International Journal of Advanced Research in Biological Sciences
www.ijarbs.com

Research Article

Changes in the level of protein and glycogen in liver of one week old *Plymouth Rock* broilers during experimental aflotoxicosis

K. Sandhya, G. Sakunthala, P.J.R. Nathanael* and V.V.Vardhani

Department of Zoology and Aquaculture, Acharya Nagarjuna University
Nagarjuna Nagar. Andhra Pradesh, India

*Corresponding author e-mail: *nathanael_pj@yahoo.co.in*

Abstract

Aflotoxins strictly pose as potent Carcinogenic, hepatotoxic and mutagenic bioagents causing deleterious effects in the poultry sector. Aflotoxicosis in poultry birds causes various acute and chronic ailments and even mortality. The present investigation are carried in one week old (wt 170-180gm) *Plymouth Rock* strain of broilers to identify the changes in the protein and glycogen content in liver. Two groups (A&B) of broilers were orally intubated with varied doses of AFB1 ,and another group (C) was kept as control for comparison. Broilers were sacrificed at day 1, 3, 8 and 11 of infection and the samples from liver were collected and processed for protein and glycogen estimation. The results showed the AFB1 brought alterations in the liver metabolism which eventually influenced the content of protein and glycogen.

Keywords: *Plymouth Rock* broilers, Aflotoxicosis, AFB1, protein, glycogen liver.

Introduction

Toxigenic strains of Aspergillus fungi exudes its metabolites referred as aflotoxins, of it the most potent and widely distributed is AFB1. It emanates wide range of toxicity, mutagenicity and it contributes itself to group I carcinogens (Buchi and Rae , 1969). AFB1 contamination in broilers causes reduced performance, anorexia, excessive liver damage, hepatomegaly, fatty liver syndrome, cirrhosis, severe odema, rectal prolapse and even death hailing severe economic down fall in poultry enterprise (Anjum et al., 1989; Toro et al., 2000). AFB1 causes profound immunosupression in broilers effecting the cell mediated immune response causing atrophied thymus and decreased peripheral T - lymphocyte numbers thereby the complement activity (Nathanael and Vardhani, 2011). AFB1 sensitizes the Gastro-intestinal tract, bursa fabricus , thymus and cecal tonsils and worsen the immune resistance mechanism and paves the broilers exposed to opportunistic infections like fowl typhoid , cecal coccidiasis and Marek's disease (Besaratinia et al., 2009). Hence a new vista has been opened to determine the level of protein and glycogen in one week old broilers infected with two doses of AFB1.

Materials and Methods

One week old *Plymouth Rock strain* (wt 170-180gm) broilers were procured and kept in open

litter system and fed with balanced standard diet. AFB1 suspension was orally intubated to two groups of experimental broilers (group A; AFB1 @ 0.01ng/ml/bird); (group B; AFB1@ 0.25ng/ml/bird) and group C was kept as control for comparison. All the experimental and control animals were sacrificed on day 1, 3, 8 and 11 of infection. Liver tissues were collected and processed for estimation of protein and glycogen content following the method of Lowry *et al*., (1951) and Kemp *et al.,* (1954) respectively.

Results and Discussion

The experimental broilers showed signs of severe weakness, reduced appetite, pale and enlarged /molted liver, with multiple hemorrhages. *Odematous bursa,* and consistent spleenomegaly is evident in autopsy findings. Congested kidneys and inflamed intestine is observed. The level of protein in experimental broilers of group A (AFB1 @ 0.01ng/ml/bird) showed higher response when compared to controls (group C) throughout the experimental tenure. Even though, the level of proteins are higher than the controls, there is a gradual decrease of proteins from day 1(84.1ng/ml) to 11(32.9ng/ml). The broilers of group B (which received the higher/doubled dose of AFB1) manifested higher protein levels throughout the experimental period than the controls. On day 1 and 3 the protein level is at constancy but on day 3 there was a slight decrease; but on day 11 there is an exponential raise of proteins were observed (Table 1). In case of broilers of group A; lowered content of glycogen was manifested than the controls, throughout the experimental period.

.

Table 1. Protein content in the liver of control (group C) and AFB1 intubated (group A, 0.01ng/ml/bird ; group B, 0.1ng/ml/bird) one week old broilers at different days of experimental period. Values are expressed in mean derived from 5 observations.

Days of necropsy	Group A (ng/ml)	Group B (ng/ml)	Group C (ng/ml)
1	84.1	42.6	26.3
3	57.3	42.6	26.5
8	42.0	33.4	26.4
11	32.9	159.6	26.2

Table 2. Glycogen content in the liver of control (group C) and AFB1 intubated (group A, 0.01ng/ml/bird ; group B, 0.1ng/ml/bird) one week old broilers at different days of experimental period. Values are expressed in mean derived from 5 observations.

Days of necropsy	Group A (mg/gm)	Group B (mg/gm)	Group C (mg/gm)
1	6.44	7.71	6.42
3	6.20	7.62	6.49
8	5.91	6.63	6.51
11	5.20	6.59	6.46

On the other hand the broilers which received high dose of AFB1 suspension (group B) showed slightly elevated levels of glycogen than the control broilers throughout the experimental duration. Though it is said to be slightly elevated levels; there is a gradual decrease is found from day 1 to 11 (Table. 2).

The decreased content of glycogen clearly indicates the malfunctioning of liver and onset of chronic hepatic ailments due to AFB1 exposure as suggested by Zimmerman (1970) and Viveka Vardhani and Nathanael (2011).{iscap paper}. It is clear from the above manifestations that AFB1 when orally intubated caused the disturbances in protein and DNA synthesis mechanisms by producing an intermediary metabolite epoxide thus breaking the DNA double strands in the liver which

resulted in aberrational changes in the levels of protein and DNA as suggested by Shivachandra et al., (2004) and Nathanael and Vardhani (2008). The disturbance in protein /glycogen metabolism in liver (the target organ) might have resulted from DNA strand breakage and release of ROS species due to severe stress manifested by broilers of group A and B caused by AFB1. These findings may be similar with that of Nathanael and Vardhani (2014), who reported increased SOD levels in liver of mice treated with Gene VacB vaccine during experimental hepatitis B.

As aflotoxins acts as biosynthetic inhibitors; AFB1 inhibits glycogensis, transport of glucose to liver and glucogenolysis; this resulted in disturbance and decrease in liver glycogen metabolism in both the experimental groups. These findings correlate with that of Madhuri *et al*., (2009) who suggested that there is an increased level of serum ALT and AST which is caused by the leakage of transaminases into the serum due to the destruction of hepatocytes in the liver due to AFB1. These findings opens a wide spectrum of innovate thoughts to emulate on the other focal dimensions of AFB1 altered mechanisms in broilers which impede the poultry sector and renovate it to its prime estate.

Acknowledgements

The Author (Nathanael PJR) expresses his gratitude to Prof. Viveka Vardhani, former Head Department of Zoology and Aquaculture for providing laboratory facilities. The author dedicates this paper to his beloved Grandparents Jesuratnam and Esther Vedanayakam for their earnest prayers and blessings.

References

Anjum, A.D., Sabbri, M.A. and Iqbal, Z. 1989. Hydro pericardium syndrome in broiler chicken in Pakistan. Veterinary Record. 124:247-248.

Besaratinia, A., Kim, S.I, Hainaut, P, and Pfeifer, G.P. 2009. In vitro recapitulating of TP53 mutagenesis in hepatocellular carcinoma associated with dietary Aflotoxin B1 exposure. Gastroenterol. 137(3):1127-37.

Buchi, G., and Ian. D Rae. 1969. The structure and chemistry of the aflatoxins. In "Aflatoxin" edited by L.A. Goldblatt Academic Press, New York.pp. 55-73.

Kemp, A., Vankits and Haljnimgem, A.J.M. 1954. A colorimetric method for the determination of glycogen in tissue. Biochem. J. 646-648.

Lowry, O.H., Rosebrough, N.J, Farr, A.L and Randall, R. J. 1951. Protein measurement with folin phenol reagent. J.Biol. Chem. 177 : 751 – 766.

Nathanael, P.J.R., and Vardhani, V.V. 2008. The pathogenesis of experimental Aflotoxicosis on protein and DNA content of liver in broilers. Asian J. of Microbio. Biotech. Env. Sci. 10 (4):839-841.

Nathanael, P.J.R., and Viveka Vardhani. 2011. Comparison of the protein and DNA activity in the liver and spleen of one week and two weeks old broilers during chronic aflotoxicosis. Asian. J.Microbiol.Biotech.Env.Sci. 13(3):519-523.

Nathanael, P.J.R., and Vardhani V.V. 2014. The influence of Immunex DS against experimental Hepatitis B vaccine on liver protein and DNA profile of mice. J. Biolife. 2(1):341-347.

Madhuri, N.S.K., P.J.R. Nathanael and V. Viveka Vardhani. 2009. Effect of aflotoxin B1 on serum proteins, aspartate transaminase and alanine transaminase of broilers ANU J. of Natur. Sci. 1(1):96-100.

Shivachandra, S.B., Singh, S.D, Kataria, J.M. and Manimaran, K. 2004. Comparative pathological changes in aflotoxin fed broilers infected with hydropericardium Syndrome. Ind.J. of Ani. Sci. 74(6) : 600-604.

Toro, H., Gonzabez, C, Cerdal, Hess, M, Reyes, E and Geisse C. 2000. Chick anemia virus and fowl adenovirus: Association to induced inclusion body hepatitis/hydropericardum syndrome. Avian dis., 44: 51-58.

Viveka Vardhani, V., and Nathanael, P.J.R. 2011. The panoramic view of changes in physiology and biochemical aspects in broilers suffering due to experimental aflotoxicosis: a review Biohelica: Ind.J. Comp. Ani. Physiol. 29 :1-7.

Zimmerman, H.J., and Seeff, L.B. 1970. Enzymes in hepatic disease. In coodley, EL(ed): Diagnostic enzymology, Philadelphia, Lea and Febiger.1-38.

.