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**Liver Histopathology of Turkey Poults Fed With Aflatoxin- Contaminated Diets
Supplemented With Nevatox Binder**

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ABSTRACT

Aflatoxin has become a nightmare in poultry industry affecting the birds as a result of various diseases caused by ingestion of the toxin. Clay minerals has however shown some potency in neutralizing the effect of the disease on broilers and other class of poultry, while such information on turkey management is scanty. This study was conducted to investigate the efficacy of Nevatox, a clay mineral in ameliorating aflatoxicosis in turkey poults. A total of 80, 21-d-old turkey poults were randomly allotted to five dietary treatments with four replicates of four poults per replicate in a completely randomized design. Treatment 1 is positive control diet with no aflatoxin and no nevatox, treatment 2 is the negative control with 0.2mg/kg of aflatoxin, treatment 3 is the negative control with 2.0g/kg Nevatox, treatment 4 is the negative control with 0.4g/kg Nevatox, treatment 5 is the negative control with 6.0g/kg Nevatox. The feeding trial lasted for 21 days. At the end of the feeding trial, 2 birds per replicate were killed and their liver, harvested, for histopathology examination. There was no observable lesion in the liver of turkey poults that were fed with diet 1(positive control). However in treatment 2 to 5, varying degree of induced pathological lesions ranging from mild to severe were observed in the liver of the turkey poults. Supplementation of the diet with Nevatox at 2, 4 and 6g/kg did not reduce liver toxicity of the birds at 0.2mg/kg of aflatoxin B1.

KEYWORDS: Aflatoxin B1, Nevatox, Turkey poults, Supplement, Histopathology

INTRODUCTION

Aflatoxins are secondary toxic metabolites of 2 fungal species namely *Aspergillus flavus* and *Asperillus parasiticus* (Wilson and Payne, 1994). Aflatoxins are of different types and forms but among the different types of aflatoxins, aflatoxin B1 is the most prevalent and the most carcinogenic and is often found in cereal grains and peanut meal (Gowda *et al.*, 2004). Aflatoxicosis in poultry causes mortality, listlessness, anorexia, decrease growth rates, negative feed conversion, fatty liver, decreased egg production among other problems (Leeson *et al.*, 1995).

One important way to reduce the effect of aflatoxin in poultry is the use of adsorbing agent such as bentonite or hydrated sodium and calcium aluminosilicates (HSCAS) (Oguz and Kurtoglu, 2000). While there are several studies in the past to determine the potencies of these adsorbing agents in broilers and other livestock species, information on effects on histopathology of liver in turkey is scanty. Therefore this study was conducted to determine specifically, the ameliorative effect of Nevatox adsorbing agent on liver histopathology of turkey poult.

MATERIAL AND METHODS

Experimental Site

The experiment was carried out at the poultry unit of the Teaching and Research Farm of the University of Ibadan. Histopathology was carried out at the department of Veterinary Pathology, University of Ibadan, Ibadan Nigeria.

Experimental Animals and Design

A total of 80 1-d-old turkey poults were used for this study. The turkey poults were brooded for three weeks. The poults were weighed at the third week and randomly allotted to five dietary treatments with four replicates and four poults per replicate in a completely randomized design as follows: Diet 1 (Positive control with no aflatoxin or Nevatox); Diet 2 (negative control with 0.2mg/kg aflatoxin); Diet 3 (negative control + 2.0g/kg Nevatox); Diet 4 (negative control + 4.0g/kg Nevatox) and Diet 5 (negative control + 6.0g/kg Nevatox).

Preparation of aflatoxin contaminated maize

A pure culture of *Aspergillus flavus* (N3228 strain) was obtained from International Institute of Tropical Agriculture (IITA), Ibadan. Maize inoculum was prepared by a method described by Shotwell *et al.* (1966).

Histopathology

At the end of the 21 days feeding trial, 2 poults per replicate were slaughtered and the liver harvested for histopathology. Sections of the liver, kidney were collected and fixed in neutral 10% formalin, embedded in paraffin, and cut into 4 - μ m thick sections. The sections were stained with hematoxylin-eosin (H&E) method for microscopic examination (Bancroft and Stevens, 1996). Sections of liver with no, mild, moderate and severe lesions were given scores of 0, 1, 2 and 3, respectively (Gowda *et al.*, 2008).

RESULTS AND DISCUSSION

Liver is considered the target organ for aflatoxin B₁ because it is the organ where most aflatoxins are bioactivated to reactive 8, 9 epoxide form which is known to bind DNA and proteins, damaging the liver structures and increasing liver weight (Miazzo *et al.*, 2005) Aflatoxin has been known to cause liver congestion during aflatoxicosis due to increased lipid deposits (Hsieh, 1988).

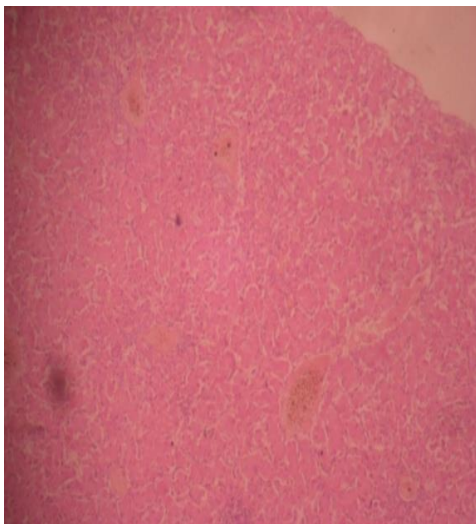


Plate 1: The photomicrograph of liver of turkey poult fed with control diet without aflatoxin showing no significant lesion

Turkeys that were fed with aflatoxin diets showed mild and moderate degeneration of hepatocytes with loss of hepatic cord arrangement in some cases despite the addition of Nevatox binder. Similar lesions have been reported during aflatoxicosis in chickens (Endrington *et al.*, 1997). Significantly higher scores of liver lesion occurred due to aflatoxicosis from AFB₁ administration and these were not present in the control diet.

The aflatoxin B₁ is the most potent hepatocarcinogen known. It is capable of inducing liver cancer in many animal species. The Aflatoxin B₁ can cause malignant hepatocellular

carcinomas at carcinogenicity amounts as low as 0.01mg/kg in the diet of trout. This makes it one of the most abundant, most toxic and the most potent naturally occurring carcinogenic substance known (Jones *et al.*, 1994).

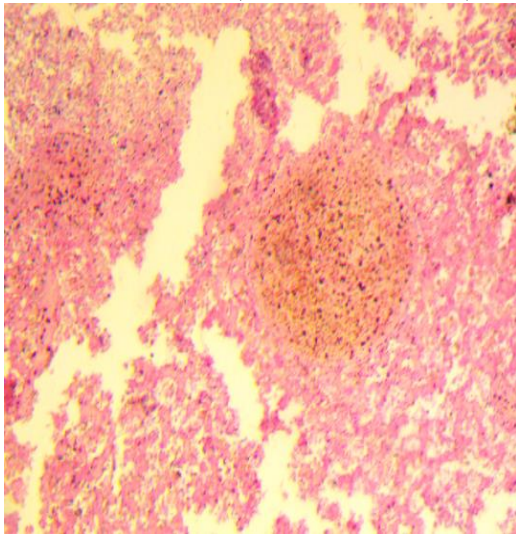


Plate 2: The photomicrograph of liver of turkey poult fed with diet 2 (0.2mg/kg aflatoxin) showing vascular congestion and areas with loss of hepatic cord arrangement (H & E x40)

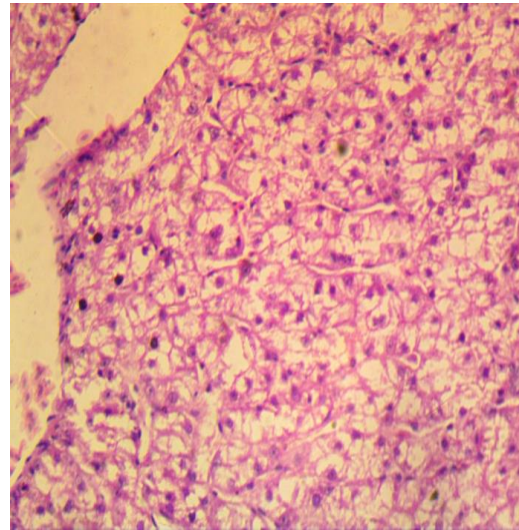


Plate 4: The photomicrograph of liver of turkey poult fed with diet 4 (0.2mg/kg + 4.0g/kg Nevatox). White arrow shows widespread vascular congestion with hepatic degeneration (H & E x40)

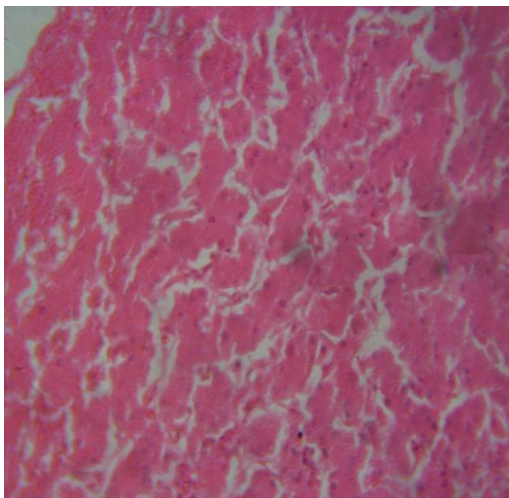


Plate 3: The photomicrograph of liver of turkey poult fed with Diet 3 (0.2mg/kg aflatoxin + 2.0g/kg Nevatox) showing widening of the sinusoids and thinning of cords observed here (H & E x 40)

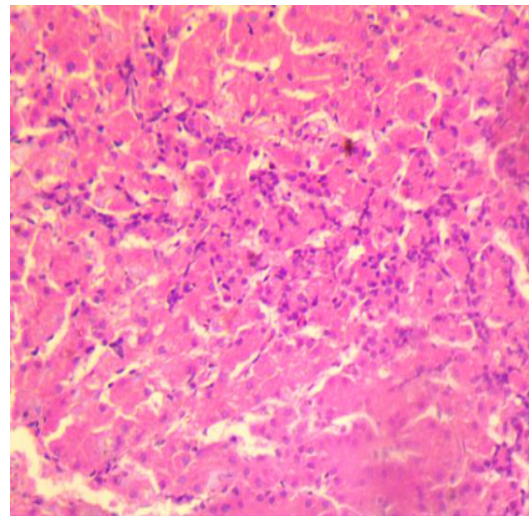


Plate 5: The photomicrograph of Liver of turkey poult fed with diet 5 (0.2mg/kg aflatoxin + 6.0g/kg Nevatox) showing dissociation of hepatic cords, dilated sinusoid (H & E x 40)

The mutagenicity of aflatoxin B₁, is considered to arise as a result of the formation of a reactive epoxide at the 8, 9 position of the terminal furan ring and its subsequent covalent binding to nucleic acid (Chrevatidis *et al.*, 2003). Aflatoxin act, after bioactivation in the liver by binding of biological molecules such as essential enzymes, blockage of RNA polymerase and ribosomal translocase (inhibiting protein synthesis) and formation of DNA adducts (Hsieh and Alkinson, 1990). Hence the lesions observed in the liver.

CONCLUSION AND OUTLOOK

It can be concluded from the findings here that 2, 4, 6g/kg of Nevatox binder could not prevent the effect of 0.2mg/kg aflatoxin B₁ on the liver. Further studies is recommended to determine if lower concentration of the aflatoxin or higher concentration of the Nevatox binder could make an impact in ameliorating the effect of aflatoxin on liver cancer.

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