

# Disturbances in Serum Enzymes and Total Protein in Aflatoxicated Chickens

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## ABSTRACT

This study was conducted to investigate the effect of induced aflatoxin (B<sub>1</sub>; @ 1.5 mg/kg body weight subcutaneously) toxicity on the serum enzymes activity in 21 days old Fayoumi chickens. Aflatoxicosis caused a significant rise in the activity of Aspartate Amino Transferase, Alanine Amino Transferase, Alkaline Phosphatase and Lactate Dehydrogenase; and significantly depressed the total proteins of the chickens in comparison to un-treated control.

**Key Words:** Aflatoxin B<sub>1</sub>; Serum Enzymes; Chicks; Total protein

## INTRODUCTION

Mycotoxicosis causes great economic losses. Evaluation of the role and extent of mycotoxin contamination in feed-stuffs and foods is a valuable area of research. More than 200 toxic compounds produced by moulds and fungi have been recognized. However, their full toxicological significance is not yet fully known (Audrey *et al.*, 1994). There are nearly 12 different genera of fungi which are involved in mixed poultry feed, the commonest being *Aspergillus*, *Fusarium*, *Penicillium* and Yeast which produce aflatoxins B<sub>1</sub>, B<sub>2</sub>, G<sub>1</sub>, G<sub>2</sub>, Ochratoxins A,B, Citrinin and Kojic acid etc. Their pathological lesions in broilers consist of enlarged heart, liver, gall bladder and kidneys with occasional blood spots on the liver and body musculature. Usually lipid metabolism is impaired with the continued feeding of contaminated feed leading to a fatty liver like condition. The presence of aflatoxins increases the dietary requirements for proteins, and vitamins like A and D (Murthy *et al.*, 1986). In view of the seriousness of aflatoxicoses in chickens, the present study was undertaken to assess serum enzyme levels in chickens affected with aflatoxin B<sub>1</sub>.

## MATERIALS AND METHODS

Fifty, day-old chicks of Fayoumi breed were reared at Feed Testing Laboratory, Poultry Research Institute, Rawalpindi under optimal managerial conditions. The chicks were fed starter ration *ad libitum* and had free access to clean and fresh drinking water. The birds were divided into two groups, at 21 days of

age. The birds in group B were injected with aflatoxin B<sub>1</sub> dissolved in corn oil (@ 1.5 mg/kg body weight), while those in group A were given injections of corn oil only to serve as control. After 24 hours, the birds of both groups were slaughtered and the blood samples were drawn, centrifuged to collect serum and stored at -20°C. These blood samples were analysed for total proteins, aspartate aminotransferase (ASAT), alanine aminotransferase (ALAT), alkaline phosphatase (AP) and lactate dehydrogenase (LDH) by chemical kit method (Frankle *et al.*, 1970; Bergmeyer & Bernt, 1974) on double beam spectrophotometer (UVIDEC 430B/JASCO-JAPAN).

## RESULTS AND DISCUSSION

The results showed that ASAT levels in blood samples of control and aflatoxin treated groups were 167.515 and 169.071 unit/L, respectively (Table I). The elevation of ASAT may be due to disruption of hepatic cells as a result of necrosis or as a consequence of altered membrane permeability (Coles, 1974). Increased level of ALAT in aflatoxin affected (213.554) verses normal (184.746) birds could be associated with hepatocellular damage in affected birds (Moss & Butterworth, 1974). Boyd (1983) concluded that ASAT and ALAT are the serum enzymes which are sensitive specific indicator of liver damage.

The average values of AP in control and infected birds were 339.102 and 700.108 units/L, respectively. The increased AP activity in infected birds may be due to the adverse effect of aflatoxin on kidneys and liver function. Stigbrand *et al.* (1984) discovered that the

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**Table 1. Mean  $\pm$  SD (n=25) of serum enzymes (unit/L) and total protein (mg/dl) in aflatoxicated and control Fayoumi chicks**

Para.	Control	Treated	Prob. of "t"
↓	Mean $\pm$ SD	Mean $\pm$ SD	↓
ASAT	167.515 $\pm$ 15.236 (148.89-200.43)	169.071 $\pm$ 49.936 (114.54-302.24)	0.8844 n.s.
ALAT	184.742 $\pm$ 38.669 (128.78-268.78)	213.557 $\pm$ 51.692 (150.68-332.59)	0.0399 *
AP	339.102 $\pm$ 80.351 (147.89-439.63)	700.105 $\pm$ 69.557 (494.64-793.34)	0.000 **
LDH	137.496 $\pm$ 65.480 (33.02-327.27)	144.946 $\pm$ 53.846 (94.23-342.54)	0.6114 n.s.
TP	5.718 $\pm$ 1.201 (3.51-7.73)	4.414 $\pm$ 0.923 (3.15-5.96)	0.005 **

Values in paranthesis are ranges; \*Significant (P<0.05);

\*\*Highly significant (P<0.01); n.s.=Non-significant

Para= Parameters; Prob.= Probability; ASAT= Aspartate aminotransferase; ALAT= Alanine aminotnsferase; AP= Alkaline phosphatase; LDH= Lactate dehydrogenase; TP= Total protein

activity of AP was significantly (P<0.01) increased in bone and liver diseases. The results further showed that LDH activity was 137.496 and 144.946 units/L in the un-treated control and experimentally infected birds, respectively. The increase in LDH activity due to mycotoxicosis was found to be significant (P<0.05) as Benjamin (1978) stated that LDH may be elevated in many disease processes in which there is cell necrosis. The average values of total proteins in the control and infected birds were found to be 5.718 and 4.414 mg/dl, respectively. The results indicated that mycotoxicosis caused loss of protein which appeared to be the cause of reduced weight gain in affected birds.

The findings of the present study are in agreement with those of Gylstorff and Rolf (1982), and Balachandran and Ramakrishnan (1988) who reported changes in concentration of enzymes due to aflatoxin toxicity in birds.

## REFERENCES

- Audrey H. E., J.E. Konlade and J.R.K. Robson, 1994. Poisons (Aflatoxins). In: *Food and Nutrition Encyclopedia*. 2nd ed., 2: 1790-8.
- Balachandran, C. and R. Rama-krishnan, 1988. Influence of dietary aflatoxin on certain serum enzyme levels in broiler chickens. *Mycopathologia*, 101: 65-7.
- Benjamin, M.M., 1978. *Outline of Veterinary Clinical Pathology*. 3rd ed., Iowa State University Press, Ames, Iowa, USA.
- Bergmeyer, H.U. and S. Bernt, 1974. *Methods of Enzymatic Analysis*. pp: 727-33. Academic Press, New York, USA.
- Boyd, B.A., 1983. Clinical Enzymology. In: *Clinical Biochemistry of Domestic Animals*. 4th ed., pp: 338-63. Academic Press, Inc., USA.
- Coles, E.H., 1974. *Veterinary Clinical Pathology*. 3rd ed., W.B. Saunders Co., Philadelphia, USA.
- Frankle, S., S. Reitman and A.C. Somenwirth, 1970. *Gardwhol's Clinical Laboratory Methods and Diagnosis*. 7th ed., 2: 113-35.
- Gylstorff, I. and J. Rolf, 1982. Influence of Mycotoxins on some clinico-chemical parameters and on enzyme activities in fresh liver tissue of young chickens. *Deutshe Tieraztlich Wochenschrift*, 89: 16-9.
- Moss, D.W. and P.J. Butterworth, 1974. *Enzymology and Medicine*. 1st ed., pp: 139-51. Pitman Medical Co., London.
- Murthy, A.S., M. MaHender and P.R.Rao, 1986. Vascular lesions in experimental aflatoxicosis in chicken. *Cheiron*, 15: 74-7.
- Stigbrand T., J.L. Millan and W.H. Fishman, 1984. Human Alkaline Phosphatase. In: *Clinical Biochemistry of Domestic Animals*. 4th ed., pp: 338-63. Academic Press Inc., New York, USA.

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