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L-CARNITINE, A NEW PROSPECTIVE PROMOTE RECOVERY FROM ACRYLAMIDE NEUROTOXICITY IN RATS

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ABSTRACT

L-carnitine (4-*N*-trimethylammonium-3-hydroxybutyric acid) proved to be an important factor in human nutrition. It synthesizes from dietary amino acids and widely distributed in the body. Research works on the ability of carnitine in protecting biological systems against acrylamide. Acrylamide (AA) C_3H_5NO the chemical with many industrial and laboratory uses is a neurotoxicant and carcinogen agent as proved in literatures. Research has pointed to L-carnitine role in recovering AA toxicity. Three days per week, L-carnitine (300 mg/kg b.w.) was administrated orally followed on the second day by treatment with 50, 200 or 500 mg acrylamide/L in drinking water. Up to 5 weeks, L-carnitine improved feed efficiency with decreasing daily body weight gain with 50 mg AA/L. L-carnitine showed approximate success in keeping liver and testis weight comparing to normal group. L-carnitine showed amelioration in vitamin C and serum glucose in 50 mg/L AA. L-carnitine ameliorated lipid profile and risk factor LDL/HDL ratio especially with 50 mg/L AA. On the other side, AA concentrations showed highly effects on liver functions, total antioxidant, reduced glutathione and cholinesterase activity to the point where L-carnitine failed to face.

INTRODUCTION

In the recent years, scientists have discovered substances promote recovery from acrylamide neurotoxicity and its analogues of these compounds 4-Methylcatechol (Saita *et al.*, 1996) and Phenobarbital (Hashimoto *et al.*, 1981). Carnitine, the prospective nutrient supplementation, has been used to improve athletic performance in several studies (Vukovich *et al.,* 1994, Arenas *et al.,* 1991 and Veechiet *et al.,* 1990). Carnitine is a vital component in lipid metabolism through restoring high energy in *β*–oxidation and subsequent oxidative phosphorylation (Packer *et al.,* 1991). Moreover, it is essential for translocation of long-chain fatty acids into mitochondrial matrix for the shuttling of acyl groups out of the mitochondria (Bremer, 1997). Not many studies have been focus on the variable benefits of carnitine.

Acrylamide (CH_2=$CHCONH_2$) the small, water soluble, organic molecule a vinyl monomer is formed from the hydration of acrylonitrile. Acrylamide (AA) the organic solid of white, odorless, flake-like crystals is used in the production of polyacrylamide. Then polyacrylamide is used in wastewater treatment, pulp, paper production, and mineral processing. It is also used in the synthesis of paper, cosmetics, toiletry industry, dyes, adhesives, contact lenses, grouts, soil conditioners, laboratory gels and permanent press fabrics. In the environment, it has a high mobility in soil and in ground water. It can be present in a variety of food cooked at high temperature. Dybing and Sanner (2003) stated that the daily mean intake of acrylamide in some foods and coffee have been estimated in advanced

countries to be 0.49 and 0.46 g/kg body weight in males and females, respectively. Bread is the most important contributor of background acrylamide-intake for the lower percentages. From the 21st percentage French fries are the main source of acrylamide exposure. From the 55th percentage on, biscuits are the second important source of acrylamide intake. Stadler *et al.* (2002) explained that heated certain amino acids and sugars beyond 120°C can form acrylamide. Processing of food rich in starch and protein is the main source of acrylamide. Specially, glucose and the amino acids asparagines, glutamine, methionine, cysteine when heated above 120°C (Mottram *et al.,* 2002 & Stadler *et al.,* 2002).

Smith and Oehme (1991) reported that single exposure of acrylamide is toxic or harmful by all routes of administration. The effects of acrylamide prior to death related to neurotoxicity, severe effects on spermatid development, eye irritation and skin peeling. European Union (E.U.) risk Assessment Report indicates that acrylamide is carcinogenic in animals producing increased incidences in a number of benign and malignant tumours in thyroid, adrenals and testicular mesothelioma (E.U. Risk Assessment Report on Acrylamide). Inducing tumours in brain and spinal cord might due to a possible relationship with disturbed endocrine function and raise the possibility of a hormonal mechanism. International Agency for Research on Cancer (I.A.R.C., 1994) reports that acrylamide induces gene lethal mutations in mice and rats. The important findings have evoked wide attention throughout for the potential carcinogensis (Rice, 2005) and neurotoxicity (Tilson, 1981) of acrylamide. Therefore and for many aspects, our research investigates the role of L-carnitine as a modern exogenous antioxidant and neuroprotective activity in reduction or decreasing the toxicity of different concentrations of acrlyamide.

MATERIALS AND METHODS

Animals and Experimental design

Acrylamide was purchased from Aldrich Company Germany. Thirty male Sprague-Dawley rats were purchased from Agricultural Research Center, Giza, Egypt. Upon arrival, the animals were given two weeks acclimation period, during which they were fed a standard rat chow diet *ad libitum*, with alternated 12-h dark/light cycle, and the ambient temperature was held constant between 21-25°C. After two weeks of accumulation the rats were divided randomly into five groups assigned up to the following treatments;

Group (I): Control group was fed 20% casein, 5% cellulose, 5% salt and vitamins mixture, 5% corn oil, 65% starch as basal diet (Compbell, 1961).

Group (II) L-carnitine group (L-carnitine, Sigma, St. Louis, MO, USA) was fed the same basal diet for control group, with oral administration with l-carnitine in saline (300 mg/kg b.w.) for three days per week.

Group (III): Rats treated with L-carnitine as group II but followed by acrylamide solution (50 mg/L) as drinking solution for three days per week.

Group (IV): Rats treated with L-carnitine as group II but followed by acrylamide solution (200mg/L) as drinking solution for three days per week.

Group (V): Rats treated with L-carnitine as group II but followed by acrylamide solution (500mg/L) as drinking solution for three days per week.

The amounts of food consumption and acrylamide AA consumed were measured daily and body weight as well was determined once a week. The biological experiment was lasted for 5 weeks. The animals were sacrificed and the blood was collected at the end of the biological experiment from the orbital plexus.

Determination of some biological parameters:

Activity of cholinesterase (ChE) was calculated every 30 sec in serum at 405 nm to follow the inhibition of the enzyme (Unit/l) (Knedel and Bottger, 1967). Reduced glutathione was determined according to the method of Beutler *et al.* (1963); total antioxidant content measured as (µmol/ml) (Koracevic *et al.,* 2001), and vitamin C (mg/l) (Harris and Ray, 1935) were examined for rat serum. Liver function as Aspartate aminotransferase, (AST) and Alanine aminotransferase, (ALT) were measured by colorimetric method (Reitman and Frankel, 1957). Glucose was measured enzymatically and colorimetrically in serum immediately (Trinder, 1969).

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

LDL-c = *Total CHL – HDL-c – (TG/5)*

Histopathological examination

Samples were taken from the rats in different 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., Cairo University.

Statistical analysis

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

RESULTS AND DISCUSSION

To humans, acrylonitrile may cause headache, nausea and dizziness (OSHA, 1978) at relatively low levels between 20 – 150 ppm for short periods, and lethal at 500 ppm for several minutes. While, Sakurai (2000) stated that long term bioassay of rats exposed through inhalation or drinking water produced cancer at several sites with tumors of the central nervous system. Research investigated some paralyzed rat cases in all the acrylamide concentrations. In agreement, Mapp *et al.* (1977) found that AA causes muscular weakness and paraesthesia with difficulties in walking and standing. Research used L-carnitine 300 mg/kg b.w. according to previous study Dayanandan *et al.,* (2000) indicated the protective role of L-carnitine on liver and heart lipid peroxidation in atherosclerotic rats.

Carnitine which is biosynthesized in liver and kidney from lysine or methionine, increased the body weight gain and food efficiency comparing to control group (Table 1). On the other hand, Sachan and Hongu (2000) stated that carnitine and choline supplementation decreased body fat and increased fat utilization for energy. With decreasing daily feed intake for the groups treated with acrylamide, the food efficiency has been increased comparing to control and carnitine but not in the same sequence of increasing acrylamide concentration. Carnitine has a significant role in increasing the food efficiency for groups treated with both of carnitine and acrylamide. Tyl *et al.* (2000) proved that 15 mg/kg/day or greater doses reduced body weight, food consumption and showed leg splay as neurotoxic agent. In agreement with Friedman (2003) and Nail (2010) acrylamide shows body weight loss, decrease in food consumption (Gipon *et al.*, 1977) and signs of neurotoxicity as mental confusion.

Table (1): Mean of body weight gain, daily weight gain, daily feed intake and food efficiency ratio for rat.

Groups	Body weight gain (g)	Daily body weight gain (g)	Daily feed intake (g)	Food efficiency (%)
GroupI (Control)	35^{b}±2.3	1.0^{b}±2.3	15.09	6.63
GroupII (Car. Treated)	49^{c}±2.7	1.4^{c}±2.7	13.6	10.29
GroupIII (50 mg/L AA + carn.)	31.5^{ab}±2.4	0.9^{ab}±2.4	12.03	7.48
GroupIV (200 mg/L AA + carn.)	30^{ab}±3	0.857^{ab}±3	16.18	5.29
Group V (500 mg/L AA + carn.)	28.5^{ab}±2.1	0.814^{ab}±2.1	9.99	8.15
F	0.66	0.66		

Table (2) shows the acrylamide mean volume and weight consumed by one rat through the day of treatment. The average daily human intake from acrylamide AA was 1 µg/kg b.w./day and for high consumers it amounted to 4 µg/kg b.w./day (Parzefall, 2008). Exposure to acrylamide was found as adduct to the *N*-terminus of hemoglobin (Hb) to form *N*-(2-carbamoylethyl) valine as main adduct (Bergmark, 1997). In general speaking, the single exposure of acrylamide is toxic or harmful by all routes of administration (Smith and Oehme, 1991).

Table (2): Mean acrylamide AA consumed during certain days as mentioned in the experiment in biological method. * Mean AA daily consumed by rat only in three days a week treated with acrylamide through 5 weeks.

Groups treated with AA in drinking water	AA solution concentration (mg/l)	AA Daily gain (ml)	Mean* AA daily consumed (mg)
AA 50 mg/l	50	17.23	0.861
AA 200 mg/l	200	14.71	2.942
AA 500 mg/l	500	9.41	4.705

The data presented in table 3 indicated changes in weights of different organs of rats in different groups. Different concentrations of acrylamide decreased body organ weights except for high AA concentration for liver weight percentages. It seems that liver enlargement has been occurred by treating with high AA concentration. Carnitine decreased liver weight comparing to control liver and the same is true for spleen weights, but increased kidneys, heart and testis. Carnitine ameliorates the liver enlargement for the low and middle AA concentration. In the same field, Bergmark *et al.* (1993) discovered acrylamide-hemoglobin adducts in blood of rats fed fried feed.

Table (3): Mean and standard deviation for different rat organs weight through the biological experiment.

Groups	Liver wt		Kidnies wt		Spleen wt		Testis wt	
	(g)	%	(g)	%	(g)	%	(g)	%
GroupI (Control)	3.83^{abc}±0.6	2.54	1.02^{a}±0.2	0.65	0.51^{a}±0.1	0.37	1.53^{a}±1.0	1.04
GroupII (Car. Treated)	3.22^{a}±0.6	2.11	1.2^{ab}±0.2	0.77	0.55^{ab}±0.1	0.38	2.07^{ab}±1.3	1.33
GroupIII (50 mg/L AA + carn.)	3.62^{bc}±0.5	2.23	1.09^{ab}±0.2	0.67	0.46^{a}±0.1	0.22	1.88^{ab}±0.4	0.82
GroupIV (200 mg/L AA+ carn.)	3.38^{ab}±0.5	2.0	1.00^{a}±0.2	0.59	0.50^{a}±0.08	0.29	2.00^{ab}±0.5	1.18
GroupV (500 mg/L AA+ carn.)	4.06^{abc}±0.5	2.58	1.17^{ab}±0.2	0.74	0.48^{a}±0.1	0.3	1.95^{ab}±0.5	1.24
F	3.9^{**}		1.6		2.1		1.3	

There is a logic correlated relationship between acrylamide concentration and available water soluble vitamin as vitamin C concentration. Zeng *et al.* (2009) examined reducing the acrylamide formation by using water soluble vitamins in chemical models and food model systems. While, fat soluble vitamins examined only in the food models, L-ascorbic acid exerted a potent inhibitory effect (>50%) on acrylamide formation in the chemical model systems. On the other hand, vitamin C is essential to the synthesis of carnitine as been mentioned. Table (4) is showing decreasing or consumption of vitamin C significantly at all the tested concentration of comparing to both serum vitamin C of control and carnitine groups. Table (4) as well showing decreasing the glucose level for carnitine group comparing to control and carnitine with AA.

Table (4): Protective effect of carnitine against AA concentrations in mean and standard deviation values for glucose and vitamin C measurements.

Groups	Glucose mg/dl	Vitamin C mg/l
GroupI (Control)	121.5^{b}±7.5	116.6^{b}±1.2
GroupII (Car. Treated)	109.7^{a}±4.1	121.1^{b}±5.6
GroupIII (50 mg/L AA + carn.)	116.75^{ab}±3.3	116.67^{b}±1.6
GroupIV (200 mg/L AA + carn.)	140.75^{c}±4.0	116.25^{b}±6.5
Group V (500mg/L AA + carn.)	151.0^{d}±5.4	91.6^{a}±1.2
F	6.7^{**}	15.3^{**}

In table (5), total antioxidants which involve many vital factors and reduced glutathione in serum have been significantly decreased in the presence of high acrylamide concentrations. Parzefall (2008) mentioned that the variety of acrylamide is conjugated with glutathione and less is activated by cytochrome *P-450*. That might be a reason for detoxifying of acrylamide by conjugation of glutathione. As shown in result, Carnitine alone increased reduced glutathione and the total antioxidants insignificantly. Dayanandan *et al.,* (2000) proved a marked improvement in the antioxidant status. Previous studies have shown that L-carnitine supplementation alters the biochemical changes observed during aging (Kalaiselvi and Pannerselvam, 1998 & Kumaran *et al.,* 2003). The decrease in antioxidants for acrylamide groups refers to producing free radicals and oxidative products (Zhu *et al.*, 2008). Srivastava *et al.* (1983 & 1986) and Nail (2010) proved that acrylamide increased lipid peroxidation and decreased glutathione content in a dose-dependent manner for serum and brain homogenate.

Table (5): Protective effect of carnitine against AA concentrations in mean and standard deviation values for antioxidative effect and reduced glutathione amount.

Groups	AO mM/l	GSH mmol/l
GroupI (Control)	0.30^{c}±0.1	7.9^{d}±2
GroupII (Car. Treated)	0.36^{c}±0.1	8.7^{d}±1.6
GroupIII (50 mg/L AA + carn.)	0.18^{b}±0.1	6.3^{c}±1
GroupIV (200 mg/L AA + carn.)	0.09^{ab}±0.2	5.6^{b}±1.4
Group V (500 mg/L AA + carn.)	0.05^{a}±0.1	4.6^{a}±0.3
F	10.3^{**}	15.7^{**}

Acrylamide has been shown to be neurotoxic in humans and laboratory animals (FAO/WHO, 2002). It has been classified as a Group 2A carcinogen by the International Agency for Research on Cancer (I.A.R.C., 1994). FAO/WHO Expert Consultation urges more research on acrylamide in food. But anyway, the main toxic endpoints of AA are known as neurotoxicity in humans and animals. Acetylcholine helps carrying messages between nerve cells in the brain. So, increase of cholinesterase (ChE) enzyme activity indicates the degraded effect on the brain function. In the same side, research shows increasing in the enzyme activity for the serum of rats treated with 50, 200, and 500 mg/l acrylamide comparing to control and carnitine rats'

enzymes (Table 6). Zeisel and Blusztajn (1994) mentioned that choline enhances acetylcholine synthesis. Choline and carnitine interaction has been reported in healthy humans and animals (Daily *et al.,* 1998 & Rein *et al.,* 1997). Choline supplementation promotes tissue carnitine conservation, especially in skeletal muscle.

Table (6): Mean and standard deviation for butyl cholinesterase activity in rat groups treated with carnitine and carnitine with acrylamide concentrations.

Groups	Cholinesterase U/l
GroupI (Control)	64.7^{ab}±9.9
GroupII (Car. Treated)	57.5^{a}±2.3
GroupIII (50 mg/L AA + carn.)	70.4^{b}±0.5
GroupIV (200 mg/L AA + carn.)	86^{c}±2.7
Group V (500 mg/L AA + carn.)	90.1^{c}±2.2
F	3.2^{**}

Table (7) showed decreasing in liver function enzymes for the carnitine treated group comparing to serum of control group in agreement with Darwish (2010). ALT and AST enzymes activity has increased dramatically for acrylamide high concentrations comparing to carnitine serum group. These data was similar to that for Edwards *et al.* (1978). They found increase in serum AST in long term administration AA 5 mg/kg body weight per day, indicating an impairment of liver function.

Table (7): Mean and standard deviation for liver function enzymes as a tool for measuring the protective effect of carnitine against AA concentrations.

Groups	AST U/l	ALT U/l
GroupI (Control)	82^{c}±4.4	32.7^{bc}±2.0
GroupII (Car. Treated)	55.31^{a}±2.9	28.4^{ab}±8.0
GroupIII (50 mg/L AA + carn.)	72.6^{b}±4.3	38.5^{cd}±4.0
GroupIV (200 mg/L AA + carn.)	79^{c}±3.0	43.6^{d}±3.0
Group V (500 mg/L AA + carn.)	88.6^{d}±9.0	44.5^{d}±6.0
F	6.5^{**}	10.4^{**}

Lipid profile (Table 8) showed decreasing in total glycerides and cholesterol for carnitine treatment, and acrylamide concentrations enhanced the formation of lipids. Data were showing the risk for both of LDL and vLDL which increased as the acrylamide concentration increased, and these amounts are higher than the amounts of that for carnitine serum group. On

the other hand, LDL/HDL ratio has proved the same theory of increasing the risk factor of acrylamide of different concentrations comparing to the factor of carnitine serum group. It's obvious to notice the positive effect of carnitine separately or with the acrylamide concentration groups. Result is correlated with that of Dayanandan *et al*., (2000), which considered significant reduction for carnitine in the tissue lipid peroxidations.

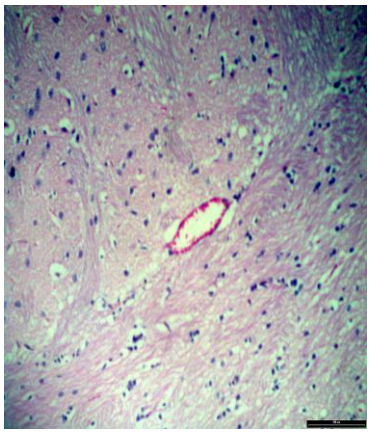
Table (8): Mean and standard deviation for lipid profile as a marker for the action of carnitine against the acrylamide concentrations in rat serum.

Groups	TG mg/dl	CHL mg/dl	vLDL-c mg/dl	LDL-c mg/dl	HDL-c mg/dl	LDL-c / HDL-c ratio
GroupI (Control)	62.6^{b}±4.9	58^{a}±2.0	12.53	15.2^{a}±2.5	30.22	0.50
GroupII (Car. Treated)	55^{a}±1.0	78^{bc}±9.0	11	16^{a}±2.4	51	0.31
GroupIII (50 mg/L AA + carn.)	56^{a}±3.5	79.8^{bc}±2.0	11.2	20^{b}±3.0	48.6	0.41
GroupIV (200 mg/L AA + carn.)	64^{b}±7.7	80.2^{c}±19.0	12.8	41.6^{cd}±15	25.8	1.61
Group V (500 mg/L AA + carn.)	74^{c}±9.6	83.2^{cd}±3.7	14.8	58^{d}±3.5	10.4	5.58
F	9.6^{**}	7.2^{**}		11.8^{**}		

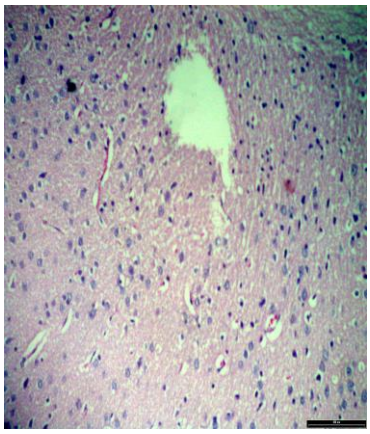
Histopathological examinations

Control group showed normal brain histological structure for the cerebral tissues, and group treated only with carnitine showed diffuse gliosis allover the cerebral tissue. Lehning *et al.* (2003) mentioned great damage of brain and nerves by acrylamide containing food in human and experimental animals. In agreement, different concentrations of AA in drinking water for rats treated with carnitine showed diffuse gliosis with penicellular oedema on cerebral tissue. On the second concentration of AA, perivascular and penicellular oedema with diffuse gliosis in the cerebrum has been showed. The higher AA concentration showed dilatation and congestion of the blood vessels with oedema in the brain matrix. Nail (2010) found correlated brain result, focal odema, inflammatory cells infiltration and congested in the blood capillaries have been shown for administrated acrylamide rat group.

Normal histological liver structure has been found in the control, rats treated only with carnitine and the lower AA concentration with carnitine groups. On the second concentration of AA, sever dilatation and congestion in the central liver vein and sinusoids have been observed. The higher AA concentration showed fibrosis with inflammatory cells infiltration and congestion of the portal vein in the portal area. The research offer L-carnitine as a protective agent for the low concentration long term of acrylamide administration Saita *et al.* (1996) stated that 4-methylcatechol improved nerve growth factor and foot movement. Hashimoto *et al.* (1981) indicated as well that phenobarbital treatment reduced neurotoxic symptoms of AA.



(a)



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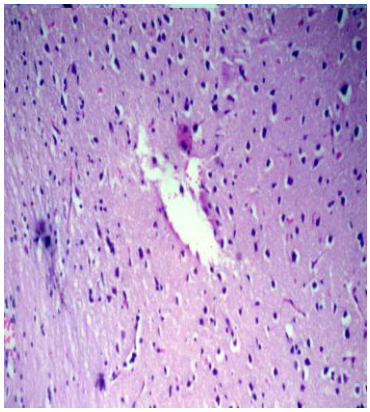

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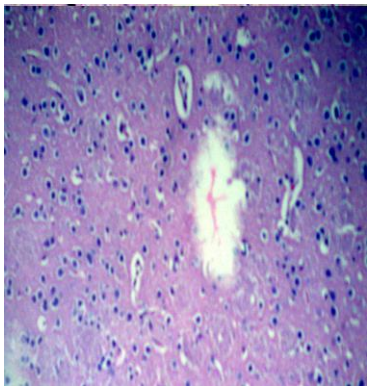

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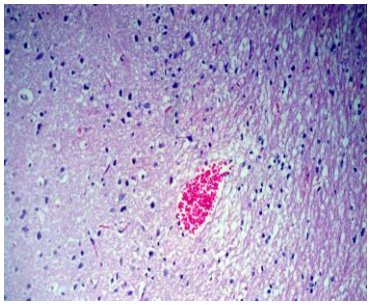

(e)

Figure 1: Histograms for brain sections for rat of control group (a), rat treated with carnitine (b), and rats treated with carnitine with 50 AA (c), 200 AA (d) and 500 AA (e) (HE X 40).

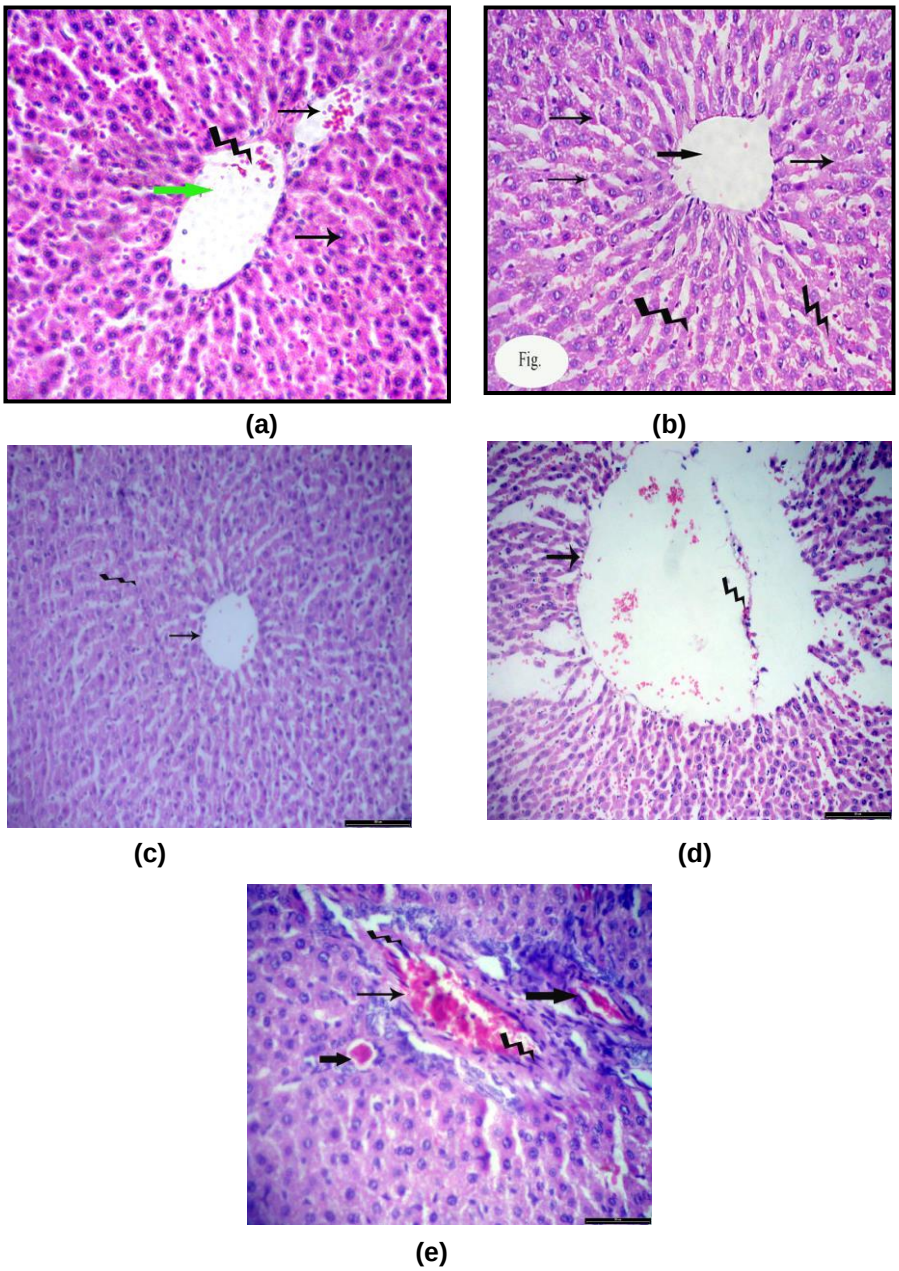

Figure 2: Histograms for liver sections for rat control group (a), rats treated with carnitine (b), and rats treated with carnitine with 50 AA (c), 200 AA (d) and 500 AA (e) (HE X 64).

Carnitine showed amelioration for rat groups treated with low concentrations of acrylamide. Comparing to control, carnitine increased food efficiency, vitamin C and declined glucose in serum. Carnitine with acrylamide treatment consumed the available vitamin C, and caused diabetic effect. On the other side, carnitine didn't show an overcome for the reduction in the antioxidants or reduced glutathione which caused by acrylamide concentrations. While, carnitine alone increased total antioxidant and reduced glutathione. Carnitine fulfilled to decrease cholinesterase activity as neurotoxic or detergent sign in the lower acrylamide concentration, but couldn't inhibit the enzyme activity in middle and high AA doses. L-carnitine decreased liver function enzymes ALT and AST. L-carnitine as well decreased cholesterol-LDL and increased cholesterol-HDL. In a biological attitude, carnitine could be as a shield as natural antioxidant against the neurotoxic effects of acrylamide at different concentrations. More research are needed to extract natural amounts from L-carnitine to apply for many polluted fields.

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ل- كارنيتين ، المادة الجديدة المأمولة للحماية من السمية العصبية من الأكريلاميد في الجرذان البيضاء

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يعتبر ل-الكارنتين (4-ثلاثى ميثيل أمونيوم –3- هيدروكسى حمض البيوتريك) من المواد المهمة في تغذية الأنسان حيث يخلق من الأحماض الأمينية الغذائية و يتوزع علي معظم اعضاء الجسم. اجريت دراسات علي قدرة الكارنيتين علي حماية الأنظمة الحيوية ضد سمية الاكريلاميد. يعتبر الأكريلاميد المادة الكيميائية ذات الأستخدامات الصناعية و المعملية الواسعة من المواد المسرطنة و المسببة للتسمم العصبي حيث أشارت الابحاث إلى دور ل - كارنيتين في علاج سمية الحادثة بواسطة الأكريلاميد. استخدم لمدة ثلاثة أيام في الأسبوع ل-كارنيتين (بمعدل 300 ملجم / كجم من وزن الجسم لجرذان التجارب) عن طريق الفم ثم في اليوم الثلي لاستخدام الكارنيتين تمت المعاملة بالتركيزات: 50 ، 200 و 500 ملجم من الأكريلاميد / لتر من مياه الشرب. لمدة تصل الى 5 اسابيع قام ل-كارنيتين بتحسين الكفاءة الغذائية للجرذان مع انخفاض وزن الجسم المكتسب يوميا لتركيز 50 ملجم اكريلاميد/لتر,كذلك أظهر الكارنيتين نجاحاً تقريبياً في حفظ الوزن النسبي لكل من الكبد والخصيتين مقارنة مع المجموعة الضابطة. وأوضح ل - كارنيتين تحسيناً بمستوي فيتامين (ج) والجلوكوز في مصل الدم لجرذان التجارب عند تركيز 50 ملجم اكريلاميد/لتر,كذلك ل-كارنيتين اظهر تحسناً في مستوي الدهون ومعامل الخطورة (النسبة بين الليبو بروتين منخفض الكثافة والليبو بروتين مرتفع الكثافة) لتركيز 50 ملجم اكريلاميد/لتر. على الجانب الآخر ، أظهرت التركيزات المرتفعة من الأكريلاميد (500 ملجم اكريلاميد/ لتر مياة شرب) آثاراً في ارتفاع وظائف الكبد ، و انخفاض مضادات الأكسدة الكلية ، وانخفاض نشاط إنزيم الجلوتاثيون المختزل و عند هذا التركيز فشل ل- كارنيتين في مواجهة تأثير الأكريلاميد الضار.

قام بتحكيم البحث

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