The Journal of Animal & Plant Sciences, 26(5): 2016, Page: 1202-1211
ISSN: 1018-7081

BINDING EFFICACY OF YEAST SLUDGE FRACTIONS AND COMMERCIAL GLUCOMANNAN AGAINST AFLATOXINS IN BROILERS

Y. A. Ditta[1], Saima[1], T. N. Pasha[1], M. Akram[2], Z. M. Iqbal[3] and S. Naseem[4]

[1]Department of Animal Nutrition, [2]Department of Poultry Production, [3]Department of Livestock Production, University of Veterinary and Animal Sciences, Lahore Pakistan
[4]Department of Animal Sciences, University of California, Davis, USA
Corresponding Author E-mail: yasirad@uvas.edu.pk

ABSTRACT

This study was conducted to compare the efficacy of sonicated yeast sludge fractions (Cell Wall and Cell Solubles) with commercial glucomannan against Aflatoxin B_1 and G_1 (AFB_1 and AFG_1) in feed. A total of 390 chicks were randomly divided into 13 treatments (three replicates / treatment and ten chicks / replicate). Negative control (NC) included no AFB_1+AFG_1 and toxin binder. The positive control contained different AFB_1+AFG_1 levels (100, 200 and 300 ppb of ech). Other treatments included yeast sludge fractions and organic toxin binder with AFB_1+AFG_1 levels. As a result of this study, depressed feed intake and body weight were observed as compared to negative control. Non-significant effect between yeast sludge cell wall and glucomannan product was observed but was significant than negative control. The aflatoxins showed significantly negative impact on weight gain, relative liver weight and serum minerals and non-significant effect on feed intake, dressing percentage and serum phosphorus. The different toxin binders showed significant effect on feed intake, weight gain, feed conversion ratio(FCR), relative liver weight, serum albumin, cholesterol and serum minerals while non-significant effect was observed on dressing percentage and serum phosphorus. The data indicated that yeast sludge cell wall showed non-significant results with commercial Glucomannan whereas Yeast Sludge Cell Soluble was showed significantly depressed effect on the production, liver and serum minerals.

Key words: Aflatoxin, Broilers, Glucomannan, Yeast Sludge Cell Wall, Yeast Sludge Cell Soluble, Sonication.

INTRODUCTION

About one third of grains are contaminated with mycotoxins in Asia-Pacific region (Zhao *et al.*, 2010) thus affecting more than 4.5 billion people chronically (Williams *et al.*, 2004). More than 100 countries have specified the limits for mycotoxins at least for AFB_1 or their sum (B_1, G_1, B_2 and G_2) (Yildirim *et al.*, 2011)in foodstuffs representing 87% of the world population (Binder, 2007; Commission, 2010). Aflatoxins (AFs) are group of closely related mycotoxins being produced by three ubiquitous (Moschini *et al.*, 2008) species of *Aspergillus; A. flavus, A. parasiticus* and the rare *A. nomius* (Magnoli *et al.*, 2011; Chen *et al.*, 2014) that grow on variety of feedstuffs, mainly maize, peanuts and cottonseed. The susceptibility of AFs to toxicity may vary with breed, species, sex, age duration / concentration of exposure, health and nutritional status of broilers (Chen *et al.*, 2013). Among AFs, the order of toxicity is $AFB_1 > AFG_1 > AFB_2 > AFG_2$ respectively. AFs mainly target liver for accumulation (Quezada *et al.*, 2000; Çelİk *et al.*, 1999) and characterized by hepatic enlargement and fatty infiltration (Rauber *et al.*, 2007).

The supplementation of different non-nutritive adsorbing materials in AFs contaminated feed is one of the most effective and economical procedures (Neeff *et al.*, 2013). Aluminosilicates, zeolite, sodium Bentonite, a cell wall derivative of *S. cerevisiae* called Glucomannan (Osweiler *et al.*, 2010) and yeast sludge (Pasha, 2008) are commonly used for the detoxification of aflatoxins (Moschini *et al.*, 2008). Yeast Sludge is produced after fermentation process of molasses with *S. cerevisiae* and is good source of glucomannan (Hashmi *et al.*, 2006) but presently, is thrown away as waste product. Since it is acidic, it can decrease soil pH if accumulated over time. *S. cerevisiae* has rigid cell wall and cytoplasm containing protein, cytoplasmic enzymes, polysaccharides etc (Liu *et al.*, 2013). The destruction of cell wall mechanically (by applying shear force) and non-mechanically (through chemical or enzymatic methods) results in the release of intracellular components (Geciova *et al.*, 2002). Mechanical methods are cheaper at industrial scale (Li and Sun, 2002). Ultra-sonication, for example, is one of the most commonly used mechanical methods for cell disruption (Chemat and Khan, 2011). Keeping in view, the current experiment presents the comparative efficacy of sonicated Yeast Sludge fractions as toxin binder and commercial glucomannan product in broilers at Poultry Research Center, University of Veterinary and Animal Sciences, Pakistan.

MATERIALS AND METHODS

Production of Aflatoxins: AFs (B_1, B_2, G_1 & G_2) were produced by using culture of *A. parasitucus* (NRL 2999) with a modified method of Shotwell *et al.* (1966). Potato dextrose agar slants were streaked with *A. parasiticus* and incubated at 28 ˚C. After 2-3 days, dark green / black spores appeared on the surface of slants. The fresh spores on the slants were transferred into flasks containing autoclaved rice and placed on orbital shaker at 225 rev / min maintaining at 28 ˚C. Autoclaved water was added aseptically in each flask. After 2-3 days of fermentation, white spots appeared on the distal ends of rice grains which grew in number with the passage of time. Thereafter, the rice grains turned from yellow to brown. Fermented rice was autoclaved, dried and ground to powder. The extraction of AFs produced was carried out by shaking for 30 minutes after mixing 30 ml chloroform / 10 g fermented rice. The mixture was then centrifuged at 5,000 rpm for 10 minutes and filtered by Whatman # 1 filter paper as reported by Oliveira *et al.* (2002). The filtrate was spotted on TLC plate and was eluted for the separation of AFB_1 and AFG_1. The separated AFB_1 and AFG_1 were then taken in chloroform and quantified. All the processes including spore formation, aflatoxins production and storage of aflatoxins were carried out in the darkness.

Sonication of Yeast Sludge: The yeast sludge (YS) was procured from Murree's Brewery Rawalpindi. The YS was washed with distilled water until the molasses was removed before drying at 71 ˚C in hot air oven. Suspensions at 1% were prepared by mixing dried YS and distilled water. The YS was fractionized by Sonicator in cycle mode (Branson Sonifier, Model No. 250, Serial No. BH50023, Manufacturing Company: Branson VWR Scientific USA, Input: 100, 200/240 VAc, 50/60 MHz, 2Amp) into two layers: a supernatant containing Cell Wall and a residue having Cell Solubles. The tip of Sonicator horn (20 mm diameter) was located in the center of solution. The temperature of solution was kept constant by the use of a cooling bath containing ice–water mixture. Ultra-sonication of yeast cells caused the disruption of cell protein (Liu *et al.*, 2013). The mixture was centrifuged at 1,500 g for ten min (height of tube 10 cm) to separate supernatant and residue (Northcote and Horne, 1952).

Experimental Diet and Chicks: Iso-caloric and iso-nitrogenous feed was formulated according to NRC (1994) recommendations (Table.1). The birds of Hubbard strain were offered with starter diet until 21st day of age and then changed to grower diet. Daily feed intake, weekly weight gain or any morbidity sign were monitored strictly on daily basis. A total of 390 day (39.29 g ± 3.07 SD) old chicks (three replicates / 13 treatment and ten birds / replicate) were procured from local hatchery and were given *ad lib* starter basal diet and water under 24 hours light. The birds were kept at 95 ˚F for brooding and temperature was reduced at the rate of 5 ˚F per week gradually until 70 ˚F. On the 8th day of age, the birds were randomly categorized into different pens on the floor and were given various treatments (8th to 28th day).

The experimental diet for each treatment was as follow: 1. Basal diet (NC), 2. NC + 100ppb AFB_1 and 100 ppb AFG_1 (PC1), 3. NC + 200ppb AFB_1 and 200 ppb AFG_1 (PC2), 4. NC + 300ppb AFB_1 and 300 ppb AFG_1 (PC3), 5. PC1 + 1% YS Cell wall (YSCW1), 6. PC1 + 1% YS Cell Soluble (YSCS1), 7. PC1 + 0.1% Glucomannan (GLUCO1), 8. PC2 + 0.1% YS cell wall (YSCW2), 9. PC2 + YS Cell Soluble (YSCS2), 10. PC2 + Glucomannan (GLUCO2), 11. PC3 + YS Cell Wall (YSCW3), 12. PC3 + YS Cell Soluble (YSCS3), 13. PC3 + Glucomannan (GLUCO3). All YCW and YCS were added at 1% and glucomannan were added 0.1% of total feed.

Minerals Estimation: Calcium, magnesium, zinc, copper, sodium and potassium of serum were analyzed by Atomic Absorption Spectrophotometer (Perkin-Elmer, AA400). Phosphorus was analyzed by spectrophotometer (AOAC, 1990).

Biochemical Parameters: On slaughtering, the serum was stored at -20˚C until analyses (Magnoli *et al.*, 2011; Yildirim *et al.*, 2011). The slaughtering was done after the approval of "Ethical Review Committee for the Use of Laboratory Animals" of the University of Veterinary and Animal Sciences, Lahore Pakistan and was according to the recommendations. The total Serum protein was determined by Biuret method (Henry *et al.*, 1974). Commercial Randox Kits (Rec, 1972) were used to determine serum albumin. Serum cholesterol (mono-reagent with LCF, CHOD-PAP method), ASAT (GOT IFCC mod) and ALAT (GPT IFCC mod) were analyzed by Clinical Chemistry analyzer (Company: Elitech Group, Microlab 300 Voltage: 220, 50 / 60 Hz) using Commercial kits according to company recommendations (Basmacioglu *et al.*, 2005).

Experimental Design: The data were statistically analyzed under Completely Randomized Design using 2-way ANOVA through GLM procedure of SAS 9.1 software. The model included the main effects of AFs levels and toxin binders (Steel *et al.*, 1997). Duncan's Multiple Range test was employed to compare the significant difference of means of treatments at 0.05 level of probability (Duncan, 1955).

RESULTS AND DISCUSSION

The AFs contaminated feed ameliorate the performance of birds (Santin *et al.*, 2006). In our study,

different dietary treatments significantly ($p < 0.01$) reduced feed intake, weight gain and FCR as compared to negative control at different AFB_1 and AFG_1 levels (Table.2). Similar observations have been reported after AFB_1 and AFG_1 feeding (Santin *et al.*, 2006; Yildirim *et al.*, 2011). The present findings indicated adverse effects on body weight which is also in agreement with the previous findings (Quezada *et al.*, 2000) due to the fact that aflatoxicosis could ablate body weight. Neeff *et al.* (2013) noted birds fed AFB_1 alone had 27% reduction in weight gain. However, *S. cerevisiae* supplemented feed accelerated growth due to the fact that it increases lipid absorption in the birds as compared to the positive control (Yildirim *et al.*, 2011).

The GLUCO and YSCW performed comparatively better in feed intake to PC at different AFB_1 and AFG_1 levels (Table.2). This may be due to the presence of glucomannan contents to counteract the deleterious effects of AFs (Santin *et al.*, 2006). The current findings agree with Mogadam and Azizpour (2011) who found higher feed intake (7.4%), weight gain (24%) and better FCR (13.7%) with 0.1% yeast glucomannan addition compared with NC birds. Likewise, Raju and Devegowda (2000) found 2.26% increase in weight gain with glucomannan supplementation (0.1% / kg of feed). Probably, modified glucomannan trapped the toxins (Girish and Devegowda, 2006) in small intestine and imparted beneficial health impacts. The PC and YSCS blunted feed intake and depressed growth at different levels of AFB_1 and AFG_1 that lead to poor FCR. These observations indicated the importance of AFs binding agents in the feed.

During this 21 days trial, PC1 and dietary treatment diets with different levels of AFB_1 and AFG_1 showed non-significant effects on feed intake (Table.2), moreover, this effect was profoundly progressed in NC. The less effect on feed intake gives the clue that may be due to insufficient AFs concentration in PC1 and positive effect of toxin binders on dietary treatments. The maximum decrease in feed intake was observed in PC3 (28.9%) followed by PC2 (24.71%) causing 59.26% and 56.29% decrease in weight gain as compared to NC respectively (Table.2). Similar trend is previously documented (Neeff *et al.*, 2013; Yildirim *et al.*, 2011).

The minimum decrease in weight gain was observed in YSCW (15.5, 26.59 and 31.06%) and GLUCO (16.4, 17.5 and 28.57%) followed by maximum decrease in YSCS (35.3, 38.04 and 39.45%) at 100, 200 and 300 ppb as compared to NC respectively. The YSCS showed poor FCR (30.4, 36.17 and 38.44% less than NC) among dietary treatments at different levels of AFs. YSCW showed better FCR among other dietary treatments at 100 ppb achieving 94.88% FCR of negative control (Table.2). The dietary treatments supplemented with 0.1% Gluco showed better FCR than others and showed 10.04 and 26.63% less FCR than negative control at 200 and 300 ppb AFs, respectively. Overall, YSCW and GLUCO produced similar effect ($p > 0.05$) on FCR at different AFs levels. Contrary to this, significant effects ($p < 0.01$) were observed for feed intake, weight gain and FCR among different dietary treatments. The AFs levels showed non-significant ($p = 0.46$) results which may be attributed to the efficacy of different toxin binders on feed intake. Significant effects of toxin binders ($p < 0.01$) on feed intake also revealed their comparative efficacy supplemented with various AFs levels. In our study, the uneven adverse effects of AFs on the performance of birds at different weeks of age are supported by meta-analysis (Suganthi *et al.*, 2011). A non-significant 2-way interaction of levels of AFB_1 and AFG_1 × toxin binders was observed for feed intake ($p = 0.66$), weight gain ($p = 0.63$) and FCR ($p = 0.99$).

Slaughtering Parameters: On slaughtering at 4th week, dressing percentage ($p = 0.93$) showed non-significant effect. Different AFs levels, toxin binders and toxin binders × AFs were found to have non-significant effect ($p > 0.05$). AFs affect mainly liver (Quezada *et al*., 2000). The dietary treatments showed significantly higher relative liver weight ($p < 0.01$) than negative control (Table.2) which matches with the findings of Huff and Doerr (1981). This agrees with Yunus *et al*. (2011) who found increase in relative liver weight by AFs contamination. However, our findings regarding relative liver weight do not support earlier work of Santin *et al.* (2006) who found non- significant increase in relative organ weight. Rauber *et al.* (2007) found significant increase in liver weight. Apparently, advances in toxin binders technology enable present day toxin binders more effective than early toxin binders. Considering this factor, the current findings appeared to have contradiction with Kubena *et al.* (1993) and Abo-Norag *et al.* (1995) who reported an increase in relative liver weight. Girish and Gevegowda (2004) noted 21.72% increase in relative liver weight with AFs but glucomannan counteracted negative effects of AFs significantly. The beneficial impact of Glucomannan is also reported by Magnoli *et al*. (2011).So in our study, the AFs levels and different toxin binders showed significant effect ($p < 0.01$) on relative liver weight. The YSCS supplemented at a rate of 1% with 300 ppb were found to have non-significant effect from PC at 200 and 300 ppb AFs with NC. The YSCW and GLUCO showed 1.5 times increase in liver weight as compared to 2 times increase in PC at 300 ppb as compared to negative control. The AFs may inhibit hepatic protein synthesis and lipid metabolism which expose liver to accumulate lipids. This pathological process leading to enlarged fatty liver (Safameher, 2008).

Serum Biochemistry: The serum copper, potassium, sodium, zinc, calcium, cholesterol, total serum protein, serum uric acid, albumin, ASAT and ALAT showed significant effects ($p < 0.01$) whereas serum chloride ($p =$

0.33), magnesium ($p < 0.08$), and phosphorus ($p = 0.28$) were found non-significant at different AFs levels (Table. 3, 4 and 5). Basmacioglu *et al.* (2005) found 1.72 ± 0.14 mg / dL, 0.74 ± 0.06 mg / dL, 112.2 ± 4.27 mg / dL, 7.90 ± 0.17 mg / dL and 5.60 ± 0.24 mg / dL for AFs contaminated feed as compared to NC 2.92 ± 0.12 mg / dL, 1.25 ± 0.05 mg / dL, 143.30 ± 5.93 mg / dL, 8.53 ± 0.42 mg / dL and 6.82 ± 0.34 mg / dL for total protein, albumin, total cholesterol, calcium and phosphorus respectively. As important indicator, moderate to severe liver intoxication affects the liver function tests that lead to increase in ASAT. Consequently, there is hepatocytes degeneration and subsequent leakage of enzymes (Tessari *et al*., 2010). Yang *et al*. (2012) also found a significant increase in serum ASAT and ALT by feeding 75% and 100% contaminated corn.

Increased ASAT and ALT are diagnostic indicators of hepatic injury. The dramatic decrease in total serum protein in positive control might be due to AFs without glucomannan contents (Yildirim *et al.*, 2011). While Quezada *et al*. (2000) attributed this effect to aflatoxicosis that also lead to damage in renal tubules. Therefore, low calcium intestinal absorption or decrease in circulating parathyroid hormone (Glahn *et al.*, 1991) significantly decreased calcium level in blood serum.

The different toxin binders showed significant effects ($p < 0.01$) on albumin, copper, potassium, Cholesterol, chloride, magnesium, zinc, sodium, calcium, ASAT and ALAT except for phosphorus ($p = 0.48$). Similarly, the different levels of AFs resulted in profound effects ($p < 0.01$) on albumin, copper, potassium, Cholesterol, zinc ($p = 0.02$), sodium, total serum protein, ASAT, ALAT and serum uric acid except for magnesium ($p = 0.54$), phosphorus ($p = 0.49$) and calcium ($p = 0.17$). Since many studies have documented that AFs impaired the hepatocytes functions, consequently hindering the biosynthesis of cholesterol (Monson *et al*., 2015; Valchev *et al.*, 2014; Yildirim *et al.*, 2011). In addition to AFs effect on cholesterol, it also deprived body from protein synthesis due to low hepatic function (Rauber *et al.*, 2007). This suggests the liver as primary target for AFs having deleterious effects on metabolism and secretions (Quezada *et al.*, 2000). Since both contained glucomannan, YSCW and GLUCO showed non-significant effect on serum albumin at 100 and 200 ppb than NC whereas there was a significant effect of 300 ppb among NC, YSCW and GLUCO. YSCS showed least effect on performance followed by YSCW. Tessari *et al.* (2010) found AFs impaired protein synthesis by reduction in carbohydrate utilization enzymes. The 0.5 and 1 mg AFB_1/kg negatively affected serum protein concentrations (Chen *et al*., 2014). This may be due to reduced serum total protein or probably due to inhibition of amino acid transport and mRNA transcription which resulted in the inhibition of DNA and protein synthesis (Yang *et al*., 2012).

Birds fed with 1 % YSCS at different levels of AFB_1 and AFG_1 resulted in lowest growth and serum indices followed by PC, suggesting aflatoxicosis. However, birds fed with 1 % YSCW and 100 ppb AFs reversed the negative impact on mineral serum concentration, for example; Cu and at 100 and 200 ppb for zinc and at 300 ppb for magnesium and sodium respectively. Interestingly, birds diet supplemented with 0.1 % GLUCO at 100 and 200 ppb AFs improved cholesterol, magnesium and sodium and at 300 ppb for magnesium and zinc.

The effect of YSCW and GLUCO were found statistically similar on ASAT and ALAT level. AFB_1 and AFG_1 at 200 and 300 ppb levels significantly increased ALAT concentration whereas ASAT level was affected by AFB_1 and AFG_1 significantly even at 100 ppb. YSCW in combination with 100 ppb AFB_1 and AFG_1 partially reversed ASAT level compared with NC, while at 300 ppb AFB_1 and AFG_1 concentration, abruptly raised ASAT with different toxin binders. The birds offered 1% YSCS at 200 and 300 ppb AFB_1 and AFG_1 reduced serum uric acid as compared to other toxin binders. As far as YSCW and GLUCO are concerned, both showed non-significant effect at 200 and 300 ppb AFB_1 and AFG_1 respectively for growth and other parameters. The glucomannan contents improved the performance and serum parameters as compared to positive control (Jigang and Wenjun, 2013; Kaki *et al.*, 2012; Yildirim *et al.*, 2011).

In summary, YSCW and GLUCO supplemented diet resulted in similar effect on birds' performance. These effects may be due to the presence of glucomannan contents present in the YSCW and GLUCO. The YSCS without glucomannan contents was unable to detoxify the deleterious effects of AFs significantly. This observation shows the importance of glucomanan supplementation in the feed. Since toxins free feed should be preferred for optimum performance. YSCW can be a great asset to detoxify the deleterious effects of AFs comparatively to the commercial glucomannan products.

Table 1. The Ingredients and Nutrients Composition of Starter and Grower Feed

Ingredients %	Starter	Grower	Nutrient Composition	Starter	Grower
Corn Grain	15.0	15.0	DM %	90.19	90.10
Rice Tips	13.73	17.7	CP %	20	19
Wheat grains	20.7	21.3	ME Kcal / Kg	2800	2900
Rice Polish	4.69	5.13	CF %	5	5
Wheat Bran	8.0	8.0	EE %	3.72	3.63
Soybean meal	15.05	10.79	Methionine %	0.6	0.5
Sunflower meal	3.0	3.0	Lysine %	1.1	1.0
Canola meal	13.65	13.24	LA %	0.76	0.76
Molasses	3.0	3.0	Ca %	1.1	0.9
Calcium Carbonate	2.27	2.26	P %	0.53	0.45
Vitamin Premix	0.3	0.3			
L-Lysine	0.34	0.43			

Vitamins and minerals premix / Kg of feed: vitmain A (retinyl acetate) 8800 IU; cholecalciferol 3855 IU; vitamin E (α-tocopheryl acetate) 15 IU; vitamin K (menadione sodium bisulfite) 1.7mg; thiamine mononitrate 1.1mg; riboflavin 6.6 mg; pentothenic acid 17 mg; niacin 55 mg; vitamin C (ascorbic acid) 100 mg; cyanocobalamin 11 µg; biotin 0.2 mg; pyridoxin 2.2mg; folic acid 1.4 mg; manganese ($MnSO_4$) 105 mg; zinc ($ZnSO_4$) 110 mg; iron ($FeSO_4.\ 7H_2O$) 60 mg; copper (CuSO4) 6mg; iodine (ethylenadiamine dihydoiodine) 2 mg; selenium (NaSeO3) 0.2mg; cobalt 0.2 mg

Table 2. Effect of Different Toxin Binders at Different Levels of Aflatoxins (B_1 and G_1) on Production and Slaughtering Parameters

Treatments[1]	Feed Intake	Weight Gain	FCR	Relative Liver Weight	Dressing percentage
	----------Grams----------			g %	
NC	1768.0^{a}±11.81	1158.42^{a}±17.88	1.53^{d}±0.01	1.73^{g}±0.14	56.93±2.09
PC1	1471.2^{bc}±27.42	611.51^{fg}±38.15	2.42^{abc}±0.14	2.71^{bc}±0.11	55.54±1.1
PC2	1331.8^{dc}±94.77	506.28^{gh}±11.14	2.63^{ab}±0.15	3.47^{a}±0.23	53.15±1.9
PC3	1257.9^{d}±131.75	471.88^{h}±35.64	2.74^{a}±0.5	3.78^{a}±0.19	54.03±1.9
YSCW1	1560.1^{ab}±33.56	978.81^{b}±56.33	1.6^{d}±0.10	2.02^{gef}±0.11	55.79±1.77
YSCS1	1491.4^{bc}±56.94	749.01^{de}±3.31	1.99^{cd}±0.07	2.39^{cde}±0.06	55.04±0.9
GLUCO1	1560.8^{ab}±46.29	968.41^{b}±76.87	1.63^{d}±0.12	1.91^{gf}±0.03	57.71±2.81
YSCW2	1526.6^{ab}±49.88	850.31^{bcd}±15.79	1.79^{d}±0.09	2.16^{gef}±0.1	55.12±1.45
YSCS2	1479.7^{bc}±60.03	717.71^{fde}±33.57	2.08^{bcd}±0.18	2.95^{b}±0.19	56.16±1.68
GLUCO2	1571.8^{ab}±37.04	955.63^{bc}±105.32	1.68^{d}±0.19	2.22^{def}±0.13	54.98±2.57
YSCW3	1558.6^{ab}±2.28	798.58^{ced}±7.7	1.95^{cd}±0.02	2.66^{bc}±0.19	54.4±1.99
YSCS3	1470.7^{bc}±94.13	701.52^{fe}±30.86	2.11^{bcd}±0.22	3.64^{a}±0.003	54.79±1.08
GLUCO3	1574^{ab}±71.68	827.42^{ced}±3.56	1.9^{cd}±0.09	2.61^{bcd}±0.11	55.16±0.38
Root MSE	112.87	74.51	0.32	0.39	12.45
p-Value	< 0.01	< 0.01	< 0.01	< 0.01	0.93
Toxin Binders	< 0.01	< 0.01	< 0.01	< 0.01	0.71
AFs Levels	0.46	< 0.01	0.16	<0.01	0.49
Toxin Binders × Levels	0.66	0.63	0.99	0.18	0.95
	Main Effects		**Levels**		
0 ppb	1768.0^{a}	1158.42^{a}	1.5267^{b}	1.73^{d}	56.93
100ppb	1530.48^{b}	838.22^{b}	1.8753^{a}	2.309^{c}	55.79
200ppb	1495.10^{b}	771.38^{bc}	1.9977^{a}	2.684^{b}	55.39
300ppb	1488.77^{b}	720.79^{c}	2.1159^{a}	3.089^{a}	55.11

[1] NC: Negative Control, PC: Positive Control, YSCW: Yeast Sludge Cell wall, YSCS: Yeast Sludge Cell Soluble, Gluco: Glucomannan Product. Different superscripts in column differ significantly ($p < 0.05$).

		Toxin Binders			
NC	1768^{a}	1158.42^{a}	1.5267^{c}	1.726^{d}	56.93
PC	1353.63^{c}	529.89^{d}	2.5956^{a}	3.322^{a}	54.24
YSCW	1548.43^{b}	875.90^{b}	1.7852^{bc}	2.28^{c}	55.1
YSCS	1480.58^{bc}	722.74^{c}	2.0611^{b}	2.99^{b}	55.33
GLUCO	1568.85^{b}	917.16^{b}	1.739^{bc}	2.24^{c}	55.95

Table 3. Effect of Different Toxin Binders at Different Levels of Aflatoxins (B_1 and G_1) on Serum Biochemistry.

Treatments[2]	**Sodium**	**Potassium**	**Chloride**	**Magnesium**	**Zinc**
	----------mg / dL----------				
NC	341.82^{a}±3.96	36.06^{a}±1.83	369.39^{a}±2.54	2.49^{a}±0.06	0.62^{a}±0.07
PC1	301.7^{c}±4.39	32.58^{bdac}±1.75	360.91^{ba}±3.06	2.36^{a}±0.08	0.59^{a}±0.06
PC2	262.52^{d}±10.29	31.65^{bdac}±1.48	347.06^{b}±4.1	2.2^{ba}±0.11	0.4^{ba}±0.05
PC3	211.66^{e}±3.3	21.35^{e}±1.66	356.23^{ba}±2.93	2.01^{b}±0.16	0.24^{b}±0.04
YSCW1	342.48^{a}±7.28	34.78^{ab}±0.76	364.36^{ba}±2.99	2.4^{a}±0.08	0.63^{a}±0.09
YSCS1	319.71^{b}±7.41	33.11^{bac}±1.01	361.64^{ba}±8.59	2.37^{a}±0.15	0.52^{a}±0.05
GLUCO1	349.43^{a}±3.47	34.33^{ba}±1.55	367.59^{a}±8.38	2.48^{a}±0.03	0.62^{a}±0.11
YSCW2	335.33^{ba}±2.58	32.28^{bdac}±1.28	359.14^{ba}±2.57	2.34^{a}±0.03	0.62^{a}±0.04
YSCS2	285.96^{c}±7.27	30.09^{bdc}±1.52	353.06^{ba}±8.88	2.34^{a}±0.11	0.49^{a}±0.09
GLUCO2	339.35^{a}±5.41	33.17^{bac}±1.28	362.42^{ba}±5.14	2.39^{a}±0.05	0.6^{a}±0.079
YSCW3	337.67^{ba}±4.67	28.35^{d}±1.13	359.79^{ba}±2.6	2.33^{a}±0.04	0.54^{a}±0.04
YSCS3	261.92^{d}±8.13	21.75^{e}±1.58	354.83^{ba}±8.54	2.24^{a}±0.06	0.39^{ba}±0.09
GLUCO3	330.83^{ba}±3.64	29.52^{dc}±1.41	355.02^{ba}±4.48	2.37^{a}±0.08	0.56^{a}±0.09
Root MSE	10.32	2.48	9.68	0.16	0.13
P-Value	< 0.01	< 0.01	0.33	0.08	0.02
Toxin Binders	< 0.01	0.01	0.04	0.03	< 0.01
AFs Levels	<0.01	< 0.01	0.09	0.54	0.02
Toxin Binders × Levels	< 0.01	0.12	0.87	0.81	0.54
	Main Effects:			**Levels**	
0 ppb	341.82^{a}	36.06^{a}	369.39^{a}	2.49^{a}	0.625^{a}
100ppb	321.47^{b}	33.55^{ba}	360.37^{ab}	2.39^{ba}	0.587^{a}
200ppb	303.52^{c}	31.12^{b}	353.59^{b}	2.25^{ab}	0.527^{ba}
300ppb	286.32^{d}	25.14^{c}	353.09^{b}	2.18^{b}	0.452^{b}
		Toxin Binders			
NC	341.82^{a}	36.06^{a}	369.39^{a}	2.49^{a}	0.62^{a}
PC	258.63^{c}	28.53^{c}	354.73^{b}	2.19^{b}	0.41^{c}
YSCW	338.49^{a}	31.8^{b}	361.09^{ab}	2.36^{ab}	0.59^{ab}
YSCS	289.19^{b}	28.32^{c}	356.51^{b}	2.31^{ab}	0.47^{bc}
GLUCO	339.87^{a}	32.34^{b}	361.68^{ab}	2.42^{a}	0.59^{ab}

[2] NC: Negative Control, PC: Positive Control, YSCW: Yeast Sludge Cell wall, YSCS: Yeast Sludge Cell Soluble, Gluco: Glucomannan Product. Different superscripts in column differ significantly ($p < 0.05$).

Table 4. Effect of Different Toxin Binders at Different Levels of Aflatoxins (B1 and G1) on Serum Biochemistry.

Treatments[3]	Phosphorus	Calcium	Copper	Albumin	Cholesterol
	mg / dL		ppm	g/Dl	mg / dL
NC	6.96^{a}±0.16	10.38^{a}±0.36	0.6^{a}±0.05	1.45^{ba}±0.03	135.26^{a}±2.48
PC1	6.65^{ba}±0.04	9.97^{bdac}±0.1	0.42^{bdac}±0.04	1.07^{de}±0.06	122.15^{bdc}±3.52
PC2	6.74^{a}±0.026	9.6^{dec}±0.14	0.34^{bdc}±0.04	0.53^{g}±0.03	112.73^{gf}±3.81
PC3	6.63^{ba}±0.05	9.28^{e}±0.09	0.28^{d}±0.04	0.39^{g}±0.03	105.18^{g}±2.26
YSCW1	6.79^{a}±0.11	10.39^{a}±0.05	0.59^{a}±0.1	1.45^{ba}±0.03	127.78^{bac}±2.16
YSCS1	6.56^{ba}±0.31	9.45^{de}±0.28	0.54^{ba}±0.04	1.11^{cde}±0.12	124.96^{bdc}±2.79
GLUCO1	6.8^{a}±0.099	10.27^{ba}±0.04	0.56^{a}±0.04	1.57^{a}±0.06	130.32^{ba}±1.76
YSCW2	6.79^{a}±0.052	10.25^{bac}±0.08	0.51^{bac}±0.03	1.29^{bcd}±0.02	126.86^{bdc}±2.62
YSCS2	6.12^{b}±0.15	9.71^{ebdc}±0.36	0.51^{bac}±0.08	0.75^{f}±0.12	120.53^{fdec}±1.68
GLUCO2	6.75^{a}±0.073	10.23^{bac}±0.06	0.51^{bac}±0.1	1.33^{bc}±0.03	125.49^{bdc}±1.54
YSCW3	6.747^{a}±0.04	10.21^{bac}±0.13	0.44^{bdac}±0.04	0.9^{ef}±0.03	116.31^{fe}±2.59
YSCS3	6.97^{a}±0.51	9.37^{ed}±0.32	0.34^{dc}±0.07	0.41^{g}±0.06	120.01^{fdec}±3.48
GLUCO3	6.68^{ba}±0.09	10.16^{bac}±0.12	0.45^{bdac}±0.05	1.22^{cd}±0.13	118.83^{fde}±1.72
Root MSE	0.32	0.35	0.103	0.12	4.49
p-value	0.28	< 0.01	< 0.01	< 0.01	< 0.01
Toxin Biners	0.48	< 0.01	< 0.01	< 0.01	< 0.01
AFs Levels	0.49	0.17	<0.01	< 0.01	< 0.01
Toxin Binders × Levels	0.19	0.63	0.95	0.0552	0.64
	Main Effects:			**Levels**	
0 ppb	6.96^{a}	10.38^{a}	0.60^{a}	1.45^{a}	135.26^{a}
100ppb	6.755^{a}	10.05^{ba}	0.517^{ba}	1.24^{b}	125.79^{b}
200ppb	6.626^{a}	9.88^{b}	0.44^{bc}	0.97^{c}	120.26^{b}
300ppb	6.719^{a}	9.76^{b}	0.35^{c}	0.71^{d}	113.56^{c}
			Toxin Binders		
NC	6.96^{a}	10.38^{a}	0.6^{a}	1.45^{a}	135.26^{a}
PC	6.67^{ab}	9.62^{b}	0.35^{c}	0.67^{c}	113.36^{c}
YSCW	6.77^{ab}	10.28^{a}	0.51^{ab}	1.21^{b}	123.65^{b}
YSCS	6.55^{b}	9.51^{b}	0.46^{bc}	0.76^{c}	121.83^{b}
GLUCO	6.74^{ab}	10.22^{a}	0.51^{ab}	1.37^{a}	124.88^{b}

Table 5. Effect of Different Toxin Binders at Different Levels of Aflatoxins (B1 and G1) on serum Parameters.

Treatments[4]	ALAT	ASAT	Uric Acid	Total Serum Protein
	IU/L		mg / dL	g/dL
NC	24.3^{c}±1.49	137.33h±8.67	5.59^{a}±0.06	2.56^{a}±0.04
PC1	28.72^{abc}±4.7	180.18^{ced}±6.43	4.9^{bc}±0.05	2.05^{dce}±0.07
PC2	32.11^{ab}±1.79	207.55^{b}±5.43	4.56^{dc}±0.12	1.68^{fg}±0.03
PC3	33.91^{a}±2	236.1^{a}±8.6	4.12^{e}±0.14	1.45^{g}±0.05
YSCW1	24.43^{c}±1.89	142.86^{gh}±9.67	5.23^{b} ±0.03	2.2^{dc}±0.06
YSCS1	28.03^{abc}±1.1	173.52^{fed}±5.6	5.13^{b}±0.12	2.09^{dec}±0.06
GLUCO1	24.0^{bc}±1.06	164.36^{gef}±4.76	5.26^{ba}±0.06	2.48^{ba}±0.08
YSCW2	26.61^{bc}±1.76	154.85^{ghf}±4.19	5.02^{b}±0.05	2.02^{dce}±0.04
YSCS2	30.38^{abc}±2.08	200.75^{cb}±7.9	4.61^{dc}±0.1	2.04^{dce}±0.09
GLUCO2	26.71^{c}±0.49	155.75^{hfgi}±3.8	5.14^{b}±0.03	2.28^{bc}±0.1
YSCW3	27.17^{bc}±1.52	170.74^{fed}±10.85	4.92^{bc}±0.11	1.91^{fe}±0.026
YSCS3	33.22^{ab}±1.93	211.36^{b}±7.22	4.32^{de}±0.08	1.58^{g}±0.2
GLUCO3	27.79^{abc}±1.09	190.06^{bcd}±6.19	5.06^{b}±0.27	1.99^{de}±0.08

[3] NC: Negative Control, PC: Positive Control, YSCW: Yeast Sludge Cell wall, YSCS: Yeast Sludge Cell Soluble, Gluco: Glucomannan Product. Different superscripts in column differ significantly ($p < 0.05$).

Root MSE	3.47	12.45	0.19	0.14
p-Value	< 0.01	< 0.01	< 0.01	< 0.01
Toxin Binders	< 0.01	< 0.01	< 0.01	< 0.01
AFs Levels	0.02	< 0.01	< 0.01	< 0.01
Toxin Binders × Levels	0.99	0.11	0.098	0.29
	Main Effects:		**Levels**	
0 ppb	24.30^{c}	137.33^{d}	5.59^{a}	2.565^{a}
100ppb	26.17^{bc}	160.21^{c}	5.08^{b}	2.243^{b}
200ppb	28.65^{ba}	177.86^{b}	4.87^{c}	1.99^{c}
300ppb	30.38^{a}	198.44^{a}	4.56^{d}	1.729^{d}
		Toxin Binders		
NC	24.3^{b}	137.33^{c}	5.59^{a}	2.56^{a}
PC	31.58^{a}	207.94^{a}	4.53^{c}	1.73^{d}
YSCW	26.07^{b}	156.15^{b}	5.06^{b}	2.04^{c}
YSCS	30.55^{a}	195.21^{a}	4.69^{c}	1.9^{c}
GLUCO	26.17^{b}	170.06^{b}	5.16^{b}	2.25^{b}

[4] NC: Negative Control, PC: Positive Control, YSCW: Yeast Sludge Cell wall, YSCS: Yeast Sludge Cell Soluble, Gluco: Glucomannan Product

REFERENCES

Abo-Norag, M., T.S. Edrington, L.F. Kubena, R.B. Harvey and T.D. Phillips (1995). Influence of a hydrated sodium calcium aluminosilicate and virginiamycin on aflatoxicosis in broiler chickens. Poult. Sci.74(4): 626–632.

AOAC**.** 1990. Association of Official Analytical Chemists. 15th Ed. Washington, D.C.

Basmacioglu, H., H. Oguz, M. Ergul, R. Col and Y.O. Birdane (2005). Effect of dietary esterified glucomannan on performance, serum biochemistry and heamatology in broiler exposed to aflatoxin. Czech J. Anim. Sci.50(1): 31-39.

Binder, E.M. (2007). Managing the risk of mycotoxins in modern feed production. Anim. Feed Sci. Tech.133(1): 149–166.

Çelİk, K., A.N. Uluocak, T. Ayaşan, F. Doran and O. Öztürkcan (1999). The effects of different levels of mycotoxin (AFB_1) on Japanese quail's performance and its histopatholigycal characteristics. Proc.: VIV Poultry YUTAV'99 Uluslararası Tavukçuluk Fuarı ve Konferansı, 3-6 Haziran, İstanbul. YUTAV'99 Bildiriler Kitab, pp. S:222-230.

Chemat, F. and M.K. Khan (2011). Applications of ultrasound in food technology: Processing, preservation and extraction. Ultrason. Sonochem.18(4): 813–835.

Chen, C., X. Yang, J. Wei, X. Tan and X. Wang (2013). EU (III) uptake on rectorite in thepresence of humic acid: a macroscopic and spectroscopic study. J. Colloid Interface. Sci.393: 249-256.

Chen, X., N. Horn and T.J. Applegate (2014). Efficiency of hydrated sodium calcium aluminosilicate to ameliorate the adverse effects of graded levels of aflatoxin B_1 in broiler chicks. Poult. Sci.93(8): 1–11.

Commission, T.E. (2010). Commission regulation (EU) No. 165 / 2010 of 26 February 2010, amending regulation (EC) No. 1881 / 2006 setting maximum levels for certain contaminants in foodstuffs as regards aflatoxin. Off. J. Eur. Union. 50: 8-12.

Duncan, D.B. (1955). Multiple range and multiple F tests. Biometrics.11: 1-42.

Geciova, J., D. Bury and P. Jelen (2002). Methods for disruption of microbial cells for potential use in the dairy industry-A review. Int. Dairy J.12(6): 541–553.

Girish C. and G. Gevegowda (2004). Evaluation of modified glucomannan (Mycosorb®) and Hydrated Sodium calcium aluminosilicate to emiliorate the individual and combined toxicity of aflatoxin and T-2 toxin in broiler chickens. Poult. Sci. Symp. 16: 126-129.

Girish C.K. and G. Devegowda (2006). Efficacy of glucomannan-containing yeast product (Mycosorb) and hydrated sodium calcium aluminosilicate in preventing the individual and combined toxicity of aflatoxin and T-2 toxin in commercial broilers. Asian Australas. J. Anim. Sci. 19: 877–883.

Glahn, R.P., K.W. Beers, W.G. Bottje, R.F. Wideman Jr, W.E. Huff and W. Thomas (1991). Aflatoxicosis alters avian renal function, calcium, and vitamin D metabolism. J. Toxicol. Env. Heal. A.34(3): 309-321.

Hashmi, I., T. N. Pasha, M. A. Jabbar, M. Akram and A. S. Hashmi (2006). Study of Adsorption potential of yeast sludge against aflatoxins in broiler chicks. The J. Anim. Pl. Sci.16(1-2): 12-14.

Henry, R.J., D.C. Cannon and J.W. Winkelman**.** (1974). Princeiples and techniques. 2nd Ed. Harper and Row. 525 p.

Huff, W.E. and J.A. Doerr (1981). Synergism between aflatoxin and ochratoxin A in broiler chickens. Poult. Sci.60(3): 550-555.

Jigang, C. and W. Wenjun (2013). Effects of astaxanthin and esterified glucomannan on hematological and serum parameters, and liver pathological changes in broilers fed aflatoxin-B_1-contaminated feed. Anim. Sci. J.85(2): 150–157.

Kaki, S., M.M. Moeini and J. Cheraghi (2012). Effects of zeolite and mycosorb on serum biochemical and hematological parameters of broilers chicken aflatoxicosis. J. Blood Lymph2(2): 1-4.

Kubena, L.F., R.B. Harvey, W.E. Huff, M.H. Elissalde, A.G. Yersin, T.D. Phillips and G.E. Rottinghaus (1993). Efficacy of hydrated sodium calcium aluminosilicate to reduce the toxicity of aflatoxin and diacetoxyscirpenol. Poult. Sci.72(1): 51 – 59.

Li, B. and D.-W. Sun (2002). Effect of power ultrasound on freezing rate during immersion freezing. J. Food Eng.55(3): 277–282.

Liu, D., N.I. Lebovka and E. Vorobiev (2013). Impact of electric pulse treatment on selective extraction of intracellular compounds from *Saccharomyces cerevisiae* yeasts. Food Bioprocess Tech.6(2): 576-584.

Mogadam, N. and A. Azizpour (2011). Ameliorative effect of glucomannan-containing yeast product (Mycosorb) and sodium bentonite on performance and antibody titers against Newcastle disease in broilers during chronic aflatoxicosis. Afr J Biotechnol. 10: 17372-17378.

Magnoli, A.P., M.P. Monge, R.D. Miazzo, L.R. Cavaglieri, C.E. Magnoli, C.I. Merkis, A.L. Cristofolini, A.M. Dalcero and S.M. Chiacchiera (2011). Effect of low levels of aflatoxin B_1 on performance, biochemical parameters and aflatoxin B_1 in broiler liver tissues in the presence of monensin and sodium bentonites. Poult. Sci.90(1): 48-58.

Monson, M.S., R.A. Coulombe and K.M. Reed (2015). Aflatoxicosis: Lessons from toxicity and responses to aflatoxin B_1 in poultry. Agric5(3): 742-777.

Moschini, M., A. Gallo, G. Piva and F. Masoero (2008). The effects of rumen fluid on the *in vitro* aflatoxin binding capacity of different sequestering agents and *in vivo* release of the sequestered toxin. Anim. Feed Sci. Tech.147(4): 292-309.

Neeff, D.V., D.R. Ledoux, G.E. Rottinghaus, A.J. Bermudez, A. Dakovic, R.A. Murarolli and C.A.F. Oliveira (2013). *In vitro* and *in vivo* efficacy of a hydrated sodium calcium aluminosilicate to bind and reduce aflatoxin residues in tissues of broiler chicks fed aflatoxin B_1. Poult. Sci.92(1): 131-137.

Northcote, D.H. and R.W. Horne (1952). The chemical composition and structure of the yeast cell wall. Biochemical.51(2): 232-239.

NRC**.** (1994). Nutrient requirements of poultry. 9th Ed. (Washington). Naitonal Academy Press. 1-145 p.

Oliveira, C.A., J.F. Rosmaninho, P. Butkeraitis, B. Correa, T.A. Reis, J.L. Guerra, R. Albuquerque and M.E. Moro (2002). Effect of low levels of dietary aflatoxin B_1 on laying Japanese quail. Poult. Sci.81(7): 976–980.

Osweiler, G.D., S. Jagannatha, D.W. Trampel, P.M. Imerman, S.M. Ensley, I. Yoon and D.T. Moore (2010). Evaluation of XPC and prototypes on aflatoxin-challenged broilers. Poult. Sci.89(9): 1887-1893.

Pasha, T.N. (2008). Detoxification of aflatoxin by yeast sludge in the feed of dairy cattle and its impact on increased milk production. I. J. A. V. M.2: 18-26.

Quezada, T., H. Cuellar, F. Jaramillo-Juarez, A.G. Valdivia and J.L. Reyes (2000). Effects of aflatoxin B_1 on the liver and kidney of broiler chickens during development. Comp. Biochem. Physiol. C Toxicol. Pharmacol.125(3): 265-272.

Raju M. and G. Devegowda (2000). Influence of esterifiedglucomannan on performance and organ morphology, serum biochemistry, and haematology in broilers exposed to individual and combined mycotoxicosis (AF, ochratoxin and T-2 toxin). Br. Poult. Sci. 41: 640–650.

Rauber, R.H., P. Dilkin, L.Z. Giacomini, C.A.A. deAlmeida and C.A. Mallmann (2007). Performance of turkey poults fed different doses of aflatoxins in the diet. Poult. Sci.86(8): 1620-1624.

Rec, G.S.C.C. (1972). Estimation of serum aspartate aminotransferase. J. Clin. Chem.10(????): 182.

Safameher A. (2008). Effects of clinoptilolite on performance, biochemical parameters and hepatic lesions in broiler chickens during aflatoxosis. J. Anim. Vet. Adv. 7: 381–388.

Santin, E., A.C. Paulillo, L.S.O. Nakagui, A.C. Alessi and A. Maiorka (2006). Evaluation of yeast cell wall on the performance of broiler fed diets with or without mycototoxins. Braz. J. Poul. Sci.8(4): 221-225.

Shotwell, O.L., C.W. Hesseltine, R.D. Stubblefield and W.G. Sorenson (1966). Production of aflatoxin on rice. Appl. Microbiol.14(3): 425-428.

Steel, R.G.D., J.H. Torrie and D.A. Dickey (1997). Principles and procedures of statistics: A biometrial approach. 3rd Ed. (New York, USA). McGraw Hill Book Co. Inc. 481 p.

Suganthi, R.U., K.P. Suresh and R. Parvatham (2011). Effect of aflatoxin on feed conversion ratio in broilers: A meta-analysis. Asian-Australas. J. Anim. Sci.24(12): 1757-1762.

Tessari E.N.C., E. Kobashigawa, A.L.S.P. Cardoso, D.R. Ledoux, G.E. Rottinghaus and C.A.F. Oliveria (2010). Effects of Aflatoxin B_1 and Fumonisin B_1 on blood biochemical parameters in broilers. Toxins. *2*: 453-460.

Valchev, I., D. Kanakov, T.S. Hristov, L. Lazarov, R. Binev, N. Grozeva and Y. Nikolov (2014). Investigations on the liver function of broiler chickens with experimental aflatoxicosis. Bulg. J. Vet. Med.17(4): 302-313.

Williams, J.H., T.D. Phillips, P.E. Jolly, J.K. Stiles, C.M. Jolly and D. Aggarwal (2004). Human aflatoxicosis in developing countries: a review of toxicology, exposure, potential health consequences, and interventions. Am. J. Clin. Nutr.80(5): 1106-1122.

Yildirim, E., I. Yalcinkaya, M. Kanbur, M. Cinar and E. Oruc (2011). Effects of yeast glucomannan on performance, some biochemical parameters and pathological changes in experimental aflatoxicosis in broiler chickens. Rev. Med. Vet.162(8-9): 413-420.

Yang J., F. Bai, K. Zhang, S. Bai, X. Peng, X. Ding, Y. Li, J. Zhang and L. Zhao (2012). Effects of feeding corn naturally contaminated with aflatoxin B_1 and B_2 on hepatic functions of broilers. Poult. Sci. 91: 2792-2801.

Yunus A.W., E. Razzazi-Fazeli and J. Bohm (2011). Aflatoxin B_1 in affecting broiler's performance, immunity, and gastrointestinal tract: A review of history and contemporary issues. Toxins. 3: 566-590.

Zhao, J., R.B. Shirley, J.D. Dibner, F. Uraizee, M. Officer, M. Kitchell, A.M. Vazquez and C.D. Knight (2010). Comparison of hydrated sodium calcium aluminosilicate and yeast cell wall on counteracting aflatoxicosis in broiler chicks. Poult. Sci.89(10): 2147-2156.