EVALUATION OF THE PROTECTIVE EFFECTS OF JOJOBA EXTRACT AGAINST FUMONISIN TOXICITY IN RATS

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ABSTRACT

Aim of the work: The current study was conducted to evaluate the protective effect of the ethanolic extract of jojoba in rats treated with fumonisin. **Material and Method**: Forty mature male Sprague–Dawley rats were divided into four groups included the control groups and the groups treated with fumonisin-contaminated cultural materials (60 mg/L), the group treated orally with jojoba extract (5 mg/kg b.w) and the group treated with fumonisin and the extract. **Results**: the results indicated that fumonisin induced a significant decrease in RBCs, HB, platelets, WBCs and triglycerides accompanied with a significant increase in all the other biochemical parameters tested. Moreover, animals treated with fumonisin alone showed severe pathological and histochemical changes in the liver tissues. Animals treated with jojoba extract alone were comparable to the controls regarding the haematological, the biochemical parameters, the histological structure and histochemical picture of the liver. **In conclusion**: The extract contracted the toxic effects of fumonisin and succeeded to normalize all the parameters tested and improve the histological and histochemical structure of the liver.

Key words: Fumonisin, jojoba, antioxidant, liver, toxicity

INTRODUCTION

(*Simmondsia chinensis*) is an arid perennial shrub indigenous to Arizona, California and Northwestern Mexico **(Hogan, 1978)**. It is also grown in Australia **(Davidson, 1983)**, Brazil, Argentina and some Middle East countries **(Borlaug *et al*., 1985)**. As much as 97% of jojoba liquid wax (JLW) consists of a mixture of esters of long chain fatty alcohols and long chain fatty acids. More than 60% of this mixture of esters contains *cis*-11-eicosenoic (jojob-enoic) acid (C20) **(Bouali *et al*., 2008)**. JLW contains a natural antioxidant postulated to be an allylic derivative of hydroxytoluene **(Kampf *et al*., 1986)**. A protein rich residue remains after oil extraction of jojoba

seeds known as defatted jojoba meal. This meal contains 20–32% of protein, consisting mainly of albumins (79%) and globulins (21%) **(Shrestha et al., 2002)**. It also contains approximately 15% of a group of glucosides, known as simmondsins **(Van Boven *et al.,* 2000)**. Eight glucoside compounds (simmondsin and seven simmondsin derivatives) have been isolated and identified form jojoba seeds **(Bouali *et al.,* 2008)**.

Jojoba oil has good markets in the cosmetics and lubricant industries **(Cokelaere *et al*., 1996)**, and recently, it has been reported that the jojoba seeds possess anti-inflammatory activity **(Habashy *et al*., 2005)**. Moreover, JLW was

used in folk remedies for renal colic, sunburn, chaffed skin, hair loss, headache, wounds and sore throat (**Yaron, 1987**).

Fumonisins are a group of fungal toxins that are commonly found on corn (**Shephard** *et al.,* **1996**) and other cereals grains used worldwide in animal feed and human foods (**Visconti** *et al.***, 1991 and Pohland, 1996**). Several studies have reported that *F. moniliform* and FB_1 are hepatocarcinogenic in rats (**Gelderblom** *et al.***, 1991; Abdel-Wahhab** *et al.***, 2002 and El-Nekeety** *et al.***, 2007**). Fumonisin B_1 has been reported to

cause morphological and functional changes in chicken macrophages *in vitro* which indicate an immunosuppressing effect (**Qureshi and Hagler, 1992**). Fumonisin have been found to disrupt sphingolipids metabolism in hepatocytes from Sprague Dawley rats (**Wang et** *al.***, 1991; Abdel-Wahhab** *et al.***, 2004; Riley and Voss, 2006**), ducks (**Tran** *et al.***, 2005**), and mice (**Voss et al., 2002**). Due to its structural similarity with sphingosine (**Bezuidenhout** *et al.,* **1988; Laurent** *et al.***, 1989 and Kim** *et al.,* **2006**), FB_1 interferes with ceramide synthase leading to intracellular accumulation of sphingoid bases which mediate several key biological processes such as cell proliferation, and DNA replication. FB_1 inhibits biosynthesis of cellular macromolecules (**Abado-Becognee** *et al.***, 1998; and He** *et al.***, 2006**), and it induces lipid peroxidation in both primary rat hepatocytes (**Abel and Gelderblom, 1998 and El-Nekeety** *et al.,* **2007**) and C6 glioma cells (**Mobio** *et al.,* **2000**). The present study aimed to evaluate the antioxidant effects of the ethanolic extract of jojoba against fumonisin toxicity in rats.

MATERIAL AND METHODS Plant - material: Jojoba (*Simmondsia chinensis*) stems, leaves and seeds were obtained from Orman garden, Giza and were identified by the Department of Botany, Fac. Science, Cairo University.

Preparation of jojoba extract: The ethanolic extract (95 %) of the different organs of jojoba was prepared by macerating the powdered defatted jojopa seeds (100 g) and the aerial parts (stems and leaves) (100

g) in successive portions of ethanol 95% till exhaustion. The extract in each case was pooled and evaporated under reduced pressure to obtain a semisolid residue. A 20% (w/v) solution of each extract in biodistilled water containing few drops of Tween 80 was prepared and used for treatment of animals.

FBs production: FBs were produced through the inculcation of PDA media by *F. moniliforme* isolated from fruits samples (banana) collected from local markets in Cairo as described by **Voss** *et al.* **(1993)**. In brief, *Fusarium* isolates were suspended in sterile water and used to inoculate the liquid media (500 g) in 2-L glass flask previously autoclaved at 121 ^{O}C for 1 h on each of 2 consecutive days. Cultures were incubated in the dark at 25 ^{O}C for 21 days. FBs in the culture media was measured by high performance liquid chromatography HPLC) according to **Shephard** *et al.* **(1990)**. Briefly, FBs was extracted from a sample of the culture material with methanol–water (3:1vol/vol). The extract was purified on a strong anion-exchange cartridge, and an aliquot was derivatized with a reversed-phase column, monitored by fluorescence detection, and quantified by comparison of peak areas with those obtained with reference standards of FB_1, FB_2 and FB_3. The FBs within the cultural material consisted of 70% FB_1, 20% FB_2 and 10% FB_3 based on the total FBs in the cultural material. The cultural material was administrated orally to the experimental rats at dose of 1 ml/ rat (containing 60 mg/L total fumonisins).

Experimental animals: Three months old Sprague–Dawley male rats (100–120 g) were purchased from the Animal House Colony, Giza, Egypt and were maintained on standard lab diet (protein: 160.4; fat: 36.3; fiber: 41 g/kg and metabolizable energy 12.08 MJ) in artificial illuminated and temperature controlled room free from any other source of chemical contamination at the Nutrition Institute, Cairo, Egypt. After an acclimatization period of 1 week, the animals were divided into four groups (10 rats/group) and housed in filter-top polycarbonate cages. All animals were received humane care in

compliance with the guidelines of the Animal Care and Use Committee of the Nutrition Institute, Cairo, Egypt.

Experimental design: Experimental groups of animals were maintained on the control diets for 3 weeks and were included the untreated control group; the group treated orally with 1 ml FB_1 cultural material (60 mg FBs); the group treated orally with jojoba extract (0.5 mg/kg b.w. in 0.5 ml distilled water) and the group treated orally with FB_1 cultural material and jojoba extract at the same doses. Body weight was recorded daily allover the experimental period. At the end of the treatment period, two blood samples were collected from the retro-orbital venous plexus of all animals after fasting for 12 h. The first blood sample from each animal within each experimental group was placed in plastic tubes containing EDTA and assayed on the same day for hematological parameters using sysmex S.F 3000 (USA). The following hematological parameters were determined: hemoglobin (Hb), red blood cells counts (RBCs), white blood cell counts (WBCs) and platelets count. The second blood sample from each animal was left to clot and centrifuged at 5000 rpm under cooling for 10 min to separate the serum for other biochemical analyses using Beckman cyncron cx5 (USA). The following biochemical parameters were determined: alanine aminotransferase (ALT), aspartate aminot-ransferase (AST), alkaline phosphatase (ALP), lactic dehydrogenase (LDH), createnine kinase (CK), triglycerides, cholesterol, urea, uric acid and createnine. After blood samples were collected, all animals were sacrificed and the liver of each animal was dissected and samples were excised and fixed in 10% neutral formalin, dehydrated in ascending grades of ethanol, cleaned in xylene and embedded in paraffin. Five μm thick sections were prepared and stained with hematoxylin and eosin (Hx&E) according to **Drury *et al*. (1976)** for histological examination and other sections were stained with Mercuric-Bromphenol blue stain for demonstration the total protein **(Mazia *et al.*, 1953)** and Feulegn technique for demonstration of DNA **(Bancroft and Stevens 1990**). Three sections from each of the control and treated animals were microscopically examined. Moreover, sections stained for DNA by Feulgen reaction and total protein by Mercuric-Bromphenol blue stain were measured using a computerized image analyzer and Image Pro plus software.

Statistical analysis: All the obtained data for hematological and biochemical parameters as well as DNA and protein were statistically analyzed using the General Linear Models Procedure of the Statistical Analysis System **(SAS, 1982)** and student t-test. The significance of the differences among treatment groups was determined Waller–Duncan k-ratio **(Waller and Duncan, 1969)**. All statements of significance were based on probability of $P \leq 0.05$.

RESULTS

The results of the current study revealed that animals treated with FBs alone showed a significant decrease in the body weight compared to the control group (Fig. 1). Animals treated with jojoba extract alone were comparable to the control group. However, the extract succeeded to restore the body weight towards the normal values of the control (Fig. 1).
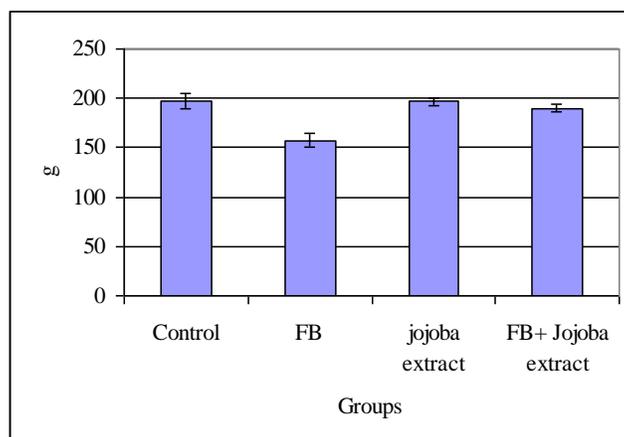
Fig. (1) Effect of Jojoba extract on the body weight in rats treated with FBs for 3 weeks

The results of the haematological study (Table 1) revealed that treatment with FB resulted in a significant decrease in count of RBCs, platelets, WBCs and Hb concentration. However, these parameters were comparable to the control group in the animals treated with the extract alone or in combination with FB. On the other hand, the biochemical results (Table 2) revealed that FB alone induced a significant increase in all biochemical parameters tested except cholesterol level which was found to decrease significantly.

The extract alone resulted in a significant decrease in ALT, LDH and CK, whereas; it did not affect significantly the other parameters. The administration of the extract to the animals treated with FB resulted in a significant improvement in all the biochemical parameters although most of them were still significantly differing than the control values. However, the combined treatment succeeded to restore ALP, triglycerides, cholesterol, urea and createnine nearly to the normal values of the control (Table 2).

Table (1). Effect of jojoba extract on some hematological parameters in rats treated with FB for 4 weeks (means ± SE)

Groups / Parameter	Control	FB	Jojoba extract	Jojoba extract + FB
RBCs (No X 10^6/µl)	6.33 ± 0.14^a	4.23 ± 0.33^b	5.75 ± 0.17^a	6.15 ± 0.15^a
Hb (g/dl)	12.25 ± 0.23^a	7.83 ± 0.61^b	12.22 ± 0.29^a	12.53 ± 0.41^a
Platelets (No X 10^3/µl)	770.83 ± 57.3^a	318.17 ± 18.75^b	784.83 ± 54.76^a	771.5 ± 32.37^a
WBCs (No X 10^3/µl)	20.15 ± 2.01^a	11.98 ± 0.68^b	20.74 ± 0.79^a	19.41 ± 0.83^a

Within each raw, means superscript with different letters are significantly different (P≤ 0.05)

Table (2). Effect of jojoba extract on biochemical parameters in rats treated with FB for 3 weeks (means ± SE)

Groups / Parameter	Control	FB	Jojoba extract	Jojoba extract + FB
ALT (IU/L)	88.33 ± 2.99^a	157.0 ± 20.49^b	68.33 ± 1.94^c	107.33 ± 3.39^d
AST (IU/L)	182.67 ± 4.87^a	410.5 ± 46.01^b	178.83 ± 9.46^a	221.67 ± 5.04^c
ALP (IU/L)	300.17 ± 15.72^a	486 ± 14.02^b	295.67 ± 13.07^a	295.17 ± 17.13^a
Triglycerides (mg/dl)	66.0 ± 0.37^a	128.33 ± 1.36^b	54.5 ± 2.28^a	64.5 ± 3.38^a
Cholesterol (mg/dl)	70.5 ± 1.18^a	47.0 ± 3.48^b	63.5 ± 4.09^a	75.64 ± 4.67^a
LDH (U/L)	4038.67 ± 251.84	6010.33 ± 476.92	3491.0 ± 228.13	3783.5 ± 182.18
CK (mg/dl)	19731.67 ± 666.74^a	32775.0 ± 814.71^b	17365.0 ± 353.94^c	17782.62 ± 367.66^d
Urea (mg/dl)	43.83 ± 2.01^a	74.17 ± 1.94^b	42.83 ± 1.38^a	43.33 ± 0.99^a
Createnine (mg/dl)	0.52 ± 0.03^a	0.94 ± 0.04^b	0.48 ± 0.05^a	0.57 ± 0.05^a
Uric acid (mg/dl)	2.93 ± 0.21^a	49.7 ± 0.18^b	3.0 ± 0.47^a	3.45 ± 0.2^a

Within each raw, means superscript with different letters are significantly different (P≤ 0.05)

The histological examination of the control liver sections revealed normal histological structure of liver lobule and hepatocytes which are forming cords radiating from the central vein (Fig 2-a). Whereas, the liver of rats treated with fumonisin alone showed marked congestion and dilatation of the portal tract, necrosis and moderate proliferation of the bile duct epithelial cells (Fig. 2-b). The hepatocytes showed hepatocytes vacuolar degeneration and periportal necrosis (Fig 2-c). The microscopic examination of the liver in the fumonisin group also revealed dilatation in sinusiod. The nuclei are pleomorphism and others showed degrees of degeneration in the form of pyknosis, karyolysis, while some of the cells nuclei appeared completely absent (Fig. 2-d). The histological examination of the liver in the animals treated with jojoba extract alone showed almost normal hepatocytes arc-hitecture, slight dilatation of blood sinusoid infiltrated by few inflammatory cells, increase in kupffer cells. Moreover, some fields showed vesicular binucleated hepatocytes (Fig. 3). The liver sections in the animals treated with fumonisin plus Jojoba extract showed marked improvement of hepatocytes architecture. The hepatocytes nuclei were vesicular and binucleated and increased in kupffer cells were also seen. Meanwhile, some dilatation and congestion of blood sinusoid were still present (Fig. 4).

DNA was histochemically demonstrated using Feulgen reaction technique (Fig. 5). The mean value per nucleus was measured by image analyzer and the results were expressed as optical density values (Fig. 6). The mean value for nuclear DNA content of hepatocytes nuclei of fumonisin-treated group (0.18 ± 0.001) was significantly decrease compared to the control group (0.23 ± 0.003). The mean DNA contents of jojoba extract alone (0.25 ± 0.002) or plus fumonisin-treated (0.25 ± 0.002) groups showed significant improvement compared to the control. The total protein content was histochemical demonstrated by Mercuric-Bromphenol blue stain (Fig 7, 8). The liver of rats treated with fumonisin only (Fig. 8-b) exhibited significant decrease in protein content in cytoplasm of hepatocytes (0.173 ± 0.001) as compared to control (0.199 ± 0.002) (Fig. 8-a). While, a significant increase in protein content was found in animals treated with extract only (0.222 ± 0.001) (Fig. 8-c) comparing to control or fumonisin groups. A significant decrease in protein content in rats treated with fumonisin plus Jojoba extract (0.187 ± 0.001) (Fig. 8-d) compared to the control or extract alone-treated group. Although it exhibited a significant increase compared to fumonisins alone-treated group (0.173 ± 0.001).
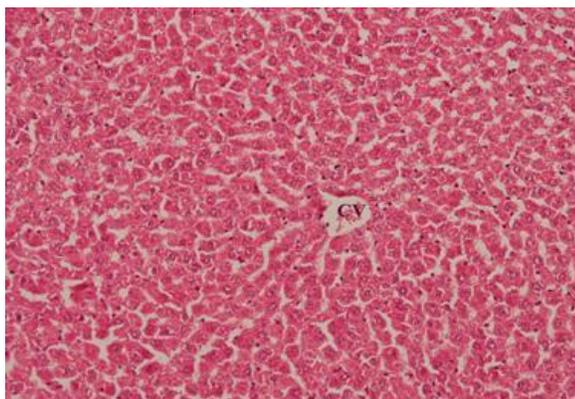
Fig. (2a). Liver sections of control animals showing the normal histological structure of liver lobule and hepatocytes which are forming cords radiating from the central vein (cv).
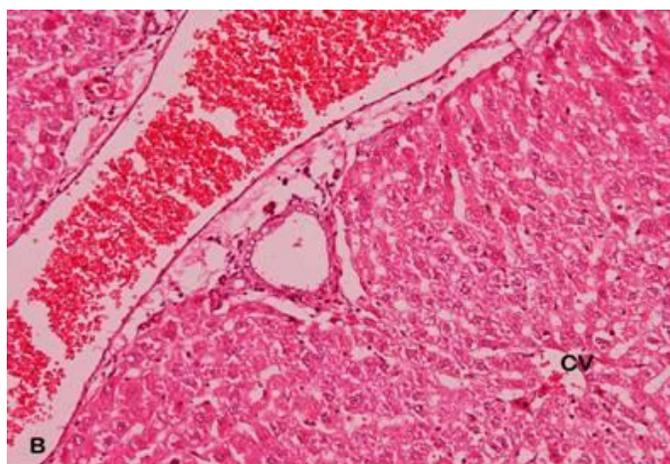
(H&E X 200)

Fig. (2b). Liver sections of rat treated with fumonisin alone showing dilated and congested hepatic portal vessel, moderate proliferation of bile duct epithelial cells.
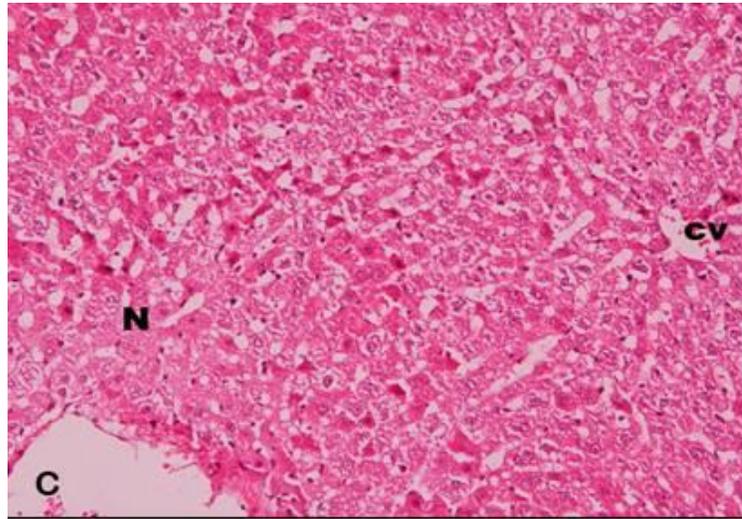
(Hx&E X 200)

Fig. (2c). Liver sections of rat treated with fumonisin alone showing hepatocytes vacuolar degeneration and periportal necrosis (N).
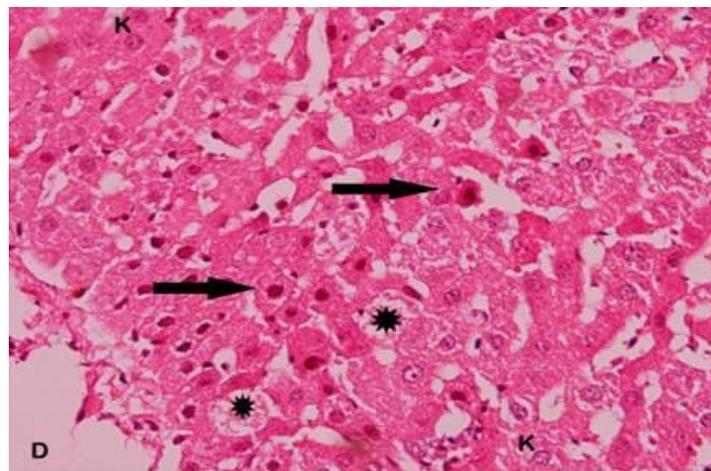
(Hx&E x200),

Fig. (2d). High magnification of previous figure showing moderate dilatation in sinusoid, necrosis, hepatocytes vacuolar degeneration, some of the nuclei showing degrees of degeneration in the form of pyknosis, (arrow) karyolysis (K) while some cells nuclei appeared completely absent (*). (H&E X 400)
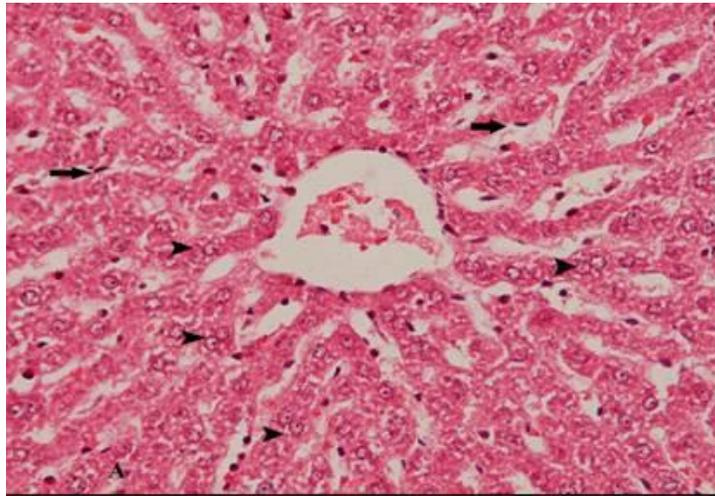
Fig. (3): liver sections of rat treated with jojoba extract alone showing almost normal architecture of hepatocytes, slight dilatation of blood sinusoid infiltrated by inflammatory cells, increase in kupffer cells (arrows) and vesicular binucleated hepatocytes (arrow head).
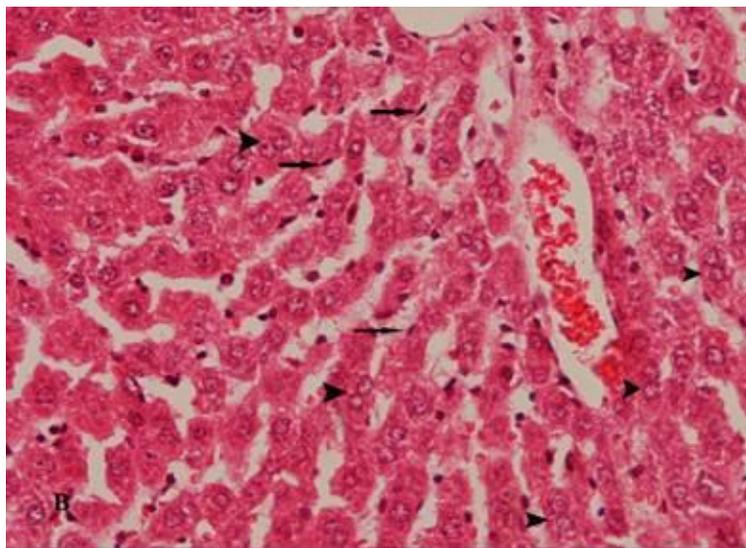
(Hx &E X 400)

Fig. (4): liver sections of rat treated with fumonisin plus Jojoba extract showing marked improvement of hepatocytes architecture, hepatocytes nuclei are vesicular and binucleated (arrow head), increase in kupffer cells (arrow). Notice: some dilatation and congestion of blood sinusoid still present.
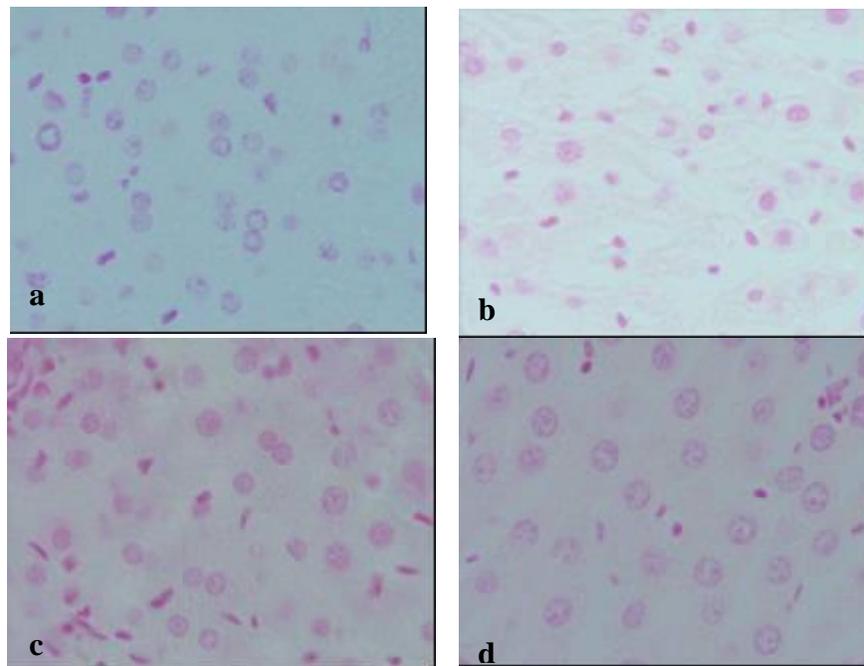
(H & E X 400).

Fig. (5): liver sections of rat showing DNA content (a): control, (b): fumonisin alone treated animals showing a decrease in DNA content, (c): jojoba extract alone-treated animals showing slight increase in DNA content compared to the control and (d) fumonisin plus Jojoba extract-treated animals showing a highly increase in DNA content compared to fumonisin-treated group.
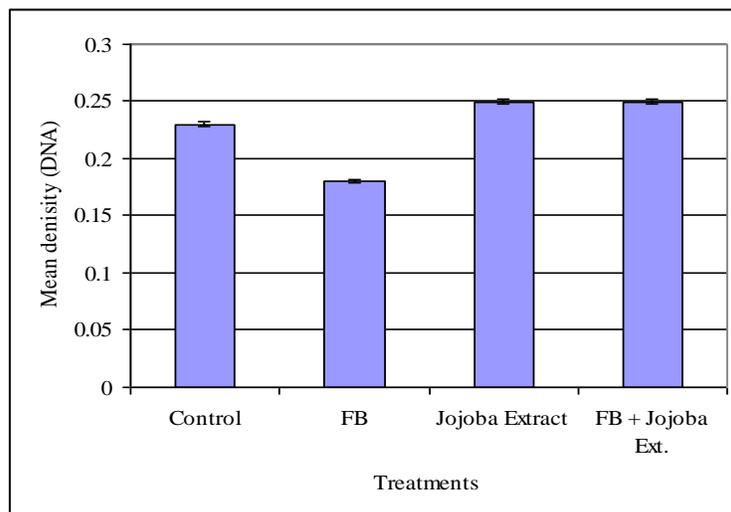(Feulgen reaction X 400)

Fig. (6): The mean optical density values of nuclear DNA in hepatocytes nuclei of different groups
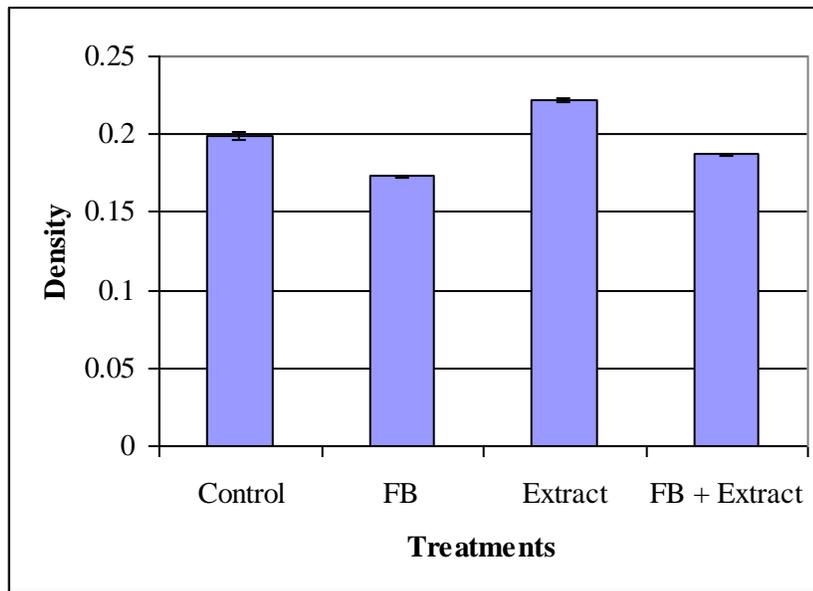
Fig. (7): The mean optical density values of protein content of hepatocytes of different groups

Fig. (8): liver sections of rat showing protein content in cytoplasm of hepatocytes (a): control showing normal distribution of protein, (b): fumonisin alone-treated animals, showing decrease in protein content, (c): jojoba extract alone-treated animals showing moderate increase in protein content compared to control and (d) fumonisin plus Jojoba extract-treated animals showing slight increase in protein content compared to toxin group. (Bromophenol blue stain X 400).

DISCUSSION

In the current study, we evaluated the ability of ethanolic extract of jojoba to protect the laboratory animals from the toxic effects of FB. The tested animals were given an extreme FB challenge to ensure induction of severe response. The selective doses of FB and extract were literature based (**Abdel-Wahhab et al., 2004 and Habashy et al., 2005**) respectively. The decrease in body weight gain reported in the current

study indicated the presence of adverse effects and toxicity in rats due to receiving FB. This decrease may indicate protein catabolism, thereby contributing to the observed kidney injury (**Abdel-Wahhab et al., 2002, 2004; Tessari et al., 2006 and El-Nekeety et al., 2007**). Similar decrease in body weight loss had been reported in rats (**Abdel-Wahhab et al., 2002 and El-Nekeety et al., 2007**), swine (**Haschek et al., 1992**) horses (**Ross et al., 1993**), broiler (**Brown et al., 1992**) and turkey poults (**Weibling et al., 1993a**) fed fumonisin. Previously, **Abdel-Wahhab et al. (2004)** and **El-Nekeety et al. (2007)** stated that administration of FB to rats enhanced lipid peroxidation which is presumably results of free-radical-mediated toxicity. **Stockmann-Juvala et al. (2004)** found that FB evoke oxidative stress, which may contribute at least in part to FB toxicity and carcinogenicity. In the current study, animals treated with FB showed a significant decrease in RBCs, Hb, platelets and WBCs. The decrease in haemoglobin concentration and total RBCs resulting in normocytic normochromic anaemia. This decrease in the haemopoietic parameters may be due to many factors, such as inhibition of protein synthesis as indicated by lower body weight gain (**Kaneko, 1989 and Abdel-Wahhab et al., 2002**). Previous reports indicated that FB decreases the total iron binding capacity (**Abdel-Wahhab et al., 2002, 2004**) and

affects the metabolism of minerals such as Cu and Zn (**Abdel-Wahhab et al., 2002 and Tardieu et al., 2008**). These results support our

findings that FB cause normocytic normochromic anaemia. Moreover, the increase in the WBC count suggested that the toxin is eliciting an inflammatory response, and in turn causes alterations in bone marrow and the function of the immune system (**Tryphonas et al., 1997 and Mexía-Salazar et al., 2008**).

The elevation in ALT, AST, ALP, triglycerides, cholesterol, uric acid, urea, createnine and CK levels and increased LDH in this group indicated necrosis or hepatocellular injury (**Abdel-Wahhab et al., 2002 and El-Nekeety et al., 2007**). FB administration enhanced the generation of free radicals which directly results of free radical-mediated toxicity (**Norred and Voss, 1994 and Abdel-Wahhab et al., 2004 and El-Nekeety et al., 2007**). The generation of free radicals is one of the main manifestations of oxidative damage and has been found to play an important role in the toxicity and carcinogenesis of many carcinogens. In this concern, **Abdel-Wahhab and Ahmed (2004)** reported that liver damage is directly related to free radical mediated toxicity. Free radicals are known to attack the highly unsaturated fatty acids of the cell membrane which considered a key process in many pathological events and is one of the reactions induced by oxidative stress (**Schinella et al., 2002**). Another mechanism of FB-induced injury was suggested by **Pinelli et al. (1999)** who stated that FB induced a down-regulation of cytoplasmic phospholipase A2 (cPLA2) activity and arachidonic acid (AA) metabolism by a mechanism involving prostaglandin production, cAMP synthesis and protein kinase (PKA) activation. Moreover, **Seegers et al. (2000)** stated that FB has inhibitor effects on ceramide synthesis, AA, prostaglandin E_2 (PGE_2) and prostaglandin A2 (PGA2). These authors suggested that FB decreased the lipid-enhanced tyrosine kinase activity, and in combination with AA, PGE2 or PGA2, FB increased the number of G2/M cells and decreased PGA2- and AA-induced p53 levels. **Meli et al. (2000)** indicated that cellular targets of FB include immune cells

and in particular macrophages and it increased the production of prostaglandin E2 in rats.

The histological findings of the liver confirmed well the biochemical results. It is clearly noticed that the extract has a protective role against FB-induced liver damage as indicated by the improvement of the histological structure, DNA and protein content in the liver tissues. Similar histological changes in the liver tissues were reported by **Abdel-Wahhab et al. (2002)** and **El-Nekeety et al. (2007)**. Moreover, **Voss et al. (1996)** and **Abdel-Wahhab et al. (2004)** stated that FB specifically disrupt cellular sphingolipid metabolism causing, among other things, increased levels of the sphinogoid base sphinganine and an increased sphinganine/sphingosine ratio. Such disruption was associated with a diversity of animal diseases. These include liver and kidney in rats **(Abdel-Wahhab et al., 2002)**, liver and brain lesions in horses **(Wang et al., 1992)**, liver and lung lesions in pigs **(Riley et al., 1993)** and liver lesions in chickens **(Weibling et al., 1993b)**. FB was reported to induce liver lesions in rats which consist one or more of the following features: single cell necrosis, hepatocellular cytoplasmic vacuolation, variation in nuclear size and staining properties, pyknosis, fibrosis and bile duct proliferation, mild to marked hepatocellular hyperplasia, mitotic figures and foci of cellular alteration were found in the more severely affected livers**(Norred and Voss, 1994; Abdel-Wahhab et al., 2002 and El-Nekeety et al., 2007)**.

According to **Kampf et al. (1986)**, jojoba contains a natural antioxidant postulated to be an allylic derivative of hydroxytoluene. It is also contains 20–32% of protein, consisting mainly of albumins and globulins **(Shrestha et al., 2002)**. Furthermore, **Van Boven et al. (2000)** stated that jojoba contains a group of glucosides, known as simmondsins and eight glucoside compounds have been isolated and identified form jojoba seeds **(Bouali et al., 2008)**. The protective effects of the extract against FB-induced hematological, biochemical, histological and histochemical changes reported herein may be due to the free radicals scavenger properties of the extract **(Abdel-Wahhab and Aly, 2003)**.

Another mechanism by which the extract may induce its protective effect through the enhancement of the synthesis of antioxidant enzymes in the liver **(Yener et al., 2009)**.

The antioxidant activity of glucoside was reported by **Mehta et al. (2009)**. Moreover, **Abdel-Wahhab et al. (2007)** concluded that simmondsin and simmondsin derivatives of glucoside decreased DNA damage and hepatocarcinogenesis induced by aflatoxin B_1 by activating the phase II enzymes glutathione *S*-transferase (GST) and GSH peroxidase (GSH-PX). These results suggest that glucoside is capable of counteracting FB toxicity by suppressing cytochromes P-450 mediated bioactivation of FB. Jojobenoic acid constitute jojoba extract has antioxidant activity and has the ability to bind metal ions represents an additional means of modulating their pharmacological responses **(Bouali et al, 2008)**. More importantly, jojoba extract itself was not toxic and did not exert any significant changes in the hematological and biochemical parameters tested or the histological structure of the liver. In this regards, previous reports showed that jojoba extract did not show any toxic manifestation on the general body metabolism and the blood serum parameters were within the normal range **(Yaron, 1987 and Habashy et al., 2005)**.

It could be concluded that the ethanolic extract of jojoba has antioxidant effects and can protect against fumonisin-induced hepatotoxicity. This action may be due to its content of several antioxidant compounds that have the ability to scavenge free radicals generated by fumonisin and consequently prevent lipid peroxidation and/or the enhancement of the antioxidant enzymes in the cell.

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تقييم التأثير الوقائي لمستخلص الجوجوبا ضد التسمم بالفيومانزين في الجرذان

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أجريت هذه الدراسة لتقييم التأثير الوقائي لمستخلص نبات الجوجوبا ضد التسمم بالفيومانزين في الجرذان حيث استخدمت أربع مجموعات من جرذان التجارب وعوملت لمدة ثلاثة اسابيع و شملت المجموعة الضابطة والمجموعة المعاملة ببيئة محتوية على الفيومانزين (60 ملجم/لتر بيئة) والمجموعة المعاملة بالمستخلص (5 ملجم/كجم وزن جسم) والمجموعة المعاملة بالفيومانزين والمستخلص معا. أثبتت النتائج أن المعاملة بالفيومانزين أدت إلى حدوث نقصا معنويا في عدد كريات الدم الحمراء والبيضاء والصفائح الدموية والهيموجلوبين والجلسريدات الثلاثية. كما سببت المعاملة نقصا معنويا في جميع القياسات البيوكيميائية الخاصة بوظائف الكبد والكلية مع حدوث تغيرات باثولوجية وهستوكيميائية في نسيج الكبد. لم تظهر المعاملة بالمستخلص بمفرده أي تغيرات في القياسات الخاصة بصورة الدم أو القياسات البيوكيميائية الأخرى أو الصورة الهستولوجية أو الهستوكيميائية للكبد. أثبتت النتائج قدرة مستخاص نبات الجوجوبا على منع التأثيرات السامة الناتجة عن الفيومانزين حيث أدى إلى تحسن في جميع القياسات الهيماتولوجية والبيوكيميائية والصورة الهستولوجية و الهستوكيميائية للكبد وكانت معظم النتائج مطابقة للمجموعة الضابطة.