

## **BENEFITING OF SOME PLANT MATERIALS IN IMPROVING LIVER FUNCTIONS FOR HEPATICALLY TOXICATED RATS WITH CCl<sub>4</sub>**

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**ABSTRACT:** *The present study investigated the effects of carrot (*Daucus carota*, L.), prickly pear (*Opuntia ficus-indica*, L.) and chicory (*Cichorium intybus*, L.) on CCl<sub>4</sub> induced hepatotoxicity in rats. Forty five male albino rats were divided into 9 groups (five rats in each group). Two groups were controls, one fed on basal diet only (negative control) and the other fed on basal diet after injection with CCl<sub>4</sub> (positive control). The other groups were injected by CCl<sub>4</sub> then received basal diet containing carrot, chicory and prickly pear at the levels 10, 15% and 15% mixture of the tested plants. Liver damage was assessed by estimation for the plasma concentration of enzymes activities of aspartate amino transferase (AST), alanine amino transferase (ALT), lipid fraction (total cholesterol and triglyceride), cholesterol fraction (HDL-c, LDL-c, VLDL-c), Uric acid, Urea nitrogen and glucose. Results showed an improvement in case of prickly pear followed by carrot and chicory for the above parameters. The best level was 15% of these plants followed by 10% of tested plants. So, this study concluded that CCl<sub>4</sub> induced liver damage in rats can be ameliorated by administration of 15% prickly pear, carrot and chicory.*

**Key words:** *Carrot - prickly pear - chicory- liver damage - Cholesterol fraction- glucose.*

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### **INTRODUCTION**

Liver diseases are among the most serious aliment. They may be classified as acute or chronic hepatitis (inflammatory liver diseases), hepatitis (non inflammatory diseases) and cirrhosis (degenerative disorder resulting in fibrosis of the liver) (Kumar *et al.*, 2011).

Carbon tetrachloride (CCl<sub>4</sub>) is a well-known hepatotoxic agent (Ilavarasan *et al.*, 2003). A single dose of CCl<sub>4</sub> (20 micro 1/kg) induced hepatotoxicity, manifested biochemically by significant elevation of serum enzymes activities, such as alanine transaminase (ALT), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH) (Mansour, 2000).

Phytotherapy is the treatment and prevention of diseases using plants or plant parts, such as leaves, flowers, roots, fruits, seeds, and rhizomes. Preparation made from them called medicinal plants, or herbs (Weiss and Fintelmann, 2000). Medicinal plants have very important place as they not only maintain the health and vitality of human beings and animals, but also cure

several disease, including liver disorders without causing any toxicity (Govind and Madhuri, 2010).

*Daucus carota*, Linn. (Family: Umbelliferae) is an annual or biennial herb, whose roots are eaten raw and also cooked in many parts of the world (Muralidharan *et al.*, 2008). Carrot polyacetylene possess allelopathic activity which may explain the historical health benefits of carrots since studies investigating β-carotene doesn't seem to adequately explain the reduced risk of certain types of cancer (Brandon and David, 2012).

Carrot could afford a significant protective action in the alleviation of CCl<sub>4</sub> induced hepatocellular injury (Bishayee *et al.*, 1995). Oral administration of carrot extract produced significant hepatoprotection against lindane induced hepatotoxicity in rats. The increase levels of serum enzymes namely aspartate transaminase, alanine transaminase, alkaline phosphatase and the levels of thiobarbituric acid reactive substances, cholesterol, triglycerides and LDL –

cholesterol in lindane administered rates were observed to be significantly decreased in the lindane (+) carrot extract group. The carrot extract also restored the depressed antioxidants and HDL- cholesterol levels to near normal (Balasubramaniam *et al.*, 1998).

Cactus pear or prickly pear, a member of the cactacea family, originated from arid and semi- arid regions of Mexico. Cactus pear fruit containing betalain pigments is a good potential for the use as a natural food colorant. This fruit contains the red- violet betacyanins in addition to the yellow betaxanthins (Salim *et al.*, 2009). The juice of prickly pear is nutritionally interesting and its dietary intake could provide protection against oxidative damage (Galati *et al.*, 2003). The cactus pear fruit extract were analyzed for determined constituents: ascorbic acid, flavonoids (quercetin, isorhamnetin, myricetin, kaempferol and luteolin), betalains, taurine, total carotenoids and total phenolics. *Opuntia ficus indica* fruit extract had strong antioxidant capacity and taurine content (Fernandez- lopez *et al.*, 2010).

*Opuntia ficus indica* fruit juice administration exerts protective and curative effects against the CCl<sub>4</sub>- induced degenerative process in rat liver (Galati *et al.*, 2005). The pears of *Opuntia* have been discovered to contain a plethora of biologically active compounds. Owing to their high nutritional value, in terms of dietary fiber, polyunsaturated fatty acid-rich oil, minerals, protein, and an assortment of other phytochemicals, the pears are gaining popularity as exotic, gourmet diet (Patel, 2013).

Chicory (*Cichorium intybus*, L.) is a perennial herb in the Asteraceae family with many commercial uses. Historically, chicory was grown by the ancient Egyptians as a medicinal plant, vegetable crop, and for animal forage (Judzntiene and Budiene, 2008). Inulin from chicory roots is considered a functional food ingredient as it affects physiological and biochemical processes resulting in better health and reduction of the risk of many diseases (Kaur and Gupta, 2002).

Esculetin, a phenolic compound found in *Cichorium intybus* investigated the possible protective effect against paracetamol and CCl<sub>4</sub>- induced hepatic damage (Gilani *et al.*, 1998). Cichorium root extract therapy led to normalization of some morphofunctional liver features (decreases glycogen content and cell of necrosis and increases the number of cells with pronounced protein synthesis activity) in rats with CCl<sub>4</sub> -induced hepatitis (Krylova *et al.*, 2006). The present study was carried out to investigate biological effects of carrot, chicory, prickly pear and mixture of the tested plants on serum parameters of liver intoxication in rats.

## **MATERIALS AND METHODS**

### **Materials**

#### **Plants**

The tested plants were chicory (*Cichorium intybus*, L.), carrot (*Daucus Carota*, L.) and prickly pear (*Opuntia Ficus indica*, L.). Chicory was purchased from herbalist of Cairo, Egypt. Carrot and prickly pear were purchased from the local markets of Shebin El-kom, Menofia Governorate, Egypt.

Carbon tetra chloride (CCl<sub>4</sub>) was used as an inducer for liver cirrhosis. It was purchased from El-Gomhorya Company, Cairo, Egypt as 10% liquid solution.

#### **Chemical reagents**

Reagent kits were purchased from Diamond Diagnostics (Egypt).

#### **Experimental animals**

Forty five white male albino rats weighing about 200 ± 5g were used as experimental animals in the present investigation. They were obtained from the animal house of Research Institute of Ophthalmology, El-Giza, Egypt. They were kept under observation for one week (as adapted period) before the onset of the experiment. The animals were housed in stainless steel cages at normal atmospheric temperature (25 ± 5°C) and had a 12 h light-dark cycle. Food and water were consumed *ad libitum*.

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### **Methods:**

#### **Induction of liver intoxication in rats**

Forty rats were treated subcutaneous injection of carbon tetra chloride in paraffin oil 50% V/V (2ml/kg b.wt) twice a week for two weeks (Jayaserkar *et al.*, 1997).

#### **Preparation of plant powder**

These plants were washed and dried in drying oven at 50°C for 3 days, then crushed and milled as a dried powder.

#### **Animals diet**

The basal diet was prepared according to AIN (1993). The vitamin mixture was prepared according to Campbell (1963), while salt mixture was prepared according to Hegsted *et al.* (1941).

#### **Experimental design**

Forty five male albino rats (200 ± 5g) were randomly divided into 9 equal groups (five rats each). All rats were fed on basal diet for one week before starting the experiment for acclimatization. After the adapted period, the initial weight was 205 ± 5g. Groups of rats were as the follows:

Group (1): Rats (n=5) were fed on basal diet only as control negative group.

Group (2): Rats (n=5) were kept without any treatment as positive control group and fed on basal diet after injection with CCl<sub>4</sub>.

Group (3): Rats (n=5) were injected by CCl<sub>4</sub> then fed on basal diet containing 10% chicory.

Group (4): Rats (n=5) were injected by CCl<sub>4</sub> then fed on basal diet containing 15% chicory.

Group (5): Rats (n=5) were injected by CCl<sub>4</sub> then fed on basal diet containing 10% carrot.

Group (6): Rats (n=5) were injected by CCl<sub>4</sub> then fed on basal diet containing 15% carrot.

Group (7): Rats (n=5) were injected by CCl<sub>4</sub> then fed on basal diet containing 10% prickly pear.

Group (8): Rats (n=5) were injected by CCl<sub>4</sub> then fed on basal diet containing 15% prickly pear.

Group (9): Rats (n=5) were injected by CCl<sub>4</sub> then fed on basal diet containing %15

of mixture chicory, carrot and prickly pear (1:1:1).

At the end of the experimental periods (35 days), rats were scarified using diethyl ether anesthesia at fasting state. Part of the blood was taken to determine the level of serum glucose and other portion of blood samples was collected and allowed to coagulate at room temperature; other portion of blood was added to it, EDTA (ethylene diamine tetracetic acid) and centrifuged at 3000 r.p.m for 15 minutes. Serum was carefully aspirated and transferred into clean covet tubes and stored frozen at -20°C until the time of analysis.

#### **Biochemical analysis:**

Serum Alkaline phosphatase (ALP) was determined according to the procedure of (IFCC., 1983). Aspartate aminotransferase (AST) or (GOT) glutamic -oxaloacetic transaminase and glutamic pyruvic transaminase (GPT) or Alanine aminotransferase (ALT) were carried out according to the method of Henry (1974) and Yound (1975). Serum uric acid was determined according to the method described by Fossati *et al.* (1980). Serum urea in plasma was determined according to the enzymatic method of Patton and Crouch (1977). Glucose was determined by enzymatic test according to Tietz (1976) and Yound (1975). Enzymatic colorimetric determination of triglycerides was carried out according to Fassati and Prencipe (1982). Total Cholesterol was determined by colorimetric method according to Allain (1974). The determination of HDL was carried out according to the method of Fnedewaid (1972) and Gordon and Amer (1977). The determination of VLDL (very low density lipoproteins) and LDL (low density lipoproteins) was carried out according to the method of Lee and Nieman (1996).

#### **Statistical analysis**

Statistical analysis were done using the Statistical Package for the Social Sciences (SPSS for WINDOWS, version 11.0; SPSS Inc, Chicago). Comparative analyses were conducted using the general linear models procedure (SPSS Inc). Values of P≤0.05 were considered statistically significant (Snedecor and Cochran, 1967).

**RESULTS**

**1-Effect of feeding different levels of chicory, carrot and prickly pear on ALP, AST and ALT levels of CCl<sub>4</sub>-intoxicated rats.**

The results in Table 1 indicated that mean value of ALP enzyme, rats injected with CCl<sub>4</sub> (C +ve group) was 230.7±3.47U/L while in normal rats (C -ve) was 99.3±1.32U/L. These results denote that there was a significant increase in the mean value of ALP enzyme of rats poisoned by CCl<sub>4</sub> as compared to normal rats. The mean values of ALP of diets from groups 3, 4, 5, 6, 7, 8 and 9 were significantly higher than control negative group. Also, it could be noticed that there is no significant differences between the values of ALP enzyme of groups 4, 5 and 7. Meanwhile, rats given CCl<sub>4</sub> then fed on diet of group 6 (rats fed on basal diet with 15% carrot) showed the lowest mean value in ALP enzyme level in the serum which was 199.4±1.35 U/L as compared to control positive group and recorded the best result

of all treatment. It could be observed that due to intoxicated rats the serum levels of AST in Table 1 showed a significant increase in control positive group as compared to normal rats represents 37.25±5.82 and 22.06±1.07 U/L, respectively. There is non significant difference between groups 5, 7 and control positive group. Also, there is no significant differences between groups 3 and 9. Meanwhile, group 6 (rats fed on basal diet with 15% carrot) showed the lowest level in serum AST and recorded the best results as compared to all treatments. The ALT, in rats given CCl<sub>4</sub> then fed on all treatments groups 3, 4, 5, 6, 7, 8, and 9, was significantly higher than control negative group. Also, there is no significant difference between group 5 and control positive group. Groups 3, 4 and 8 showed similar (P>0.05) mean values of ALT. The obtained results showed that there is no significant difference in serum levels of ALT in group 6 as compared to normal rats and the best treatment was recorded for group 6 (rats fed on basal diet with 15% carrot).

**Table (1): Effect of feeding different levels of chicory, carrot and prickly pear on ALP, AST and ALT levels of CCl<sub>4</sub>-intoxicated rats.**

Liver function	ALP (U/L)	AST (U/L)	ALT (U/L)
Animal Groups	Mean± SD	Mean ± SD	Mean ± SD
Group (1) Control – ve	99.3±1.32 <sup>E</sup>	22.06±1.07 <sup>D</sup>	9.51±0.94 <sup>D</sup>
Group (2) Control + ve	230.7±3.47 <sup>A</sup>	37.25±5.82 <sup>A</sup>	16.10±1.10 <sup>A</sup>
Group (3) 10% chicory	227.3±5.06 <sup>A</sup>	28.6±61.76 <sup>B</sup>	12.29±0.26 <sup>C</sup>
Group (4) 15% chicory	222.4±3.13 <sup>B</sup>	24.87±0.42 <sup>C</sup>	11.79±2.28 <sup>C</sup>
Group (5) 10% carrot	222.3±7.5 <sup>B</sup>	37.52±7.22 <sup>A</sup>	15.93±1.25 <sup>A</sup>
Group (6) 15% carrot	199.4± 1.35 <sup>D</sup>	22.31±1.80 <sup>D</sup>	10.44±0.79 <sup>D</sup>
Group (7) 10% prickly pear	219.7±2.05 <sup>B</sup>	35.10±4.92 <sup>A</sup>	14.43±1.21 <sup>B</sup>
Group (8) 15% prickly pear	206.3±1.35 <sup>C</sup>	26.12±0.51 <sup>C</sup>	12.80±0.48 <sup>C</sup>
Group (9) 15% mixture of all plant	207.1±3.11 <sup>C</sup>	31.12±0.51 <sup>B</sup>	14.80±0.48 <sup>B</sup>

\*Non significant differences between the values had the same letter. Significant at p≤0.05.

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**2- Effect of feeding different levels of chicory, carrot and prickly pear on total cholesterol and triglyceride levels (mg/dl) of CCl<sub>4</sub> intoxicated rats.**

Data in Table 2 revealed that injection of CCl<sub>4</sub> led to significant (P≤0.05) increase in serum total cholesterol level in hepatotoxic rats. The mean value ± SD of serum cholesterol in hepatotoxic control +ve group was 172.55±12.38 mg/dl as compared to 89.78 ± 5.25 mg/dl in the control - ve group. The mean values of total cholesterol in rats given CCl<sub>4</sub> then fed on all diets of groups 3 , 4 , 5 , 6, 7 , 8 and 9 were significantly lower than positive control group .There is no significant differences in total cholesterol

between groups 3 and 7, as well as between groups 5, 6 and 8. Groups 6 and 8 showed the lowest levels in total cholesterol as compared to all groups. Concerning triglycerides (Table 2), data revealed that rats injected with CCl<sub>4</sub> (control positive group) had higher (P≤0.05) value of serum levels triglycerides compared to normal rats control negative group. There were nonsignificant differences between groups 3 and 7 as well as between groups 5 and 9. Meanwhile, group 8 (rats fed on diet contained 15% prickly pear) showed the lowest level in the mean value of serum triglycerides which showed 54.10 ± 4.72 mg/dl as compared to all treatment and recorded the best result.

**Table (2): Effect of feeding different levels of chicory, carrot and prickly pear on total cholesterol and triglyceride levels (mg/dl) of CCl<sub>4</sub> intoxicated rats.**

Animal Groups	Lipid Fraction	
	Total cholesterol (mg\dl) Mean ± SD	Triglyceride (mg\dl) Mean± SD
Group (1) Control – ve	89.78±5.25 <sup>F</sup>	39.40±0.96 <sup>G</sup>
Group (2) Control + ve	172.55±12.38 <sup>A</sup>	110.70±3.11 <sup>A</sup>
Group (3) 10% chicory	151.29±6.92 <sup>C</sup>	91.30±1.44 <sup>C</sup>
Group (4) 15% chicory	165.78±1.72 <sup>B</sup>	103.40±2.04 <sup>B</sup>
Group (5) 10% carrot	124.12±4.71 <sup>E</sup>	70.00±3.82 <sup>D</sup>
Group (6) 15% carrot	119.46±2.62 <sup>E</sup>	60.10±6.74 <sup>E</sup>
Group (7) 10% prickly pear	154.31±6.15 <sup>C</sup>	96.00±2.85 <sup>C</sup>
Group (8) 15% prickly pear	117.48±9.24 <sup>E</sup>	54.10±4.72 <sup>F</sup>
Group (9) 15% mixture of all plant	137.08 ±1.04 <sup>D</sup>	74.10±0.92 <sup>D</sup>

\*Non significant differences between the values had the same letter. Significant at p≤0.05.

**3- Effect of feeding different levels of chicory, carrot and prickly pear on HDL-c, LDL-c, VLDL-c and the ratio between LDL-c/HDL-c levels (mg/dl) of CCl<sub>4</sub>-intoxicated rats.**

It is obvious that rats injected with CCl<sub>4</sub> (control+ ve) had serum levels HDL-c value 28.38±5.33 mg/dl. In normal rats (control-ve) the mean value of serum levels HDL-c was 60.58±3.62 mg/dl (Table 3). These finding denote that there was a significant decrease in HDL-c in the serum of rats poisoned by CCl<sub>4</sub> as compared to normal rats (Table 3). There were no significant differences among rats given CCl<sub>4</sub> and fed with diet of groups 3, 5, 6, 7 and 9. Finally group 8 (rats fed on diet contained 15% prickly pear) showed the highest increase in serum level of HDL-c and recorded the best treatments. It could be noticed that the data in table 3 evidence that, LDL-c levels was significantly elevated in control positive group to 104.03±8.07 from 20.86±2.74 mg/dl in control negative group. All rats intoxicated with CCl<sub>4</sub> then fed on all

tested plant materials showed significant decrease in LDL-c as compared to control positive group. Group 8 (rats fed on diet contained 15% prickly pear) showed the lowest value of serum LDL-c and recorded the best results as compared to all treatments. Data presented in table 3 also indicated the effect of feeding CCl<sub>4</sub> intoxicated rats with chicory, carrot, and prickly pear on the serum levels of VLDL-c. There were nonsignificant differences between normal rats (negative control group) and rats fed with diet of group 7, 8, 9. Group 8 (rats fed on diet contained 15% prickly pear) showed the lowest decrease in serum level of VLDL-c and recorded the best results as compared to all groups. Regarding rats injected with CCl<sub>4</sub> without treatment (control positive ), the serum LDL-c/HDL-c increase dramatically from 0.34 ± 0.04 for control negative group to 3.67±1.03 for control positive group. Rats fed on basal diet contained 15% prickly pear showed the lowest level in the serum LDL-c/HDL-c and recorded the best results as compared to all treatments.

**Table (3): Effect of feeding different levels of chicory, carrot and prickly pear on HDL-c, LDL-c, VLDL-c and the ratio between LDL-c/ HDL-c levels (mg/dl) of CCl<sub>4</sub>-intoxicated rats.**

Lipid fraction Animal Groups	HDL-c (mg/dl) Mean±SD	LDL-c (mg/dl) Mean± SD	VLDL-c (mg/dl) Mean±SD	LDL-C/HDL-c (mg/dl) Mean±SD
Group (1) Control – ve	60.58±3.62 <sup>A</sup>	20.86±2.74 <sup>H</sup>	7.92±0.19 <sup>E</sup>	0.34±0.04 <sup>G</sup>
Group (2) Control + ve	28.38±5.33 <sup>E</sup>	104.03±8.07 <sup>A</sup>	22.20±0.64 <sup>A</sup>	3.67±1.03 <sup>A</sup>
Group (3) 10% chicory	50.05±4.08 <sup>C</sup>	62.97±8.48 <sup>D</sup>	11.42±0.94 <sup>D</sup>	1.26±0.22 <sup>C</sup>
Group (4) 15% chicory	43.38±4.36 <sup>D</sup>	74.85±6.59 <sup>C</sup>	15.00±0.76 <sup>B</sup>	1.73±0.19 <sup>B</sup>
Group (5) 10% carrot	49.55±1.64 <sup>C</sup>	83.30±3.71 <sup>B</sup>	13.16±0.76 <sup>C</sup>	1.68±0.13 <sup>B</sup>
Group (6) 15% carrot	52.81±7.63 <sup>C</sup>	46.07±6.61 <sup>F</sup>	10.87±0.58 <sup>D</sup>	0.87±0.57 <sup>E</sup>
Group (7) 10% prickly pear	52.03±4.16 <sup>C</sup>	57.88±8.58 <sup>E</sup>	8.86±0.28 <sup>E</sup>	1.11±0.26 <sup>D</sup>
Group (8) 15% prickly pear	56.46±1.94 <sup>B</sup>	35.67±6.61 <sup>G</sup>	7.88±0.58 <sup>E</sup>	0.63±0.52 <sup>F</sup>
Group (9) 15% mixture of all plant	52.46±1.04 <sup>C</sup>	45.67±0.61 <sup>F</sup>	8.08±0.38 <sup>E</sup>	0.86±0.12 <sup>E</sup>

\*Non significant differences between the values had the same letter. Significant at p≤0.05.

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**4- Effect of feeding different levels of chicory, carrot and prickly pear on glucose levels (mg/dl) of CCl<sub>4</sub>intoxicated rats.**

Control positive group had significantly higher mean value  $\pm$ SD of glucose than normal rats, it was being  $140.14 \pm 0.02$  and  $80.05 \pm 2.11$  mg/dl, respectively (Table 4). In rats given CCl<sub>4</sub> then fed on all treatments, there were a significant increases in the glucose levels as compared to normal group. There is no significant difference between groups 3, 6 and 8, as well as between groups 4, 5, 7 and 9. Finally, group 8 (rats fed on diet contained 15% prickly pear) showed the lowest increase in glucose level which were  $111.9 \pm 0.12$  and recorded the best treatment.

**5- Effect of feeding different levels of chicory, carrot and prickly pear on Uric acid and Urea nitrogen levels (mg/dl) of CCl<sub>4</sub> intoxicated rats.**

Results revealed that, treated rats with CCl<sub>4</sub> -intoxicated diet control positive group led to a significant increase ( $P \leq 0.05$ ) in serum uric acid when compared with control negative group. The mean values of uric acid of groups 4, 5, 6, 7, 8 and 9 were significantly lower than positive control group (Table 5). Nonsignificant differences were observed between groups 3 and control positive group. Meanwhile, group 9 (rats fed on diet contained 15% mixture of all plant materials) showed the lowest level in serum uric acid among all treatment and recorded the best results compared to normal group .

For urea nitrogen, there is nonsignificant difference between group 3 and control positive group. Also, group 4 was similar ( $P > 0.05$ ) to normal group. Groups 5, 6, 7, 8 and 9 showed lower ( $P \leq 0.05$ ) urea nitrogen than both control groups. Finally, group 9 (rats fed on basal diet with 15% mixture of all plant materials) showed the lowest level of urea nitrogen among all treatment groups.

**Table (4): Effect of feeding different levels of chicory, carrot and prickly pear on glucose levels (mg/dl) of CCl<sub>4</sub>intoxicated rats.**

<b>Animal Groups</b>	<b>Glucose (mg\dl) Mean<math>\pm</math> SD</b>
Group (1) Control – ve	$80.05 \pm 2.11^D$
Group (2) Control + ve	$140.14 \pm 0.02^A$
Group (3) 10% chicory	$117.14 \pm 0.59^C$
Group (4) 15% chicory	$130.13 \pm 0.21^B$
Group (5) 10% carrot	$130.12 \pm 0.85^B$
Group (6) 15% carrot	$116.11 \pm 0.14^C$
Group (7) 10% prickly pear	$129.11 \pm 0.20^B$
Group (8) 15% prickly pear	$111.9 \pm 0.12^C$
Group (9) 15% mixture of all plant	$125.79 \pm 0.72^B$

\*\*Non significant differences between the values had the same letter. Significant at  $p \leq 0.05$

**Table (5): Effect of feeding different levels of chicory, carrot and prickly pear on Uric acid and Urea nitrogen levels (mg/dl) of CCl<sub>4</sub> intoxicated rats.**

Kidney function	Uric acid (mg\dl) Mean ± SD	Urea Nitrogen (mg\dl) Mean ±SD
Animal Groups		
Group (1) Control – ve	1.05±0.11 <sup>C</sup>	14.7±0.9 <sup>B</sup>
Group (2) Control + ve	1.14±0.12 <sup>A</sup>	15.6±2.2 <sup>A</sup>
Group (3) 10% chicory	1.13±0.11 <sup>A</sup>	15.6±1.4 <sup>A</sup>
Group (4) 15% chicory	1.14±0.09 <sup>A</sup>	14.9±0.6 <sup>B</sup>
Group (5) 10% carrot	1.12±0.05 <sup>B</sup>	13.6±1.9 <sup>C</sup>
Group (6) 15% carrot	1.11±0.15 <sup>B</sup>	14.1±0.8 <sup>C</sup>
Group (7) 10% prickly pear	1.11±0.10 <sup>B</sup>	13.8±0.9 <sup>C</sup>
Group (8) 15% prickly pear	0.9±0.72 <sup>D</sup>	13.1±1.2 <sup>D</sup>
Group (9) 15% mixture of all plant	0.79±0.72 <sup>E</sup>	10.1±1.2 <sup>E</sup>

\*\*Non significant differences between the values had the same letter. Significant at  $p \leq 0.05$

### Discussion:

The reactive electrons species from CCl<sub>4</sub> induces rat liver cirrhosis that resembles the human disease, and it can serve as a suitable animal model for studying human liver cirrhosis (An *et al.*, 2006).

Toxicity experienced by the liver during CCl<sub>4</sub> poisoning results from the production of a metabolite, CCl<sub>4</sub> which is a direct hepatotoxin responsible for change in cell permeability and it inhibits mitochondrial activity followed by cell death (Ambrose *et al.*, 2009). It has also been reported that chronic CCl<sub>4</sub> exposure produced cirrhosis in rats (Chieli and Malvadi, 2008).

An obvious sign of hepatic injury is the leakage of cellular enzyme into plasma (Schmidt *et al.*, 1975). When the liver cell plasma membrane is damaged, a variety of enzymes normally located in the cytosol are released into blood stream. Their estimation in the serum is a useful quantitative marker for the extent and type of hepatocellular damage (Ansari *et al.*, 1991). ALT and AST

are the most often used and most specific indicators of hepatic injury and represent markers of hepatocellular necrosis. These liver enzymes catalyze transfer of alpha-amino group aspartate and alanine to the alpha-ketoglutaric acid. Whereas ALT is primarily localized to the liver, AST is present in a wide variety of tissue, including heart, skeletal, kidney, brain, and liver. AST is present in both the mitochondria and cytosol of hepatocytes, but ALT is found only in the cytosol. In an asymptomatic person with isolated elevation of AST or ALT level, diagnostic clues can be garnered from the degree of elevation (Rosen and Keeffe, 1998).

Results of the current study revealed that administration of CCl<sub>4</sub> caused significant increases in the levels of aspartate aminotransferase, alanine aminotransferase, glucose levels, lipid profile and kidney enzymes and these are in agreement with Túnez *et al.* (2005). On the other hand, the current study demonstrated that the

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treatment with Cactus pear extract caused marked ameliorations of transaminase enzymes activity (ALT and AST). The results are in accordance with Tapiero *et al.* (2002) who showed the effect of carrot extracts on carbon tetrachloride-induced hepatotoxicity in rats.

The mechanism by which the cactus pear fruit induces its hepatoprotective activity is not certain. However, it is possible that  $\beta$ -sitosterol, a constituent of cactus pear, which is at least partly responsible for the protective activity against  $\text{CCl}_4$  hepatotoxicity (Tesoriere *et al.*, 2004). An additional and important factor in the hepatoprotective activity of any drug is the ability of its constituents to inhibit the aromatase activity of cytochrome P-450, thereby favoring liver regeneration. On that basis, it is suggested that flavonoids in cactus pear could be a factor contributing to its hepatoprotective ability through inhibition of cytochrome P-450 aromatase (Kowalska *et al.*, 1990). In addition, the recorded content of vitamin C in the chicory (35 -38 mg per 100 g) may also play a role in hepatoprotection. Previous *in vivo* studies indicate that hepatic microsomal drug metabolism decreases in ascorbic acid deficiency and is augmented when high supplements of the vitamin are given to guinea pigs (Burtis and Ashwood, 2001).

Blood glucose concentration is known to depend on the ability of the liver to absorb or produce glucose. The liver performs its glucostatic function owing to its ability to synthesize or degrade glycogen according to the needs of the organism, as well as via gluconeogenesis (Ahmed *et al.*, 2006). The blood sugar level after overnight fasting in cirrhotic patients is believed to decrease only in severe hepatic failure (Kruszynska and McIntyre, 1991). This is confirmed by our data that indicate that glucose levels in cirrhosis decreased.

### **Conclusions**

The study clearly demonstrates that 15% of tested plants and its mixture have potential for treatment and prevention of  $\text{CCl}_4$  -induced hepatic cytotoxicity. This study, along with other research, targets

cactus pear as a potentially safe and effective fruit that has important medicinal values and benefits.

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## الاستفادة من بعض المواد النباتية في تحسين وظائف الكبد لدى الفئران المصابة بالتسمم الكبدى برابع كلوريد الكربون

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### الملخص العربى

تهدف هذه الدراسة إلى معرفة تأثير الجزر والتين الشوكى والهندباء على الفئران المصابة بالتسمم الكبدى. قسمت الفئران (45 فار ذكور بيضاء) إلى 9 مجموعات بكل مجموعة 5 فئران. تم استخدام مجموعتان ضابطتان غذيت المجموعة الأولى على الوجبة الأساسية فقط كمجموعة ضابطة سالبة والمجموعة الثانية فتغذيت على الوجبة الأساسية بعد حقنها برابع كلوريد الكربون كمجموعة ضابطة موجبة ، أما المجموعات الأخرى فتم حقنها برابع كلوريد الكربون ثم تغذيت على الوجبة الأساسية محتوية على نسبة 10% ، 15% من الجزر والتين الشوكى والهندباء و15% خليط من النباتات المستخدمة . تم تقدير وجود تلف الكبد عن طريق تقدير نشاط وتركيز الإنزيمات فى البلازما وهى انزيم اسبرتات ترانس امينيز والانين امينو ترانسفيريز والكوليستيرول الكلى والتراى جلسريد والليوبروتينات منها الليوبروتين عالى الكثافة والليوبروتين منخفض الكثافة و الليوبروتين منخفض الكثافة جدا وحمض اليوريك واليوريا والجلوكوز. أظهرت النتائج وجود تحسن لهذه القياسات باستخدام الجزر والتين الشوكى والهندباء وكانت أفضل النتائج باستخدام نسبة 15% يليها نسبة 10% من هذه النباتات. لذلك نستخلص من هذه الدراسة ان تلف الكبد بالفئران الناجم عن رابع كلوريد الكربون يمكن تحسنه باستخدام 15% من التين الشوكى يليه الجزر والهندباء.