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HISTOPATHOLOGICAL AND HAEMATOLOGICAL STUDIES ON RATS FED ON YELLOW CORN GRAINS CONTAMINATED WITH ASPERGILLUS FLAVUS AND / OR WITH AFLATOXIN

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ABSTRACT

Eight groups of rats which represent eight treatments were fed on yellow corn grains infected with a sore spension of *A. flavus* (2500-3000 sores/g. grains). Weight of rats was significantly reduced in all groups contained diets contaminated with aflatoxins, spores or both of them when compared with the control (non-infested). The reduction in weight of rats was increased by increasing concentration of the aflatoxins. Also, the rats fed on diets contained high concentration of aflatoxins showed some clinical changes such as falling of their body hair.

Some alteration in liver as, severe congestion (hyperaemia) of the central vein, sinusoides, and degenerative changes in the form of vascular and hydropic degeneration were show in the groups fed on diets containing different concentration of aflatoxins (B1 and/or B2) and/or spores of *A.flavus*.

Significantly reduced count of red blood cells (RBC's) was recorded in rats fed on diets contained high concentration of aflatoxins, while significantly increased count of white blood cells (WBC's) was found in these group. Concentration of hemoglobin (Hb) and activity of serum glutamate pyruvate transaminase (SGPT) and serum glutamate oxalacetate transaminase (SGOT) were raised in the blood.

INTRODUCTION

The increased need of corn in recent years put the corn among the major grains produced in the world for food and feed. Corn as a feed is the second component that has commercial value. It is used mainly as a feed stuff for animals and poultry wealth. The importance of reliable quantitative information on aflatoxins arising from the bad conditions of corn storage has increased considerably in recent years.

Aflatoxins are mycotoxins that has acute, chronic and sometimes mutant effects. Aflatoxins B1, B2, G1, and G2 are the main toxins produced by *Assergillus falvus* and *A.parasticus* (Diener and Davis, 1986). Bearing in mind all the above mentioned points, the present study was conducted to

study the histopathologyical and the hematological effects of the aflatoxins on experimental rats.

Delvi (1986) reported that, factors influenced the toxic affects of aflatoxin were age, sex, nutrition, evironmental stresses and exposure to chemicals (duration and dose) including other mycotoxins.

Hamilton (1986) stated that, aflatoxin has many effects on farm animals, including malabsorption of various nutrients, decreased tissus integrity, poor growth, poor efficiency of feed conversion, enhanced susceptibility to infection, vaccine failure, drug failure, reproducetive problems in males and females and increased sensitivity to temprature extremes. Toxic residuse of aflatoxin in animal products were harmful to public health. The minimum effective dose (MED) of aflatoxin determined according to epidemiological studies coupled with laboratory experiments and mathematical corrections, was found to be less than 10 p.p.b. It may be assumed that no level of aflatoxin is free of risk.

Panangala *et al.* (1986) found that, weanling swine when fed on 500 p.p.b dietary aflatoxins caused reduction in growth rate and feed efficiency, while feed containing 300 p.p.b aflatoxins could affect growth rate when feeding process was prolonged.

Pier (1986) reported that, aflatoxicosis of mammalian animals caused a variety manifestations, due to the ability of aflatoxin to impair protein synthesis, react with macromolecules and cellular organells and interfere with normal production of cellular regulators. Acute aflatoxicosis caused hepatic necrosis, dearrangement of hepatic functions. Coagulopathy and extensive hemorrhagic lesions, resulting death of the animal. Subacute or chronic aflatoxicosis caused fatty changes in liver, enlargement of the gall bladder, periportal fibrosis with proliferative changes in bileduct epithelium, icterus and also reduced rate of growth production. In addition to liver, the thymus gland is also a primary target organ.of aflatoxin. Consumption of aflatoxin caused thymic aplasia and marked suppression of cell-mediated immune responses, as well as non sepcific factors of the native defense mechanisms, such as macrophages and production of compiement (C4), T. cell poplation of the peripheral blood and antibody titers are usually normal. Immunosupperssive effects are thought to arise from effects on antigen presentation and lymphokine production. Acut aflatoxicosis has pathognomic signs and hence it was relatively diagnosed easly. Major economic losses in animals were assocaited with the subacute or chronic forms of aflatoxicosis, which are the most difficult ones to recognize. Carcinogensis is an important aspect of aflatoxicosis in animals.

Baker and Green (1987) showed that, the coagulation defect caused by aflatoxicosis is primarily due to diminished hepatic synthesis of coagulation factors, except when hepatic necrosis is sever enough to strat interavascular coagulation and consnotion of coagulation factors.

Coppock *et al.* (1989) found that, aflatoxicosis was diagnosed in 600 pigs (2500-3500μg aflatoxin/kg of feed) of which 400 were died, 150 were destroyed and 50 were markedly affected.

Roger *et al.* (1991) found that, cereal brans had no longterm effect on body weight as a nutrient during rat experiment, but influenced differently each of total serum lipid levels and intestinal weights.

Slowik *et al.* (1985) mentioned a clear correlation between morphological changes in the lymphatic system and that in the liver.

Harvey *et al.* (1988) analysis of blood samples drawing from pigs at the 28th days of feeding diets containing 0, 1, 2, 3 or 4mg aflatoxin/kg showed that, haemoglobine decreased in aflatoxin-treated pigs comparing with control ones. Examination of bone marrow samples collected after slaughtering (at the 29th days) showed a decrease in granulocytic cellularity in aflatoxin feeding pigs.

Browine (1988) reported that, serum aspartate aminotransferase (SGOT) actvities were indicative of hepatotoxicity. Lactate dehydrogenase (LHD) activity was significantly increased under treatment with aflatoxin B1.

Harvey *et al.* (1989) noticed changes in enzymatic activities of alkaline phosphatase, aspartate traneaminase (GOT), creatine kinase, and δ-glutamyl transferase (GPT) in blood serum of pigs treated with aflatoxin B1.

Steele and Winsch (1989) reported that, many enzyme are synthesized by liver cells, but not all of them have been found useful in diagnosis of hepatoma and hepatocirrhosis. Some enzymes have been found useful in this respect such as, serum glutamate pyruvate transaminase (SGPT).

In order to check the hazardus of aflatoxins and/or spores of *Aspergillus flavus* on the microscopic structure of tissues (specially liver tissues) and on some blood components, this study was conducted.

MATERIALS AND METHODS

Experimental animals and diets:-

For histopathological examination, the method described by (Pearse, 1968) was used. Eight groups, each with six male rats weighing approximately 70-75 g. were used. The diet consisted of corn (represent 60% treated with *A. flavus*,) Casein, Oil, Vitamin B+K, Mineral mixture, Cellulose, Charbohydrate and Methionine. Each rat fed in a single cage and was weighed daily during the experiment for 25 days. Eight groups of rats which representer eight treatments were tabulated in table (1) as follows:-

Table (1): Groups of treatments and their components.

Group	Treatment	Grade *	Moisture content	** Concentrations of the produced aflatoxins		Presence of spores
				B_1(p.p.b)	B_2(p.p.b)	
1	Commercially stored corn for 30d	3	17	54.8	27.4	+
2	Stored corn at # 28°C. for 30d	3	17	10.3	-	+
3	Stored corn at 18°C. for 180d.	3	17	-	-	+
4	Stored corn at 10°C. for 180d.	3	17	-	-	+
5	Stored corn at 28°C. for 3.	3	23	986.3	219.0	+
6	Stored corn at 18°C. for 180d.	3	23	174.0	-	+
7	Stored corn 10°C. for 180d.	3	23	-	-	+
8	Control	3	23	-	-	-

d =day B2= Aflatoxin B2 produced by *A. flavus*.
\+ =Presence of spores in diet - = Absence of spores in the diet.
B1 = Aflatoxin B1 produced by *A. flavus*. # = AACC, 1962
* = USFGS, 1990. **= AOAC Methods, 1990

Histopathological examination:-

At the end of the experiment all rats were sacrificed and samples. Samples were fixed in 10% formaline solution, followed by washing in tap water, then dehydrated by different grades of alcohol (70,85,96 and 99%). and finally cleared by xylene and embedded in paraffine wax. The paraffine embedded blocks were six-micron cutted, stained by hematoxyline and eosin and then subjected to a histopathological. (Pearse, 1968).

Haematological examination:

For haematological examination, blood samples were taken from vein of rat's eye at the end of experiment in Wasserman test tubes containing anticoagulant. Counting of red blood cells (RBC's) and white blood cells (WBC's) were done according to the method of Jain (1986). Haemoglobin was determined according to Van Kampen and Zijlstra (1961).

To determine the activity of serum glutamate pyruvate transaminase (S.G.P.T.) and Serum glutamate oxalacetate transaminase (S.G.O.T.), the method described by Karmen, (1955) was carried out.

Data obtained were subjected to the proper analysis of variance (Snedecor and Cochran, 1980).

RESULTS AND DISCUSSION

The histopathological effects:-

With respect to, effect of feeding on diets contaminated with aflatoxins and/or spores of *A. flavus* on the weight of experimental rats, data in the present study indicated that, weight of rat was significantly reduced in all groups contained diets contaminated with aflatoxin, spores or both when

compared with control. In the same time, the reduction in rat weight was increased with increase of aflatoxin concentration. Also, rats fed on diets contained high concentrations of aflatoxin (B1 and B2) showed some clinical changes sush as, falling of body hairs. These results are in agreement with these stated by Smith and Hamilton (1970); Garlich and Hamilton (1971); Washburn and Britton (1971); Thurston *et al.*, (1972); Pier (1973); Wyatt *et al.*, (1973); Brigg *et al.*, (1974); Osborne *et al.*, (1976); Howarth and Wyatt (1980); Gianbrone *et al.*, (1985); Hamilton (1986); Panagala *et al.*, (1986); Pier (1986) and Rosiles (1986).

Table (2): Effect of contaminated diets with aflatoxin and / or spores of *A. flavus* on weight of experimental rats.

Group	Sample	Aflatoxin		Other content	Weight at the beging of the Experi-mental (g)	Weight at the end of the Exepri-mental (g)	Reduction in the Weight (g)
		B1	B2				
Group 1	G3 TN M17 (30 days)	54.8	27.4	Spores	74.0	68.9	5.1
Group 2	G3 T28 °C M17% (30 days)	10.3	-	Spores	82.6	74.7	7.9
Group 3	G3 T18 °C M17% (180 days)	986.3	219.0	Spores	76.3	66.46	9.8
Group 4	G3 T10 °C M17% (180 days)	-	-	Spores	75.2	70.1	6.1
Group 5	G3 T28 °C M23% (30 days)	174.7	-	Spores	84.3	75.2	8.83
Group 6	G3 T18 °C M23% (180 days)	-	-	Spores	72.3	64.96	7.3
Group 7	G3 T10 °C M23% (180 days)	-	-	Spores	81.3	74.26	7.04
Group 8	Control	-	-	Free	69.3	8.7	0.6

Where:-

G3 = Grade 3 M = Moiscontent TN =Natural temperatur

L.S.D. at 5% for reduction in weught = 5.0

Results showed that, liver in group No. 8 which was fed on control diet (containing aflatoxins - free corn) was normal. On the other hand, the groups of experimental rats (groups No. 1-7) which were fed on diets containing different concentrations of aflatoxins (B1 and B2) and / or spores of *A. flavus* showed some alterations in liver such as, severe congestion (hyperaemia) of the central vein and sinusoids (Fig.1) and degenerative changes in the form of vacuolar and hydropic degeneration (Fig. 2).

These results are in accordance with those reported by Salmon and Newberne (1963); Buttler (1966); Thurston *et al.,* (1972); Edds (1973);

Fig (1): Liver showing severe congestion of the central vein and sinusoids (H. & E. X 450)
1- Normal central vein 2- Congested central vein

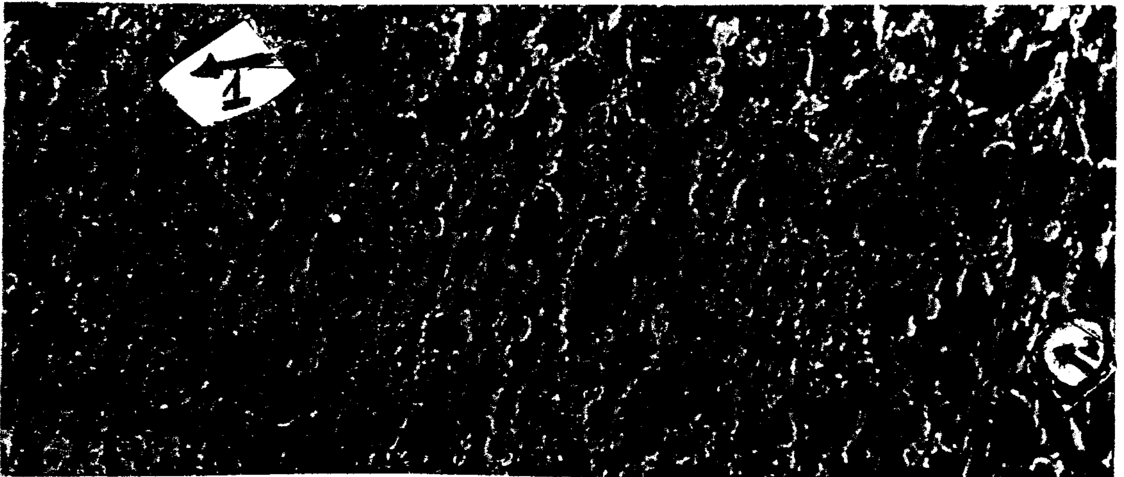

Fig (2): Liver showing degenerative changes in the form of vacuolar and hydropic degeneration (H. & E. X 450).
1- Normal cell showing nucleus and cytoplasm.
2- Abnormal cell showing nucleus and vaculated (hydropic)cytoplasm.

Chen *et al.* (1985); Hamilton (1986); Pier (1986); Rosiles (1986); Baker and Green (1987) and Hamza *et al.* (1989).

The haematological effects:-

Data presented in Table (3), generally showed that, no obvious differences were observed in the count of red blood cells in all tested groups, except in groups 5 (where there was high concentration of aflatoxins) there was significant decrease in count of red blood cells (RBCS), indicated a case of erythrocytopenia, in comparison with either experimental control or normal one. Also, no significant differences were noticed in count of white blood cells (WBCS) in all tested groups, except in group 5 (where there was a high concentration of aflatoxins) there was significant increase in count of white blood cells (WBCS), indicated a case of leucocytosis, in comparison with either experimental control or the normal one. In the same time, the concentration of hemoglobin showed unsignificant differences in all tested rat groups, except in group (5) where there was significant increase in concentration of haemoglobin (Hb) in comparison with either experimental or normal controls. With respect to the tested blood enzymes (SGPT and SGOT), the present data showed that, there were no significant differences in concentrations of either (SGPT) or (SGOT) in all tested groups, except in group (5) where the differences were significant in comparison with either experimental or normal controls.

Generally aflatoxins (B1 and B2) in high concentrations significantly reduced count of red blood cells (RBCS) and significantly increased count of white blood cells (WBCS), concentration of haemoglobin (Hb) and activity of either SGPT or SGOT. Also these results are in accordance with the histopathological results and may be attributed to the hazardous effects of aflatoxins on liver as formerly indicated, or on the bone marrow as the main generator of blood components, or on both. SGPT and SGOT are liver enzymes and their presence in blood in high concentrations mean that liver was exposed to some histopathological changes and so could be used as indicators of such changes.

Lun (1978); Slowik *et al.* (1985); Sova *et al.* (1985); Dafalla *et al.* (1987); Browine (1988); Hamza *et al.* (1989); Harvey *et al.* (1989) and Steele and Winsch (1989). Also, the haematological results in this study are in accordance with the histopathological results Lun (1978) stated that, SGOT measuring activity is useful for identifying inflammation and necrosis of the liver SGOT enzyme is located in the microsomal mitochondrial portion of the hepatic cell and also is present in the epidermis of the skin, heart, skeleton muscle, pancreas and kidneys. Increased enzyme SGOT activity in blood may be due to the damage of the hepatic cell as a results of inflammation or hepatocellular infection.. Slowik *et al.* (1985) mentioned to a clear correlation between morphological changes in the lymphatic system and that in the liver. Steele and Winsch (1989) reported that, some enzymes have been found useful in diagnosis of hepatoma and hepatocirrhosis, such as, serum glutamate pyruvate transaminase (SGPT)

Table (3): Hematological changes by aflatoxins and/or spores of *A.flavus* in male rats.

Red blood cells (RBCs).

RBC (10^6 / µl.) Mean ± SD (P=0.05)

Group / State	G1 Mean ± S.D.	G2 Mean ± S.D.	G3 Mean ± S.D.	G4 Mean ± S.D.	G5 Mean ± S.D.	G6 Mean ± S.D.	G7 Mean ± S.D.
Treated	5.14 ± 0.461	5.36 ± 0.601	5.42 ± 0.523	5.52 ± 0.623	3.96 ± 0.327	4.82 ± 0.391	4.98 ± 0.426
Control (G8)	5.72 ± 0.53	5.72 ± 0.53	5.72 ± 0.53	5.72 ± 0.53	5.72 ± 0.53	5.72 ± 0.53	5.72 ± 0.53
Normal	5.37 ± 0.39	5.37 ± 0.39	5.37 ± 0.39	5.37 ± 0.39	5.73 ± 0.39	5.37 ± 0.39	5.37 ± 0.39
(T) test	N. S.	N. S.	N. S.	N. S.	S.	N. S.	N. S.

White blood cells (WBCs)

W.B.C. (10^3 / µl.) Mean ± SD (P = 0.05)

Group / State	G1 Mean ± S.D.	G2 Mean ± S.D.	G3 Mean ± S.D.	G4 Mean ± S.D.	G5 Mean ± S.D.	G6 Mean ± S.D.	G7 Mean ± S.D.
Treated	7800 ± 2261.7	7100 ± 2989.3	5700 ± 421.9	6950 ± 3691.7	10300 ± 1639.2	9650 ± 2371.2	4650 ± 726.7
Control (G8)	8350 ± 2371.4	8350 ± 2371.4	8350 ± 2371.4	8350 ± 2371.4	8350 ± 2371.4	8350 ± 2371.4	8350 ± 2371.4
Normal	7860 ± 428.9	7860 ± 428.9	7860 ± 428.9	7860 ± 428.9	7860 ± 428.9	7860 ± 428.9	7860 ± 428.9
(T) test	N. S.	N. S.	N. S.	N. S.	S.	N. S.	N. S.

Hb (g / 100ml.) Mean ± SD (P = 0.05)

Group / State	G1 Mean ± S.D.	G2 Mean ± S.D.	G3 Mean ± S.D.	G4 Mean ± S.D.	G5 Mean ± S.D.	G6 Mean ± S.D.	G7 Mean ± S.D.
Treated	8.26 ± 0.248	8.93 ± 0.369	10.69 ± 0.561	8.87 ± 0.298	11.36 ± 0.44	9.63 ± 0.372	8.312 ± 0.329
Control (G8)	9.81 ± 0.326	9.81 ± 0.326	9.81 ± 0.326	8350 ± 2371.4	9.81 ± 0.326	8350 ± 2371.4	9.81 ± 0.326
Normal	10.41 ± 0.429	10.41 ± 0.429	10.41 ± 0.429	10.41 ± 0.429	10.41 ± 0.429	10.41 ± 0.429	10.41 ± 0.429
(T) test	N. S.	N. S.	N. S.	N. S.	S.	N. S.	N. S.

G group

Table (3) Continued.

Serum glutamate pyruvate transaminase (SGPT)

SGPT (I.u/ml) Mean ± SD (P = 0.5)

Group	G1	G2	G3	G4	G5	G6	G7
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Treated	131± 7.1	129± 5.65	119± 2.1	113± 5.56	139± 12.7	124± 2.1	106± 10.6
Control (G8)	121	121	121	121	121	121	121
Normal	154	154	154	154	154	154	154
(T) test	N.S.	N.S.	N.S.	N.S.	S.	N.S.	N.S.

Serum glutamate oxalacetate transaminase (SGOT)

SGOT (I.u/ ml) Mean ± SD (P = 0.05)

Group	G1	G2	G3	G4	G5	G6	G7
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Treated	231± 9.9	225± 5.56	173± 31.1	209± 5.56	229± 8.5	219± 1.4	143± 52.3
Control (G 8)	217	217	217	217	217	217	217
Normal	195	195	195	195	195	195	195
(T) test	N.S.	N.S.	N.S.	N.S.	S.	N.S.	N.S.

G group

which is useful in identifying inflammation and necrosis of the liver. Acute hepatocellular inflammation increased SGPT. SGPT enzyme released in the blood when liver was damaged. Enzyme activity has been used to monitor drugtoxicits.

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دراسات هستوباثولوجيه وهيماتولوجيه لفئران غذيت على حبوب أذره صفراء ملوثه بالأسبرجلس فلافس و/أو بالأفلاتوكسين

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٢ المعمل المركزى للأغذية والأعلاف - مركز البحوث الزراعيه - الجيزه - مصر.

أجرى هذا البحث لدراسة بعض التغيرات النسيجيه وبعض التغيرات فى مكونات الدم لفئران تجارب تمت تغذيتها على حبوب صفراء ملوثه بالأفلاتوكسينات و/أو الفطر اسبرجلس فلافس. وقد أدت تغذية فئران التجارب على غذاء ملوث بالأفلاتوكسينات أو الجراثيم أو بكليهما (للفطر أسبرجلس فلافس) إلى خفض معنوى فى الوزن مقارنا بالكنترول. وقد لوحظت زيادة الإنخفاض فى وزن الفئران بزيادة تركيز الأفلاتوكسين فى الغذاء الملوث بالأفلاتوكسين B1 (٩٨٦,٣ ميكروجرام /كجم حبوب) و B2 (٢١٩ ميكروجرام/كجم حبوب) + جراثيم الفطر أسبيرجلس فلافس وأيضا فإن الفئران التى تغذت على هذه العليقه أظهرت بعض التغيرات المرضيه مثل سقوط شعر الجسم.

أظهر الفحص الهستوباثولوجي لقطاعات فى نسيج الكبد أن كبد مجموعة فئران التجارب المقارنه والتى تغذت على غذاء خال من الأفلاتوكسينات كان طبيعياً. وعلى الجانب الأخر فإن مجموعات فئران التجارب والتى تغذت على غذاء يحتوى تركيزات مختلفه من الأفلاتوكسينات (B1, B2) و/ أو جراثيم الفطر أسبرجلس فلافس أظهرت بعض التغيرات فى تركيب نسيج الكبد مثل وجود إحتقان فى الوريد المركزى والأوعيه والشعيرات الدمويه وإستسقاء فى خلايا الكبد.

أظهرت إختبارات الدم أن التركيز العالى من الأفلاتوكسينات أدى إلى خفض معنوى لعدد كرات الدم الحمراء ، كما أدى إلى زيادة معنويه لكل من عدد كرات الدم البيضاء وتركيز الهيموجلوبين وتركيزات بعض إنزيمات الكبد والتى تفرز فى الدم مثل جلوتامات بيروفات ترانس اميناز السيرم (SGPT) جلوتامات أوكسال ترانس اميناز السيرم (SGOT) والتى تستخدم كدلائل على التغيرات الهستوباثولوجيه فى الكبد.