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# Altered glucose and lactic acid contents in the blood and urine of rabbits during aflatoxicosis

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**Abstract**

Blood and urine samples were collected on 0,15,30,45,60,75 and 90 days of treatment. Concentration of glucose in both blood and urine were decreased upto 30 days followed with significantly increased value from 45th day onwards. Concentration of lactic acid in blood and urine were significantly increased on 15$^{th}$ day onwards. Increased concent~ations of ~lucose and lactic acid in blood during aflatoxicosis was followed with its increased appearance in urine. It clearly indicates that uptake of glucose and lactic acid by tissues particularly by liver is greatly hampered.

**Keywords:** Aflatoxin, aflatoxicosis, glucose,lactic acid,blood, urine.

## Introduction

The only nutrition that the brain, retina, and gonad germinal epithelium can use in large enough amounts to provide them with the energy they need is glucose. Thus, one of the body's most intricately controlled homeostatic mechanisms in which the liver plays a significant role is the maintenance of a normal blood glucose level. A "Soskin effect" has been noted in numerous species. wherein the liver absorbs glucose

when the portal glucose concentration climbs above a particular threshold in response to variations in the concentration. and, when it drops below a particular threshold, releasing glucose.

The body retains the large amount of lactic acid produced during anaerobic glycolysis. but once oxygen is available once more. Lactic acid can be utilized directly as fuel or it can be transformed back into glucose. The liver is where the majority of this reconversion takes place.

Aspergillus flavus produces secondary hazardous fungal compounds called aflatoxins. When aflatoxin-contaminated food or feed is consumed, aflatoxicosis, an extremely toxic condition, develops. According to Busby and Wogan,[1] aflatoxin is a strong hepatotoxin that causes hepatocarcinogenesis in a number of species and may be connected to liver cancer in men. [2,3,4,5] Further more, dietary toxicosis has been linked to biochemical alterations in the liver,[6] heart,[7] kidney 8 and skeletal muscle[9] in rabbits, resulting in hyperglycemia and a decreased red blood cell count containing PCV.[10] Since concentrations of blood glucose and lactic acid are controlled by interactions of various organs including liver, kidney skeletal muscle. The present investigation was undertaken.

**Materials and Methods**

Aspergillus parasiticus (NRRL 3240 obtained from I.A.R.I. ,New Delhi, India) was grown on SMKY liquid medium at 280c for 10 days.[11] Pooled culture filtrate were extracted with analytical grade chloroform and qualitatively analyzed for different types of aflatoxins on TLC plates[12] aflatoxin content was quantified[13] using a shimazu UV160A spectrophotometer

The crude aflatoxin concentrate in chloroform was mixed with feed (7.5 mg aflatoxin / kg) and left overnight to allow for chloroform evaporation. The presence of toxin in such toxin mixed ration was ensured by taking random sample and analysis. Feed for control rabbit was prepared in similar way except for addition of toxin.

Young adult New Zealand strain of rabbits (Oryctolagus cuniculus) weighing approximately 1.2 kg were maintained under laboratory condition and fed with rations and water ad-libitum. Ten rabbits were randomly segregated in two groups and fed with either control or test feed continuously for 90 days.

Blood (from ear pinna) and urine samples of rabbits were collected for analysis on 0, 15, 30, 45, 60, 75 and 90 days of treatment. The concentrations of glucose[14] and lactic acid[15] were measured in deproteinized samples.

The data were statistically analysed using student's 't' test.

**Results**

*Glucose:*

The concentration of glucose in blood of control rabbits was 120.63 mg/dl. Feeding aflatoxin contaminated diet caused initial decline (upto 30 days) followed with a rise. Changes at 15. 30. 45. 75 and 90 days of treatments were found significantly different from control. Concentration of glucose in urine of control rabbits was 93.124 mg/dl. Feeding aflatoxin contaminated diet caused initial decline (upto 30 days) followed with increased appearance of glucose in urine. Changes at 15. 30. 60. 75 and 90 days were found significantly different from control.

**Table 1:** Effect of feeding aflatoxin contaminated diet on concentration of glucose and lactic acid in blood and urine in rabbits during aflatoxicosis.

| Days of treatment | Concentration of glucose (mg/dl) | | Concentration of lactic acid (mg/dl) | |
|---|---|---|---|---|
| | blood | urine | blood | urine |
| 0 (Control ) | 120.63 ± 7.53 | 93.12± 6.87 | 12.27 ± 1.59 | 6.16 ± 1.26 |
| 15 | 87.71c ± 8.17 | 65.91d ± 6.72 | 21.17 ± 0.95 | 7.03 ± 0.21 |
| 30 | 71.39b ± 8.15 | 52.61b ± 6.57 | 25.06a ± 0.45 | 8.56 ± 0.21 |
| 45 | 91.75c ± 5.05 | 86.24 ± 6.87 | 27.31a ± 1.34 | 11.54b ± 0.39 |
| 60 | 137.21 ± 7.19 | 148.28a ± 7.02 | 33.56a ± 2.17 | 17.54a ± 1.29 |
| 75 | 152.01d ± 7.85 | 169.50a ± 7.17 | 39.56a ± 3.54 | 21.75a ± 1.11 |
| 90 | 162.44b ± 8.69 | 198.20a ± 7.17 | 50.64a ± 1 .61 | 25.31a ± 1.29 |

Values are mean ± S.E.M.; n = 5.
Significant at : a = P < 0.001 ; b = P <0.01 ; c = P <0.02 ; d = P <0.05

*Lactic acid:*

Concentrations of lactic acid were 12.27 mg/dl in blood and 6.16 mg/dl in urine of control rabbits. Time dependent increased concentration of lactic acid were observed in aflatoxin fed rabbits. Significantly increased concentrations were recorded in the blood and urine after 30 and 45 days of treatment respectively.

**Discussion**

Aflatoxin fed dtet caused initial decline in concentration of blood glucose upto 30 days. It may be due to decreased feed and water consumption in treated rabbits or due to digestive disturbances,[16] a similar condition to starvation during aflatoxicosis.

But after 30 days of treatment. blood glucose level gradually increased which could be a result of reduced uptake of glucose by tissues. as evidenced by decreased glycogen concentration in liver,[6] heart [7] and Kidney. [8] Smith and Ross[17] also reported depletion of liver glycogen during aflatoxicosis. Time dependent alteration in blood glucose suggests cumulative toxicity during aflatoxicosis. These changes may arise as an effect of aflatoxins on synthetic enzymes or by inhibition of glycogenesis. acceleration of glycogenolysis and depression of glucose transport into hepatocytes. Intraperitioneal administration of toxin to a day-old chick reduce the level of UDP-glucose-glycogen transglucosylase activity.[18]

Mechanisms of increased glucose concentration during aflatoxicosis is not clearly understood but it might be due to alteration in hormones or due to stress condition which occurs during aflatoxicosis. and is known to increase blood

sugar by altering insulin action. Hyperglycaemia may be greater in degree and more prolonged in the presence of cirrhosis and some other liver disease.[19] Induced aflatoxicosis in rabbits caused rise in blood glucose level, decrease in glucose uptake by liver and other tissues followed by its appearance in urine. Glucose concentration in the urine of control rabbits was higher; it could be due to voracious feeding nature of rabbits or any other unknown mechanism.

Aflatoxin contaminated diet caused significantly increased concentration of glucose in urine of treated rabbits. It indicates tubular injuries and reduced their reabsorptive capacity. Aflatoxin induced renal injuries, desquamation of epithelial cells of the proximal tubules and necrosis of glomeruli have been reported in kidney during aflatoxicosis.[20,21,22,23]

Feeding aflatoxin contaminated diet cause increased concentration of lactic acid in blood which could be due to enhanced rate of anaerobic metabolism in muscle followed by its release into blood stream. Bioclinically. rabbits during aflatoxicosis became lethargic and showed respiratory troubles.[24] Aflatoxin induced. significant decrease in RBC count. Hb. PCV,[10] cytotoxicity on RBC in vitro,[25] Hyperbilirubinaemia[26] and alteration in skeletal muscle.[9]

LDH. which converts lactate to pyruvate. registered an increase in liver during aflatoxicosis.[6] This suggests an increased capacity of liver to utilize lactate produced intracellularily brought over to the liver through blood. Aflatoxin is well known hepatotoxic agent and altered histoarchitecture. Although LDH activity increased. liver is unable to utilize elevated concentration of circulating lactic acid in the process of Cori cycle causing its accumUlation in the blood followed by excretion in the urine.

It is concluded that aflatoxin contaminated diet caused alterations in carbohydrate metabolism during chronic aflatoxicosis in rabbits.

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