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Toxin and toxin-binders affecting the performance, organs, haematology and histological characteristics of broilers fed with infected diets

Rafiu T. A.[1*], Babatunde G. M.[1], Ibrahim O. O. K.[2], Akanbi A. O.[1] and Ojelade R. A.[1]

[1]Animal Production and Health Department, Faculty of Agricultural Sciences, Ladoke Akintola University of Technology, Ogbomoso, Nigeria.
[2]Department of Pathology, Faculty of Basic Medical Sciences, University of Ilorin Teaching Hospital (UITH), Ilorin, Nigeria.

The potency of four varieties of toxin binders (labeled A, B, C and D) on the physiological status of broiler birds fed with diets infected with aflatoxin was investigated. Six experimental diets were formulated: Diet 1 was infected with aflatoxin without binders, Diets 2 to 5 were infected but binders A, B, C or D were added, while Diet 6 was the control diet without infection. 180 birds were equally and randomly assigned to the 6 treatments and raised for 8 weeks. Their weights and feed consumptions were monitored. After eight weeks, 3 birds per replicate were randomly selected, starved and slaughtered. Their organs were obtained and weighed. Their blood and liver samples were collected for histological examination. Only the animals fed with control diet had significant difference (p<0.05) in the average daily feed intake at starter phase. While T5 (infected diet + activated charcoal) had significantly higher (p<0.05) feed intake and best feed conversion ratio (3.44) at finisher phase. Kidney proportion of birds from T1 was significantly (p<0.05) higher than others while control had the least value (0.53%). Hemoglobin, lymphocyte and heterophils were not influenced (p>0.05) among blood parameters. Histological observation of liver revealed inflammation of hepatocyte. Conclusively, the use of activated charcoal as toxin binder gave optimum performance compared to the other binders.

Key words: Aflatoxin, infected feed, physiology, toxin-binder.

INTRODUCTION

Poultry is by far the largest group of livestock and is estimated to be about 14,000 million consisting mainly of chickens, ducks, and turkeys (Udoh and Etim, 2007). It is the most commonly kept livestock and over 70% of those keeping livestock are reported to be keeping chickens (Udoh and Etim, 2007). Poultry production is of considerable significance to the rural as well as national

economy; it is an important source of animal protein (FOS, 1999). Chicken species constitute about 98% of the total poultry production in Africa (Guèye, 2003). This has however prompted researchers to look into broiler rearing and its feeding. Mycotoxins are toxic secondary metabolites of certain fungi and cause illness or death when ingested by animals or human beings (Qazi and

Fayyaz, 2006) especially when the ingestion is above the lethal/tolerance level. Mycotoxin contamination is very costly for the animal industry and it is of significant importance to investigate its safety concern because of the potential mycotoxin residue in meat, dairy and egg (Pandey and Chauhan, 2007; Denli et al., 2009). The most significant economic cost of mycotoxin in poultry is reduced growth rate and high mortality rate in farm animal (Alaa et al., 2015). It is also classified as human carcinogen (Talebi et al., 2011). Cereal grains and their by-products are important ingredients in poultry feed ration; however many of supplied cereals intended for animal feed are frequently contaminated with mycotoxins (Gowda et al. 2008).

One of the most toxic mycotoxins is aflatoxin. Aflatoxins (AFs) are toxic secondary metabolites produced by *Aspergillus* spp. They are natural contaminants of poultry feed ingredients such as maize, millet, rice, peanut meal, and cottonseed meal (Reddy et al., 2000). The increasing knowledge and awareness of aflatoxin as a potent source of health hazards to both man and farm animals make producers, researchers and government organizations to intensify efforts on preventive management and decontamination technologies to minimize the content of aflatoxin in food and feed (Galvano et al., 2005). In order to reduce the toxic and economic impact of aflatoxin, established regulations and legislative limits have been set for aflatoxin in poultry feed. Many countries follow a maximum acceptable level of 20 ppb for aflatoxin in poultry feed (CAST, 2003). Pre and post-harvest contamination can be reduced by using appropriate agricultural practices. However, the contamination is often unavoidable and remains a serious problem associated with many agricultural commodities, and this emphasizes the necessity for a suitable process to inactivate the toxins (Galvano et al., 2005). Since the beginning of the 1990s, adsorbent-based studies have been reported to be effective in minimizing aflatoxin contamination in feed (Ibrahim et al., 2000).

A possible strategy to minimize unavoidable effect of afflatoxins could be the use of toxin binders in feed. The active ingredient(s) are mostly synthetic, not readily available and differ from one binder to the other (Rafiu et al., 2014); this makes their effectiveness varies from one to the other. The present investigation focuses on evaluation of potency of various available toxin binders in poultry diets compared to activated charcoal which is more available and affordable even in rural areas.

MATERIALS AND METHODS

Test ingredients

Various toxin binders were procured and labeled as A, B, C and D. According to the manufacturers, Binder A contains Hydrated Sodium Calcium Aluminosilicates (HSCAS) as the active ingredient; Binder B contains Bentonite/Montmorillonite Yeast cell walls, Binder C contains Aromatic Polyphenols and Binder D contains Activated charcoal.

Toxin production and analysis

Toxin was produced by the inoculation of fungus *Aspergillus flavus* which was carried out using semovita. Moistened semovita was stored in a dark cupboard to enable rapid spoilage. The organism was then isolated and cultured on a Petri dish using potato dextrose agar (PDA) as the growing medium. It was incubated at 27°C for 6 days. Semi-synthetic medium containing 2 g yeast extract and 20 g sucrose in every 100 ml of distilled water (all inside fermentation bottle) was used as basal fermenting medium for the organism to produce toxin. Fermentation bottles were sterilized in an autoclave at 121°C for 15 min to remove or eliminate any form of contamination. The fermenting medium was allowed to cool to about 45°C following sterilization after which the organisms were inoculated in a sterile environment, placed in a shaker and allowed to stand for 6 days.

Experimental diets

Six experimental diets containing the same crude protein and metabolizable energy (MJME/kg) were formulated for the starter phase and finisher diets (Table 1). Five diets (1- 5) were infected with the prepared toxin. Binders A, B, C and D were incorporated into diets 2, 3, 4 and 5, respectively. The toxin binders (binders A, B, C and Activated charcoal) were included in the diets at the rate of 1, 1.5, 2.25 and 10 g/kg of A, B, C and D (activated charcoal) respectively as recommended by the respective manufacturers.

Experimental birds

One hundred and eighty (180) days old commercial broiler chicks were procured and weighed. They were randomly and equally allotted into six (6) treatments of 3 replicates of ten chicks each, making a total of 30 birds in each treatment under a completely randomized design.

Data and samples collection

Average feed intake, body weight gain and feed conversion ratio were estimated from related data that were recorded.

Blood samples

At the end of the experiment, blood samples were collected from three birds per replicate and drained into two differently labeled tubes for haematological and serum investigation. The blood samples for haematological parameters were collected into tubes pretreated with Ethylene Diamine Tetra Acetic acid (EDTA) anti-coagulant, while samples for serum indices were collected into tubes without EDTA pre-treated. Investigated serum biochemical indices include total protein (TP), albumin, cholesterol, alanine amino transferase (ALT), aspartate amino transferase (AST), alkaline phosphatase (ALP) while analyzed haematological indices include white blood cell (WBC) counts, red blood cell (RBC) counts, packed cell volume (PCV), lymphocyte, platelet and hemoglobin.

Liver samples and organs weight

Liver from sampled birds was obtained, fixed in 20% formalin solution, taken to the laboratory, stained and further processed for

Table 1. Gross composition of experimental broiler diets (starter and finisher).

Ingredients	Starter diets						Finisher diets					
	T_1	T_2	T_3	T_4	T_5	T_6	T_1	T_2	T_3	T_4	T_5	T_6
Maize	55.00	55.00	55.00	55.00	55.00	55.00	50.00	50.00	50.00	50.00	50.00	50.00
Soy bean meal	15.00	15.00	15.00	15.00	15.00	15.00	14.00	14.00	14.00	14.00	14.00	14.00
Wheat offal	10.00	10.00	10.00	10.00	10.00	10.00	15.00	15.00	15.00	15.00	15.00	15.00
Groundnut cake	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00
Fish meal	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00	3.00
Bone meal	2.50	2.50	2.50	2.50	2.50	2.50	4.50	4.50	4.50	4.50	4.50	4.50
Oyster shell	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10
Methionine	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10	0.10
Lysine	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25
Premix	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25	0.25
Salt	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30	0.30
Aflatoxin	+	+	+	+	+	-	+	+	+	+	+	-
Binders	-	*	**	***	****	-	-	*	**	***	****	-
Cal. Analysis												
Crude protein	20.61	20.61	20.61	20.61	20.61	20.61	20.09	20.09	20.09	20.09	20.09	20.09
Metabolizable energy	2868.9	2868.9	2868.9	2868.9	2868.9	2868.9	2654.00	2654.00	2654.00	2654.00	2654.00	2654.00

histological characteristic. The weights of the organs (liver, kidney, spleen, whole gizzard, empty gizzard, heart, proventriculus and lungs) were measured using a sensitive weighing balance. Then their relative weights to live body weight were determined and documented.

Statistical analysis

All data collected were subjected to one- way analysis of variance (ANOVA) using SAS (2000) software package; significant means were separated using Duncan's multiple range test of the same package.

RESULTS

Performance of the birds fed with infected diets incorporated with various binders

Table 2 shows the average daily weight gain

among the treatments at starter phase. The obtained values varied from 35.12 g in treatment 4 (diet infected with mycotoxin + binder C) to 21.85 g in treatment 3 (infected diet + binder B). There was no significant difference (p>0.05) in the average daily feed intake at starter phase for all treatments except treatment 6 (control) where the feed intake was significantly lower (p<0.05). Different trend was observed at finisher phase, where feed intake of treatment 6 (control) was statistically similar (p<0.05) to the infected diets except treatment 5 which had a significantly higher feed intake (173.57 g). Feed conversion ratio of treatment 3 (infected diet + binder B) was statistically higher (p<0.05) than others while the best value (2.83) was recorded from treatment 4. At finisher phase, no statistical (p>0.05) difference was observed in all the treatments. However,

treatment 5 (infected diet + binder D) had a superior value (3.44). Likewise best average daily feed intake was obtained from treatment 5 though statistically similar (p>0.05) to other except treatments 4 and 6.

Organ characteristics of birds fed with infected diet with different toxin binders

The live weight of the birds from treatment 3 (Binder B) was significantly lower (p<0.05) than treatments 5 (Binder D), 4 (Binder C) and 6 (Control) but statistically similar (p>0.05) to treatments 1 (negative control) and 2 (infected diet + Binder A). However, treatment 6 (control) recorded the highest value (2116 g) while the least (1698 g) was obtained from treatment 3

Table 2. Performance characteristics of broiler chicken under treatments.

Parameter	T$_1$	T$_2$	T$_3$	T$_4$	T$_5$	T$_6$	SEM(±)
			Starter				
ADWG (g)	27.93[b]	31.17[ab]	21.85[c]	35.12[a]	32.61[ab]	30.49[ab]	1.04
ADFI (g)	96.12[a]	96.49[a]	98.21[a]	97.98[a]	99.93[a]	78.96[b]	1.47
FCR	3.52[b]	3.11[b]	4.63[a]	2.83[b]	3.13[b]	2.75[b]	0.15
			Finisher				
ADWG (g)	43.29[ab]	47.21[ab]	45.51[ab]	42.54[ab]	50.39[a]	39.86[b]	1.19
ADFI (g)	164.05[ab]	165.71[ab]	164.40[ab]	152.50[b]	173.57[a]	149.64[b]	2.81
FCR	3.79	3.51	3.61	3.58	3.44	3.75	0.09

[abc]Means on the same row with different superscripts were significantly (<0.05) different.
ADWG: average daily weight gain, **ADFI:** average daily feed intake, **FCR:** feed conversion ratio.
SEM: standard error of mean **T1:** Infected diet, **T2:** Infected diet + binder A **T3:** Infected diet +binder B**T4:** Infected diet + binder C**T5:** Infected diet + binder D**T6:** Control diet (uninfected).

(infected diet + Binder B).

Liver proportion had no significant difference (p>0.05) for all treatments except treatment 2 (infected diet + Binder A) which had significantly higher (p<0.05) value (2.67%) compared to others. The spleen and the proventriculus showed no difference for all treatments as it could be observed from Table 3. There was significant (p<0.05) difference in the values for whole gizzard and empty gizzard with infected + Binder B based diet (treatment 3) having the highest values of 3.41 and 2.61%, respectively while least values of 2.57 and 1.98% were obtained from treatment 4 (infected + Binder C) and treatment 5 (infected + Binder D), respectively. The obtained values for heart revealed a significant difference (p<0.05) among all treatments. Treatment 1 (infected diet without binder) recorded the highest value of 0.87% for kidney proportion while treatment 6 (control) had the least value of 0.53% and the treatments were significantly different (p<0.05).

Haematological and serum characteristics of broiler birds fed infected diets with inclusion of different binders

Table 4 revealed that there was significant difference (p<0.05) among treatments mean values of packed cell volume (PCV). The highest value (26.00%) was obtained from treatment 3 (infected diet + Binder B) and was statistically higher (p<0.05) than treatments 2, 4, 5, and 6. However, the least value (19.00%) was recorded by treatment 6 (control). Red blood cell (RBC) count values obtained from all treatments were statistically similar (p>0.05) except treatment 1 (infected diet without binder) that had least value of 3.19×10^6 mm^3 which was significantly lower (p<0.05) than others (Table 4). Meanwhile, a different trend was observed in white blood cell (WBC) count where treatments 1, 3 and 5 were

significantly higher (p<0.05) than other treatments including the control. Monocyte count showed significant difference (p<0.05) among the treatment means. The highest value (3.50%) was obtained from treatment 4 (infected + Binder C), followed by treatments 6 (control) and 2 having a value of 3.00%. The least value (1.00%) was obtained from treatment 1 (infected diet without binder).

A significant difference (p<0.05) in albumin values was recorded only between negative control and the infected diets (Table 4). The total protein value of treatment 4 (Binder C) was significantly higher (p<0.05) than treatments 1, 3 and 6 but similar (p>0.05) to treatments 2 and 5 which were also similar to treatments 1 and 6. Least value (2.11 g/dl) was obtained from treatment 3. Creatinine was significantly different (p<0.05). Treatment 5 recorded the highest value (0.87 mg/dl) while treatment 3 (Binder B) had the least value (0.74 mg/dl). Treatments 1, 3, 4 and 6 had similar (p>0.05) cholesterol level while treatments 2 and 5 were also observed to be similar (p>0.05); however, treatment 1 (infected diet without binder) and treatment 4 (infected diet + Binder C) recorded significantly (p<0.05) higher values compared to others while treatment 5 (Binder D; Activated charcoal) had the least value for cholesterol (87.18 mg/dl).

Histological observation of the liver

Generally, there were alterations in the histological structure of the liver samples which ranged from gradual hepatic cell inflammation, mild inflammation to severe inflammation of the hepatocytes. Although the morphological alteration was observed in all the livers, the inflammations are limited to certain portions as other areas showed normal liver hepatocytes. Liver sample obtained from birds placed on infected diet without binder showed a characteristic inflammation of hepatocytes

Table 3. Organ characteristics of broiler chicken fed contaminated diet with inclusion of different toxin binders.

Parameter	T_1	T_2	T_3	T_4	T_5	T_6	SEM(±)
Live weight (g)	1840.00[ab]	1941.67[ab]	1698.00[b]	2011.00[a]	2062.67[a]	2116.00[a]	42.65
Liver (%)	2.26[b]	2.67[a]	2.24[b]	2.07[b]	2.07[b]	2.17[b]	0.05
Spleen (%)	0.11	0.15	0.16	0.13	0.15	0.14	0.01
Proventiculus (%)	0.38	0.40	0.44	0.36	0.49	0.41	0.02
Wgizzard (%)	2.63[b]	2.95[ab]	3.41[a]	2.57[b]	2.72[b]	3.07[ab]	0.78
Egizzard (%)	2.20[ab]	2.33[ab]	2.61[a]	2.07[b]	1.98[b]	2.04[b]	0.70
Heart (%)	0.46[b]	0.47[ab]	0.48[ab]	0.61[a]	0.34[bc]	0.28[c]	0.26
Kidney (%)	0.87[a]	0.69[b]	0.73[b]	0.60[bc]	0.70[b]	0.53[c]	0.02
Lungs (%)	0.74[a]	0.60[b]	0.67[ab]	0.60[b]	0.67[ab]	0.74[a]	0.01

[abc]Means on the same row with different superscripts were significantly different (p<0.05).
WGizzard: Whole gizzard, EGizzard: Empty gizzard.

Table 4. Haematological indices of broiler chicken fed contaminated diet with inclusion of different toxin binders.

Parameter	T_1	T_2	T_3	T_4	T_5	T_6	SEM(±)
	Haematological indices						
PCV (%)	23.00[ab]	21.50[bc]	26.00[a]	22.50[bc]	20.00[bc]	19.00[c]	0.58
Hb (g/dl)	9.45	8.60	8.00	9.15	9.70	8.90	0.25
RBC ($10^6 mm^3$)	3.19[b]	3.88[a]	3.97[a]	4.03[a]	4.25[a]	3.94[a]	0.09
WBC ($10^3 mm^3$)	21.60[a]	16.68[bc]	20.23[a]	14.00[c]	22.13[a]	19.80[ab]	0.64
Lymph (%)	63.50	59.00	61.00	60.50	63.50	63.50	1.19
Heterophil (%)	32.00	34.50	36.00	31.00	29.50	29.50	1.07
Monocyte (%)	1.00[c]	3.00[ab]	1.50[c]	3.50[a]	2.50[b]	3.00[ab]	0.18
Eosinophil (%)	3.50[a]	2.00[b]	1.50[b]	4.00[a]	4..00[a]	4.00[a]	0.26
Platelet(10^6/dl)	301.00[a]	137.50[cd]	224.50[b]	120.50[d]	199.00[bc]	131.50[cd]	13.74
	Serum parameter						
TP (g/dl)	2.17[bc]	2.35[abc]	2.11[c]	2.70[a]	2.56[ab]	2.23[bc]	0.06
Alb (g/dl)	1.30[a]	1.17[b]	1.22[b]	1.17[b]	1.20[b]	1.18[b]	0.01
AST (IU)	154.95[c]	163.08[bc]	154.97[c]	171.46[abc]	185.33[a]	176.17[ab]	2.78
ALT (IU)	6.56[ab]	6.40[ab]	8.56[a]	5.44[b]	5.20[b]	5.88[ab]	0.39
CRT (mg/dl)	0.78[bc]	0.82[ab]	0.74[c]	0.78[bc]	0.87[a]	0.84[a]	0.01
ALP (g/dl)	310.27[a]	302.15[a]	281.69[ab]	212.18[c]	251.52[b]	260.36[b]	7.25
CHOL (mg/dl)	115.69[a]	96.79[bc]	108.34[ab]	116.67[a]	87.18[c]	104.17[ab]	2.71

[abcd]Means on the same row with different superscripts were significantly different (p<0.05).
PCV: Packed cell volume, **Hb**: Haemoglobin, **RBC**: Red blood cell, **WBC**: White blood cell, **Lymph**: Lymphocyte **TP**: total protein, **Alb**: Albumin, **AST**: Aspartate amino transferase, **ALT**: Alanine amino transferase, **CRT**: Creatinine, **ALP**: Alkaline phosphatase, **CHOL**: Cholesterol.

around the portal vein, as it could be rightly observed at x100 magnification under electronic microscope (Figure 1). Figure 2 show liver from birds placed on infected diet + Binder A. There was a severe inflammation of hepatocytes around the portal vein. Figure 3 reveals the histological changes of liver obtained from birds placed on infected diet + Binder labeled B. It shows mild and gradual inflammation of hepatic cells, under electronic microscope with a view at x100 magnification. Figure 4 shows the observation of liver from birds placed on infected diet +Binder C based treatment. The sample showed little inflammation of hepatic cells at the portal vein at x100 magnification level. Figure 5 reveals the observation of liver from birds placed on infected diet + activated charcoal. The sample showed mild inflammation of hepatic cells around the portal vein. Figure 6 shows cross sectional view of broiler liver fed non-infected diet (control or treatment 6). It showed a gradual inflammation of hepatocytes around the portal vein at x100 magnification, despite not infected.

Figure 1. Section of broiler liver fed infected diet without binder showing inflammation of hepatic cells (X 100 stained with H and E, Haematoxylin & Eosin).
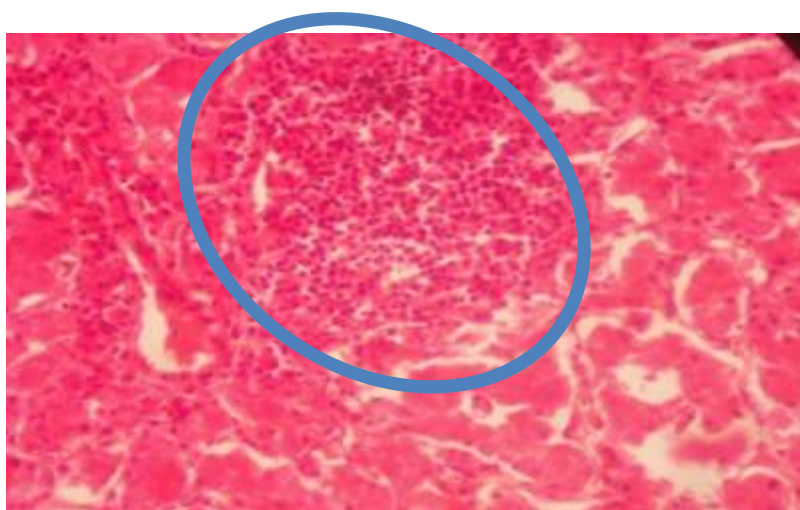

Figure 2. Section of broiler liver fed infected diet + Binder A (HSCAS) showing severe inflammation of hepatocytes. (X 100 stained with H and E, Haematoxylin & Eosin).

DISCUSSION

There is loss of energy availability in the feed when feeding birds with aflatoxin infected diet (Verma et al., 2007). When there is loss of energy in feed, the birds tend to consume more in order to meet up with their energy requirement. Probably, this was one of the factors responsible for the high feed intake observed in all the infected diets at starter phase. But at finisher phase, consumption rate of birds on infected diets was observed to be in a definite trend. Treatment 1 (infected diet

without binder) was statistically similar (P>0.05) to other dietary treatments. This suggested that the birds might have developed resistance to the effect of mycotoxin or that the administration of anti-oxidants such as vitamins as anti- stress during the period of feeding trial might have helped in reducing the effect of aflatoxin as it was reported that antioxidants seem to be chemo-preventive against common mycotoxin (Gowda and Ledoux, 2008). Treatment 5 (infected diet + activated charcoal) had the least feed conversion ratio with a value of 3.44 though statistically similar to others. It made the best use of the
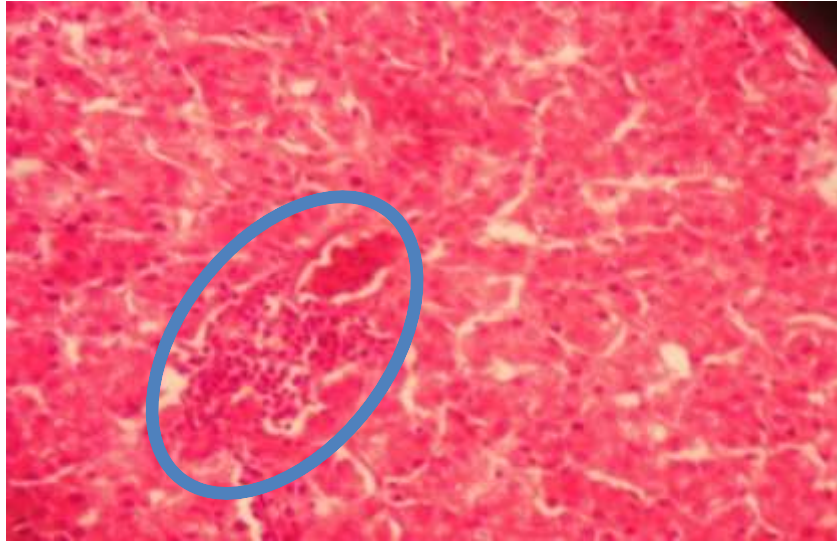
Figure 3. Section of broiler liver fed infected diet + Binder B (Bentonite/Montmorillonite Yeast cell walls) based diet showing a gradual inflammation of hepatocytes. (X 100 stained with H and E, Haematoxylin & Eosin).

Figure 4. Section of broiler liver fed infected diet + Binder C (Aromatic Polyphenols) Showing little inflammation of hepatic cells around the portalvein. (X 100 stained with H and E).

feed having the leading value of average daily weight gain of 50.39 g. Growth rate is the most significant cost/effect of mycotoxin in poultry (Gowda et al., 2008). Despite the moderate consumption rate obtained from treatment 1(infected diet with no binder), it had the highest feed conversion ratio (3.79) expressing the negative effect of the toxin and toxin concentration in the diet (Qazi and Fayyaz, 2006). This supported the work of Tedesco et al. (2004) that contamination of aflatoxin in broiler feed causes poor feed conversion ratio (FCR) and

poor feed utilization. However, the infected diets with incorporated binders had better FCR values, even than the control diet (uninfected), notably expressing the positive effects of various binders on feed utilization.

The observed growth rate showed a reduction in treatments 1 up to 5 when compared to treatment 6 and justified the report of Shi et al. (2009) that aflatoxin contaminated feed causes aflatoxicosis and is characterized by decreased weight gain of the livestock. The higher proportion ($p<0.05$) of the kidney to live
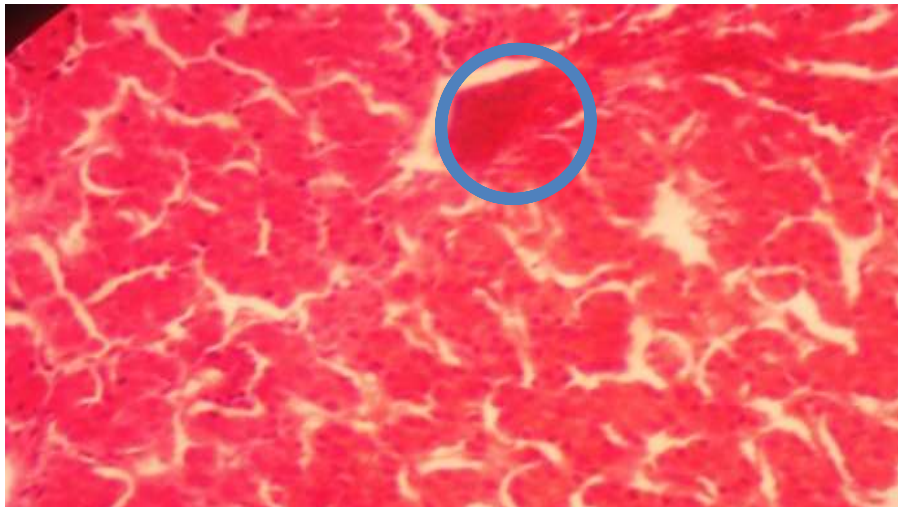
Figure 5. Section of broiler liver fed infected diet + Binder D (Activated Charcoal) based diet showing mild inflammatory cells. (X 100 stained with H and E, Haematoxylin & Eosin).

Figure 6. Section of broiler liver fed non- infected diet (control) showing gradual inflammation of hepatocytes around the portal vein. (X 100 stained with H and E, Haematoxylin and Eosin).

weight obtained from treatment 1 (infected diet without binder) and similar variation obtained from other treatments fed infected diets were suggested to be as a result of aflatoxin consumption, as aflatoxin can cause productive deterioration which is associated with kidney abnormalities and impaired immunity (Oguz et al., 2003). The liver is the main target and a key player of aflatoxicosis in poultry (Diaz and Murcia, 2011). The relative weights of liver were statistically similar (p>0.05)

for all the treatments except treatment 2; which translates that the infected diets, those incorporated with binders as well as the control were the same. Researches have revealed that relative weight of liver increased as the aflatoxin ingestion was 200 ppb and more significantly increased at higher aflatoxin level (Saleh et al., 2013). Therefore, the little or no change in the liver proportion of the dietary treatments compared to the control could be as a result of the low exposure level which had little

(p>0.05) effect on the liver proportion.

Red blood cells are made up of proteins. The range of red blood cell count (4.25 - 3.19) recorded in this work was within the range of 2.90- 4.10 ($x10^6 mm^3$) recommended by Paggot (1992). However, lower red blood cell observed in treatment 1 might be the effect of aflatoxin consumption as it has been reported by Cassel et al. (2012) that the level of protein and vitamins in feed should be increased as toxin affects protein synthesis which in turn is the building block of the RBC component of the blood. Monocytes play important role in healing process thus; they increase in number during healing process. The average monocyte count for poultry bird is 2.00% (Paggot, 1992). It was therefore suggested that the increase in monocyte count above 2.00% in treatments 2, 3, 4, 5 which were infected diets with inclusion of the binders, activated charcoal inclusive and treatment 6 (Control) probably indicated that healing process was demanding though may be mild as no mortality was recorded in these treatments. However, the least monocyte count (1.00%) obtained from treatment 1 (infected diet with no binder) is a pointer that the birds were more stressed by the effect of the toxin ingested.

There was a significant (p<0.05) difference in albumin value of all the treatments with the highest value recorded at treatment 1 (negative control). The value of total protein obtained showed significant difference (p<0.05). However, all fall within the range of 2.11 to 2.70 g/dl. This range was found to be in accordance with the report of Cassel et al. (2012) that the level of protein and vitamins in feed should be increased as toxin binds vitamins and affects protein synthesis. Cholesterol is mostly synthesized by the liver, it aids efficient metabolism, that is, it is essential for the body to produce vitamin D. Consumption of infected diet by birds has a significant influence on the liver; the normal synthesis of cholesterol is thereby affected as it was observed from treatment 1 (infected diet without binder) where the value obtained was significantly (p<0.05) higher which consequently leads to a higher cholesterol deposition than other treatments. Out of all the binders, activated charcoal seemed to be more efficient when blood cholesterol is considered. Blood cholesterol indicates the level of deposition of fat into the adipose muscles in the liver.

The histological effects of toxicosis include liver necrosis, hepatic cell degeneration and inflammation of hepatocytes. The varying degree of alterations (inflammations) in the histological structure of all the liver samples (as observed from the plates) could be due to the level of aflatoxin in the diet, which may or may not be sufficient enough to cause morphological actions such as necrotic lesions and cell degeneration. This is because the biological effects of mycotoxins depend on the ingested amounts, number of occurring toxins, duration of exposure to mycotoxins and animal sensitivity (Yiannikouris and Jonany, 2002).

CONCLUSION AND RECOMMENDATION

The results of the study had shown that inclusion of activated charcoal as a binder in an aflatoxin contaminated diet gave an encouraging performance and feed conversion value than the other binders. Although all the liver samples showed varying degree of inflammations, samples from birds placed on activated charcoal proved to have reduced inflammatory cells. It could therefore be recommended that the use of activated charcoal available at 10 g/kg diet should be more encouraged rather than other synthetic binders. Alternatively, the binder could be used instead of none especially when the status of the diet could not be ascertained as it has proved to be the most efficient and economical approach of preventing and counteracting aflatoxicosis in poultry.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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