

Protective Effects of Apricot and Plum Kernel Powder in Carbon Tetra Chloride-Induced Liver Disorder in Rats

Adel A. Abd El-Mouty, Yousif A. Elhassaneen and Samia R.H. Tahoon

Department of Nutrition and Food Science, Faculty of Home Economics, Menoufia University, Shebin El-Kom Egypt

Abstract

Effects of different concentrations 2.5 and 5 % of apricot, plum and their mixture as powder on biological and biochemical changes of hepatic rats were investigated. Eighty four male albino rats were used and divided to 8 groups, each group (6) rats. The rats treated with carbon tetra chloride (CCl₄). Results indicated that the highest GOT liver enzyme of treated group recorded for group fed on 2.5 % apricot kernel powder but, the lowest value recorded for group fed on 5% mixture apricot and plum with a significant difference at ($P \le 0.05$). The mean values were 71.50 and 53.70 U/L, respectively. The highest GPT liver enzyme of treated group recorded for group fed on 2.5 % plum kernel powder but, the lowest value recorded for group fed on 5% mixture apricot and plum with significant difference at ($P \le 0.05$). The mean values were 16.05 and 11.80 U/L, respectively. The lowest triglyceride and total cholesterol level recorded for group fed on 5% mixture apricot and plum kernel. On the other hand, the highest HDL-c of treated group recorded for group fed on 5% mixture of apricot and plum kernel powder. The verse versa recorded for LDL-c and VLDL-c. Also, the lowest glucose, urea, uric acid and creatinine levels recorded for 5% mixture plum and apricot kernel powder with significant differences. As conclusion, 5.0 % mixture of apricot and plum kernel powder recorded the best levels for protective, improvement lipid profile, kidney functions and glucose levels. Therefore, apricot and plum kernel powder and their mixture could be used in our beverages and daily dishes.

Key words: Fruits by product, protective effect and Rats.

Introduction

Liver is a major site of metabolism and excretion. It is continuously exposed to xenobiotics which result in a variety of serious liver disorders. Plant based formulations are frequently employed for the liver diseases, but there are few effective suitable drugs available (Chaterrjee, 2000).

The liver is a vital organ that plays a central role in transforming and clearing chemicals and is susceptible to the toxicity from these agents; therefore it is an important target organ of the toxicity of drugs, xenobiotics, and oxidative stress. Hepatotoxicity is presently the most widespread pathology worldwide, representing up to 83% of all cases and the most serious health problems. Free radicals and reactive oxygen species are increasingly believed to play a crucial role in the initiation and progression of liver diseases, independent of the original causal agent. Carbon tetrachloride (CCl₄) is a selective hepatotoxic chemical agent that is metabolized by the cytochrome P450 into highly reactive metabolites including trichloromethyl free radical (CCl₃•) and trichloromethylperoxy radical (CCl₃O₂•) (**Al-Harbi** *et al.*, **2014**).

The plant-based hepatoprotective agents or drugs contains diversity of major active constituents such as phenols, coumarins, lignans, terpenoids, carotenoids, glycosides, flavonoids, organic acids, alkaloids and xanthenes. Several phytomolecules have been reported as having potent hepatoprotective principles. So, an investigation into the lead molecules, that may produce better therapeutic effects, is required to overcome the pharmaceutical imbalance between remedies that protect the liver and drugs that induce hepatotoxicity (Ahmed *et al.*, **2008**).

Epidemiological studies have shown that consumption of fruits and vegetables is associated with reduced risk of chronic diseases. Hepatic dysfunction due to ingestion or inhalation of hepatotoxins such as acetaminophen, cadmium chloride, ethanol, carbon tetrachloride (CCl₄) and allyl alcohols are increasing worldwide. Carbon tetrachloride is metabolized by cytochrome P450 in the liver cell endoplasmic reticulum leading to the generation of an unstable complex of CCl3 radical, which reacts rapidly with O_2 to yield highly reactive hepatotoxic trichloromethyl peroxy radical (**Cha** *et al.***, 2010**).

Carbon tetrachloride is used as a solvent in synthetic chemistry research. It is one of the most potent hepatotoxins and is widely used in

scientific research to evaluate hepatoprotective agents (Seifert et al., 1994).

Apricot (*Prunus armeniaca*, L.) is a fruit which has a high content of carotenoids, mainly b-carotene. B-carotene is the source of provitamin A. A quantity of 250 g fresh or 30 g dried apricots provides 100 % of the recommended daily allowance of provitamin A. Apricot also contains vitamins C and E (**Ruiz et al., 2005**).

Ozturk (2009) reported that long-term apricot feeding shows beneficial effects on CCl_4 -induced liver steatosis and damage in rats because of its high radical-scavenging capacity. Its effect occurred probably due to its high phytochemicals and vitamin content. It is possible to say that apricot as a natural food could have beneficial effects on non-alcoholic hepatic steatosis.

Bayram and Ismail, (2011) evaluated the hepatoprotective effect and antioxidant role of sun, sulphited-dried apricot and its kernel against ethanol-induced oxidative stress. The results showed that apricot has a hepatoprotective effect in rats with ethanol, probably acting by promoting the antioxidative defense systems.

Fleshy fruits of several species of genus Prunus, including that of *P. domestica*, *P. salicina*, *P. americana*, are called Plum. More than 100 species of plum are cultivated in the temperate zones throughout the world since prehistoric times. Commonly, dried plums are called prunes. China is leading plum producing country in the world. Plants are small to medium sized trees. Leaves are ovate or elliptical with acute or obtuse tips, short petioles and crenulate margins. Flowers are small, white and have longer pedicels, mostly born in umbel-like clusters of 2-3 individuals on short spurs, and solitary or 2-3 in axils of 1 year old wood. Fruits are fleshy, oval or round to conical having glaceous surface. Fruits come in variety of colors and sizes (**Donovan** *et al.*, **1998**).

Plums and prunes are rich source of polyphenolic compounds. Total phenolic contents of different plum cultivars have been reported between 282-922 mg/100 g of fruit. Phenolic compounds of prunes consist mainly of chlorogenic acid, neochlorogenic acid, caffeic acid, coumaric acid, rutin and proanthocyanidin (**Kimura** *et al.*, 2008).

Daily ingestion of prune juice by human volunteers was found to have mild laxative effect and significant reduction in serum activities of alanine transaminase and serum alkaline phosphatase. Therefore, prune juice may be useful as mild laxative and beneficial in hepatic diseases (Ahmed *at al.*, 2008).

Raj *et al.*, (2016) worked out the hepatoprotective effect of *Prunus armeniaca* in paracetamol intoxicated rats. They found that the extract of the leaves of *Prunus armeniaca* in methanol and water decreased the liver toxicity by decreasing the levels of SGOT, SGPT, ALP and bilirubin.

This work was conducted to study the effect of different concentrations of apricot, plum kernels and their mixture as powder on biological and biochemical changes of hepatic rats.

Material and methods

Materials

Apricot and plum kernel

Commercially dried and ground apricot (*Prunus armeniaca*, L), plum (*Prunus domestica*, L) kernels and their mixture were obtained from local market in 2019 from local market at Menoufia Governorate, Egypt. Pure white crystalline cholesterol powder and saline solutions were purchased from SIGMA Chemical Co., (USA). Casein, cellulose, choline chloride powder, and DL methionine powder, were obtained from Morgan Co. Cairo, Egypt. Chemical kits used in this study (TC, TG, HDL-c, ALT, AST, ALP, bilirubin, urea, creatinine, albumin) were obtained from Al-Gomhoria Company for Drugs, Chemical and Medical Instruments, Cairo, Egypt. While, malondialdehyde kits was obtained from SIGMA Chemical Co., Cairo, Egypt.

Experimental animals

A total of 48 adult normal male albino rats Sprague Dawley strain weighing 140±10 g were obtained from Vaccine and Immunity Organization, Ministry of Health, Helwan Farm, Cairo, Egypt.

Methods

Preparations of apricot and plum kernel

To prepare the dried apricot and plum kernel and their mixture powder, were washed thoroughly under running tap water, summarize dried, and ground to a fine powder using an air mill, high speed mixture (Molunix, Al-Araby company, Benha, Egypt) and then serving as powder seize.

Experimental design

Eighty four adult male white albino rats, Sprague Dawley Strain, 10 weeks age, weighing (140±10g) were used in this

experiment. All rats were fed on basal diet (casein diet) prepared according to American Institute of Nutrition (AIN) (1993) for 7 consecutive days for adaptation. After this adaptation period, rats were divided into 6 groups, six rats per each as follows: group (I): rats fed on basal diet as negative control. Group (2): injected by 0.2 ml/100 g body weight of 40 ml/l CCl₄ (Morgan Chemical Factory, Egypt) dissolved in paraffin oil (Morgan Egypt) (Dong al., Chemical Factory, et 2005). Carbon tetrachloride was injected three times per week for 6 consecutive weeks and used as a positive control group. Group (3): group hepatic rats fed on apricot kernel as powder by 2.5% of diet. Group (4): group hepatic rats fed on apricot kernel as powder by 5% of diet Group (5): group hepatic rats fed on the plum kernel 2.5% of diet. Group (6): group hepatic rats fed on the plum kernel 5% of diet. Group (7): group hepatic rats fed on the mixture (1:1) of apricot and plum kernel 2.5% of diet. Group (8): group hepatic rats fed on the mixture (1:1) of apricot and plum During the experimental kernel 5% of diet. period, the experiment continued for 28 days, at the end of the experimental period each rat weight separately then, rats are slaughtered and blood samples collected.

Blood sampling

After fasting for 12 hours, blood samples were obtained from hepatic portal vein at the end of each experiment. Two kinds of blood samples were taken. The blood samples were collected into a dry clean centrifuge glass tubes and left to clot in water bath (37°C) for 30 minutes, then centrifuged for 10 minutes at 3000 rpm to separate the serum, which were carefully aspirated and transferred into clean cuvette tube and stored frozen in deep freezer till analysis.

Biochemical analysis

Serum lipids profile

Serum total cholesterol was determined according to the colorimetric method described by **Thomas (1992)**. Serum triglycerides were determined by enzymatic method using kits according to the **Young, (1975) and Fossati and Principle, (1982)**. HDL-c was determined according to the method described by **Fredewaid (1972) and Grodon and Amer (1977)**.

- VLDL-c was calculated in mg/dl according to Lee and Nieman (1996) was using the following formula: VLDL-c (mg/dl) = Triglycerides / 5
- LDL-c was calculated in mg/dl according to Lee and Nieman (1996) as follows:

LDL-c (mg/dl) = Total cholesterol – HDL-c – VLDL-c Liver functions

Determination of serum alanine aminotransferase (ALT), serum asparatate aminotransferase (AST), serum alkaline phosphatase (ALP) were carried out according to the method of **Clinica Chimica Acta**, (1980), Hafkenscheid (1979) and Moss (1982), respectively.

Kidney functions

Serum urea was determined according to the enzymatic method of **Patton and Crouch**, (1977). Serum uric acid was determined calorimetrically according to the method of **Barham and trinder** (1972). Creatinine was determined according to kinetic method of **Henry**, (1974).

Serum glucose

Enzymatic determination of plasma glucose was carried out calorimetrically according to the method of **Tinder (1969).**

Statistical analysis

The data were analyzed using a completely randomized factorial design (SAS, 1988) when a significant main effect was detected. The means were separated with the Student-Newman-Keuls Test. Differences between treatments at $P \le 0.05$ were considered significant using Costat Program. Biological results were analyzed by One Way ANOVA.

Results and discussion

Effect of plum, apricot kernel and their mixtures powder on liver functions of diabetic rats

Data given in Table (1) show the effect of plum, apricot kernel and their mixtures on (GOT) and (GPT) of diabetic rats. The obtained results indicated that GOT liver enzyme of positive control rats group recorded the higher value when compared with negative control group with significant difference at (P \leq 0.05). The mean values were 95.50 and 49.20 U/L, respectively. While, the highest GOT liver enzyme of treated group recorded for group fed on 2.5 % apricot kernel powder but, the lowest value recorded for group fed on 5% mixture apricot and plum with significant difference at (P \leq 0.05). The mean values were 71.50 and 53.70 U/L, respectively.

On the other hand, the GPT liver enzyme of positive control rats group recorded the higher value when compared with negative control group with significant difference at ($P \le 0.05$). The mean values were 110.0 and 52.15 U/L, respectively. While, the highest GPT liver enzyme of treated group recorded for group fed on 2.5 % plum kernel powder but, the lowest value recorded for group fed on 5% mixture apricot and plum with significant difference at (P≤0.05). The mean values were 86.50 and 55.75 U/L, respectively. These results are in agreement with Abdel-Rahman (2011) who demonstrated that ground apricot kernel (GAK) administration specifically (1.5 mg/kg/BW/rat) can effectively improve liver fibrosis caused by DMN, and may be used as a therapeutic option and preventive measure against hepatic fibrosis. Furthermore, a human trial would be applied specially GAK is a part of Egyptian diet. The act of why high amounts of GAK was improved biochemical values as compared to low or moderate levels tested in this study may be due to increase levels of oleic acid and other polyphenols in apricot kernels.

Groups	(GOT)	(GPT)
	U/L	U/L
G ₁ C (-)	$49.20 \pm 1.10^{\rm f}$	52.15 ± 0.80^{g}
G ₂ C (+)	95.50 ± 1.35^a	110.00 ± 0.40^{a}
G ₃ (2.5% Plum kernel)	60.50 ± 2.05^{d}	86.50 ± 1.20^{b}
G ₄ (5% Plum kernel)	57.00 ± 0.60^{d}	$74.85 \pm 0.90^{\circ}$
G ₅ (2.5% Apricot kernel)	71.50±1.25 ^b	82.50 ± 0.50^{b}
G ₆ (5% Apricot kernel)	68.00 ± 0.9 ^c	70.50 ± 0.60^d
G ₇ (2.5% Plum+ apricot mixture)	60.00 ± 0.90^{d}	60.35 ± 0.60^{e}
G ₈ (5% Plum+ apricot mixture)	$53.70 \pm 0.90^{\circ}$	$55.75 \pm 0.60^{ m f}$
LSD (P≤0.05)	2.25	1.34

Table (1) : Effect of plum, apricot kernel and their mixtures on liver functions of hepatic rats

Each value represents the mean \pm SD of three replicates.

Effects of plum, apricot kernel and their mixtures on serum lipid profile of hepatic rats

The effect of plum, apricot kernel and their mixtures on total cholesterol and triglycerides of diabetic rats are shown in Table (2). The

obtained results indicated that the triglyceride of positive control group recorded the higher value when compared with negative control group with significant difference at (P \leq 0.05). The mean values were 104.0 and 68.0 mg/dl, respectively. While, the lowest triglyceride recorded for group fed on 5% mixture apricot and plum kernel while, the highest value recorded for 5 % plum kernel powder with a significant difference at (P \leq 0.05). The mean values were 72.40 and 92.00 mg/dl, respectively.

In the other hand, the total cholesterol levels of positive control group recorded the higher value when compared with negative control group with significant difference at ($P \le 0.05$). The mean values were 132.00 and 98.0 mg/dl, respectively. While, the lowest total cholesterol levels recorded for group fed on 5% mixture apricot and plum kernel, while the highest value recorded for 2.5% apricot kernel powder with significant difference at (P≤0.05). The mean values were 127.0 and 105.0 mg/dl, respectively. These results are in agreement with Yakubu et al., (2008), they reported that changes in the levels of major lipids such as cholesterol and triacylglycerol could provide useful information on the predisposition of the heart of animals to atherosclerosis and its associated coronary heart disease. The significant reduction in triacylglycerol may be associated with impaired lipolysis while reduction in HDL-C at all doses investigated may not be clinically beneficial to the animals since the rate at which plasma cholesterol are carried to the liver will be also decreased. Also, Torres-Duran et al., (1998) they reported that levels of TG and TC in the liver also have been estimated to explain the status of liver. High level of TG and TC in the liver is the indication of the liver injury. They also indicated that TC and TG increased in CCl₄-induced fatty liver. In the similar studies Kutlu et al., (2009) they examined the effects of apricot kernel oil on some of the hepatic oxidative parameters of the experimental animals. The results demonstrated that oral administration of apricot kernel oil prevented the high-cholesterol diet-induced elevation of MDA and resulted in a significantly (p < 0.05) decrease in MDA content of liver homogenates.

Data presented in Table (3) show the effect of plum, apricot kernel and their mixtures on the serum lipid profiles of diabetic rats. The results indicated that the HDL-c of negative

Table	(2):	Effect	of	plum,	apricot	kernel	and	their	mixtures	on
triglycerides and total cholesterol of hepatic rats										

Groups	Triglycerides	Total cholesterol
Groups	(TG) mg/dl	(TC) mg/dl
G ₁ C (-)	68.00 ± 0.20^{e}	98.00 ± 0.10^{g}
$G_2 C (+)$	104.00 ± 2.21^{a}	132.00 ± 1.40^{a}
G ₃ (2.5% Plum kernel)	88.50 ± 1.30^{b}	$122.00 \pm 0.30^{\circ}$
G ₄ (5% Plum kernel)	$92.00 \pm 0.50^{\circ}$	$111.00 \pm 0.40^{\rm e}$
G ₅ (2.5% Apricot kernel)	85.50 ± 0.15^{b}	127.00 ± 0.30^{b}
G ₆ (5% Apricot kernel)	$83.00 \pm 0.60^{\circ}$	115.00 ± 0.10^{d}
G7 (2.5% Plum+ apricot	79.50 ± 0.10^{d}	$109.00 \pm 0.20^{\rm e}$
G ₈ (5% Plum+ apricot mixture)	72.40 ± 0.30^{e}	$105.00 \pm 0.30^{ m f}$
LSD (P≤0.05)	2.60	2.10

Each value represents the mean \pm SD of three replicates.

 Table (3): Effect of plum, apricot kernel and their mixtures on lipid profile of hepatic rats

	Parameters				
Groups	(HDL-c)	(LDL-c)	(VLDL-c)		
	(mg/dl)	(mg/dl)	(mg/dl)		
G ₁ C (-)	45.00 ± 1.40^{a}	39.40 ± 0.11^{g}	13.70 ± 0.16^{d}		
G ₂ C (+)	$29.50 \pm 1.20^{\circ}$	81.70 ± 1.35^{a}	20.80 ± 1.10^{a}		
G ₃ (2.5% Plum kernel)	43.50±1.15 ^b	60.80 ± 1.91 ^c	17.70± 1.52 ^b		
G ₄ (5% Plum kernel)	41.50±0.30 ^b	51.10±0.23 ^e	$18.40 \pm 0.10^{\text{ b}}$		
G ₅ (2.5% Apricot kernel)	39.60±0.50 ^b	70.30±1.30 ^b	17.10 ± 1.40^{b}		
G ₆ (5% Apricot kernel)	42.50 ± 1.20^{a}	55.90 ± 1.12^{d}	16.60 ± 0.50^{b}		
G ₇ (2.5% Plum+ apricot mixture)	43.40 ± 1.50^{a}	49.70±1.13 ^e	$15.90 \pm 0.20^{\circ}$		
G ₈ (5% Plum+ apricot mixture)	44.00 ± 1.00^{a}	$46.52 \pm 1.10^{\text{f}}$	$14.48 \pm 1.20^{\circ}$		
LSD (P≤0.05)	3.02	3.00	2.30		

Each value represents the mean \pm SD of three replicates.

Control rats group recorded the higher value when compared with positive control group with significant difference at (P \leq 0.05). The mean values were 45.00 and 29.50 mg/dl, respectively. While, the highest HDL-c of treated group recorded for group fed on 5% mixture of apricot and plum kernel but, the lowest value recorded for group fed on 2.5% apricot kernel powder with significant difference at (P \leq 0.05). The mean values were 44.00 and 39.60 mg/dl, respectively.

On the other hand, the LDL-c of positive control rats group recorded the highest value when compared with negative control group with significant difference at (P \leq 0.05). The mean values were 81.70 and 39.40 mg/dl, respectively. While, the highest LDL-c of treated group

recorded for group fed on 2.5% apricot kernel powder but, the lowest value recorded for group fed on 5% mixture of apricot and plum kernel with significant difference at (P \leq 0.05). The mean values were 70.50 and 46.52 mg/dl, respectively.

In case of VLDL-c, the positive control rats group recorded the highest value when compared with negative control group with significant difference at (P≤0.05). The mean values were 20.80 and 13.70 mg/dl, respectively. While, the highest VLDL-c of treated group recorded for group fed on 5 % plum kernel powder but, the lowest value recorded for group fed on 5% mixture of apricot and plum kernel powder with significant difference at (P≤0.05). The mean values were 18.40 and 14.48 mg/dl, respectively. These results are in agreement with Tanwar et al., (2018), they reported that blood lipid profile demonstrated that the detoxified apricot kernel group exhibited significantly (p < 0.05) increased levels of HDL-cholesterol (48.79%) and triglycerides (15.09%), and decreased levels of total blood cholesterol (6.99%), LDL-C (22.95%) and VLDL-C (7.90%) as compared to that of the raw (untreated) kernel group. Overall, it can be concluded that wild apricot kernel flour could be detoxified efficiently by employing a simple, safe, domestic and cost-effective method, which further has the potential for formulating protein supplements and value-added food products.

Effect of plum, apricot kernel and their mixtures powder on glucose level of hepatic rats:

Data presented in Table (4) show the effect of plum, apricot kernel and their mixtures as powders on glucose level of hepatic rats. The obtained results indicated that the higher glucose level recorded for positive control group, while the lower level recorded for negative control group with significant differences (P \leq 0.05). The mean values were 128.00 and 88.00 mg/dl, respectively. On the other hand, rats fed on 2.5 % plum kernel powder recorded the highest level, while the lowest glucose level with significant differences at (P \leq 0.05) recorded for 5% mixture plum and apricot kernel as powder, the mean values were 113.0 and 91.50 mg/dl. It could be concluded that increasing plum, apricot kernel and or mixture levels showed highest reduction in glucose level. These results are in agreement with **Ravi** *et al.*, (2004), who reported that oral administration of black plum kernel extract showed hypoglycemic activity in STZ-induced diabetes in experimental rats,

better than that of black plum whole seed or glibenclamide treatment, due to the presence of excessive amounts of hypoglycemic active principles in the kernel.

8-meese iever of inepatie rais			
Groups	Glucose (mg/dl)		
G ₁ C (-)	$88.0\pm0.30^{\rm f}$		
$G_2 C (+)$	128.0 ± 1.10^{a}		
G ₃ (2.5% Plum kernel)	113.00 ± 0.50^{b}		
G ₄ (5% Plum kernel)	$106.00 \pm 0.30^{\circ}$		
G ₅ (2.5% Apricot kernel)	$108.50 \pm 0.60^{\circ}$		
G ₆ (5% Apricot kernel)	100.00 ± 0.10^{d}		
G ₇ (2.5% Plum+ apricot mixture)	97.00 ± 0.40^{d}		
G ₈ (5% Plum+ apricot mixture)	$91.50 \pm 0.60^{\circ}$		
LSD (P≤0.05)	3.10		

 Table (4): Effect of plum, apricot kernel and their mixtures on glucose level of hepatic rats

Each value represents the mean \pm SD of three replicates.

Effect of plum, apricot kernel and their mixtures on kidney functions of hepatic rats:

Data presented in Table (5) show the effect of plum, apricot kernel and their mixtures on urea, uric acid and creatinine of hepatic rats. The obtained results indicated that the urea level of positive control rats group recorded the higher value when compared with negative control group with significant difference at (P \leq 0.05). The mean values were 71.05 and 34.00 mg/dl, respectively. While, the highest urea level of treated group recorded for group fed on 2.5 % apricot kernel powder but, the lowest value recorded for group fed on 5% mixture of apricot and plum kernel powder with significant difference (P \leq 0.05). The mean values were 57.60 and 38.60 mg/dl, respectively.

On the other hand, the uric acid level of positive control rats group recorded the higher value when compared with negative control group with significant difference at (P \leq 0.05). The mean values were 3.30 and 1.20 mg/dl, respectively. While, the highest uric acid level of treated group recorded for group fed on 2.5 % plum kernel powder but,

the lowest value recorded for group fed on 5% mixture of apricot and plum kernel powder with significant difference at (P \leq 0.05). The mean values were 2.05 and 1.40 mg/dl, respectively.

In case of creatinine, the level of positive control rats group recorded the higher value when compared with negative control group with significant difference at ($P \le 0.05$). The mean values were 66.00 and 28.00 mg/dl, respectively. While, the highest creatinine level of treated group recorded for group fed on 2.5 % plum kernel powder but, the lowest value recorded for group fed on 5% mixture of apricot and plum kernel powder with significant difference ($P \le 0.05$). The mean values were 58.00 and 32.10 mg/dl, respectively. In similar studies, Vardi et al., (2013) reported that apricot diet had a clearly protective effect against lipid peroxidation and reduced renal MDA production. Also, they demonstrated that pretreatment apricot diet significantly decreased the apoptotic cell ratio when compared to the MTX-treated group. Also, Huang et al., (2008) indicated that lipid peroxidation and impairment of antioxidant status may be involved in the sequence of events leading to methotrexate (MTX)-induced renal damage. Additionally, increased serum creatinine and urea levels may reflect renal dysfunction and an activation of apoptotic cell markers, such as PARP, which also possibly contribute to MTX-caused kidney injury. Prophylactic administration of apricot may provide new therapeutic implications for the treatment of kidney diseases, which are characterized by apoptotic cell death and renal failure.

Kitiley functions of nepatic fats				
Groups	Urea	Uric acid	Creatinine	
	mg/dl	mg/dl	mg/dl	
G ₁ C (-)	34.00 ± 1.10^{e}	1.20 ± 0.30^{b}	28.20 ± 0.42^{f}	
G ₂ C (+)	71.05 ± 0.20^{a}	3.30 ± 0.10^a	66.00 ± 0.50^{a}	
G ₃ (2.5% Plum kernel)	55.75 ± 0.50^{b}	2.05 ± 0.30^{b}	58.00 ± 3.04^{b}	
G ₄ (5% Plum kernel)	$48.15 \pm 0.40^{\circ}$	1.54 ± 0.40^{b}	56.35 ± 1.20^{b}	
G ₅ (2.5% Apricot kernel)	57.60 ± 0.20^{b}	2.00 ± 0.40^{ab}	$42.50 \pm 1.30^{\circ}$	
G ₆ (5% Apricot kernel)	$46.38 \pm 0.30^{\circ}$	$1.70 \pm 1.50^{\circ}$	$40.10 \pm 1.50^{\circ}$	
G ₇ (2.5% Plum+ apricot mixture)	41.90 ± 0.50^{d}	$1.65 \pm 1.40^{\circ}$	38.50 ± 1.40^{d}	
G ₈ (5% Plum+ apricot mixture)	38.60 ± 0.40^{d}	$1.40 \pm 1.30^{\circ}$	32.10 ± 1.30^{e}	
LSD (P≤0.05)	3.00	1.21	3.13	

 Table (5): Effect of plum, apricot kernel and their mixtures on kidney functions of hepatic rats

332

References

- Abdel-Rahman, M.K. (2011): Can apricot kernels fatty acids delay the atrophied hepatocytes from progression to fibrosis in dimethylnitrosamine (DMN)-induced liver injury in rats. Lipids in Health and Disease, 10 (114): 2-10.
- Ahmed, B.; Khan, S.; Masood, M.H. and Siddique, A.H. (2008): Antihepatotoxic activity of cichotyboside, a sesquiterpene glycoside from the seeds of *Cichorium intybus*. Journal of Asian Natural Products Research, 10 (3-4): 223-231.
- AIN (1993): American institute of nutrition purified diet for laboratory Rodent, Final Report. J. Nutrition, J. Essential Oil Res., 8 (6): 657-664.
- Al-Harbi, N.O.; Imam, F.; Nadeem, A.; Al-Harbi, M.M.; Iqbal, M. and Ahmad, S.F. (2014): Carbon tetrachloride-induced hepatotoxicity in rat is reversed by treatment with riboflavin. Int. Immunopharmacol., 21: 383–388.
- Barham, D. and Trinder, P. (1972): Determination of uric acid. Analyst, 97: 142.
- Bayram, Y. and Ismail, C. (2011): Hepatoprotective effect and antioxidant role of sun, sulphited-dried apricot (*Prunus armeniaca*, *L*.) and its kernel against ethanol-induced oxidative stress in rats. Food and Chemical Toxicology, 49 (2): 508-513.
- Cha, Y. ; Park, C. and Young,S. C. (2010): Hepatoprotective effect of chicory (*Chicorium intybus*) root extract against orotic acidinduced fatty liver in rats. Food Science and Biotechnology, 19 (4): 865-871.
- Chaterrjee, T.K. (2000): Medicinal plants with hepatoprotective properties. Herbal Options. (Books & Applied Allied P Ltd., Calcutta), 143.
- Clinica Chimica Acta (1980): Chemical kits.105: 147-172.
- Dong, C. Z.; Jun, X. L.; Rui, C.; Wei, H. Y.; Xiu, M. Z.; Shu, N.L. and Peng, X. (2005): Bone marrow derived mesenchymal stem cells protect against experimental liver fibrosis in rats. World J. Gastroenterol., 11 (22): 3431-3440.
- **Donovan, J.L.; Meyer, A.S. and Waterhouse, A.L. (1998):** Phenolic composition and antioxidant activity of prunes and prune juice (*Prunus domestica*). J. Agric. Food Chem., 46: 1247-1252.

Fossati, P. and Principle, I. (1982): Chemical Kits. Clin. Chem., 28: 2077.

Friedwaid, W.T. (1972): Determination of HDL. Clin. Chem. 18: 499.

- Grodon, T. and Amer, M. (1977): Determination of HDL. Clin. Chem., 18: 707.
- Hafkenscheid, J.C. (1979): Determination of GOT. Clin. Chem., 25:155.
- Henry, R.J. (1974): Clinical Chemist: Principles and Techniques, 2nd Edition, Hagerstoun (MD), Harcer, ROW,P 882.
- Huang, S.M.; Chuang, H.C.; Wu, C.H. and Yen, G.C. (2008): Cytoprotective effects of phenolic acids on methylglyoxalinduced apoptosis in Neuro-2A cells. Mol. Nutr. Food Res., 52: 940-949.
- Kimura, Y.; Ito, H.; Kawaji, M.; Ikami, T. and Hatano, T. (2008): Characterization and antioxidative properties of oligomeric proanthocyanidin from prunes, dried fruit of *Prunus domestica*, L. Biosci. Biotechnol. Biochem., 72: 1615-1618.
- Kutlu, T.; Durmaz, G.; Ates, B. and Erdoğan, A. (2009): Protective effect of dietary apricot kernel oil supplementation on cholesterol levels and antioxidant status of liver in hypercholesteremic rats. Journal of Food, Agriculture & Environment, 7 (3&4): 61-65.
- Lee, R. and Nieman, D. (1996): Nutrition Assessment. 2nd Ed., Mosby, Missouri, MI.
- Moss, D.W. (1982): Alkaline phosphatase isoenzymes. Clin. Chem., 28: 2007-2016.
- **Ozturk, F.; Ozturk, I.C. and Batcioglu, K. (2006):** The effect of melatonin on 7,12-dimethyl-benz[a]anthracene injury in comparision with itamin E b selenium in mouse kidneys. Fundam Clin Pharmacol 20: 359-364.
- Patton, C.J. and Crouch, S.R. (1977): Enzymatic determination of urea. J. of Anal. Chem., 49: 464-469.
- Raj, V.; Mishra, A. K.; Mishra, A. and Khan, N. A. (2017): Hepatoprotective effect of *Prunus armeniaca* L. (Apricot) leaf extracts on Paracetamol induced liver damage in Wistar rats. Pharmacognosy Journal, 8 (2): 154-158.
- Ravi, K.; Sivagnanam, K. and S. Subramanian, (2004): Anti-Diabetic Activity of *Eugenia jambolana* Seed Kernels on Streptozotocin-Induced Diabetic Rats. J. Med. Food 7 (2): 2004, 187-189.

- Ruiz, D.; Egea, J. and Tomas-Barberan, F.A. et al., (2005): Carotenoids from new apricot (*Prunus armeniaca* L.) varieties and their relationship with flesh and skin color. J. Agric. Food Chem., 53: 6368-6374.
- **SAS (1988):** SAS Users Guide: Statistics version 5th Ed. SAS. Institute Inc., Cary N.C.
- Seifert, W.F.; Bosma, A.; Brouwer, H.F. and Hendriks, P.J. (1994): Vitamin A deficiency potentiates carbon tetrachloride induced liver fibrosis in rats. Hepatology. 19: 193-201.
- Tanwar, B.; Modgil, R. and Goyal, A. (2018): Anti-nutritional factors and hypocholesterolemic effect of wild apricot kernel (*Prunus armeniaca*, L.) as affected by detoxification. Food Funct., 9: 2121-2135.
- Thomas, L. (1992): Labor and Diagnose, 4th Ed., (Chemical Kits).
- **Tinder, P. (1969):** Determination of triglycerides. Ann. Clin. Biochem., 6: 24-27.
- Torres-Duran, P.V.; Miranda-Zamora, R.; Paredes-Carbajal, M.C.; Mascher, D.; Daaz-Zagoya, J.C. and Juarez-oropeza, M.A. (1998): Spirulina maxima prevents induction of fatty liver by carbon tetrachloride in the rat. Biochem. Mol. Biol. Int., 44: 787-793.
- Vardi, N.; Parlakpinar, H.; Ates, B.; Cetin, A. and Otlu, A. (2013): The protective effects of *Prunus armeniaca*, L (apricot) against methotrexate-induced oxidative damage and apoptosis in rat kidney. J. Physiol. Biochem., 69: 371-381.
- Yakubu, M.T.; Akanji, M.A. and Oladiji, A.T. (2008): Alterations in serum lipid profile of male rats by oral administration of aqueous extract of *Fadogia agrestis* stem. Res. J. Med. Plant., 2: 66-73.
- Young, D.S. (1975): Determination of GOT. Clin. Chem., 22 (5): 1-21.



التأثير الواقى للكبد لمسحوق بذور المشمش والبرقوق على اضطرابات الكبد الفئران المستحث برابع كلوريد الكربون

عادل عبد المعطى أحمد ، يوسف عبد العزيز الحسانين ، سامية رشدى حسنى طاحون قسم التغذية و علوم الأطعمة - كلية الأقتصاد المنزلى - جامعة المنوفية - شبين الكوم - مصر .

الملخص العربى

تم در اسة تأثير تركيز ات مختلفة 2.5 ، 5٪ من بذور المشمش والبرقوق على شكل مسحوق على التغير ات البيولوجية والكيميائية الحيوية في الفئر ان المصابة بالكبد بو اسطة ر ابع كلوريد الكربون . حيث تم استخدام ثمانية وأربعون من ذكور الفئران من نوع الألبينو وقسمت إلى 8 مجموعات، كل مجموعة بها (6) فئران فئران. وأشارت النتائج المتحصل عليها أن أعلى قيم لإنزيم الكبد (GOT) من المجموعات المصابة بالكبد سجل مع تركيز 2,5٪ من مسحوق بذور المشمش، في حين سجلت أقل قيم سجلت مع مخلوط بذور المشمش والبرقوق بتركيز 5٪ مع وجود فرق معنوى حيث كان متوسط القيم 53,70، 51,50 وحدة /لتر على التوالى ببينما سجلت أعلى قيم لانزيمات الكبد (GPT) مع تركيز 2,5٪ من بذور البرقوق، في حين سجلت أقل قيمة مع تركيز 5٪ من مخلوط بذور المشمش والبرقوق مع وجود فرق معنوي. حيث كان متوسط القيم 11.80، 16,05 وحدة /لتر على التوالي . سجلت أقل قيم من الدهون الثلاثية و الكوليسترول في الدم من المجمو عات المصابة بالكبد مع تركيز 5٪ من مخلوط بذور المشمش والبرقوق مع وجود فروق معنوية. من ناحية أخرى، سجلت أعلى مستويات الكولسترول عالى الكثافة من المجموعات المصابة بالكبد مع تركيز 5٪ من مخلوط بذور المشمش والبرقوق مع وجود فرق معنوي. والعكس صحيح مع قيم الكولسترول منخفض الكثافة و الكولسترول منخفض الكثافة جدا. كما سجلت أقل قيمة من مستويات كلا من الجلوكوز اليوريا وحمض اليوريك والكرياتينين مع مخلوط بذور المشمش والبرقوق بتركيز 5٪ مع وجود فروق معنوية مع وجود فروق معنوية. وخلاصة القول أن تركيز 5٪ من مخلوط بذور المشمش والبرقوق سجلت أفضل مستويات للمحافظة على صحة الكبد، وتحسين صورة دهون الدم ووظائف الكلى ومستوى الجلوكوز وقد خلصت الدراسة إلى إمكانية استخدام مسحوق بذور المشمش والبرقوق و مخلوطهم معا كمشروب أو إلى الوجبات اليومية.

الكلمات الدالة: المنتجات الثانوية للفاكهة - التأثير الواقى للكبد - الفئران.