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Biochemical and Toxicological Studies on Raw and Roasted Peanut Naturally Contaminated With Aflatoxins

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ABSTRACT

Aflatoxins are common contaminants of peanut and peanut–based products. Survey for aflatoxins in commercially available peanut was carried out in Great Cairo, Egypt. One hundred and four samples (52 raw peanut sample and other 52 roasted peanut samples) were purchased from markets of all over Cairo. The samples were analyzed for aflatoxins and quantified by liquid chromatography equipped with fluorescence detector. The results indicated that 33% of the raw peanut samples were naturally contaminated with aflatoxin and 21% of the samples were violated the maximum residue limit (MRL). While only 10% of the samples of roasted peanut were naturally contaminated and 0.02% of the samples were violated the MRL. The results were integrated with consumption data to develop a dietary exposure for Egyptian consumers. The percentages of estimation daily intake (EDI) of aflatoxin B1 were 8233 and 2948% for raw and roasted peanut, respectively.

Despite of the previous data the toxicological parameters analyzed i.e. total protein, albumin, ALT activity and AST activity, as well as the histopathology studies showed contrarily results when groups of rats were fed on raw and roasted peanut free of aflatoxins and raw and roasted peanut naturally contaminated with aflatoxins. The parameters were studied after zero time, two, four and six weeks. The changes in the last group of rats were irreversible, whereas changes in the other groups were reversible. From the biochemical

analysis and histopathology logical lesions it was found that roasted peanut naturally contaminated with aflatoxins was more effective on liver function than raw peanut.

INTRODUCTION

Egypt, like many other developing countries is faced with the challenge of providing adequate food supply for its teeming population. To meet this challenge, policies and programs aimed at boasting Agriculture and Food Production have been actively promoted. While battling to meet this quantitative demand for the population, it is also faced with the issue of safety in the foods. It has to contend with the problem of contamination and chemical residues in foods with other attendant social, economic, health and cost problems.

Great care has been directed today towards improving and ensuring the quality of analytical data on mycotoxins in agricultural commodities. The data are used for assessing risk from consumer's exposure (food surveillance), and for food control (regulatory monitoring) or for monitoring standards for trading purposes (FAO, 2006).

Mycotoxins have been linked to cancer and possible disruption in small children's growth and immune system development; hence food producers and regulators in most countries have been aggressive in their attempts to keep mycotoxins out of the food supply or at least at barely traceable levels (Anonymous, 2003).

Aflatoxins cause cancer in the liver of laboratory animals by damaging the DNA Hayes (1986). They have also been linked to cancer in a number of developing countries where some foods that are an important part of the diet can contain high levels of aflatoxins. Deaths of a large number of people have also been reported in Kenya after eating maize that was highly contaminated by aflatoxins (Anonymous, 2003).

The aim of this study was to determine the presence of aflatoxins in raw and roasted peanut and to produce information that can be used for assessment of the dietary exposure of Egyptian people to aflatoxins by consumption of peanut in raw and roasted cases. To assess the risk to public health from consumption of mycotoxins, the exposure of consumers to these toxins can be compared to safety guidelines such as Tolerable Daily Intakes (TDIs).

As well as to evaluate the effects of raw and roasted contaminated peanut on rats by means of serum biochemistry and histopathology.

MATERIALS AND METHODS

One hundred and four samples of raw peanut and roasted peanut, fifty-two samples for each commodity were determined for the four types of aflatoxins (B1, B2, G1 and G2). One kilogram of every studied commodity was collected weekly from Great Cairo Markets from March 2006 to March 2007. Samples were mixed according to the guidelines of CAC (1993)

Determination of aflatoxins:

Determination of total aflatoxin and aflatoxins B_1, B_2, G_1 and G_2 were carried out by high performance liquid chromatography (HPLC) as the procedure of AOAC (2005).

Preparation of aflatoxins standard solutions:

Aflatoxin stock solutions, aflatoxin mixture working solution standards were prepared according to AOAC (2005).

Preparation of sample:

The samples prepared according to the guidelines of Codex Alimentarius (1992) and AOAC (2005).

Extraction:

Fifty g of homogenized sample were weighted into spherical flask 500 ml with stopper. Then 200 ml of methanol-water (80:20) solution were added and the flask was shacked for 30 min. The extract was filtered through medium fast filter paper (Whatman No.1).

Partitioning:

Forty ml of filtrate were transferred into 500 ml separating funnel. Forty ml (10%) sodium chloride solution and 50 ml n-hexane were added to the funnel. The funnel was shacked gently for one min. and the phases were let to separate. The aqueous lower layer was drained into another 500 ml separating funnel and 50 ml chloroform were added and shacked gently for 1 min. The phases were let to separate and lower layer was drained through anhydrous sodium sulfate (15 g) into 250 ml flask. The aqueous layer was washed with two portions of 25 ml chloroform and shacked gently for one min. each time. The received chloroform was evaporated to dryness by

using rotary evaporator. The dry film residue was dissolved in 2ml dichloromethane and transferred to silica gel clean up column.

Clean-up column:

A small boll of glass wool was put into 5 ml plastic syringe to make a bed for 0.5 sodium sulfate. Three ml of dichloromethane were add to the syringe then 0.5 g of silica gel was added and dichloromethane was rinsed out of the column, after that another 0.5 g of sodium sulfate was added. One ml of dichloromethane was added to the column AOAC (2005).

Derivatization of the sample extracts:

Two hundred µl of hexane and 50 µl Trifluroacetic acid (TFA) were added to the column extract. The tube was capped and shacked on vortex-mix vigorously for 30 s. (exactly). The tube was let stand for 5 min and 1.950 ml acetonitril-water (1:9) were added. The tube was shacked on vortex-mix for 30 s. (exactly) and the layers were let to separate for 10 min. The lower aqueous layer was used for HPLC AOAC (2005).

Detection by HPLC:

HPLC measurements

Hewlet Pachard (HP) 1100 equipped (HPLC) with reversed phase Chrompack C_{18} column (5 um, 250 mm Length, and 4.6 mm id.), pump, auto sampler and programmable fluorescence detector. Mobile phase water- methanol-acetonitrile (60: 25: 15 v/v /v) was also used and the amount of water might be changed for improving the resolution between G_{2a} and B_{2a}. Flow rate of mobile phase was ranged from 0.8 – 1 ml/min with maximum pressure 3000 psi. Twenty five µl loop of injection was also used with detection conditions (excitation 360 nm, emission 440 nm with high gain).

Calculation:

Concentration of sample Cs (µg/g) was calculated as follows:

$$Cs = \frac{Hs}{Hst} X \frac{Ast}{Ws} X \frac{V}{VA} X \frac{D}{I}$$

Where: Cs = sample concentration, Hs = Peak height of sample, Hst = Peak height of standard, Ast = Standard amount (µg), Ws = Sample weight (g), V = Extract volume (ml), Va = Taken volume for partitioning (ml), D = Final dilution (ml), I = Injected volume (ml).

Dietary intake and estimation daily intake (EDI) of aflatoxins B1:

Dietary exposure to mycotoxins can be estimated for an average consumer and for high level consumers using data derived from National and Nutrition Surveys. These estimates can be compared with tolerable daily or provisional tolerable weekly intake (TDI or PTWI) established by expert committees.

Calculation the estimation daily intake (EDI) is reflecting the situation of dietary intake and it is calculated by using the following equation to get the real number of toxin by ng/kg body weight /day Codex Alimentarius (2006).

$$\textbf{EDI} = \frac{\text{food consumption of the product} \times \text{mean of contamination}}{\text{60 kg (average of body weight)}}$$

Animal feeding experiments

Experimental animals

A total of 28 male albino rats, (Sprague Dawely strain), weighing 200 to 250 g, were obtained from Helwan Station for experimental animals, Helwan, Cairo, Egypt. The animals were housed in stainless steel cages and raised in the animal house of biochemistry department, faculty of agriculture, Cairo University. The rats were kept under normal healthy laboratory condition. The animals were adapted on free access of water and fed basal diet for two weeks before the initiation of the experiments.

Diets:

The composition of the standard diet was made according to AOAC (2005) and Experimental design A total of 28 rats were divided into four groups as follows:

Group I (7 Rats), fed basal diets supplemented with raw free aflatoxin peanut .Group II (7 Rats), fed basal diet supplemented with roasted free aflatoxin peanut. Group III (7 Rats), fed basal diet supplemented with raw peanut naturally contaminated with aflatoxins (Aflatoxin B1 = 104.70 µg/ kg diet, B2 = 93.60 µg/ kg diet). Group IV (7 Rats), fed basal diet supplemented with roasted peanut naturally contaminated with Aflatoxins (Aflatoxin B1 = 52.97 µg/ kg diet, B2 = 27.55 µg/ kg diet).

Experimental procedure.

The feeding experiment continued for 45 days. Blood samples were collected every two weeks, and at the end of the experiment, rats were killed by decapitation, and blood sample of each rat was collected in dry clean centrifuge tube, let stand for 30 min., and then centrifuged at 3000 rpm to separate the serum. The clear serum was then pipett into epindorff tubes and stored at – 20°C until biochemical analysis. The livers were rinsed in the situs with formalin solution (10%) and kept at 4°C for histopathological analysis.

Biochemical analysis.

Serum total protein was measured according to Gomal *et al.* (1949). The level of serum albumin were measured according to the method of Doumas *et al.* (1971). Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities were measured colorimetrically in serum according to Reitman and Frankel (1957).

Statistical analysis.

All data were expressed as mean± standard error. Data analysis was made by analysis variance (ANOVA P <0.05). The statistical packaged for social science **S.P.S.S. (1999)** Program version 10 was used for all analysis.

RESULTS AND DISCUSSION

Biochemical survey of aflatoxins in raw and roasted peanut:

Data in Table (1) show the number of naturally contaminated samples and the mean, minimum, maximum, number of violated samples comparing with Maximum Residue Limits (MRL) for the four types of aflatoxins and total aflatoxin. Seventeen samples were naturally contaminated with aflatoxin B1 and B2 with means 47.5 and 6.9 µg/kg, respectively when only 21% of the analyzed samples were violated with B1. The minimum and maximum of B1 were 0.55 and 349.4 µg/kg, respectively while, the minimum and maximum of found aflatoxin B2 were 0.50 and 52.6 µg/kg, respectively. No violation for aflatoxin B2 because of there is no MRL. All the samples were free from any detectable amount of aflatoxin G1and G2 while the mean, minimum and maximum of total aflatoxin were 54.4, 1.05 and 402 µg/kg, respectively. These data reflect that the most mycotoxin found in raw peanut was aflatoxin B1.

Table (1): Number of naturally contaminated samples, mean, minimum, maximum, number of violated and percentages of violation in 52 analyzed raw peanut samples.

Type of toxin	No. of contaminated samples	Mean (µg/kg)	Minimum (µg/kg)	Maximum (µg/kg)	MRL	No. of violated samples	Violation %
Aflatoxin B1	17	47.5	0.55	349.4	5	11	21
Aflatoxin B2	14	6.9	0.50	52.6	-	-	-
Aflatoxin G1	0	-	-	-	-	-	-
Aflatoxin G2	0	-	-	-	-	-	-
Total Aflatoxin	17	54.4	1.05	402.0	10	10	19

LOQ= Limit of quantitation for B1 & B2 = 0.5 µg/kg, for G1 & G2 = 1 µg/kg
MRL= Maximum Residue Limit as seated in Egyptian regulation
- = Less than Limit of Quantitation (LOQ)

As in Table (2) roasted peanut samples were less than raw peanut in the content of aflatoxins. Five samples from 52 analyzed samples were naturally contaminated with aflatoxins B1 and mean, minimum and maximum amounts were 17.01, 0.51 and 78.47µg/kg, respectively. When one sample was violated with aflatoxin B1 with violation percentage 1.9%. Only two samples were contaminated with aflatoxin B2 and mean, minimum and maximum amounts were 3.11, 0.92 and 5.29µg/kg, respectively. As in the raw peanut there was not any contamination with aflatoxin G1and G2.

Table (2): Number of naturally contaminated, mean, minimum, maximum, number of violated and percentages of violation in 52 analyzed roasted peanut samples.

Type of toxin	No. of contaminated samples	Mean (µg/kg)	Minimum (µg/kg)	Maximum (µg/kg)	MRL (µg/kg)	No. of violated samples	Violation %
Aflatoxin B1	5	17.01	0.51	78.47	5	1	1.9
Aflatoxin B2	2	3.11	0.921	5.29	-	-	-
Aflatoxin G1	0	-	-	-	-	-	-
Aflatoxin G2	0	-	-	-	-	-	-
Total aflatoxins	5	20.12	1.43	83.76	10	1	1.9

LOQ= Limit of quantitation for B1 & B2 = 0.5 µg/kg, for G1 & G2 = 1 µg/kg
MRL= Maximum Residue Limit as seated in Egyptian regulation.
- = Less than LOQ

Tables 1 and 2 show that all samples of raw peanut and roasted peanut are free from any detected traces of aflatoxin G1 and G2. The results of this study are almost agreed with the results of Shundo *et al.* (2003).

Dietary intake and estimation daily intake (EDI) of aflatoxins B1:

Dietary exposure estimates set by scientific committees are based on threshold levels identified during toxicological studies, below which the toxins are considered not to cause adverse effects. A (TDI) represent an estimate of the amount of a contaminant, expressed on a body weight basis, which can be ingested daily over a lifetime without appreciable health risks.

Table (3): Estimated daily intake (EDI) of aflatoxin B1 adults consuming peanuts.

Commodity	Mean conc. µg/kg	Consumption (g/person day) [a]	Estimated Daily Intake (EDI) In (ng /kg body weight /day) [b] Calculated value from mean	PMDI [c] (Af B1)	Rate % [d] Calculated value from mean
Raw peanut	47.5	0.26	12.35	0.15	8233
Roasted peanut	17.01	0.26	4.42	0.15	2948

a= Food and Agriculture Organization (FAO) 2006

b = EDI (ng /kg b.w./day) Calculated for each toxin by multiplying estimated values (µg/kg) by consumption (g/person/day) and divided by weights of adult person in average 60 kg

C = PMDI Provisional Maximum Daily Intake seated by CSHPF, 1999

d = Rate percentages calculated by dividing EDI by PMDI and multiplied by 100.

From Tables 1 and 2 mean concentrations of aflatoxin B1 in naturally contaminated samples of raw and roasted peanut used with food consumption data of Egypt FAO (2006) on peanut (nuts) which is 0.26 g /day/ person, (this number is include peanut and peanut products) for calculation of estimation daily intake as in Table (3). Because of the provisional maximum daily intake (PMDI) seated by Consell Supérieur hygiéne Publique de France CSHPF (1999) for aflatoxin B1 and there is not any certain number of acceptable daily

intake (ADI) for aflatoxin B1, PMDI number was used in calculation of EDI (Table 3). There is not available data of ADI or PMDI for aflatoxins B2, G1, G2 and total aflatoxins the EDI's of them were not calculated.

European countries decreased the maximum residue limits for mycotoxins especially aflatoxins in peanut because of the high health risk caused by mycotoxins. Whereas Joint FAO/WHO Expert Committee on Food Additives JECFA did not establish any values of ADI of aflatoxins and also there are no values of no observed effected level (NOEL) and low observed effected level (LOEL) of aflatoxins. For calculation of dietary intake and estimation daily intake of aflatoxins we used value of PMDI seated by CSHPF (1999) for aflatoxin B1. Data in Table (3) represent the intake amounts of aflatoxins and EDI aflatoxin B1 due to consuming peanut. Only aflatoxin B1, B2 and total aflatoxins were found in studied peanut samples.

The EDI and the percentages rate values were calculated in raw peanut and roasted for aflatoxin B1, the values were 12.35 and 4.42 (ng/kg body weight/day), respectively. and the percentages values were 8233 and 2948%, respectively. From these results it is appear that the Egyptian persons who are living in Great Cairo are exposure to about thousands percentages more than the maximum exposure limit seated by **CSHPF (1999).** However the consuming amounts are varied among persons due to their consuming habits.

Toxicological studies of aflatoxin in roasted peanut naturally contaminated with aflatoxins:

1-Determination of toxicological parameters:

The present study investigated the effects of different diets supplemented with raw and roasted peanut naturally contaminated or not with aflatoxins on total protein, albumin, aspartate transaminase (AST) activity and alanin transaminase (ALT) activity in serum as summarized in Table (4). Data illustrate that, aflatoxins significantly increased serum total protein level in both raw and roasted peanut groups. After two weeks the increase in the concentration of total protein was small and still in the normal value, but serum total protein level was greatly increased from 5.94 g/dl and 5.39g/dl in raw peanut group to 7.43g/dl and 7.14g/dl in raw peanut naturally contaminated with aflatoxins group and from 5.35g/dl and 5.50g/dl in roasted peanut

group to 7.22g/dl and 6.99g/dl in roasted peanut naturally contaminated with aflatoxins after two and four weeks, respectively. From these results one may suggest that aflatoxins contaminated diets were more effective than aflatoxins free diets on serum total protein. As similar as in serum total protein, there was no significant different in serum albumin between all groups after two weeks.

After continuous feeding for four weeks on the different diets, the concentration of serum albumin was significantly increased in rats fed raw and roasted peanut contaminated with Aflatoxins by 29.76% and 27.70% compared with that fed free aflatoxins raw and roasted peanut, respectively. While after six weeks serum albumin concentration was significantly increased by 23.08% and 23.89%, respectively. Statistical analyses by Pearson correlation show that, there are very good linear positive correlation between serum total protein and serum albumin (Pearson Correlation= 0.944, $P<0.000$). It means that the increase in serum albumin level may lead to the increase in serum total protein level. There finding are in a good agreement with data published by Pozzi *et al.* (2000).

The present data recorded in Table (4) show also that, feeding normal rats on roasted peanut free aflatoxins diet directly increased serum alanin aminotransferase (ALT) activity of 2.01 and 2.03- times compared with that fed raw peanut free aflatoxins diet after four and six weeks , respectively .In case of raw peanut naturally contaminated with aflatoxins diet, ALT activity was increased of 2.33, 1.95, and 2.87- fold than that of the raw peanut free aflatoxins diet after two, four and six weeks, respectively .The activity of serum ALT in roasted peanut naturally contaminated with aflatoxins diets was also significantly increased of 2.4, 3.1 and 3.38- fold than that of the raw peanut free aflatoxins diet, and of 2.27, 1.54 and 1.67- fold than of the roasted peanut after two, four and six weeks, respectively. The highest value of ALT activity was recorded for rats fed diet supplemented with roasted peanut naturally contaminated with aflatoxins after six weeks (88.00 U/L). Also, increasing in the activity of aspartate aminotransferase (AST) was observed, but with only minor variation between treated groups. It must be noticed that the activity of AST in all groups was significantly increased compared with raw peanut free aflatoxins group especially after four and six weeks.

As similar as in ALT activity, the highest value of AST activity was recorded for rats fed diets supplemented with roasted peanut naturally contaminated with aflatoxins (38.68 U/L). In the present study, there were positive correlation between serum ALT and AST activities (Pearson Correlation=0.832, P<0.000). The activities of ATL and AST were increased in roasted peanut group and also aflatoxins contaminated peanut groups. It is indicated that not only aflatoxins, but also roasting treatment, is a rate limiting factor, for liver function.

Table (4): Serum total protein and albumin levels as well as alanin aminotransferase (ALT) and aspartate aminotransferase (AST) activities of rats fed diets supplemented with raw peanut, roasted peanut, raw peanut naturally contaminated with aflatoxins or roasted peanut naturally contaminated with aflatoxins for six weeks.

Treatment	Total protein (g/dl)				Albumin (g/dl)				ALT activity (U/L)				AST activity (U/L)			
	Zero time*	Two weeks	Four weeks	Six weeks	Zero time*	Two weeks	Four weeks	Six weeks	Zero time*	Two weeks	Four weeks	Six weeks	Zero time*	Two weeks	Four weeks	Six weeks
Raw Peanut (control)	6.43 ± 0.13	6.42[a] ±0.09	6.94[b] ±0.23	6.39[a] ±0.19	3.56 ± 0.23	3.78[a] ±0.21	3.83[a] ±0.32	3.84[ab] ±0.29	25.33 ± 2.44	26.73[a] ±2.15	23.20[a] ±1.91	26.80[a] ±2.00	24.67 ± 1.53	21.33[a] ±0.67	24.33[a] ±1.67	25.67[a] ±2.06
Roasted peanut		6.78[b] ±0.09	6.35[a] ±0.14	6.58[a] ±0.03		3.86[a] ±0.27	3.91[a] ±0.24	3.39[a] ±0.29		27.33[a] ±1.77	46.67[b] ±3.53	52.67[b] ±5.21		26.00[a] ±1.16	33.33[ab] ±1.86	36.34[b] ±1.20
Raw peanut contaminated with aflatoxins		8.49[b] ±0.42	7.43[a] ±0.28	7.14[a] ±0.30		4.21[a] ±0.10	4.97[b] ±0.05	4.68[b] ±0.22		68.00[b] ±3.06	45.33[a] ±2.67	74.67[b] ±1.33		25.00[a] ±0.58	28.33[a] ±0.33	38.34[b] ±0.33
Roasted peanut contaminated with aflatoxins		6.82[a] ±0.02	7.22[a] ±0.42	6.95[a] ±0.21		3.98[a] ±0.15	4.61[b] ±0.01	4.28[ab] ±0.07		62.00[b] ±4.17	72.00[b] ±6.12	68.00[b] ±2.31		25.00[a] ±0.58	36.00[b] ±2.00	38.68[b] ±1.77

***= Before any treatment**

Each value represents the mean ± Standard Error.

The mean values with different letters within a column indicate significant differences (P< 0.05).

Such increase in ALT and AST activity can be attributed to cell necrosis, changes in cell membrane permeability or impairment of biliary excretion Pozzi *et al.* (2000). It should be noted, however, that this effect was more marked in the groups treated with Aflatoxins containing diets and affected especially ALT and AST, indicating a hepato-biliary dysfunction. As mention by Saleh *et al.* (2007) aflatoxins attack different cells in the body, especially liver cells. The activities of liver enzymes namely ALP, ALT and AST of animals

given aflatoxins –contaminated diet were significantly increased in serum. This is likely due to liver damage and the release of there enzymes into the circulation Lynch *et al.* (1971). The results are in a good agreement with data published by Gyamfi and Aniya (1998), (Pozzi *et al.* (2000) and Kocabas *et al.* (2003).

2-Histopathological findings of treated rat's liver:

The combination between aflatoxin and roasted peanut comparing with non roasted and contaminated peanut was studied and the results were as follows in liver figures from 1 to 6.

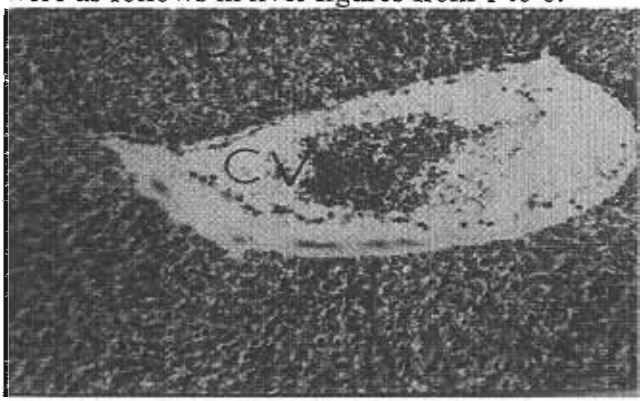

Fig. (1): liver section of rats fed on raw free aflatoxin peanut (gp-1).

Showing dilatation and congestion of central vein (CV) with degeneration change in The surrounding hepatocytes (D) (H.P. E X 40)

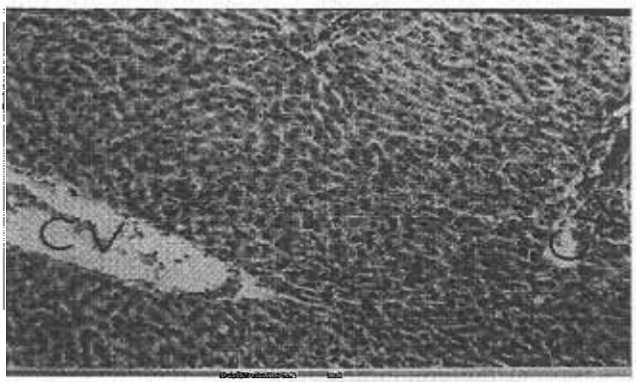

Fig. (2) **Liver section of rats fed on roasted free aflatoxin peanut (gp2)**

Showing severs congestion in the central vein (CV).). **(H.P. E X 160).**

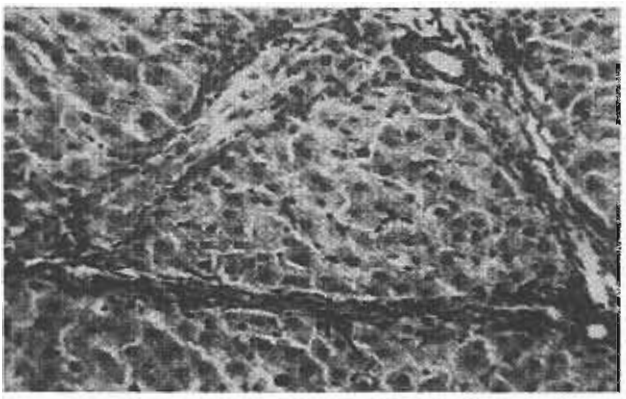

Fig. (3) Liver section of rats fed raw peanut naturally contaminated with aflatoxins. (gp-3).

Showing magnification of to identify triangular inflammatory reaction (arrow) and diffuse kupffer cells proliferation in between the hepatocytes (k). **(H.P.E X 64)**

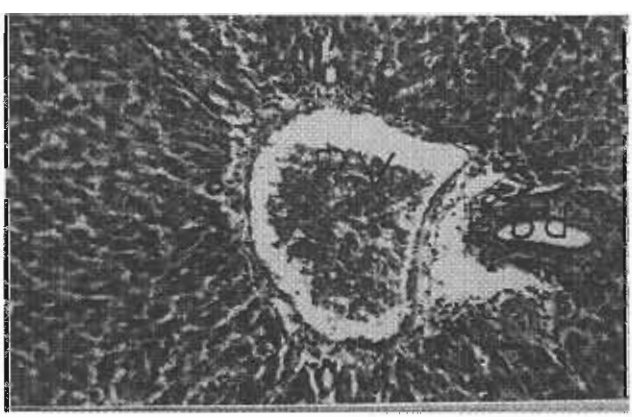

Fig. (4) Liver section of rats fed on roasted peanut naturally contaminated with aflatoxins (gp-4).

Showing Sever dilatation of portal vein (PV) with periductal inflammatory cells infiltration and fibroblastic cells proliferation surrounding the bile duct (bd) in portal area. **(H.P.E X 40)**

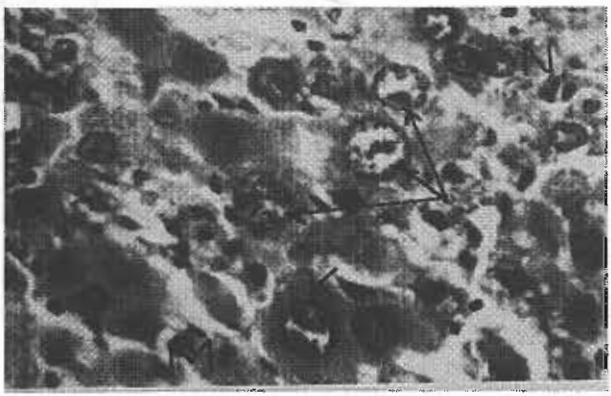

Fig. (5) Liver section of rats fed on roasted peanut naturally contaminated with aflatoxins (gp-4)

Showing cytomegaly and karyomegaly in the hepatocytes (arrows) with inflammatory cells infiltration (M) and kupffer cells proliferation (k) in between. **(H.P.E X 160)**.

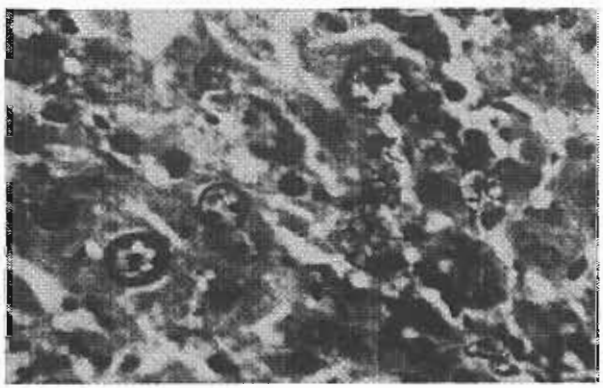

Fig.(6) liver section of rats fed on roasted peanut naturally contaminated with aflatoxins (gp-4).

Showing multinucleated giant cells formation. **(H.P.E X 160)**.

Figures from 1 to 6 concluded that the combination between aflatoxin and roasted peanut for long duration administration is leading to dysplastic activity of the hepatocytes characterized by karyo and cytomegaly associated with the toxic syndrome characterized by degenerative changes and inflammatory reaction in diffuse manner allover the hepatic tissue as well as in focal manner in

the portal area the changes in the five group are irreversible however changes in the other group are reversible. The results are matching with data of Karakilick *et al.* (2004) study on rabbit. The toxicological effects of roasted peanut contaminated with aflatoxins are due to forming of free radical reactions which formed as results of increasing of enzymes activities, increasing temperature. From the biochemical analysis and histopathology logical lesions it can be found that roasted peanut contaminated with aflatoxins is more effective on liver function than raw peanut.

It could be recommended that toxicological parameter must put into account if we should make decision for any commodity free or not free of aflatoxin specially which is mad from contaminated raw materials. Also, food consumption and total diet study must be established as soon as possible in Egypt to avoid more diseases. In addition, risk exposure must be done for commodities which are heavy consumed.

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دراسات كيميائية حيوية و سمية على الفول السودانى الخام و المحمص الملوث طبيعيا بالسموم الفطرية الأفلاتوكسينات

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يشيع التلوث بالأفلاتوكسينات (ب1، ب2 ، ج1 &ج2) في الفول السوداني و منتجاته . لذا فإن الحصر القومي للأفلاتوكسينات للفول السوداني التجاري تم عمله على مستوى أسواق القاهرة الكبرى في مصر. مائه و أربع عينات من الفول السوداني تم تجميعهم من أسواق القاهرة الكبرى . و استخلصت العينات لتحليل الأفلاتوكسينات و تم التقدير باستخدام باستخدام جهاز التحليل الكروماتوجرافي السائل العالي الكفائة المزود بكاشف فلوريسني. أظهرت النتائج أن 17 عينة من مجموع 52 عينة من الفول السوداني الخام و التي تم تحليلهم كانوا ملوثين بالأفلاتوكسينات و 11 عينة تعدوا الحد الأقصى المسموح. بينما عدد 5 عينات فقط من عدد 52 من الفول السوداني المحمص كشف التحليل بتلوثهم بالأفلاتوكسينات و عينة واحدة فقط كانت قد تعدت الحد الأقصى المسموح به. تم دمج هذه النتائج إلى نتائج الأستهلاك الغذائي من نبات الفول السوداني في مصر و ذلك لتقييم الخطر الذي يتعرض له المستهلك و تم ذلك في حالة الأفلاتوكسين ب1 ووجد أن نسبة ما يتناوله المستهلك المصري يوميا من الأفلاتوكسين ب1 عند تناوله الفول السوداني الخام هي 8233 بينما في حالة استهلاك الفول السوداني المحمص بنفس معدل الأستهلاك تصبح النسبة 2948 %.

بالرغم من النتائج السابقة فعند تحليل عناصر قياس السمية على النظم الحيوية (المجموع الكلي للبروتين – الألبيومين – نشاط ألانين امينو ترانسفيراز – نشاط أنزيم اسبرتات امينو ترانسفيراز) و دراسة التغييرات النسيجية و جد تعارض في النتائج عند تغذية الفئران على فول سوداني خام غير ملوث ، فول سوداني محمص و غير ملوث ، فول سوداني خام ملوث طبيعيا بالأفلاتوكسينات و فول سوداني محمص ملوث بالأفلاتوكسينات. تم دراسة عناصر السمية السابقة بعد المعاملة مباشرة و بعد اسبوعين ثم أربع ثم ست أسابيع. من تحليلات الكيمياء الحيوية و دراسة التغيرات النسيجية نجد أن الفول السوداني الملوث طبيعيا بالأفلاتوكسينات و محمص أكثر خطورة على وظائف الكبد. وأوضحت نتائج دراسة التغيرات النسيجية لكبد مجموعات الفئران محل الدراسة أن وجود الأفلاتوكسينات مع عملية التحميص و التغذية عليها مدة طويلة أدت إلى نشاطات خطيرة على خلايا الكبد. جميع التغيرات غير قابلة للشفاء بالرغم أن التغيرات التى فى المجاميع الأخرى كانت قابلة للشفاء.