L-Carnitine a protective natural agent against high loaded fat or frying oil diets in male rats

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ABSTRACT

The effect of oral L-carnitine administration to rats fed high ratio of corn oil, or fried oil diets has been studied for 35 days. Rats were equally divided into 6 groups, control CG, carnitine Car. (300 mg/kg body weight three times per week), high fat HF, HF + Car., frying oil FO, and FO + Car. Liver function activity, lipid profile, antioxidant status, cholinesterase level and liver, testis histograms were investigated. Carnitine alone increased significantly body weight and improved feed efficiency. Carnitine with FO decreased body weight significantly. Carnitine alone, or with HF or FO diet decreased significantly ALT, and AST. Also, research showed an improvement for either carnitine alone or with HF or FO in lipid profile (significant reduce in triglycerides, LDLc and risk factor). Vitamin C and glucose levels showed no and little changes, respectively with carnitine oral treatments. Carnitine alone or in protected groups has highly improvement on antioxidant status and cholinesterase activity as compared to the corresponding groups without carnitine. The research showed the dangerous effects of loaded fat and fried oil on antioxidant content and histograms of liver and testis sections. However, carnitine as natural food supplements ameliorates these bad effects of loaded fat and fried oil.

Key words: *Carnitine, high fat, fried oil diets, liver, testis, rats.*

INTRODUCTION

High-fat diet has been reported to have an adverse affect on human and animal health (Ghosh *et al.,* 2001). Abnormal lipid metabolism is a main cause of dyslipidemia, which is a major risk factor for cardiovascular disease, obesity, cholestasis and overall mortality (Rizvi *et al.,* 2003). High levels of fat increase fat-mediated oxidative stress and decrease antioxidative enzymes activity (Slim *et al.,* 1996). On the other hand, there are various reports indicating the beneficial effects of antioxidant supplementation in preventing dyslipidemia and cardiovascular disease (Mary *et al.,* 2003).

The concern has been raised about the safety ingestion of oxidized frying oil (OFO), since the crisp and aromatic fried foods are popular for consumers worldwide. Clark and Serbia (1991) suggested that heating fat or oil form antinutritional compounds as enzyme inhibitors, vitamin destroyers, lipid oxidized products, free radicals, gastrointestinal irritants and mutagens. Nawar (1997) stated that oil used repeatedly at elevated temperature, caused a wide variety of chemical reactions. These lead to accumulate the decomposition products which affect the fried food quality, harm the human health and nutrition, and accelerate tumor growth (Mori *et al.,* 2001). Besides that, the activities and

levels of mRNAs coding for lipogenic enzymes are reduced (Eder and Kirchgessner, 1998). Moreover, triglycerol (TG) levels in the liver and plasma (Eder *et al.*, 2003), very low-density lipoprotein, and adiposity are being reduced in fried oil-fed rats (Chao *et al.*, 2007).

L-Carnitine is a natural endogenous cofactor for the translocation of long-chain fatty acids from the cytoplasmic compartment into the mitochondria, where β-oxidation enzymes are located. L-Carnitine is synthesized mainly in the kidney and liver and can be obtained exogenously from dietary sources (mainly red meat and dairy products) (Carter *et al.*, 1995). L-Carnitine shows potential protective effects against many mitochondrial toxic agents (Arrigoni-Martelli and Caso, 2001). Also, L-carnitine used recently in anorexia, chronic fatigue, coronary vascular disease, diphtheria, hypoglycemia, male infertility and muscular myopathies (Kelly, 1998).

In a study include healthy men receiving dietary carnitine, plasma free carnitine rose significantly in individuals following a high fat, low-carbohydrate diet. While, no change in carnitine level was observed in men fed on a high-carbohydrate, low-fat diet (Kelly, 1998). Renal excretion of carnitine increased only on the high fat diet. This evidence suggests that high-fat; low-carbohydrate diet might be capable of boosting endogenous synthesis of carnitine and its metabolites (Cederblad, 1987). Maccari *et al.* (1987) found that oral carnitine administration significantly decreased triglycerides, cholesterol, phosph-olipids levels and very low density lipoproteins in the blood through promotion of β-oxidation. While, low density lipoprotein levels were not affected and high density lipoproteins were found to be decreased by 20%. Carnitine decreased in correlation with plasma free fatty acid levels.

Due to the importance of L-carnitine in fatty acid transfers and body energy improve-ement, this research work concentrates on the role of L-carnitine in relieving the risky effects produced from the dietary high fat content and the oxidized oil or reused-frying oil in food.

MATERIALS AND METHODS

Animals and Experimental design

Thirty six male Sprague-Dawley rats were purchased from Agricultural Research Center, Giza, Egypt on the summer of 2008. Upon rat arrival, they were aged approximately 8 – 9 weeks. They were housed individually in special healthy standard cages and divided equally into six groups. They were given two weeks acclimation period, during which they were fed a standard rat chow diet *ad libitum* which contains 17% protein, with alternated 12-h dark/light cycle. The ambient temperature was held constant between 20-25°C. The six rats per treatment with mean weight 125±5g were randomly assigned as the following;

Group (1) control group (CG) was fed a basal diet contained 20% casein, 5% cellulose, 5% salt and vitamins mixture, 5% corn oil and 65% starch (Compbell, 1961). Food intake was recorded weekly, after giving each rat calculated 20 g daily.

Group (2) L-Carnitine group (Car.) was fed the same basal diet for control group, with oral administration of carnitine (L-carnitine, Sigma, St. Louis, MO, USA) in saline solution (300 mg/kg body weight) three days per week.

Group (3) high fat group (HF) rats were fed modified diet containing 20% casein, 5% cellulose, 5% salt and vitamins mixture, 20% corn oil, 50% starch (Cha *et al.*, 1999). This group was considered as control for group 4.

Group (4) high fat with L-carnitine group (HF+Car.) rats were fed the modified diet of group (3) and orally administrated with L-

carnitine (300 mg/kg body weight three days per week).

Group (5) frying oil group (FO) rats were fed the basal diet in which the corn oil was substituted with hard frying corn oil which previously boiled many times until getting dark colour. This group as well was considered as a control for group 6. Corn oil obtained from Minia Local Market was boiled several hours at 180°C/24 h until the spoiled dark colour appeared.

Group (6) frying oil with L-carnitine group (FO+Car.) rats were fed the diet of group (5) and orally administrated with L-carnitine (300 mg/kg body weight three times per week).

The animals were sacrificed at the end of the biological experiment (35 days), the blood was collected from the orbital plexus under ether anesthesia. Blood was allowed to clot and then centrifuged at 3000 rpm for 15 min, and serum kept at -20°C until required. Food consumption was monitored daily, and body weight was determined once a week.

Determination of biochemical para-meters

Triglycerides TG, Cholesterol CHL and HDL cholesterol were colorimetrically determined in rat serum using the enzymatic colorimetric methods (Fassati and Prencipe, 1982; Richmond, 1973, and Lopes-Virella *et al.*, 1977, respectively). LDL cholesterol was calculated (Friedewald *et al.* (1972) (mg/dl) as follows:

LDLc = *Total CHL – HDLc – (TG/5)*

Activity of butyrylcholinesterase BChE was calculated every 30 sec in serum at 405 nm to follow the inhibition of the enzyme (Unit/l) (Knedel and Bottger, 1967). Antioxidant determinations; total antioxidant AO content measured as (µmol/ml) (Koracevic *et al.*, 2001), and vitamin C (mg/l) (Harris and Ray, 1935) were examined for rat serum. Reduced glutathione (GSH) was measured colorimetrically in fresh heparinized blood (Beutler *et al.*, 1963). Liver function as aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were measured with colorimetric method (Reitman and Frankel, 1957). Glucose was measured enzymatically and colorimetrically in serum immediately (Trinder, 1969).

Histological examination

Autopsy samples were taken from the rats in different experimental groups. Then, samples were fixed in 10% formol saline solution for twenty four hours. Washing was done in tap water then serial dilutions of absolute ethyl alcohol were used for dehydration. Specimens were cleared in xylene and embedded in paraffin at 56°C in a hot air oven for twenty four hours. Paraffin bees wax tissue blocks were prepared for sectioning at 4 microns thickness by slidge microtome. The obtained tissue sections were collected on glass slides, deparaffinized and stained by hematoxylin and eosin stain for histopathological examination through the light microscope (Banchroft *et al.*, 1996). Histopathological examinations have been done and explained by Prof. Dr. A. Khlosy, Pathology Dept., and Cairo Univ.

Statistical analysis

Means of results were calculated among 6 replicates, with their standard errors (SE) for each group. Analysis of variance was used to make statistical comparisons (ANOVA) with Dunnett's post hoc test. SPSS computer program (SPSS, 1990) was used to calculate the significance between groups at the same experiment at 1% and 5% probabilities.

RESULTS AND DISCUSSION

Rats treated with carnitine alone showed the highest body weight gain and the best feed efficiency ratio (Table 1). Control group showed a significant decrease in the body weight gain comparing to the group fed carnitine alone.

Table (1): Effect of L-carnitine on performance of rats fed high-fat or deep-frying corn oil diets.

Group	Body weight gain (g) Mean ± SE	Daily body weight gain (g) Mean ± SE	Daily feed intake (g) Mean	Feed efficiency Ratio Mean
CG	35^{b}±0.93	1.0^{b}±0.93	15.09	0.0663
Car.	49^{d}±1.10	1.4^{d}±1.10	13.60	0.1029
HF	38.4^{c}±1.06	1.097^{c}±1.06	14.17	0.0774
HF + Car.	36^{bc}±1.10	1.028^{bc}±1.10	11.94	0.0861
FO	36^{bc}±1.10	1.028^{bc}±1.10	16.07	0.0640
FO + Car.	30^{a}±0.93	0.857^{a}±0.94	16.08	0.0533
F	0.66	0.66		

Each value represents mean of 6 replicants ±SE.
The mean values with different letters within a column indicate significant difference at P<0.05.

Table (2): Effect of L-carnitine on liver, kidnies, heart, spleen, testis weight and their ratio of rats fed high-fat or deep-frying corn oil diets.

Group	Liver wt		Kidney wt		Heart wt		Spleen wt		Testis wt	
	(g)	%	(g)	%	(g)	%	(g)	%	(g)	%
CG	3.83^{abc}±0.24	2.54	1.02^{a}±0.08	0.65	0.49^{a}±0.04	0.32	0.51^{a}±0.05	0.37	1.53^{a}±0.41	1.04
Car.	3.22^{a}±0.24	2.11	1.2^{ab}±0.08	0.77	0.57^{a}±0.08	0.37	0.55^{ab}±0.05	0.38	2.07^{ab}±0.02	1.33
HF	4.67^{c}±0.29	2.78	1.30^{b}±0.05	0.79	0.59^{a}±0.05	0.38	0.64^{ab}±0.04	0.39	2.27^{ab}±0.12	1.37
HF + Car.	4.17^{bc}±0.24	2.66	1.15^{ab}±0.09	0.73	0.56^{a}±0.08	0.36	0.53^{a}±0.05	0.37	2.12^{ab}±0.16	1.34
FO	4.70^{c}±0.32	2.86	1.28^{b}±0.06	0.78	0.61^{a}±0.05	0.39	0.77^{b}±0.16	0.46	2.41^{b}±0.24	1.47
FO + Car.	4.59^{c}±0.16	2.79	1.22^{ab}±0.09	0.77	0.59^{a}±0.05	0.38	0.67^{ab}±0.08	0.42	2.25^{ab}±0.20	1.35
F	3.9^{**}		1.6		1.4		2.1		1.3	

Each value represents mean of 6 replicants ±SE. The mean values with different letters within a column indicate significant difference at P<0.01 % is a percentage of organs to rat body weight.

Carnitine administration decreased significantly body weight in frying oil containing diet comparing to similar group without carnitine treatment. Feed efficiency was increased with the high fat diet, and decreased with frying oil without carnitine compared with normal control. In agreement, Galal *et al.* (1992) evaluated the fried oil used for potatoes frying, and found a decrease in the feed efficiency by 60%. That might due to both of diet digestibility and absorption. Korkina *et al.* (1989) reported that carnitine accelerated body weight gain and disappeared latent fatigue with increasing mental performance. L-Carnitine decreased significantly body weight gain when added to groups fed high fat or frying oil (Table 1). These results are in agreement with that of Ghoniem (2007), who found that food conversion showed a significant body weight improve in group fed on antioxidant diets compared with frying oil group.

L-Carnitine alone in the diet enhanced body organ weights: kidney, testis and spleen. The increase was insignificant for heart

weight, but there was a significant decrease in liver weight comparing with normal control group. Liver and kidney weights were significantly increased in group fed on 5% frying oil comparing to fresh oil (Table 2). These results agreed with the study of Izaki *et al.* (1984). They studied the peroxidative effect of 15% thermal oxidized rapeseed oil for 13 weeks in comparison with rats fed fresh oil. They reported that liver and kidney were adversely affected in proportion to the degree of oil deterioration.

L-carnitine has a significant decrease in liver function enzymes activity comparing to control, high fat or frying oil groups (Table 3). The marked increase in liver enzymes AST (GOT) and ALT (GPT) may be revealed to the cellular damage. Hyperlipidemia as well might lead to liver tissue injury and enzyme disorders. When cell membrane gets damage, enzymes located in the cytosol leak in the blood stream which affect liver and other tissues. AST (GOT) has a significant increase in high fat diet group comparing to control, which was in agreement with the data of Shyamala *et al.* (2003). Galal *et al.* (1992) pointed to the effect of using oil many times on all blood parameters except for GOT/GPT ratio that caused a significant decrease in liver malfunction. On the other side, data obtained herein show increasing GOT/GPT ratio from 1.49 for control and 2.11 for frying oil groups.

Table (3): Effect of L-carnitine on ALT and AST activity in rats fed high-fat or deep-frying corn oil diets.

Group	(GPT) ALT U/l	(GOT) AST U/l	GOT/GPT ratio
CG	$32.72^{gh}\pm1.96$	$82^{cd}\pm1.79$	2.506
Car.	$28.37^{ef}\pm1.42$	$55.31^{a}\pm1.18$	1.949
HF	$37.54^{h}\pm3.47$	$89.43^{d}\pm1.18$	2.332
HF + Car.	$24.8^{abc}\pm3.14$	$55.4^{a}\pm1.83$	2.233
FO	$30.4^{fg}\pm1.92$	$78.8^{c}\pm1.75$	2.592
FO + Car.	$25.35^{def}\pm1.18$	$69.57^{b}\pm1.79$	2.749
F	10.4^{**}	6.5^{**}	

Each value represents mean of 6 replicants ±SE.
The mean values with different letters within a column indicate significant difference at $P<0.01$

Effective action for L-carnitine is expected to arise in the high fat diet group (Table 4), since it is exhausted in lipid metabolism to avoid the accumulation of lipid in liver or blood. In the same time, carnitine as a food supplement and antioxidant can produce effective results with the fried oil. Administration of L-carnitine caused a significant reduce in accumulation of triglycerides and LDLc in groups II, IV and VI, where HDLc was increased in the same groups comparing with control groups (I, III and V) (Table 4). Maccari *et al.* (1987) found that oral carnitine administration significantly decreases triglycerides, cholesterol, phosphorlipid levels and very low-density lipoproteins in the blood through promotion of β-oxidation. Results obtained herein showed complete agreement in carnitine treatment (Table 4). While, Maccari *et al.* (1987) showed that low density lipoprotein level was not affected and high density lipoproteins were found to decrease by 20%. Carnitine also decreased plasma free fatty acid levels as well. Risk factor LDLc/HDLc has been decreased with using carnitine for control group of rats, rats fed high-fat or frying oil diets.

Table (4): Effect of L-carnitine on lipid profile TG, CHL, LDLc ,vLDLc, HDLc and risk value LDLc/HDLc in rats fed high-fat or deep-frying corn oil diets.

Group	TG mg/dl	CHL mg/dl	LDLc mg/dl	vLDLc mg/dl	HDLc mg/dl	LDLc/HDLc ratio
CG	116.17^{ef}±0.8	180.5^{abc}±6.5	24.63^{ab}±2.6	23.23	132.64	0.18
Car.	38.39^{a}±1.6	172.5^{ab}±0.4	23.61^{ab}±0.7	7.68	141.21	0.17
HF	65.8^{bc}±0.8	245.8^{cd}±6.9	46.28^{d}±4.9	13.16	186.36	0.25
HF+Car.	52.7^{ab}±4.8	234.2^{bcd}±4.5	16.39^{a}±0.4	10.54	207.27	0.08
FO	125^{f}±5.7	267^{d}±3.2	29.4^{b}±2.9	25	212.6	0.14
FO+Car.	96.2^{de}±5.7	251.4^{cd}±4.1	16.8^{a}±2.5	19.24	215.36	0.08
F	13.5^{**}	3.3^{**}	8.3^{**}			

Each value represents mean of 6 replicants ±SE.
The mean values with different letters within a column indicate significant difference at P<0.01.

Several investigators reported a beneficial impact of L-carnitine administration on plasma glucose and insulin levels following intravenous infusion of glucose. Carnitine administration in the present results decreased significantly the glucose values when compared with control groups (Table 5). Negro *et al.* (1994) observed that addition of both 2 g and 4 g of L-carnitine to 500 ml solutions of 5% and 10% glucose reduced the increase in plasma glucose levels. Grandi *et al.* (1997) reported a similar improvement in glucose metabolism following the addition of 2 g of L-carnitine to a 5% glucose solution. Whether these observations would translate to a beneficial clinical effect in individuals with a tendency to reactive blood sugar and that is not currently known. However, due to the safety of L-carnitine and its tendency to improve fatigue (a common concomitant symptom of individuals with reactive blood sugar), a clinical trial with L-carnitine seems warranted.

Vitamin C level was enhanced insignificantly with using carnitine in the normal rats and oxidative stress group or the frying oil (Table 5). While using carnitine, didn't enhance vitamin C content for group fed high lipid. Enhanced ascorbic acid status may be attributed to L-carnitine acting as a chelator (Rauchova *et al.,* 1998) and hence decreases the amount of iron available to induce oxidative damage. Since ascorbic acid is one of the cofactors in carnitine biosynthesis, supplementation of L-carnitine spares ascorbic acid and thereby elevates its level (Kalaiselvi and Panneerselvam, 1998).

Table (5): Effect of L-carnitine on serum antioxidant capacity, vitamin C amount and glucose level in rats fed high-fat or deep-frying corn oil diets.

Groups	Vitamin C mg/l	Glucose mg/dl
CG	116.6^{b}±0.48	121.5^{b}±3.06
Car.	121^{b}±2.29	109.7^{a}±1.67
HF	120.3^{b}±1.83	156.2^{d}±1.84
HF + Car.	120.3^{b}±1.96	144.8^{c}±2.85
FO	96.3^{a}±1.92	157.2^{d}±1.63
FO + Car.	101^{a}±3.47	129.2^{b}±1.22
F	15.3^{**}	6.7^{**}

Each value represents mean of 6 replicants ±SE.
The mean values with different letters within a column indicate significant difference at P<0.01

Total antioxidant levels (Table 6) showed a significant increase when carnitine was administrated in control or in high fat or frying oil groups. Ghoniem (2007) as well showed significant increases in malondialdehyde (MDA) and decreases in Glutathione (GSH) and superoxide dismutase (SOD) levels in rats fed frying oil. In agreement, data obtained herein show mostly a significant decrease in AO and GSH in rats fed high fat diet or frying oil compared to control (Table 6). In agreement, Rani and Panneerselvam (2001) suggested that L-carnitine acts as free radical scavengers, protecting cells from reactive oxygen species (ROS) and preventing hypercholesterolaemia in rabbits (Sayed *et al.*, 2001). Combination of both L-carnitine and N-acetyl cysteine (NAC) gives the most significant elevation in GSH level. The energy enhancing action of L-carnitine may be responsible for the increase in GSH status (Kumaran *et al.*, 2003 and Ramadan, 2007). Reduced glutathione in the oxidation reduction cycle is catalyzed by glutathione peroxidase. Glutathione (GSH) is a reducing agent for H_2O_2, and breaks the chain reaction forming highly reactive hydroxyl radical from superoxides. Therefore, GSH acts as a natural scavenger for superoxide anion to protect protein thiol groups against oxidation and maintain cellular integrity. GSH reactivates free radical scavengers and antioxidant vitamins to their reduced state (Stein *et al.*, 1990). Acetylcholine helps for carrying messages between nerve cells in the brain. Increase of cholinesterase (ChE) enzyme activity indicates the degraded effect on the brain function. L-carnitine is a naturally occurring compound widely distributed in the body. Carnitine availability as shown (Table 6) decreases the enzyme activity, since choline supplementation decreases carnitine synthesis (Daily and Sachan, 1995). Rani and Panneerselvam (2001) proved that carnitine plays an important role in the translocation of acetyl moieties from the mitochondria into the cytoplasm for acetylcholine synthesis in the brain. Other studies have shown that L-carnitine suppresses oxidative damage during aging.

Table (6): Effect of L-carnitine on serum reduced glutathione and cholinesterase activity in rats fed high-fat or deep-frying corn oil diets.

Group	AO (mM l)	GSH(mg dl)	Cholinesterase(IU l)
CG	0.19^{a}±0.01	7.9^{a}±0.81	60.05^{a}±1.55
Car.	0.27^{b}±0.008	8.7^{a}±0.65	38.6^{c}±.41
HF	0.05^{d}±0.008	4.7^{b}±0.12	94.9^{b}±1.14
HF + Car.	0.17^{c}±0.01	5.7^{b}±0.61	58.6^{c}±3.06
FO	0.05^{d}±0.004	1.2^{c}±0.20	63.9^{c}±1.96
FO + Car.	0.2^{b}±0.008	6.4^{b}±0.41	40.2^{c}±0.65
F	9.5	15.7	14.2

Each value represents mean of 6 replicants ±SE.
The mean values with different letters within a column indicate significant difference at P< 0.01

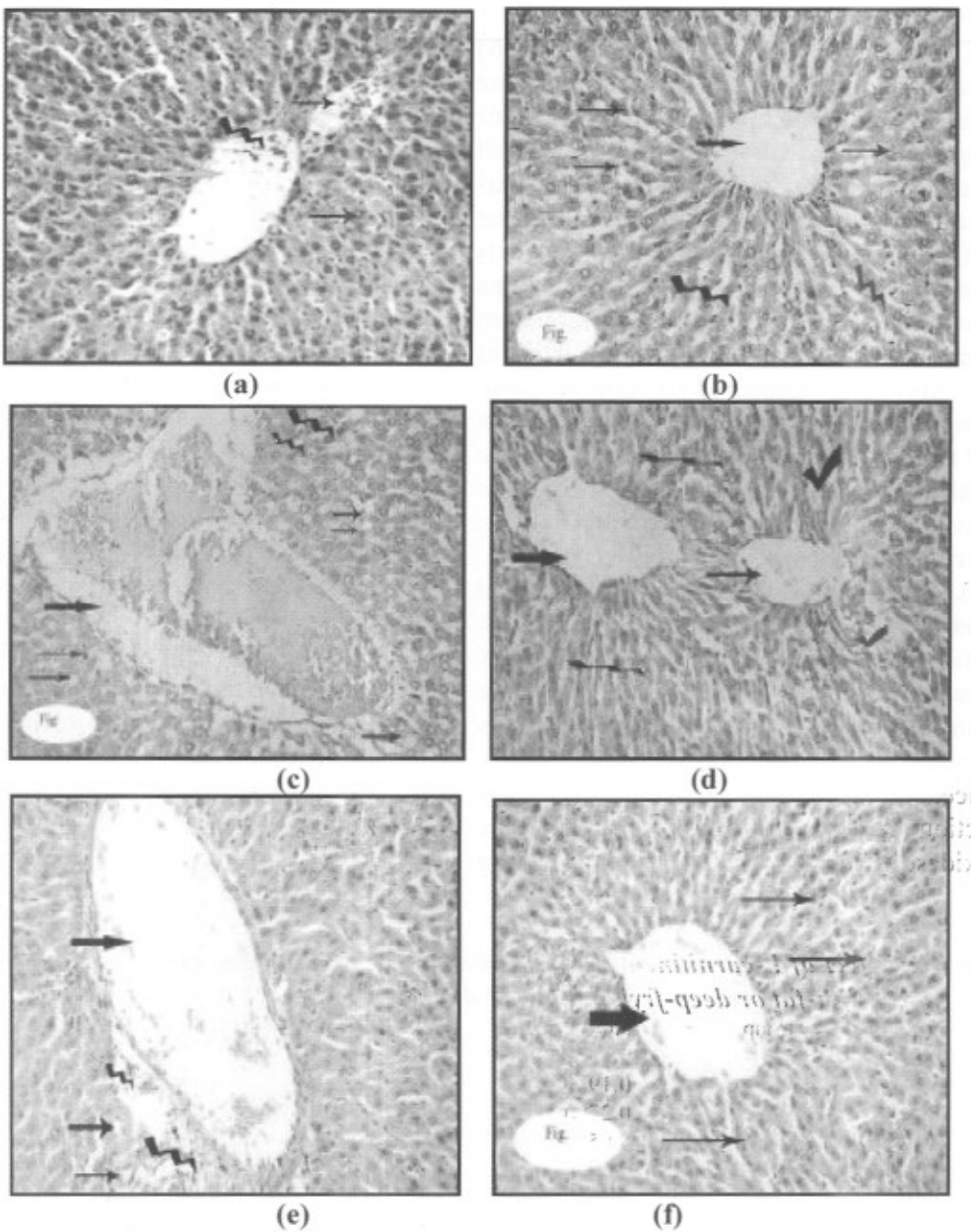

Fig. (1): A photomicrograph of the liver sections of the control rat (a), L-carnitine (b), high fat diet (c), high fat diet with carnitine group (d), frying oil group (e), and frying oil with carnitine group (f) (H&E, x 100).
Thin arrows; red blood hepatocytes or vaculation – thick arrows; congested central vein – zigzag arrows; congested blood.

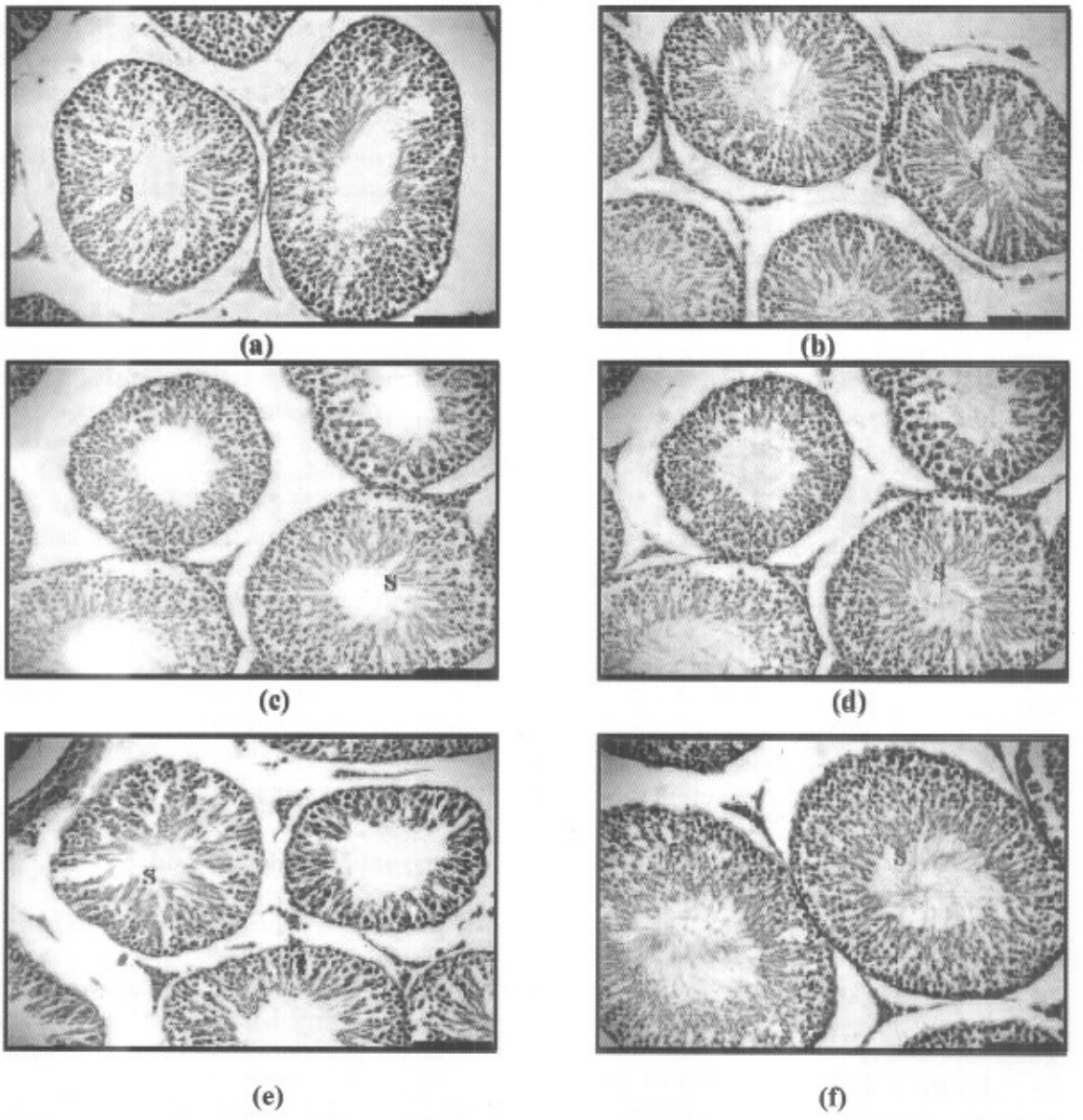

Fig. (2): A photomicrograph of the testis sections for control (a), L-carnitine (b), high fat diet (c), high fat diet with carnitine group (d), frying oil group (e), and frying oil with carnitine (f) groups (H&E, x 160)(S; Spermatogenic series).

Histopathological examinations

Histopathological examination for liver and testis of the different groups were compared with each others. Figure (1a) showed the normal liver section properties as hepatocytes (thin arrows), central vein (thick arrows), with blood sinusoid with congested blood in the central vein (zigzag arrows). L-carnitine group (Figure 1b) showed hepatocytes with homogenous cytoplasm. The group fed high fat diet (Figure 1c) showed congested blood sinusoid (thick arrow) with little number of Kupffer cells and red blood cells appeared in the liver sinusoids (thin arrows) and vaculation (zigzag arrow). Treating the high fat diet group with L-carnitine (Figure 1d) showed hepatocytes with homogenous cytoplasm in the congested central vein (thick arrow), and appearance of blood sinusoids (thin arrows) and Kupffer cell. In the same side, feeding rats with deep-frying oil (figure 1e) showed congested central vein (thick arrow), vaculation (thin arrow) and Kupffer cell (zigzag arrow). Again, the treatment of L-carnitine (Figure 1f) showed homogenicity of cytoplasm in the hepatocytes of central vein (thick arrow). El Zawahry *et al.* (1992) reported the liver histopathological effects of feeding adult male rats on 15% sunflower seed oil for 10 weeks. They found irreversible changes as necrosis (cell nuclei destruction), portal tract fibrosis, vascular congestion and hemorrhagic zones.

Figure 2 showed the effect of carnitine on male fertility or sperm counting, where increasing in spermatocytes was noticed by carnitine treatments. As shown in Figure (2b), spermatocytes or spermatogenic series (S) were increased, comparing to free carnitine group (Figure 2a). The same effect has been noticed by using carnitine with high fat diet (Figure 2d) comparing to Figure 35c. As well, using carnitine with diet containing frying oil (Figure 2f) enhanced the spermatocytes status (S) comparing to group feed with frying oil in diet (Figure 2e). In agreement, carnitine proved to be significantly more active than testosterone in improving nocturnal penile tumescence and International Index of Erectile Function score. It proved to be active drug of symptoms associated with male aging (Cavallini *et al.*, 2004).

Biochemical and histopathological investigations (in the biological experiment) explained the importance of using L-carnitine to defend against the dangerous effects produced from the habites of increasing high fat ratio or using oxidized frying oil in diet. The different biological investigations showed obvious improvements by using carnitine as protective natural subject or even carnitine alone. It can be recommended to use carnitine for its properties as natural antioxidant agent for curing many diseases as liver fibrosis, and moreover as mentioned in other studies for enhancing male fertality.

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الملخص العربي

ل – كارنيتين المادة الطبيعية الواقية ضد تغذية ذكور الجرذان على علائق مرتفعة فى نسبة الدهن او محتوية على زيوت مغلية

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اجريت الدراسة الحالية لتقييم تأثير استخدام ل-كارنيتين علي الجرذان المغذاة علي عليقة مرتفعة في نسبة زيت الذرة او تلك المغذاة علي زيت مغلي و استخدم فيها 36 من ذكور الجرذان لمدة 35 يوماً. قسمت الجرذان الي 6 مجموعات : مجموعة ضابطة عادية، مجموعة الكارنيتين، مجموعة تغذت علي دهن مرتفع، مجموعة تغذت علي دهن مرتفع+الكارنيتين، مجموعة تغذت علي زيت مغلي، و مجموعة تغذت علي زيت مغلي + الكارنيتين. تم تقدير كل من وظائف الكبد, محتوي الدهون, حالة مضادات الأكسدة, مستوي الكولين استيريز و كذلك الفحص الهستولوجي لانسجة الكبد و الخصية. و كان من اهم النتائج المتحصل عليها: الكارنتين بمفرده يزيد معنوياً من وزن الجسم ويحسن من الكفاءة الغذائية له. بينما الكارنيتين مع الزيت المغلي قلل بشكل معنوي من وزن الجسم. كذلك الكارنيتين بمفرده أو مع الدهن المرتفع أو الزيت المغلي قلل بشكل معنوي من نشاط انزيمات الكبد الناقلة لمجموعة الأمين. أيضاً اوضحت الدراسة تحسناً لكل مجموعات الكارنتين سواءً بمفرده او مع الدهن المرتفع او الزين المغلي علي محتوي الدهون من انخفاض معنوي في تركيز الدهون الثلاثية، الليبوبروتين منخفض الكثافة (الضار) و معامل الخطورة (نسبة الليبو بروتين مرتفع الكثافة الي الليبوبروتين منخفض الكثافة). لم يظهر تغيير فى تركيز فيتامين ج بينما كان التغيير طفيفاً لمستوي الجلوكوزمع استخدام الكارنيتين. أظهرت مجموعات الكارنيتين الثلاثة زيادة معنوية في مستوى مضادات الأكسدة و نشاط الكولين استيريز مقارنة بالمجموعات الثلاثة الأخرى التي لم يستخدم فيها الكارنيتين. أوضحت الدراسة الخطر الحقيقي من استخدام غذاء مرتفع في نسبة الدهن أو به زيت سبق غليه علي محتوي مضادات الأكسدة و الفحص الهستولوجي لانسجة الكبد و الخصية. في النهاية فان الكارنيتين كمادة طبيعية مضافة للغذاء يمكن ان تحسن من هذه التأثيرات الضارة للدهن.