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Performance depression due to ingestion of aflatoxin in poultry

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ABSTRACT

Aflatoxin (AF) (0.5ppm) was tested in an in vivo study forming 2 dietary treatments each with three replicates on a total of 336 on broiler chicks up to six weeks. Results showed that chicks receiving AF contaminated feed had suppressed body weight and improved feed consumption. The serum antibody titers against ND and IBD vaccination were significantly depressed by AF. The serum concentration of total protein, uric acid and albumin were not affected in AF fed supplemented group. The activity of serum GGT significantly increased in AF fed group. Compared with control, activity of serum ALT was not affected in AF or control supplemented groups.

Key Words: Aflatoxin, broilers, performance.

INTRODUCTION

Cereal grains and associated by-products constitute important sources of energy for poultry. There is increasing evidence that global supplies of cereal grains for animal feedstuffs are commonly contaminated with mycotoxins. Aflatoxins are secondary toxic metabolites produced by certain strains of fungi, e.g. *Aspergillus flavus* and *Aspergillus parasiticus* species. Aflatoxin B1 (AFB1), the most toxic of all aflatoxins (AFB1, AFB2, AFG1 and AFG2), is produced by certain strains of fungi in greater quantities than in others. In poultry, aflatoxin ingestion leads to "Aflatoxicosis" syndrome which is characterized by retardation of growth, feed consumption, feed conversion efficiency, bruising, immunosuppression and mortality. Co-contamination of cereal grains with mycotoxins produced by different fungal genera, including *Fusarium* and *Aspergillus* has been reported to increase the toxicity symptoms in poultry [1].

MATERIALS AND METHODS

Experimental animals and design

Three hundred and thirty six, unsexed one-day old commercial broiler chicks were wing banded, weighed and assigned to a 2X1 factorial arrangement of one levels of Aflatoxin AF (0 and 0.5ppm) in a Completely Randomized Design manner, forming a total of 2 dietary treatments each with 3 replicates.

Experimental housing, management and test diet

Each replicate group of chicks was housed in an independent pen in an open sided deep litter conventional house. Chicks in all the replicate groups were reared up to five weeks of age under uniform standard conditions throughout the study. Brooding was done until three weeks of age using incandescent bulbs. Each pen was fitted with an

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automatic bell type drinker and a hanging tubular feeder. Chicks were provided continuous light throughout the study.

Aflatoxin was produced using the pure culture of *Aspergillus parasiticus* MTCC 411 grown on potato dextrose agar. Then toxin produced on rice was then extracted [2] and quantified by thin layer chromatography (TLC) [3].

The experimental diets were prepared by the addition of required quantities of rice containing aflatoxin to arrive at the levels of 0 and 0.5ppm of aflatoxin B_1 . To

Basal diet was formulated and compounded to meet the nutrient requirements of commercial broilers during the starter (0-3 wks) and finisher (4-5 wks) phases. Chicks were provided *ad libitum* supply of feed and water throughout the study. Feeding of test diets commenced at zero day of age and continued until the termination of the experiment at five weeks of age. Chicks were vaccinated against Newcastle Disease (ND) on the 7th day using F_1 strain and against Infectious Bursal Disease (IBD) on the 14th day using intermediate strain. Both vaccines were given via the ocular route.

Data collection

At the end of the trials, body weight, feed consumption and mortality, if any were recorded and gain in weight and feed efficiency were calculated. Six birds from each replicate were sacrificed by cutting the jugular vein at the end of the trial. Blood was collected in non-heparinized tubes from six birds in each treatment (3 males and 3 females) by puncturing the brachial vein during the 5th week of age. Serum was separated after 8 to 10 hours as per the standard procedures [4] and was stored at -20 °C for subsequent analysis. The individual serum samples were analyzed for total protein, serum albumin, uric acid and the activities of gamma glutamyl transferase (GGT) and alanine amino transferase (ALT) using an automatic analyzer (Boehringer Mannhein Hitachi 704 automatic analyzer, Japan), antibody titers against Newcastle disease (ND) and Infectious Bursal Disease (IBD) using ELISA technique.

Statistical analysis

The experimental data were analyzed statistically by using the General Linear Model procedure of the Statistical Analysis System (SAS[®]) software [5]. Overall data were analyzed by repeated measures design. The Duncan multiple range test was used to compare means [6]. The result of this study was subjected to one way ANOVA test.

RESULTS AND DISCUSSION

Body weight, feed consumption, feed conversion ratio and mortality data for broilers fed control and different experimental diets at fifth week of age are presented in Table1. Chicks receiving AF contaminated feed had significantly (P<0.05) suppressed body weight, feed consumption and efficiency of feed utilization compared to chicks fed the control diet. Efficiency of feed utilization which was decreased significantly with addition of 0.5ppm AF inclusion. High mortality rate of 14.20 per cent was observed in the group fed with diet containing 0.5ppm AF.

The decreased body weight, feed consumption and increased feed conversion ratio due to AF are consistent with the findings of other scientists [7, 8, 9 and 10]. The growth depression effects of AF may be due to their inhibitory action on protein synthesis and nutrient utilization [11].

AF (ppm)	Body weight (g)	Feed consumption (g/bird)	Feed Conversion Ratio	Mortality (%)
0	1301.2±0.06 ^a	2452.1±5.07 ^a	1.89±0.0 ^c	3.90
0.5	1086±0.03°	2211±2.82 ^b	1. 91±0.05 ^a	11.10

Means bearing at least one common superscript in a column do not differ significantly (P < 0.05)

The effect of Aflatoxin supplementation on the antibody titers against New Castle Disease (ND) and Infectious Bursal Disease (IBD), serum protein, serum albumin, uric acid, the activities of gamma glutamyl transferase (GGT) and alanine amino transferase (ALT) are presented in Table2. A significant (P<0.05) decrease in antibody titer values against ND and IBD vaccine was observed upon feeding AF. This depression in titer values is a clear indication of immunodepressing effects of AF on humoral antibody response. These findings agree with the

M. R. Pourelmi

previous reports [12]. The reduction of antibody titers could be due to inhibition of DNA and protein synthesis by aflatoxin through impairment of amino acid transport and m-RNA transcription, resulting in lowered level of antibody production [13].

Table 2: Effect of Aflatoxin on the antibody titers against New Castle Disease (ND) and Infectious Bursal Disease (IBD), serum protein, serum albumin, uric acid, the activities of gamma glutamyl transferase (GGT) and alanine amino transferase (ALT) in broilers.

Means bearing at least	one common supers	cript in a column	do not differ	significantly (P<0.0)

AF (ppm)	ND titer	IBD titer	Serum protein (g%)	Serum Albumin (g%)	Uric acid (µg/dl)	GGT (IU/L)	ALT (IU/L)
0	4134.7±77.05 ^{ab}	4194.0±0.08 ^a	2.25±0.11 ^a	1.16 ± 0.16^{a}	635.9±6.23 ^a	8.94 ± 1.06^{d}	26.16±0.60 ^a
0.5	3198±16.3 ^e	3428±6.72 ^d	1.05±0.14 ^{bc}	1.09±0.17 ^a	594.4±5.43 ^a	16.7 ± 1.02^{ab}	24.13±1.36 ^a

REFERENCES

[1] Arvind, K.L, Patil V.S, Devegowda, G, Umakantha, B, Ganpule, S.P. 2003. Poult. Sci., 82: 571-576.

[2] Chaturvedi, V.B, and Singh, K.S. 2004. Anim. Nut. Feed Technol. 4: 187-195.

[3] Daoud, A.Z, 2002. Iraqi J. Vety. Sci. 16:161-165.

[4] Devegowda, G, Arvind, B.I.R, Rajendra, K, Morton, M.G., Baburathna, A, Undareshan, C. **1994.** Proc. Altech's 10th Ann. Symp., Ky, USA, pp. 235-245.

[5] AOAC, 1995. Association of Official Analytical Chemists, Washington, D.C.

[6] Duncan, D.B, **1955.** *Biometrics*. 11:1-42.

[7] Gupta, K, and Amarjit Singh, 2003. Ind. J. Vety. Pathol. 27: 5-7.

[8] Ibrahim, I.K, Shareef, A.M, and Joubory, K.M.T, 2000. Res. in Vet. Science, 69:2, 119-122; 35 ref.

[9] Kurnick, A.A, Reid, B.K, 1989. Feedstuffs, 32: 34.

[10] Marquardt, D.R, and Frohlich, A, 1992. J. Anim. Sci.70: 3968-3988.

[11] Miazzo, R, Pevalta, M.F, Magnoli, C, Salvano, M, Ferrero S, Chiacchiera S.M, Carralno E.C.Q, Rosa C.A.R, Dalcero, A. **2005**. *Poult. Sci.* 84:1-8.

[12] Perozo, F, and Rivera, S, 2003. Ind. Vet. J. 80: 1218-1221.

[13] SAS Institute (2000). SAS Institute Inc., Cary, NC,