

The ameliorative effect of mycosorb in aflatoxin contaminated diet of broiler chickens

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Abstract This experiment was conducted to study the effect of Aflatoxins (AF) on broiler performance, organ weight, plasma characteristics, protein digestibility, and metabolizable energy of the diets. Dietary treatments were: 1) control diet based on corn and soybean meal; 2, 3 and 4) control diet plus 1 mg kg⁻¹ AF, 1 mg kg⁻¹ AF and 0.25% mycosorb, and 0.25% Mycosorb, respectively. The results in the Aflatoxins and Aflatoxins+ mycosorb treatments showed that dietary AF, significantly decreased the feed intake and weight gain as compared to the control diet (P<0.001). Treatment 2 resulted in inferior feed conversion ratio in comparison to the control diet (p<0.05). Uric acid and total protein in the second and third, and phosphorous only in the third treatment were significantly lower than the control (P<0.05). The *in-vivo* protein and dry matter digestibility, apparent metabolizable energy (AME) and nitrogen corrected-apparent metabolizable energy (AMEn) were not affected by the treatment (P>0.05). Aflatoxin resulted in the proventriculus enlargement, increased liver weight and AF deposition in the liver and breast muscle (P<0.05). This study clearly demonstrated that Mycosorb was effective in alleviating the adverse effects of dietary aflatoxins in broiler chickens.

Keywords: aflatoxin, broiler, mycosorb, protein digestibility

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Introduction

Aflatoxins are a class of mycotoxins produced by *Aspergillus* species including, *A. flavus*, *A. parasiticus*, and *A. nomius* (Kim et al., 2000). Seventeen aflatoxins have been isolated, but only B1, B2, G1 and G2, are the chief toxic metabolites produced by these fungi (Hussein and Brasel, 2001). These toxins occur worldwide in contaminated wheat, corn, soybean, and sorghum which are generally used in poultry rations (Miller, 1995). Aflatoxin B1 (AFB1) is one of the most important carcinogenic toxins. Several adverse effects including liver damage, poor performance and immunosuppression may occur by aflatoxins, and especially by AFB1 in poultry (Kubena et al., 1993). Liver characteristically becomes pale and enlarged as a result of aflatoxicosis, with microscopic changes including fatty

change, necrosis, and biliary hyperplasia (Hoerr, 1997). Aflatoxicosis in poultry may be manifested by a decrease in serum concentrations of total protein, albumin, cholesterol, triglyceride, glucose (Harvey et al., 1993; Edrington et al., 1997; Kubena et al., 1998), uric acid (Abo-Norag et al., 1995; Kececi et al., 1998), inorganic phosphorus and calcium (Fernandez et al., 1994).

Contamination of grains and other feedstuffs by fungi reduces the content of the protein, fat and energy up to 7%, 63% and 50% respectively. Moreover, lower protein and dry matter retentions and metabolizable energy were reported in poultry fed with moldy diets (Chen, 2003).

Decontamination processes of AF have been focused on degrading, destroying, inactivating or removing AF by physical, chemical or biological methods. Zeolites (Harvey et al., 1993; Kececi et al., 1998), such as hydrated sodium calcium aluminosilicate, bentonite (Santurio et al., 1999) a natural phyllosilicate (Kubena et al., 1993; Kubena et al., 1998; Ledoux et al., 1999) and activated charcoal (Edrington et al., 1997) have been used as adsorbents for reduction of AF toxicity in

Abbreviations:

AF: aflatoxin,
AME: apparent metabolizable energy,
AMEn: nitrogen corrected-apparent metabolizable energy,
AFB1: Aflatoxin B1,
BWG: body weight gain,
ELISA: enzyme -linked immunosorbent assay.

broiler chickens. However, a large-scale, practical, and cost-effective procedure is not currently available. Mycosorb application is another alternative proposed for this purpose. Mycosorb is a fine pale brown powder, insoluble in water which is made for addition to feed, not for direct dosing to animals. Mycosorb, a patented glucomannan-containing yeast product derived from yeast cell wall, has been known to adsorb different mycotoxins and alleviate their adverse effects in farm animals. Therefore, this study investigated the effect of mycosorb adsorbent in an AF-contaminated broiler diet on performance, organ weights, serum biochemical parameters, metabolizable energy and dry matter and protein digestibility.

Materials and methods

Animals and diets

This study was carried out at the Animal Research Station of Bu-Ali Sina University in March 2014 in Hamadan-Iran. All experimental protocols were approved by the Bu-Ali Sina University Animal Care Act.

Four hundred one-day-old Ross 308 broiler chicks were reared under control temperature and lighting conditions. A pre-starter diet was fed up to 7 days of age, after which the chicks were randomly allocated to one of four treatments containing four replicates (pens) of 25 birds per pen (11.2×1.2 meters). The dietary (NRC, 1994) treatments were: corn-soybean meal as a control; control diet plus 1mg kg⁻¹ AF; control diet plus 1mg kg⁻¹ AF and 0.25% mycosorb adsorbent; and control diet plus 0.25% mycosorb adsorbent (Table 1). The experiment lasted 42 days. Starter and grower diets were offered *ad libitum*, beginning at 8 and 22 days of age, respectively. Feed intake, body weight gain, feed conversion ratio (FCR), broiler production index (daily weight gain (g) × survival (percent) / 10 × FCR) and mortality were determined weekly. Finally, 2 birds from each replicate were weighed and slaughtered at 21 and 42 days of age to determine the effect of treatment on the weight of the liver, kidney, spleen, pancreas, heart, abdominal fat, proventriculus and gizzard. Proventriculi were quantitatively evaluated for enlargement. In fact, when their length were increased, the organ was enlarged. Gizzard erosion was numerically determined according to the presence of local lesions on the gizzard surface. Aflatoxin was also measured in the liver and breast muscle.

Preparation of aflatoxins

Aspergillus parasiticus ATCC 143473 was supplied by

Table 1. Composition and calculated nutrient contents of diets

Ingredient (%)	Starter ^a	Grower ^a
Corn	56.36	66.37
Soybean meal	32.94	26.46
Fish meal	3.5	2
Sunflower oil	3.54	1.62
DCP ^b	0.97	0.74
Oyster shell	1.18	1.29
Salt	0.26	0.27
Mineral premix ^c	0.25	0.25
Vitamin premix ^d	0.25	0.25
AF- Contaminated corn ^e	-	-
mycosorb adsorbent	-	-
Filler ^f	0.75	0.75
Total	100.00	100.00
Calculated composition (%)		
ME (kcal kg ⁻¹)	3000	3000
Crude protein	21.56	18.75
Calcium	0.42	0.33
Available Phosphorus	0.94	0.33
Lysine	1.28	1.05
Methionine	0.52	0.44

^aThe dietary treatments were T₁: Control diet as per NRC requirement; T₂: T₁ + 0.32 g of AF- contaminated corn; T₃: T₂ + 0.25% mycosorb in diet; T₄: T₁ + 0.25% mycosorb in diet.

^bDCP: Dicalcium phosphate.

^cMineral premix supplied per kg diet: MnSO₄ H₂O, 64 g; ZnCO₃, 44 g; FeSO₄ 7H₂O, 100 g; CuSO₄ 5H₂O, 16 g; KI, 0.64 g.

^dVitamin premix supplied per kg of diet: thiamin, 3.3 g; riboflavin, 0.72g; menadione dimethylpyrimidinol bisulfate, 1.6 g; dl- α -tocopherol acetate, 14.4 g; cholecalciferol, 7 g; retinyl acetate 7.7 g; D-Ca-pantothenate acid, 12 g; pyridoxine, 6.2 mg; B₁₂ 14.4 g; choline, 440 mg.

^e1mg AFs was added to 0.32 g of corn.

^fA filler (straw) was used up to 0.75% in diets.

the Veterinary school of Tehran University, Iran. Aflatoxin was incubated on 10 plates containing with potato dextrose agar (PDA) medium, and the plates were kept in an oven at 30°C for 5 days (OSK 95000; electric oven, Ogawa Seiki Co. Ltd., Tokyo, Japan). Ground corn in four flasks was used for AF production. The flasks were kept in an autoclave at 120°C (OSK, Ogawa Seiki Co. Ltd., Tokyo, Japan). The proliferated fungi were then added and completely mixed with the specimens under sterile conditions. Contaminated corn samples were kept in an incubator at 26°C and 50% moisture for 30 days. The contents of the flask were dried in an oven (OSK 95000; electric oven, Ogawa Seiki Co. Ltd., Tokyo, Japan) at 50°C for 72 hours and ground into powder; The aflatoxin level was measured by a direct competitive ELISA (Barabolak, 1977) and mixed according to the calculation to get the desired level of aflatoxin in the feed. The diets were analyzed again to confirm their AF content.

Serum biochemical analysis

At the end of the experiment, 3 mL of blood was obtained from the brachial vein of 8 chickens from each treatment. Serum total cholesterol, uric acid, total protein and glucose concentration were determined by kits (Pars Azmoon and Ziest Chem, Tehran, Iran).

Determination of aflatoxin in the liver and breast muscle

Aflatoxin contents of the liver and breast muscle at 21 and 42 days of age were determined by ELISA (Barabolak, 1977). At first, 2 g of each sample was weighed and extracted with 10 ml of 70% methanol. This mixture was homogenized for 10 min at 18 °C and then the resultant deposit was centrifuged. The supernatant (100 µL) was diluted with 600 µL of phosphate buffer. This solution (50 µL) or standard solution (50 µL), 50 µL of the aflatoxin-peroxidase conjugate and 50 µL of the mouse antibody solution against aflatoxin were added to each well of the used plate. In fact, plate is a part of kit (8 wells + 12 strips). Then all samples were incubated for 30 min at 18 °C in darkness. At the Next stage, the plate was emptied and washed with phosphate buffer. Then, tetramethylbenzidine (50 µL) and urea peroxide (50 µL) were added and incubated again for 30 min in darkness. Finally, stop reagent (100 µL) was add to terminate reaction. Solution absorbance was also obtained at a wavelength of 450 nm, using an ELISA reading apparatus. At lastly, aflatoxin content of each sample was calculated according to the standard curve which was previously prepared.

Protein and dry matter digestibility; and metabolizable energy determination

Two 21- days-old broiler chicks were randomly placed per cage, to measure metabolizable energy, protein and dry matter digestibility. Then, cages (experimental units) wererandomly assigned to treatments. Chromic oxide was included in the diets as an indigestible marker. The diets and water were offered *ad libitum* for 7 days. Excreta were collected twice daily during for the last 3 days and frozen (-20°C). The excreta from each replicate were pooled and dried in an oven at 50 °C for 16 hour prior to grinding and analysis (Newkirk et al., 2003).

The excreta digestibility of diet protein based on uric acid was calculated as described by Marquardt method (1983). The excreta samples were analyzed for uric acid content. The protein content of excreta was

corrected based on uric acid as follows:

$$\left(\frac{\% \text{ nitrogen in excreta} - \% \text{ nitrogen of uric acid in excreta}}{\% \text{ nitrogen of uric acid in excreta}} \right) \times 6.25 \quad (1)$$

With these corrected data, excreta digestibility of protein based on uric acid was calculated with the following formula:

$$DC_{diet} = 1 - \left[\left(\frac{M_{diet}}{M_e} \right) \times \left(\frac{C_e}{C_{diet}} \right) \right] \quad (2)$$

DC_{diet}, digestibility coefficient of protein in diet; M_{diet}, marker concentration in diet; M_e, marker concentration in excreta; C_{diet}, protein concentration in diet; C_e, protein concentration in excreta.

Apparent metabolizable energy and nitrogen corrected-apparent metabolizable energy were measured according to Newkirks (2003).

$$AME_{diet} = GE_{diet} - \left[\left(\frac{M_{diet}}{M_e} \right) \times GE_{excreta} \right] \quad (3)$$

AME_{diet}, is the apparent metabolizable energy of the diet; GE_{diet}, gross energy of the diet; M_{diet}, marker concentration in the diet; M_e, marker concentration in excreta; GE_{excreta}, gross energy of excreta.

$$AMEn_{diet} = AME_{diet} - \left[N_{diet} - \left(\frac{M_{diet}}{M_e} \times N_{excreta} \right) \right] \times 8.73 \quad (4)$$

AMEn_{diet}, nitrogen corrected-apparent metabolizable of the diet; AME_{diet}, Apparent metabolizable energy of the diet; N_{diet}, nitrogen content of the diet; M_{diet}, marker concentration of the diet; M_e, marker concentration in excreta; N_{excreta}, nitrogen content in excreta; 8.73, constant coefficient.

Chromic oxide was measured by the method of Fenton and Fenton (1979). Gross energy contents of diets and excreta were determined using a traditional adiabatic bomb calorimeter.

Statistical analysis

The experiment was set up as completely randomized designs (CRD). Data were analyzed by one-way analysis of variance (ANOVA) using the General linear Model (GLM) procedure (SAS Institute 2004). A chi-squared test was applied to gizzard erosion incidence data to determine the differences between samples isolated from AFs-contaminated treatments. When the F test was significant (P<0.05), the means were compared using the Duncan's multiple range test.

Results

Feed intake, body weight gain, and broiler production

index of AF diet were significantly lower ($P < 0.001$) than the Mycosorb and control diets (Table 2). The inclusion of Mycosorb to the contaminated diet significantly increased ($P < 0.001$) the feed intake, body weight gain and broiler production index as compared to the aflatoxin treatment (Table 2). In AF contaminated diets, FCR was higher ($P < 0.05$) than the control diet. Feed conversion ratio in the diets containing AF was higher ($P < 0.05$) than the control diet. In contrast, FCR was not influenced ($P > 0.05$) by mycosorb treatment compared with the control. Serum uric acid ($P < 0.01$), total protein ($P < 0.001$) and phosphorous ($P < 0.05$) levels were observed at 42 days of age in the chickens fed with AF contaminated diets in comparison with the control diet (Table, 3). No significant differences were observed in the serum concentration of cholesterol, glucose and calcium.

With the exception of gizzard and liver relative weights, no differences were found in the weight of organs at 42 days of age (Table 4). Proventriculus enlargement was increased ($P < 0.05$) by AF treatment (1 mg kg^{-1}) compared with the control at 21 and 42 days of age (Table 4). Gizzard erosion score was numerically increased by feeding AF-contaminated diet (AF and AF plus mycosorb treatments) at 21 and 42

days of age (Table 4; Figure 1).

No differences were recorded in AME and AMEn contents, and *in-vivo* protein and dry matter digestibility among treatments. The mean values for AME and AMEn values were 2779.9 ± 27 and $2775.2 \pm 32 \text{ kcal}^{-1} \text{ kg}$, and for *in vivo* protein and dry matter digestibility were 64.5 and 78.1%, respectively. Aflatoxin contents ($\mu\text{g kg}^{-1}$) in the liver and breast muscle of chickens fed the AF diet (without Mycosorb) were ($P < 0.05$) higher than the other treatments at 21 and 42 days of age (Table 5).

Discussion

The most prevalent symptoms of aflatoxicosis in poultry are reduced growth rate and poor performance. This study showed a decrease in feed intake and growth rate in chickens receiving 1 mg kg^{-1} AF and 1 mg kg^{-1} AF plus 0.25% mycosorb in the diet in comparison with the control diet. The lower feed intake may be attributed to the presence of aflatoxin, which can depress appetite and ultimately reduce the growth rate (Miazzo et al., 2000). These results are in line with the findings of Edds and Bortell (1983) and Kubena et al. (1990).

The improvement in feed intake, weight gain and

Table 2. Effect of alfatoxin-contaminated diets with or without mycosorb on broiler performance at 42 days of age

Treatments	FI (g)	WG (g)	FCR	BI	Mortality (%)
Control	4192.2 ^a	2077.5 ^a	2.1 ^b	659.3 ^a	0.0
Aflatoxin	3839.6 ^c	1847.5 ^c	2.2 ^a	559.7 ^c	2.3
Aflatoxin + mycosorb	3964.0 ^b	1943.8 ^b	2.2 ^{ab}	602.3 ^b	2.0
mycosorb	4195.0 ^a	2117.5 ^a	2.1 ^{ab}	681.7 ^a	0.0
SEM	19.45	21.43	0.02	6.72	0.76
P value	***	***	*	***	ns

FI: Feed intake; WG: Weight gain; FCR: Feed conversion ratio;

BI: Broiler index = Daily weight gain (g) × Survival (percent) / 10 × FCR;

SEM: Standard error of the mean.

^{a-c} Values within a column with common superscript(s) do not differ. ns: Not significant ($P > 0.05$); *: ($P < 0.05$); ***: ($P < 0.001$).

Table 3. Effect of alfatoxin-contaminated diets with or without mycosorb on serum biochemical parameters in broilers at 42 days of age

Treatments	Ch	UA	TP	Gl	Ca	P
Control	103.4	10.2 ^a	3.4 ^a	246.3	8.3	3.9 ^a
Aflatoxin	100.5	8.2 ^c	2.8 ^b	241.3	7.8	3.3 ^b
Aflatoxin + mycosorb	101.9	8.7 ^{bc}	2.8 ^b	243.9	8.1	3.6 ^{ab}
mycosorb	106.3	9.8 ^{ab}	3.4 ^a	245.0	8.2	3.8 ^a
SEM	3.03	0.37	0.10	4.49	0.33	0.13
P value	ns	**	***	ns	ns	*

Ch: Cholesterol, (mg/dL); UA: Uric acid, (mg/dL); TP: Total protein, (g/dL); Gl: Glucose, (mg/dL);

Ca: Calcium, (mg/dL); P: Phosphorous, (mg/dL); SEM: Standard error of the mean.

^{a-c} Values within a column with common superscript(s) do not differ. ns: Not significant ($P > 0.05$); *: ($P < 0.05$); ***: ($P < 0.001$).

Table 4. Effect of aflatoxin-contaminated diets with or without mycosorb on organ and abdominal fat relative weights (g/100 g of live weight) of broilers at 42 days of age (%)

Treatments	21 days		42 days										
	Ge ¹	Pe ²	Ge ¹	Pe ²	G	P	L	k	H	S	P	F	T
Control	-	2.7 ^b	-	3.6 ^b	1.6 ^b	0.4	2.4 ^b	0.5	0.6	0.1	0.3	2.9	0.5
Aflatoxin	3	2.9 ^a	3.0	3.8 ^a	1.8 ^a	0.4	2.7 ^a	0.7	0.7	0.1	0.3	2.9	0.5
Aflatoxin + mycosorb	2	2.8 ^{ab}	3.0	3.7 ^{ab}	1.7 ^a	0.4	2.6 ^a	0.6	0.6	0.1	0.3	2.9	0.5
mycosorb	-	2.6 ^b	-	3.6 ^b	1.5 ^b	0.4	2.3 ^b	0.5	0.6	0.1	0.3	2.3	0.5
SEM	0.49	0.07	0.53	0.05	0.05	0.05	0.07	0.03	0.46	0.03	0.02	0.23	0.01
P value	ns	*	ns	*	**	ns	*	ns	ns	ns	ns	ns	ns

Ge: Gizzard erosion; Pe: Proventriculus enlargement; G: Gizzard; P: Proventriculus ; L: Liver; K: kidney ; H: Heart; S: Spleen; P: Pancreas; F: Fat ; T: Tibia; ¹gizzard erosion (number) ; ²Proventriculus enlargement (cm); ³g/100 g body weight. SEM: Standard error of the mean.

^{a-b} Values within a column with unlike superscripts differ significantly. ns: Not significant (P>0.05); *: (P<0.05); **: (P<0.01).

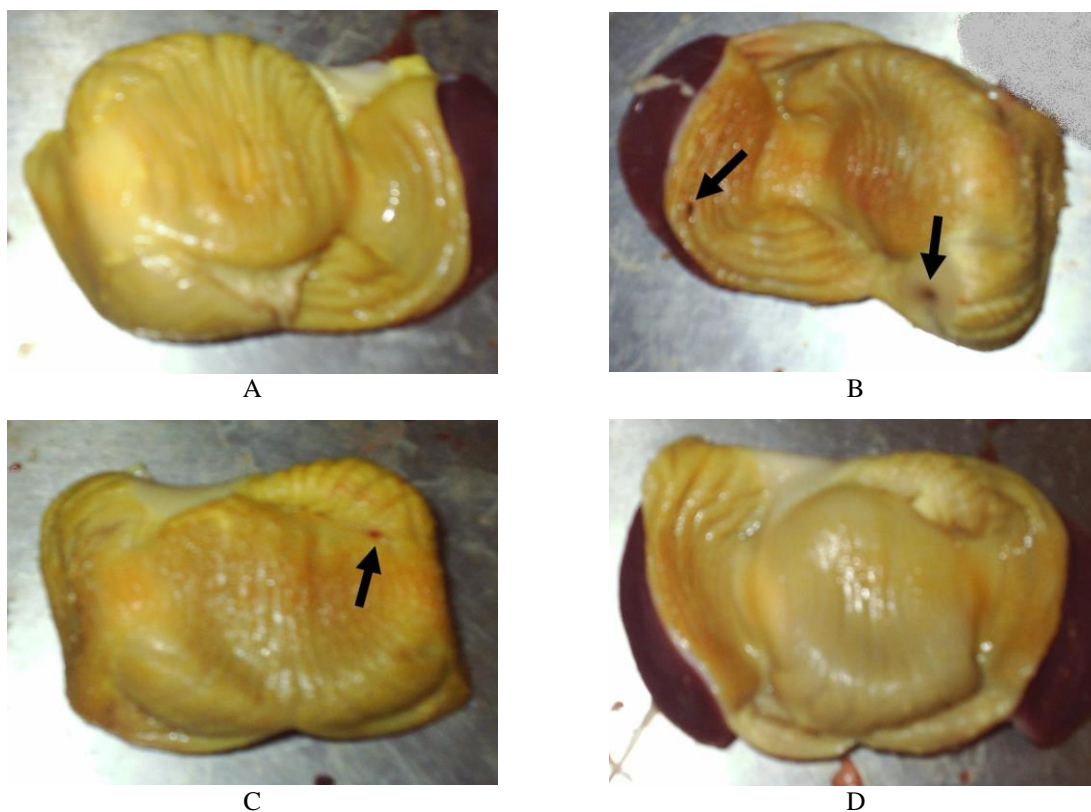


Figure 1. Gizzard erosion is shown with arrows (A: Control, healthy gizzard); (B: Control and 1 mg kg⁻¹ AFs, gizzard erosion); (C: Control, 1 mg kg⁻¹ AFs and 0.25% mycosorb absorbent, gizzard erosion); (D: Control and 0.25% mycosorb absorbent, healthy gizzard).

broiler production index of chickens due to mycosorb supplementation to AF-contaminated diet indicated that mycosorb may be a suitable aflatoxin absorbent and can form a stable complex to reduce the toxic effects of AFs and improve performance.

Significant decrease in total serum protein in birds fed the AF diets (treatments 2 and 3) could be attributed to reduced hepatic protein synthesis (Tung et al., 1975) resulting from the hepatotoxicity seen in aflatoxicosis (Tung et al., 1975; Huff et al., 1986).

Decreased serum uric acid concentration as a result of feeding AF-contaminated diet is in agreement with the findings of Harvey et al. (1993) and Ledoux et al. (1999) not others (Abdel-Rahman et al., 2002; Fatemi et al., 2006; Scholl et al., 2006). It is well established that reduced hepatic protein synthesis can decrease utilization of dietary amino acids resulting in increased uric acid synthesis as amino acids are oxidized as a source of energy (Tung et al., 1975). In the other words, there is a negative correlation between total

Table 5.Effect of aflatoxin-contaminated diets with or without mycosorb on aflatoxin concentration in liver and breast muscle at 21 and 42 days of age ($\mu\text{g kg}^{-1}$)

Treatments	21 days		42 days	
	L	Bm	L	Bm
Control	0.00 ^{lb}	0.00 ^b	0.00 ^b	0.00 ^b
Aflatoxin	0.42 ^a	0.03 ^a	0.60 ^a	0.11 ^a
Aflatoxin+ mycosorb	0.00 ^b	0.00 ^b	0.18 ^b	0.00 ^b
mycosorb	0.00 ^b	0.00 ^b	0.00 ^b	0.00 ^b
SEM	0.03	0.002	0.04	0.02
P value	***	***	***	***

L: Liver; Bm: Breast muscle; SEM: Standard error of the mean.

^{a-b} Values within a column with unlike superscripts differ significantly.

ns: Not significant ($P>0.05$); *: ($P<0.05$); **: ($P<0.01$).

serum protein and uric acid concentration (Tung et al., 1975) while adverse response was recorded in the present study. Aflatoxin is an oxidant (Shaaban et al., 2010) and uric acid is an antioxidant substance (Machin et al., 2004); therefore, it seems that a portion of uric acid was used for modulating free radicals which are caused by aflatoxin (Table 3).

In the current work, there were no significant differences in serum glucose levels among treatments, indicating that AF treatment had no negative effect on carbohydrate metabolism. The result is consistent with Tedesco et al. (2004) and Zhao et al. (2010) who used lower than 1 mg kg^{-1} AF in the diet. Contrary to this, Ledoux et al. (1999) and Zhao et al. (2010) reported an increase in serum glucose level when AF was used at levels of 2 and 4 mg kg^{-1} . Therefore, it seems that serum glucose level could be increased at concentrations greater than 2 mg kg^{-1} of AFs in poultry diets.

Compared with the control, chickens fed AF alone had ($P<0.05$) lower serum phosphorous concentration which is agreement with previous studies (Ledoux et al., 1999; Eraslan *et al.*, 2002; Zhao et al., 2010). It has been shown that AF toxicity in broilers may be manifested by decreased serum concentrations of phosphorus and calcium values (Harvey et al., 1993).

The weight of liver and gizzard increased by feeding AF-contaminated diets consistent with previous reports (Huff and Doerr 1981; Huff et al., 1986; Miaz-zo et al., 2005; Pasha et al., 2006) that showed liver and gizzard relative weights were affected by AF. The relative weight of kidney was numerically higher for broilers fed the AF-contaminated diets as also shown previously (Huff and Doerr 1981; Kubena et al., 1995; Tung et al., 1975); this may be due to slight renal damage (Mckenzie et al. 1998).

Protein digestibility and metabolizable energy values were not affected by AF. Contradictory reports have been published in this regard. For instance, it was

reported that dry matter, nitrogen and amino acid digestibilities and metabolizable energy were negatively affected by AF (Devegowda and Murthy, 2005; Surai and Dvorska, 2005). In contrast, other studies did not show any differences in digestibility and metabolizable energy content of diet (Nelson et al., 1982; Applegate et al., 2009).

The presence of AF residues in the edible tissues including the liver and breast muscle of the chicken fed the AF-contaminated diet (0.03 to $0.60 \mu\text{g kg}^{-1}$ by feeding 1 mg kg^{-1} AF in the diet) was confirmed in this study. The liver AF level obtained in the present study was comparable with that reported in few studies. Residues of AFB1 in the liver of broilers and layers varied from no detection to $3.0 \mu\text{g kg}^{-1}$ by feeding 250 – $3310 \mu\text{g kg}^{-1}$ AFB1 for different periods (Gregory et al., 1983; Chen et al., 1984; Wolzak et al., 1986; Bintvihok et al., 2002). The wide variations in AF concentration suggested that these levels might be influenced by many factors including the dietary AF level, duration of administration, age, and type of birds. In this study, the effect of AF was modulated by Mycosorb which was reflected in broiler organs. Dietary contamination of aflatoxins poses a big risk to human health including acute aflatoxicosis, hepatocellular carcinoma, hepatitis B virus infection, and growth impairment in some regions, particularly in Asian and African countries (Wild and Gong, 2010). The European community and many other countries have imposed 2 ng/g AFB1 as maximum tolerance level in human food products (Anonymous, 2004). Nonetheless, the level of AF in human food should be kept as low as possible to reduce the incidence of health problem in human.

Finally, the adverse effects of AF on feed intake, weight gain, broiler index and AF content in edible tissues including the liver and breast muscle were ameliorated by inclusion of mycosorb (2.5 g kg^{-1}) to the AF-contaminated diet.

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References

- Abdel-Fattah, S., Abosrea, Y.H., Shehataf, E., Flourage, M.R., Helal, A.D., 2010. The Efficacy of thyme oil as anti-toxicant of aflatoxin (s) toxicity in sheep. *Journal of Animal Science* 6, 948-960.
- Abdel-Rahman, S.S., Sheble, M.A., Abdel-Azeem, A., Rawi, S.M., 2002. Biochemical and pathological studies on hepatic and renal alterations of male albino rats given aluminum chloride and/or aflatoxin B1. *Egyptian Journal of Clinical Pathology* 15, 57-73.
- Abo-Norag, M., Edrington, T.S., Kubena, L.F., Harvey, R.B., Phillips, T.D., 1995. Influence of hydrated sodium calcium aluminosilicate and virginiamycin on aflatoxicosis in broiler chicks. *Poultry Science* 74, 626–632.
- Applegate, T.J., Schatzmayr, G., Prickett, K., Troche, C., Jiang, Z., 2009. Effect of aflatoxin culture on intestinal function and nutrient loss in laying hens. *Poultry Science* 88, 1235–1241.
- Barabolak, R. J., 1977. Improved procedure for quantitative determination of aflatoxin in corn and wet milled corn products. *Journal of the Association of Official Analytical Chemists*, 60, 308-310
- Chen, Y.J., 2003. Mould and mycotoxin: Control from grain to feeding. Feed International. 6th Ed. December. Volume 24, pp. 22-26.
- Devegowda, G., Murthy, T.N.K., 2005. Mycotoxins: Their effects in poultry and some practical solutions. In: Diaz, D.E. (Eds.), the Mycotoxin Blue Book. Nottingham University Press, Nottingham, UK, pp. 25–56.
- Edds, G.T., Bortell, R.A., 1983. Biological effects of aflatoxins: poultry aflatoxin and *Aspergillus flavus* in corn. In: Bulletin of the Alabama Agricultural Experiment Station. (Eds), U.L. Diener, R.L. Asquit, J.W. Dickens. Alabama, AL., Auburn University, pp. 64–66.
- Edrington, T.S., Kubena, L.F., Harvey, R.B., Rottinghaus, G.E., 1997. Influence of a superactivated charcoal on the toxic effects of aflatoxin or T-2 toxin in growing broilers. *Poultry Science* 76, 1205–1211.
- Fatemi, F., Allameh, A., Dadkhah, A., Forouzandeh, M., Kazemnejad, S., Sharifi, R., 2006. Changes in hepatic cytosolic glutathione S-transferase activity and expression of its class-P during the prenatal and postnatal period in rats treated with aflatoxin B1. *Archives of Toxicology* 80, 572-579.
- Fenton, T. W., & FENTON, M. (1979). An improved procedure for the determination of chromic oxide in feed and feces. *Canadian Journal of Animal Science* 59, 631-634.
- Fernandez, A., Verde, M., Gascon, M., Ramos, J., Gomez, J., Luco, D.F., Chavez, G., 1994. Variations of clinical, biochemical parameters of laying hens and broiler chickens fed aflatoxin-containing feed. *Avian Pathology* 23, 37–47.
- Harvey, R.B., Kubena, L.F., Ellissalde, M.H., Phillips, T.D., 1993. Efficacy of zeolitic ore compounds on the toxicity of aflatoxin to growing broiler chickens. *Avian Disease* 37, 67–73.
- Hoerr, F.J., 1997. Mycotoxicosis. In: Calnek, B.W., Barnes, H.J., Beard, C.W., McDougald, L.R., Saif, Y.M. (Eds.), Diseases of Poultry. Iowa State University Press, Ames, IA. pp. 958–962.
- Huff, W.E., Doerr, J., 1981. Synergism between aflatoxin and ochratoxin an in broiler chickens. *Poultry Science* 60, 550-555.
- Huff, W.E., Kubena, L.F., Harvey, R.B., Corrier, D.E., Mollenhauer, H.H., 1986. Progression of aflatoxicosis in broiler chickens. *Poultry Science* 65, 1891–1899.
- Hussein, H.S., Brasel, J.M., 2001. Review: Toxicity, metabolism, and impact of mycotoxins on humans and animals. *Toxicology* 167, 101–134.
- Kececi, T., Oguz, H., Kurtoglu, V., Demet, O., 1998. Effects of polyvinylpyrrolidone, Synthetic zeolite and bentonite on serum biochemical and hematological characters of broiler chickens during aflatoxicosis. *British Poultry Science* 39, 452–458.
- Kim, B., Kim, D., Park, R., Kwon, K., Ryu, D., Kim, Y., Kim, N., Jeong, S., Kang, B., Kim, K., 2000. Effect of an extract of the root of *Scutellaria baicalensis* and its flavonoids on aflatoxin B1 oxidizing cytochrome P450 enzymes. *Planta Medica* 67, 396–399.
- Kubena, L.F., Edrington, T.S., Kamps-Holtzapple, C., Harvey, R.B., Ellissalde, M.H., Rottinghaus, G.E., 1995. Influence of fumonisin B1, present in *Fusarium moniliform* culture material, and T-2 toxin on turkey poult. *Poultry Science* 74, 306–313.
- Kubena, L.F., Harvey, R.B., Bailey, R.H., Buckley, S.A., Rottinghaus, G.E., 1998. Effects of hydrated sodium calcium aluminosilicate (T-Bind™) on mycotoxicosis in young broiler chickens. *Poultry Science* 77, 1502–1509.
- Kubena, L.F., Harvey, R.B., Phillips, T.D., Clement, B.A., 1993. Effects of hydrated sodium calcium aluminosilicate on aflatoxicosis in broiler chicks. *Poultry Science* 72, 651–657.
- Kubena, L.F., Harvey, R.B., Phillips, T.D., Corrier, D.E., Huff, W., 1990. Diminution of aflatoxicosis in growing chickens by the dietary addition of a hydrated, sodium calcium aluminosilicate. *Poultry Science* 69, 727–735.
- Ledoux, D.R., Rottinghaus, G.E., Bermudez, A.J., Alanso-Debolt, M., 1999. Efficacy of hydrated sodium calcium aluminosilicate to ameliorate the toxic effects of aflatoxin in broiler chicks. *Poultry Science* 78, 204–210.

- Machin, M., Simoyi, M.F., Blemings, K.P., Klandorf, H., 2004. Increased dietary protein elevates plasma uric acid and is associated with decreased oxidative stress in rapidly-growing broiler. *Comparative Biochemistry and Physiology* 137, 383-390.
- Mckenzit, K.S., Kubenta, L.F., Denvir, A.J., Rogers, T.D., Hitchens, G.D., Bailey, R.H., Harvey, R.B., Buckley, S.A., Philips, T.D., 1998. Aflatoxicosis in turkey poult is prevented by treatment of naturally contaminated corn with ozone generated by electrolysis. *Poultry Science* 77, 1094-1102.
- Miazzo, R., Peralta, M.F., Magnoli, C., Salvano, M., Ferrero, S., Chiacchiera, S.M., Carvalho, E.C.Q., Rosa, C.A.R., Dalcerro, A., 2005. Efficacy of sodium bentonite as a detoxifier of broiler feed contaminated with aflatoxin and fumonisin. *Poultry Science* 84, 1-8.
- Miazzo, R., Rosa, C.A.R., Queiroz Carvalho, E.C., De Magnoli, C., Chiacchiera, S.M., Palacio, G., Saenz, M., Kikot, A., Basaldella, E., Dalcerro, A., 2000. Efficacy of synthetic zeolite to reduce the toxicity of aflatoxin in broiler chicks. *Poultry Science* 79, 1-6.
- Miller, J.D., 1995. Fungi and mycotoxins in grain implications for stored product research. *Journal of Stored Products Research* 31, 1-16.
- National Research Council, 1994. Nutrient Requirements of Poultry. 9th rev. ed. National Academy Press, Washington, DC.
- Nelson, T.S., Johnson, Z.B., Kirby, L.K., Beasley, J.N., 1982. Digestion of dry matter and amino acids and energy utilization by chicks fed molded corn containing mycotoxins. *Poultry Science* 61, 584-585.
- Newkirk, R.W., Classen, H.L., Edney, M. J., 2003. Effects of prepress-solvent extraction on the nutritional value of canola meal for broiler chickens. *Animal Feed Science and Technology* 104, 111-119.
- Pasha, T.N., Farooq, M.U., Khattak, F.M., Jabbar, M.A., Khan, A.D., 2006. Effectiveness of sodium bentonite and two commercial products as aflatoxin absorbents in diets for broiler chickens. *Animal Feed Science and Technology* 71, 624-636.
- Santurio, J.M., Mallmann, C.A., Rosa, A.P., Appel, G., Heer, A., Dageforde, S., Bottcher, M., 1999. Effect of sodium bentonite on the performance and blood variables of broiler chickens intoxicated with aflatoxin. *British Poultry Science* 40, 115-119.
- SAS Institute. 2004. SAS User's Guide, Version 8. SAS Institute, Inc., Cary, NC.
- Scholl, P.F., McCoy, L., Kensler, T.W., Groopman, J.D., 2006. Quantitative analysis and chronic dosimetry of the aflatoxin B1 plasma albumin adduct lys-AFB1 in rat by isotope dilution mass spectrometry. *Chemical Research in Toxicology* 19, 44-49.
- Surai, P.F., Dvorska, J.E., 2005. Effects of mycotoxins on antioxidant status and immunity. In *The Mycotoxin Blue Book*, Diaz, D.E., Ed.; Nottingham University Press: Nottingham, UK, pp. 93-137.
- Tedesco, D., Steidler, S., Galletti, S., Tameni, M., Sonzogni, O., Ravarotto, L., 2004. Efficacy of silymarin-phospholipid complex in reducing the toxicity of aflatoxin B1 in broiler chicks. *Poultry Science* 83, 1839-1843.
- Tung, H.T., Wyatt, R.D., Thaxton, P., Hamilton, P.B., 1975. Concentrations of serum proteins during aflatoxicosis. *Toxicology and Applied Pharmacology* 34, 320-326.
- Zhao, J., Shirley, R.B., Dibner, J.D., Uraizee, F., Officer, M., Kitchell, M., Vazquez-Anon, M., Knight, C.D., 2010. Comparison of hydrated sodium calcium aluminosilicate and yeast cell wall on counteracting aflatoxicosis in broiler chicks. *Poultry Science* 89, 2147-2156.

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