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ON THE EFFECT OF AFLATOXIN ON LIPID METABOLISM IN THREE STRAINS OF BROILER CHICKEN

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Presence of 250 ug aflatoxin B_1 /kg feed caused a decrease in the liver weight of all the three strains of broiler chicken by the end of the first week. During the second week however, the liver weight started increasing and by the end of the third week, the increase in weight became more pronounced as compared to the controls. Reverse was the case with serum lipid and serum cholesterol. Possible reasons for these changes are discussed.

Key words: Aflatoxin, Chicken serum lipids, Serum cholesterol, Liver weight.

Introduction

Aflatoxicosis in commercial poultry is a serious problem in places with high humidity as it favours growth of aflatoxin-producing fungi in the feed. Karachi, the coastal city of Pakistan and major seat of poultry husbandry in the country, has high levels of humidity for most part of the year and therefore, fraught with the danger of aflatoxicosis.

Aflatoxins are bisfuranocoumarins produced as secondary metabolites by strains of *Aspergillus flavus* and *Aspergillus parasiticus*. It has been shown that the presence of aflatoxins in the poultry feed can cause a significant increase in the lipid content of the excreta of chicken [1], decrease in bile salts and pancreatic lipase concentration affect the digestion and absorption of lipids from intestine[2]. Further more, it can cause disturbances in the metabolism of essential nutrients [3] and an enlargement of liver possibly due to accumulation of lipids [4-8]. In this study effects of 250 ug/kg of aflatoxin B_1 (AFB) in the feed were observed on the liver weight, serum lipids and cholesterol, of the three strains of broiler chickens.

Materials and Methods

Three strains of healthy, unvaccinated day-old broiler chicks were obtained from the three well established commercial hatcheries of Karachi. Each set of strain comprising of 40 chicks was marked with different colours and designated as "A", "B" and "C". The chicks weighing between 30 and 40 gms were randomly divided into the control groups and those which were to receive contaminated feed. The birds were housed in litter-based pens under continuous illumination. Feed and water were provided *ad-libitum*. A pure culture of *Aspergillus flavus* NRRL 3357 was obtained from North Regional Research Laboratory Peoria, Ill., USA. Aflatoxin was produced on broken rice [9], and incorporated in the feed to make it toxic. A balance of broken rice was maintained in both the control and treated feeds. Ten samples from both the feeds were analysed with CB method [10], quantified by comparison of standards and confirmed by triflouroacetic acid derivatives and by spraying 50% H_2SO_4. The control feed had no aflatoxin within detectable limit, while the toxic (treated) feed contained 250 ug AFB_1/kg of feed.

Three birds were randomly removed from each set of strain 'A', 'B' and 'C' both from among the controls and the test groups and sacrificed to collect the blood and the liver after intervals of 7, 14 and 21 days. Blood was collected and serum was pooled for determining the total serum lipids and cholesterol. Livers were immediately excised, dried with filter paper to remove blood and weighed on pooled basis. Photometric determinations of total serum lipids and cholesterol were carried out using diagnostic kits, Merckotest 3321 and 2312 respectively obtained from E.Merck of West Germany.

Results

Liver weight. During the first week, the mean liver weight in all the treated chicks was less than controls. From second week onwards, the weight in all the treated chicks started increasing and at the end of the third week, the increase in the liver weight became highly significant (Table 1).

Serum total lipids. All the three strains 'A', 'B' and 'C' showed an initial increase in total serum lipids at the end of the 1st week. However, at the end of the 2nd week, there was a drastic reduction in the total lipids content of the serum of all the three strains. Thus, there was a 3.5, 1.4 and 2.5 fold decrease in the total lipids in the strains 'A', 'B' and 'C' respectively, at the end of the 2nd week. At the end of the 3rd week, this trend of reduction persisted and amounted to 2.1, 2.4 and 2.3 folds respectively for the above strains (Table 2).

Serum cholesterol. The three strains of broiler chicken 'A', 'B' and 'C' also showed a rise in the serum cholesterol levels amounting to 116, 43 and 12%, respectively, at the end

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TABLE 1. EFFECTOF DIETARY AFLATOXIN (250 µg/kg FEED ON LIVER WEIGHT (POOLED LIVER OF 3 CHICKS) OF 3 STRAINS OF BROILER CHICKS

	Liver weight (gm)								
	Strain A			Strain B			Strain C		
Week	Control	Treated	%Increase	Control	Treated	%Increase	Control	Treated	%Increase
1st	9.82	7.65	-22.1	11.17	10.57	-5.4	11.63	10.87	-6.5
2nd	21.30	23.02	8.1	23.55	24.90	5.7	22.80	27.60	3.3
3rd	32.17	49.72	54.6	36.07	50.10	38.9	35.85	53.77	50.0

TABLE 2. EFFECT OF DIETARY AFLATOXIN (250 µg/kg FEED), ON CHOLESTEROL AND TOTAL LIPIDS, IN THE SERUM (POOLED SERUM OF 3 CHICES IN EACH GROUP) OF 3 STRAINS OF BROILER CHICKENS.

Parameters	Week	Strain A			Strain B			Strain C		
		Control	Treated	Decrease (x folds)	Control	Treated	Decrease (x folds)	Control	Treated	Decrease (x folds)
Cholesterol	1st	108	234	--	203	291	--	107	120	--
mg/100 ml	2nd	116	21	5.5	130	24	5.4	127	13	9.8
	3rd	140	51	2.7	142	67	2.1	127	46	2.8
Total lipids	1st	805	863	--	561	562	--	602	637	--
mg/100 ml	2nd	760	219	3.5	525	372	1.4	686	274	2.5
	3rd	594	287	2.1	538	223	2.4	649	287	2.3

of the 1st week. By the end of the 2nd week, however, the treated chicks of strains 'A', 'B' and 'C' showed a 5.5, 5.4 and 9.8 fold reduction in their serum cholesterol levels. At the end of the 3rd week, the cholesterol levels in the treated birds somewhat improved but were still higher than controls by 2.7, 2.1 and 2.8 folds for strains 'A', 'B' and 'C', respectively (Table 2).

Discussion

The results show that all the three strains were equally sensitive to the effect of aflatoxins on liver weight and the serum lipids. During the 1st week, the liver weight of the treated chicks of all the three strains decreased as compared to their respective controls, whereas, the serum total lipids and serum cholesterol increased implicating that lipids and cholesterol transport from the liver into the blood was faster than their control counterparts. This does indicate an onset of liver malfunction due to the effect of aflatoxin. However, in the 2nd and 3rd week, the aflatoxin exerts its full effects on lipids and cholesterol metabolism resulting in lipid accumulation in the liver and hence increase in the weight of the liver. Increase in liver weight and lesions in the liver of poultry as a result of aflatoxin ingestion, has been previously reported by a number of workers [6, 11-17]. Liver lesions comprise of degenerative changes in the liver parenchymal cells and hyperplasia of the bile duct [18]. These changes lead to a break-down of the normal lipid metabolism in the liver and lipids start accumulating in it causing much enlargement of liver.

Biochemical studies have shown that aflatoxin interferes with the lipid metabolism and lipid transportation in a complex manner. Thus it has been demonstrated that the effect of aflatoxin on hepatic lipidiosis is primarily mediated, through inhibition of the synthesis of phospholipids and cholesterol [3]. This, in turn, affects the trans portation of lipids from the liver and this break-down of cholesterol mediated lipid transport system in the liver is the primary effect in aflatoxin toxicity [3,19,20].

It has been observed that aflatoxins inhibit transport of the three major lipid classes, i.e. triglycerides, phospholipids and cholesterol from liver into the blood [6]. Furthermore, there may be a specific inhibition of hepatic cholesterol biosynthesis [21] and/or a negative feed back control of lipogenic enzyme activity [22]. These factors may explain the low levels of cholesterol and total lipids observed in the serum of the treated chickens in the present study.

It is, therefore, concluded that all the three strains of broiler chicken are equally sensitive to the effect of aflatoxin B_1 and efforts should be made to keep the poultry feed free of

aflatoxin producing fungi by adopting proper drying and storage facilities.

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References

1. D.J. Osborne and P.B. Hamilton, Poult. Sci., **60**, 1398 (1981).
2. P.B. Hamilton, Fed. Proceed., **36**, 1899 (1977).
3. F.S. Chu, Appl. Microbiol., **83**(7), (1977).
4. J.W. Smith and P.B. Hamilton, Poult. Sci., **49**, 207 (1970).
5. P.B. Hamilton and J.D. Garlich, Poult. Sci., **50**, 800 (1971).
6. H.T. Tung and W.E. Donaldson, Poult. Sci., **52**, 80 (1972).
7. W.E. Donaldson, H.T. Tung and P.B. Hamilton, Comp. Biochem. Physiol., **41**, 843 (1972).
8. D.N. Reddy, P. V. Rao, V.R. Reddy and B. Yadgiri, Indian J. Anim. Sci., **54**, 68(1984).
9. O.L. Shotwell, C.W. Hesseltine, R.D. Stubblefield and W.G. Sorenson, Appl. Microbiol., **14**, 425 (1968).
10. (Association of Official Analytical Chemists 1984) *Official Methods of Analysis*, 14th ed., Chapter 26.
11. M. Newberne and W.H. Butler Cancer Res., **29**, 236 (1969).
12. R.D. Wyatt, P. Thaxton and P.B. Hamilton, Poult. Sci., **54**, 1065 (1975).
13. M.A. Rajion and D.J. Ferrell, Br. Poult. Sci., **17**, 79 (1976).
14. H.T. Tung, W.E. Donaldson and P.B. Hamilton, Poult. Sci., **50**, 1637 (1972).
15. W.L. Bryden, Proceed. First Austral. Poult. Stock Feed Convent., Melborne, **2**, 317 (1976).
16. Q.K. Iqbal, P.V. Rao and S.J. Reddy, Indian. J. Anim. Sci., **53**, 1277 (1983).
17. C.O. Ubosi, P.B. Hamilton, E.A. Dannington and P.B. Seigel, Poult. Sci., **64**, 1065 (1985).
18. J.G. Heathcote and J. Hibbert, *Aflatoxins*, ed. (Elservier Sci. Publishers, Amsterdam Oxford and New York, 1977).
19. R.C. Shank and G.N. Wogan, Toxicol. Appl. Pharmacol. **9**, 468 (1966).
20. R. Kato, K. Onod and Y. Omeri, Experimentia., **25**, 1026 (1969).
21. B. J. Horton, J. D. Hortons and J.R. Sabine, Eur. J. Cancer., **8**, 437 (1977).
22. W.L. Bryden , R. B. Cumming and D. Balnave, Br. J. Nutr., **41**, 529 (1979).