)=1 , Ruffled feather+Diarrhea(R)=2 , R+D+Bone strength(B)=3 , R+D+B+Inhomogenous growth=4 , Mortality=5); Score lesion index: total lesion score / total number of birds.

Chart (1): body weight in different groups over 4 weeks
**Chart (2):** food conversion rate in different groups

**Fig 10.1:** Liver chickens (mycotoxicosis) showing minute area of coagulative necrosis in the hepatic parenchyma.

**Fig 10.2:** Liver of chicken ("mycotoxicosis"), showing proliferative bile duct epithelium and lymphocytic aggregation in the portal area.

**Fig 10.3:** Kidney of chickens ("mycotoxicosis"), showing necrotic tubularepithelial haemorrhage and lymphocytic aggregation of chickens.

**Fig 10.4:** Kidney of chickens (mycotoxicosis), showing necrotic tubules haemorrhage blood casts, arranged beside vasculated vascular media

**Fig 10.5:** Spleen of chickens (mycotoxicosis); showing diffuse coagulative necrosis and depletion of white pulps.

**Fig 10.6:** High power of previous figure showing white pulps and proliferation of RE cells.
Fig (10.7): Bursa of fabricus of chickens (mycotoxosis) showing intense lymphoid depletion of follicles and interfollicular fibroplasia.

Fig (10.8): High power of previous figure to show depleted cortex and medulla of follicles and replaced by minute spaces.

Fig (10.9): Intestine of chickens (mycotoxosis); showing blunted and denuded villi with fusion and reduction in villus length.

Fig (10.10): Intestine of chickens (mycotoxises) showing depleted lymphoid follicles and degenerated intestinal (crypts).

Fig (10.11): Liver of chickens (treated group); showing interstitial lymphocytic aggregation and apparently normal hepatic cells.

Fig (10.12): Kidney of chickens (treated group); showing regenerated attempts from tubular epithelia and apparently normal renal tubules.
**Fig (10.13):** Spleen of chicken of (treated group); showing activation hyperplasia of white pulps.

**Fig (10.14):** High-power of the previous figure to show overpopulation of white pulps by lymphocytes.

**Fig (10.15):** Bursa of chickens of (treated group); showing activated and hyperplastic lymphoid follicles and normal covering epithelium.

**Fig (10.16):** High power of the previous figure to show hyperplasia of follicles represented by over population with less demarcation of cortico medullary junction.

**Fig (10.17):** Intestine of chickens (treated) showing long intestinal villi with mucosal lymphocytic and proliferative intestinal crypts.

**Fig (10.18):** Intestine of chicken (treated) intense proliferation and elongation of intestinal crypts in submucosa.

**Discussion**

Aflatoxin is the most common mycotoxins in poultry feed (Pattison et al., 2008). Aflatoxin is the most dangerous one and associated with several economic losses to poultry industry including decreasing immunity in flocks and enhancing susceptibility to various infections, decrease feed consumption, loss of weight gains and decreased meat production (Pimpukdee et al., 2004). The United States Food and Drug Administration (US FDA) regulated the permissible limit of aflatoxin level in poultry feed as 20 ppb (US FDA, 2019). The most harmful four aflatoxins that are produced naturally are B1, B2, G1, and G2; among them, B1 Subtype is the most found in high concentrations in feed (Sweeney and Dobson 1998; Vaamonde, 2003). This study focused on evaluating the level of Aflatoxin contamination in some locally produced feedstuff formulation in Egypt and determining if it exceeded the permissible level for healthy poultry production or not. Also, the study discussed the prevalence, performance, and impact of the aflatoxin on growth rate and mortality on broilers either in treated or non-treated ration groups. In treated ration groups with HASCS as chemical anti-mycotoxins binder, Propower as prebiotic, Byg 35 as yeast extracts, AVI5 bac as probiotic confirmed the protective effects of different feed additives as growth promoting and/or as protective factors against mycotoxins. Two ration samples were chosen for the detection of aflatoxin fragments (B1, B2, G1, and G2) by HPLC. B1 aflatoxin represents the higher percentage in the tested samples by 88% and 85% respectively; This result agreed with Rodriguesa et al. (2011) who found that the presence of aflatoxin B1 in evaluated poultry feed samples varied from 0 to 94%. And agree also with Chen X et al., (2013) and Wacoo AP et al. (2014) who explained that aflatoxins are...
produced by Aspergillus species of fungi as secondary metabolites and found in four main forms (aflatoxin B1, B2, G1 and G2) in contaminated grains. Aflatoxin B1 is the most common and potent toxin. It accounts for about 75% of all aflatoxin contamination of food and feeds in the world. Mycotoxins are very difficult to remove from contaminated commodities, so preventing them from accumulating in agricultural commodities is the most effective strategy to combat the problem. The addition of some feed additives may protect the poultry from the hazardous effect of these mycotoxins; Nevertheless, excessive mycotoxin levels may occur despite all preventive measures. 

(Schatzmann et al., 2003; Molnar et al., 2004; and Vekiru et al., 2010) reported that Yeast strains with anti-mycotoxin, chemical, and lactobacillus were capable of detoxifying ochra and aflatoxins. Clinicopathological signs and mortalities were more prominence in group A (fed higher mycotoxins ration) in compared to other treated groups, it showed increased water consumption and decrease in feed intake leads to significant weight loss, diarrhea, dullness, stunting growth, ruffled poor appearance and broken feather, paleness, trembling ataxia, lameness, paralysis of leg and lameness gasping, prostration and death; similar results previously detected (Okoye et al., 1988; Khan et al., 1990; Rao and Joshi 1993; lesson et al., 1995 and Kubena et al., 1998) all those researchers described thriftiness, increased water intake, anorexia and mortality as general changes during aflatoxicosis. During toxicity, increased water intake could be an effort to dodge desiccation thus regaining water loss from the body due to diarrhea as reported (Kubena et al., 1998; Hussain et al., 2008; Khan and Zahoor, 2014) Postmortem examination for birds in Group A showed hemorrhage in different organs/tissues, enlarged liver with distended gallbladder, enlarged kidneys with an accumulation of urates, reduced bone firmness, and poor pigmentation. Similar lesions in the liver have been reported by Khan et al. (2014) in the layer breeders. Also, Resanovic (2009) reported that a minimum amount of ochratoxin (OTA) causes reduced bone firmness and poor pigmentation; Acutely intoxicated birds are depressed, dehydrated, and often polyuria and die in acute renal failure; Survivors will be poorly feathered, have delayed sexual maturity, increased clotting times, anemia and immunosuppression. Dragan et al. (2011) reported that mycotoxins are one of major factors suppressing poultry productivity causing substantial losses among Birds due to decrease body weight, reduced feed efficiency (FCR) decrease immunity of the birds leading to decreased resistance to infectious diseases, liver damage, bile duct proliferation, kidney damage. The treated groups showed
no obvious clinical signs and postmortem lesions compared with group (A) similar findings were clarified by lesion score and lesion score index, the best-treated group was (D) followed by group (C) and (B) respectively. In addition to no mortalities were recorded in both groups D and E all over the experiment period attributed to the usage of AV15 and Byg 35 to myotoxic ration, Similar results were obtained previously, when added probiotic and prebiotic additives, these additives could prevent the absorption of mycotoxins during their passage in the gastrointestinal tract and eliminate in the feces (Bueno et al., 2006; El-Nezami et al., 2000; Gratz et al., 2007). Also, Peltonen et al. (2000) and Peltonen et al. (2001) reported that probiotic microorganisms had a wide range of binding capacities to aflatoxins. Group A showed significant decrease in body weight and a significant increase in feed conversion ratio all over the experimental period, this result agreed with previous reports whom recorded a significant decrease in body weight of broiler chicks below 21 days of age fed up to 5 mg/kg aflatoxin (Tessari et al., 2006; Jakhar and Sadana, 2004; Dos Anjos et al., 2016 Duff et al., 1987. The treated Groups with different feed additives and probiotics showed an increase in body weight, FCR, decreased in morbidity, and mortality rates; this was due to immunomodulatory agents by activating specific and non-specific host immune response in chickens, which in turn help in prevention and control of various infectious diseases. These results were confirmed by histopathological examination of groups A and D, which support the previous finding (Fuller, 1999 and Koenen et al., 2004). the liver in G-A revealed severe destruction and necrosis of hepatic parenchyma accompanied with intense congestions of blood vessels and hepatic sinusoids were common. Also, the kidney in G-A showed areas of hemorrhages associated with necrosis of renal tubules and fibroblast proliferation were common in glomeruli interstitial aggregations of lymphocytes. The observed findings agreed with the results reported by Galab (1999). Also, Karaman et al. (2005) detected aplasia of thymus, spleen, bursa of Fabricius in chicken and suppression immunoglobulins during immunization fed on aflatoxin rations. Group D showed Significant results in FCR, body weight, low mortality, and morbidity due to intestinal (distal jejunal) morphometry with a significant increase in villus length to crypt depth ratio compared to birds in group A (the contaminated group with aflatoxin), where mycotoxins are able to affect activity and proliferating cells, damage epithelial tissue, increase intestinal permeability and therefore result in weak immune system.
Consequently, when pathogen enters the organism and propagate, result in inefficient immune response and stronger clinical signs; as previously reported by Antonissen et al. (2014). The need for continuous detection and control of these mycotoxins in the chicken ration, consider important Oguz (2011). Therefore, continuous monitoring is essential and efficient detoxification strategies are needed to deal with such outbreaks.

Conclusion
From the results of the present work, it can be concluded that, the using of food additives silica, biological probiotic, yeast extracts and probiotic has protective effect against hepatotoxic and nephrotic toxicity of aflatoxins in broiler chicks.

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دراسات باثولوجية على تأثير البروبيوتك والبريبيوتك على سمية الافلاتوكسين في كلناك الروم أمان

داليا منصور حامد 1، دعاء سليم الحلوس 2، اسماعيل أحمد محمد 1، وائل محمد الفيل 1

كلية الطب البيطري جامعة قنا السويس 1، معهد بحوث صحة الحيوان بالإسماعلية 2

يشير التلوث المشترك لعينات العلف بالسموم الفطرية المستقلبات بتركيزات مختلفة إلى مخاطر صحية محتملة على صناعة الدجاج الاحض. تم تطبيق طرق تحليلية مختلفة للكشف عن السموم الفطرية في اعلاف الدواجن التجارية، وكان HPLC أكثر فائدة في الكشف عن جزيئات الافلاتوكسين المختلفة عند تعريج دجاج تسمين على نسب عالية من السموم الفطرية الافلاتوكسين أظهرت اكتئاباً وتكدساً في الرئتين وتشقية الكبد مع تمدد القناة الصفراوية وتضخم الكلى بالإضافة إلى الأداء السيئ للغاية والانتفاخات هستوباثولوجية. في حين أن إضافة بعض مضادات الأعلاف (HASCS، الخميرة، البروبيوتك والزنك) إلى اعلاف ملوثة بالسموم الفطرية كانت لها التأثير وقائي للغاية ضد هذه السموم الفطرية. من الصعب للغاية إزالة السموم الفطرية من السلع الملوثة، لذا فإن المراقبة المستمرة ضرورية، وهناك حاجة إلى منتجات وقائية فعالة للتعامل مع مثل هذه الفاشيات