Effect of Almond Seeds Oil Extract and Some Antioxidant Agents on lipid profile and Oxidative Stress in Induced Diabetes Mellitus in Rats

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***Abstract*—** In the present study 30 male albino rats have been utilized. Diabetes mellitus was induced of rats with alloxan.

The animals were divided into 6 groups, control rats, diabetic rats (model), group was treated daily with almond oil, group was treated with vitamin-E, group was treated with L-carnitine, and the last group was treated with vitamin -E + L-carnitine daily for five weeks. At the end of the treatments the levels of serum glucose, cholesterol, and Triacylglcerol and serum malondialdehyde were significantly increased in comparison to non treated diabetic rats. Serum catalase and Serum superoxide dismutase level were significantly reduced in non-treated. Feed of almonds, Vitamin-E, L-carnitine, and (L-carnitine +Vitamin- E) for five weeks were significantly, elevated serum superoxide dismutase, reduced the levels of serum TG and serum malondialdehyde. While the diabetic rats treated with Vitamin-E, (Vitamin-E+L-carnitine) and almond, showed significant reducing serum glucose and cholesterol..

***Keywords*—** Almond oil, Diabetes, Alloxan, Antioxidant.

I. Introduction

Diabetes mellitus is one of the main widespread health problems in the world and it is getting worse particularly in the developing countries, and all over the world, thus the disease constitutes a major health concern, presently, it is an incurable metabolic disorder which affects about 2.8% of the global population (Etuk, 2010). Oxidative stress results from an imbalance between free radical-generating and it scavenging systems, such as elevated of free radical production or reduced activity of antioxidant defenses or both, oxidative stress is included in the pathogenesis of diabetes **(**Kangralkar *et al.*, 2010). Antioxidant enzymes are consisting of superoxide dismutase (SOD), catalase, glutathione peroxidase (GPx), and glutathione reductase (GR) (Abd El-Aal, 2012). Catalase was the first antioxidant enzyme that catalyses the two stages conversion of hydrogen peroxide to water and oxygen. The superoxide dismutase catalyses the dismutation of superoxide to hydrogen peroxides. The hydrogen peroxide must then be removed by catalase or glutathione peroxidase, (Kefer *et al*, 2009).

Nuts are contain high levels of fiber, arginine, magnesium, polyphenolic compounds, vitamin- E, and monounsaturated fatty acids, specifically oleic acid (Fraser, 1999). Some studies revealed association between frequent nut consumption and reduced risk of diabetes mellitus. Therefore, the present study is done to evaluate the effects of almond oil seeds extract on sperm quality in alloxan induced diabetic rats.

Vitamin-E is the lipid soluble vitamin that protects biological membranes and lipoprotein by direct action of cellular responses to oxidative stress during modulation of signal transduction pathway (Bansal and Gurmail, 2009).

II. Materials and Methods

Adult male laboratory rat used in the present study, (8-10 weeks) in age, weighing (200-250) gm, kept in the animal house at the Department of Biology, Faculty of Science and Education Science, Sulaimani University/Iraqi Kurdistan-Region, in precise environment that was maintained under a 12 hour light/dark cycle, a temperature of 22 ± 2 Cº and The rats supplied with a standard pellet diet and water ad-libitum (Abo-Ghanema *et al.*, 2012).

III. Induction of diabetes

After fasting over night (access to water only) the male rats were given a single S.C injection of freshly prepared alloxan monohydrate (120 mg/kg of body weight) using saline solution (0·9% w/v), (Bahnak and Gold, 1982). Alloxan injected rats were given 5% glucose overnight to prevent rapid fatal hypoglycemia resulted from insulin release due to alloxan action. After 72 hours diabetes was confirmed by testing of serum glucose obtaining blood from rat tails. Rats having fasting serum glucose more than 200 mg /dL was considered diabetics (Johnson *et al*., 2013), after 4 weeks, living rats that blood glucose more than 200 mg / dl measured by glucometer (Accu-check Roche Diagnostics GmbH, Mannheim, Germany), were regarded as diabetes (Gidado *et al*., 2005), because of the high volume of urine produced, diabetic rats are housed sex per cage and the bedding changed daily.

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IV. EXPERIMENTAL DESIGN

Thirty-eight (30) adult male rats used in the current study, after one month of induction of diabetes mellitus, they were separated into two main groups; diabetic and nondiabetic (control) groups. Diabetic group divided into five subgroups (Table1).

At the end of experimental period, (5 weeks), blood samples were collected, from fasted rats, control and diabetic rats, using anesthetic with ketamine hydrochloride (50 mg/ Kg/ b.w.) (Alp *et al.,* 2012), and sacrificed, blood sample was taken by heart puncture, put into chilled tubes without EDTA later centrifuged at 3000 rpm for 15 minutes then serum separated and stored in deep freeze (-45C°) .

V. STATISTICAL ANALYSIS

Analysis of data was performed by using SPSS (Version 18). Results expressed as mean ± S.E. Statistical differences were determined by Dunnett's test for multiple comparisons after ANOVA Dunnett test treats one group as a control and compares all other groups against it.

VI. RESULTS AND DISCUSSION

A. Serum glucose

Serum glucose level was significantly (P<0.05) increased in alloxan induced diabetic rats (440.250 ± 38.937 mg/dl) when compared to control rat group (113.330 ± 3.612 mg/dl).

After five weeks of treatment with almond oil, vitamin-E, and (vitamin-E+L-carnitine), rat showed significant (P<0.05) reducing in the levels of serum glucose, (190.200 ± 12.816 mg/dl, 341.200 ± 34.343 mg/dl, 251.600 ± 21.588 mg/dl,) respectively as compared to their levels in non-treated diabetic rats (440.250 ± 38.937 mg/dl).

But diabetic rats supplemented with L-carnitine caused none significant (P<0.05) change in serum glucose level (374.400 ± 31.833 mg/dl) when compared to untreated diabetic rats (440.250 ± 38.937 mg/dl). (Table 2)

B. Serum cholesterol

Serum TC level in alloxan induced diabetic rats was elevated significantly (P<0.05) (105.225 ± 5.224 mg/dl) when compared to control rat group (69.657 ± 3.330 mg/dl).

The supplementation of diabetic rats supplemented with L-carnitine showed non significant change (P<0.05) in level of serum cholesterol (94.926 ± 3.705 mg/dl), in comparison with diabetic non-treated rats (105.225 ± 5.224 mg/dl)

But diabetic rat supplemented with almond oil, vitamin-E and (vitamin-E+ L-carnitine), caused significant (P<0.05) reducing in the levels of serum cholesterol (78.210 ± 6.767 mg/dl, 72.836 ± 5.551 mg/dl, 64.480 ± 4.776 mg/dl,) respectively, as compared to their level in diabetic non-treated rats (105.225 ± 5.224 mg/dl). (Table 2)

C. Serum triacylglycerols

Serum total triacylglycerols levels significantly (P<0.05) increased in alloxan induced diabetic rats (125.805 ± 4.163 mg/dl), when compared to control rat group (78.493 ± 8.880 mg/dl).

When diabetic rats supplemented with almond oil, Vitamin-E, L-carnitine and (Vitamin-E+L-carnitine), showed significant decrease (P<0.05)in the level of serum triacylglycerols (81.292 ± 7.795 mg/dl, 83.870 ± 4.560 mg/dl, 98.064 ± 8.798 mg/dl, 76.130 ± 2.413 mg/dl,) respectively in comparison with non-treated diabetic rats (125.805 ± 4.163) (Table 2).

D. Serum catalase

Serum catalase significantly (P<0.05) reduced in alloxan induced diabetic rats (6.472 ± 0.728 U/ml), in comparison with control rat group (15.815 ± 4.563 U/ml). Diabetic rats treated with vitamin-E and (L-carnitine + vitamin-E), Serum catalase level not change significantly (7.522 ± 0.806 U/ml, and 7.558 ± 0.849 U/ml) respectively when compared to untreated diabetic rats (6.472 ± 0 .728 U/ml).

While in diabetic rats treated with L-carnitine, almond oil, serum catalase level was significantly (P<0.05) elevated (18.280 ± 1.624 U/ml, 14.520 ± 1.174 U/ml) respectively

in comparison with diabetic control rat group (6.472 ± 0 .728 U/ml). (Table).

E. Serum superoxide dismutase (SOD)

Serum SOD level significantly (P<0.05) reduced in alloxan induced diabetic rats (92.585± 1.742 U/ml) in comparison with control group rats (104.278± 0.944 U/ml).

The treatment of diabetic rats with almond oil, vitamin-E, L-carnitine, and (L-carnitine + vitamin-E), showed significant (P<0.05) elevation in the level of serum SOD (101.748 ±1.018 U /ml, 102.4 00 ± 2.405 U /ml, 98.942 ± 0.698 U/ml, and 100.190 ± 4.833 U /ml,) respectively in comparison with un treated diabetic rat group (92.585± 1.742 U /ml). (Table 3 &).

F. Serum malondialdehyde (MDA)

Serum MDA level significantly (P<0.05) elevated in alloxan induced diabetic rats (3.960 ± 0.229 μmol/L) in comparison with control group rats (1.920 ± 0.175 μmol/L)**.**

The treatment of diabetic rats with almond oil, Vitamin-E, L-carnitine and (L-carnitine + vitamin-E), showed significant decrease in the level of Serum MDA (2.112± 0.244 μmol/L, 2.560 ± 0.191 μmol/L, 2.592 ± 0.192 μmol/L, and 1.992 ± 0.264 μmol/L) respectively in comparison with un treated diabetic rats (3.960 ± 0.229 μmol/L) (Table 3).

TABLE I
EXPERIMENTAL DESIGN

Groups	No. of rats	Treatment	Duration
Normal control	6	Tape water	5 weeks
Diabetic	4	Tape water	5 weeks
DM + Almond oil	5	1ml almond oil / kg of rat / day	5 weeks
DM +Vitamin- E	5	2000 IU/kg diet/ day	5 weeks
DM +L-Camitine	5	5 gm/kg diet/ day	5 weeks
DM + Vit- E+ L-Cami	5	2000 IU of vit-E+5gm L-camitine /kg/ day	5 weeks

TABLE II
EFFECT OF ALMOND OIL, VITAMIN-E AND L-CARNITINE ON SOME BIOCHEMICAL PARAMETERS IN DIABETIC MALE RATS

parameters / Groups	Serum glucose (mg/dl) *	Serum Cholesterol (mg/dl) *	Serum Tracylglycerols (mg/dl) *
Normal control	113.330 ± 3.612 [a]	69.657 ± 3.330 [a]	78.493 ± 8.880 [a]
Diabetic	440.250 ± 38.937 [d]	105.225 ± 5.224 [b]	125.805 ± 4.163 [b]
DM + Almond oil	190.200 ± 12.816 [b]	78.210 ± 6.767 [a]	81.292 ± 7.795 [a]
DM +Vitamin-E	341.200 ± 34.343 [c]	72.836 ± 5.551 [a]	83.870 ± 4.560 [a]
DM +L-camitine	374.400 ± 31.833 [c d]	94.926 ± 3.705 [b]	98.064 ± 8. 751 [a]
DM+L-cami.+Vit E	251.600 ± 21.588 [b]	64.480 ± 4.776 [a]	76.130 ± 2.413 [a]

Values expressed as mean ± S.E. The differences letters mean significant differences
* =p<0.05, ** =p<0.01

TABLE III
EFFECT OF ALMOND OIL, VITAMIN-E AND L-CARNITINE ON SOME BIOCHEMICAL PARAMETERS IN DIABETIC MALE RATS

parameters / Groups	Serum catalaseU /ml*	Serum SODU /ml*	Serum MDA (μmol/L) *
Normal control rat	15.815 ± 4.563 [b]	104.278 ± 0.944 [b]	1.920 ± 0.175 [a]
Diabetic	6.472 ± 0 .728 [a]	92.585 ± 1.742 [a]	3.960 ± 0.229 [b]
DM+Almond oil	14.520 ± 1.174 [b]	101.748 ± 1.018 [b]	2.112 ± 0.244 [a]
DM +Vitamin-E	7.522 ± 0.806 [a]	102.4 00 ± 2.405 [b]	2.560 ± 0.191 [a]
DM +L-camitine	18.280 ± 1.624 [b]	98.942 ± 0.698 [b]	2.592 ± 0.192 [a]
DM +L-cami +Vit E	7.558 ± 0. 849 [a]	100.190 ± 4.833 [b]	1.992 ± 0.264 [a]

Values expressed as mean ± S.E. The differences letters mean significant differences

G. Serum glucose

In the present study, the level of serum glucose significantly elevated in alloxan induced diabetic rat. This result is concurrences with other findings reported by (Mohamadin *et al*., 2011; Roy *et al*., 2013), they revealed that induced diabetic cause significant increases in the level of serum glucose, in male rats, also, they established that the levels of serum insulin were significantly inhibited in diabetic rats.

Induction of diabetes via alloxan caused damage of the beta cell of pancreas, producing radicals which have a particularly low anti oxidative defense capacity leading to increase in plasma glucose levels, and inhibition of insulin secretion (Lenzen *et al*., 1988). Sexual disturbance is frequently related with diabetes in men and experimental animals (Scarano *et al*., 2006). Several problems due to diabetes-induced defect in the peripheral nervous system, but most suggests, that central nervous system-related changes in endocrine function and may affect sexual dysfunction (McVary *et al*., 1997).

The current results demonstrate that diabetic rats treated with vitamin-E, showed significant reduction of blood glucose level. These results are in agreement with that of (Al Shamsi *et al*., 2004; Katyal *et al*., 2009), they reported that the reduction in blood glucose level by vitamin-E may be due to their antioxidant properties and reduction in oxidative stress in â-cells of islets of langerhans resulting in increased insulin secretion and decreased blood glucose levels.

Paolisso *et al*., (1994) reported that reduction action of vitamin-E in serum blood glucose level may be by modulating insulin action. Moreover, vitamin-E administration resulted in protein kinase C inhibition due to the direct interaction between á-tocopherol and protein kinase C in the cell membrane (Brigelius and Traber, 1999). Controversially (Kinalski *et al*., 2000; Bin-Jaliah *et al*., 2013) revealed that diabetic rats were found unaffected by vitamin-E treatment.

L-Carnitine supplementation does not changed blood glucose in diabetic rats, these results are supported by previous findings (Uysal *et al*., 2005; Bazotte and Bertolini, 2012). On other hand Shaker *et al*., (2009), reported that L-carnitine increased glucose utilization and uptake by stimulating the activity of pyruvate dehydrogenase and decreasing the acetyl CoA/CoA ratio so the level of glucose inhabited in diabetes. Supplementation of diabetic rats with (Vitamin-E + L-carnitine), showed significant reduction of serum blood glucose due to their high potent of free radical scavenger. It has been proposed that vitamin-E may have a role in modulating insulin action, L-carnitine treatment dramatically increased glucose oxidation rates in the diabetic rat, while there were unavailable more study about co-treatment of vitamin-E and L-carnitine till know.

The treatment of induced diabetic rats with almond oil was significantly lowering blood glucose level. These results are in accordance with previous studies (Teotia and Singh, 1997; Shah *et al*., 2011; Anwar *et al*., 2013).

Almond oil is a rich source of antioxidants and á-linolenic acid, n-3 fatty acid, further benefits of almonds may result from their high plant sterols, and associated phenolic substances (Jenkins *et al*., 2002). The oil content of almond may improve beta cell and regulate insulin action by reducing free radicals and enhances anti oxidant enzyme.

Prior in vivo studies have shown that monounsaturated fatty acid MUFA in almond oil (Berry *et al*., 1992) enhances the intestinal secretion of glucagon-like peptide-1 (GLP-1) an incretin hormone that improves the regulation of postprandial glucose disposal and insulin secretion (Perfetti *et al*., 1999). In addition, Rocca *et al*., (2001) suggested that the high oleic acid content in the almonds may improve beta-cell efficiency through enhanced intestinal secretion of GLP-1.

H. Serum TC and TG

The results of the present study showed significant elevation in levels of serum total cholesterol (TC) and triacylglycerol (TG) in induced diabetes rats. The same results are approved with previous experimental diabetes studies (Hakkim *et al*., 2007; Roy *et al*., 2013).

Mathe, (1995), mentioned that hypercholesterolemia in diabetic rat results from elevated intestinal absorption and cholesterol production also the levels of acute and chronic hyperglycemia correlate strongly with the level of cholesterol oxidation, due to role of insulin in lipid metabolism by inhibiting hormone-sensitive lipase. Also lack of insulin causes hypertriglyceridemia.

In this study the supplementation of vitamin-E to diabetic rats causes significant improvement in the concentrations of TG, and TC.

Halim *et al*., (2006) and Almeida *et al*., (2012) documented the same results. These results may be explained by Baydas *et al*., (2002) concluding a negative association between vitamin-E and serum cholesterol and triacylycerol levels. The efficacy of vitamin-E with regards reducing serum triacyglycerols and may be attributed to its protection of membrane-bound lipoprotein lipase against lipid peroxide.

Vitamin-E, due to its antioxidatant properties increases the total hepatic triacylglycerol lipase activity by elevating the lipoprotein lipase activity possibly by protecting the membrane-bound lipase against peroxidative damage (Pritchard *et al*., 1986).

In this study, in diabetic rats treated with L-carnitine the level of serum total TG was reduced significantly. Rodrigues *et al*., (1988) and Mansour, (2013) documented the same results. While in diabetic rats treated with L-carnitine, the level of serum cholesterol not changed significantly, this result agrees with previous studies by (Gonzalez *et al*., 2008; Salama, 2011).

Bazotte, and Bertolini, (2012), concluded that reduction of blood triacylglycerol obtained with the L-carnitine supplementation in the diabetic rats did not depend on an amelioration in the glycemia. One possible mechanism of the triacylglycerol lowering effect is the influx of fatty acids to the mitochondria. L-carnitine is known to promote the transport of cytosolic long-chain fatty acids into the mitochondrial matrix for -oxidation, thereby providing mitochondrial energy (Diaz *et al*., 2000).

In addition, treatment of diabetic rat group with combination of Vitamin-E+L- carnitine improved both TG and TC when compared to untreated diabetic rat group due their combination. This increase may be attributed to the fact that the antilipidemic and hypocholesterolemic effects of both treatments may have been more effective in decreasing both TG and TC.

Current study demonstrated that supplementation of diabetic rat group with almond oil significantly inhibits the level of serum TG and TC, this results documented with previous studies (Shah *et al*., 2011; Anwar *et al*., 2013).

The results of reduced cholesterol, TG, and LDL in rats received almond oil can be attributed to the relatively high content of unsaturated fatty acids in almond oil. In fact, about 90% of the total fatty acids present in almond oil are unsaturated fatty acids with oleic acid and linoleic acid being the predominant unsaturated fatty acids (jai et al, 2011). Furthermore, polyunsaturated fatty acids PUFA have been recommended over the last few years as a dietary change to lower serum cholesterol, (Fernandez *et al*., 2007).

Hyson *et al*., (2002), concluded that the lipid-lowering effect of almonds is mediated primarily by the almond oil fraction. Diabetic rats received almond oil, showed significant decrease in the levels of cholesterol, triacylglycerol, the almond oil also contains several minor components such as tocopherols with potential healthy biological properties (Miraliakbari and Shahidi, 2008). These compounds have been reported to inhibit cholesterol deposition in the arteries and prevent lipoprotein structural alterations (Basu *et al*., 2007).

Berryman *et al.*, (2011), reported that the cholesterol reduction associated with almond oil consumption has been primarily attributed to the replacement of saturated fat with unsaturated fat where the major fatty acids in almonds are oleic acid and linoleic acid, almond oil are poor in saturated fatty acids and rich in unsaturated fatty acids decreased absorption of cholesterol and bile acid, increased bile acid and cholesterol excretion and an increased LDL-cholesterol receptor activity.

The nutrients which are present in almonds regulate the enzymes which are involved in cholesterol synthesis and bile acid production. It also rich with phytosterols which may exert hypocholesterolemic effects via interactions with intracellular enzymes, namely acyl-CoA, cholesterol acyltransferase, and the rate limiting enzyme in cholesterol synthesis in almond decreases LDL cholesterol by disrupting enterohepatic circulation, thus increasing Bile acid and cholesterol excretion and up regulating the LDL cholesterol receptor (Berryman *et al.,* 2011).

Almond oil is an excellent source of α-tocopherol, this may enhances the enzyme activity including lipid degradation. In addition, the polyphenolic constituents of almonds have been characterized recently and found to possess antioxidant actions (Chen *et al*., 2006).

I. Effects of almond oil, vitamin-E and L-carnitine on antioxidant enzymes

The present study revealed that serum catalase and superoxide dismutase levels were significantly decreased in alloxan diabetes rats. These results are confirmed by previous studies (Shrilatha and Muralidhara, 2007; Akondi *et al*., 2011; Hisalkar *et al*., 2012). In contrast to our results, catalase and superoxide dismutase activity increases in diabetic rat (Bhor *et al*., 2004).

SOD is an intracellular enzyme found in every cell, it's actually represented by group of metalloenzym converts superoxide radical to hydrogen peroxide, by reducing (adding an electron) to super oxide to form hydrogen peroxide, catalase acts as main Regulator of hydrogen peroxide metabolism. Hydrogen peroxide is a highly reactive small molecule formed as natural by-product of energy metabolism

(Kakkar *et al*., 1995). Also catalase enzyme catalyzes the decomposition of hydrogen peroxide to water (H_2O) and oxygen molecule (O_2). (Evans *et al*., 2002).

Excessive concentration of hydrogen peroxide may cause significant damages to proteins, DNA, RNA, and lipids. In addition increased risk of diabetes has been documented in patients with catalase deficiency (Goth and Eaton, 2000). Also reduction in SOD activity has been shown to increase the level of superoxide, which is known to inactivate (GPx). Similarly, when GPx fails to eliminate H_2O_2 from the cells, the accumulated H_2O_2 has been shown to cause inactivation of SOD (Shrilatha and Muralidhara, 2007).

In the present study, serum catalase activity in diabetic treated rat with vitamin-E does not improved, but was higher than that in the diabetic non treated rat. This result is similar previously results (Musalmah, 2002; Bughdadi, 2013).

While serum SOD activity was elevated in alloxan induced diabetic rat, this result agree with previous studies (Hong *et al*., 2004; Ghaffari *et al*., 2011; Baragob *et al*., 2014).

Shirpoor *et al*., (2009), reported in their study that vitamin-E improve the activity of SOD, vitamin-E in general does appear to normalize the expression level of particular antioxidant enzymes under diabetic conditions. It is likely then that changes in antioxidant enzyme expression might occur as a result of oxidative stress and that vitamin-E simply prevents some of these changes through its scavenging abilities in present study the variation of SOD activity may be due to the change of some correlated enzymes and content of vitamin-E in testicular cell membrane.

Administration of L-carnitine to alloxan induced diabetic rat induced significant increase in the activities of SOD concentration. these results are agreed with that of (Uysal *et al.,* 2005; Mansour, 2013), they reported that L-carnitine have anti diabetic effects that may be mainly attributed to its potent antioxidant or due to its active role in the transport of fatty acids for energy production.

In addition in the L-carnitine-treated rats, the increase in the level of antioxidant enzymes, catalase and SOD, were in agreement with other studies (Cao *et al*., 2011; Abo-Ghanema *et al*., 2012). The antioxidant effect of L-carnitine may have been due to the role of L-carnitine in the chelation of free $Fe+^2$ ions with a subsequent reduction in free radical generation (Reznick *et al*., 1992). This may be due to its ability to improve ATP creation, which promote the overall level and activity of antioxidant enzymes in the cell.

While supplementation of (Vitamin-E+L-carnitine) to diabetic rat group unable to maintain catalase activity this may be due to dose dependant or rout of administration. While improve SOD activity when compared to untreated diabetic rat group this refers to synergetic action of (Vitamin-E+L-carnitine).

In this study, the levels of serum catalase and superoxide dismutase were elevated significantly in diabetic treated rats with almond oil. This may be due to almond oil contains antioxidant and antiradical activity, may be helpful in preventing or slowing the progress of various oxidative stress. However, to uses the extract of these phenolic compounds as antioxidant in foods (Isfahlan, 2010).

Diabetic rats treated with oral almond oil showed a significant increase in the levels of these enzymes, superoxide dismutase SOD and catalase. Which indicated the free radical scavenging property of almond oil (Jia *et al*., 2011). From these results it could be noticed that consume mainly monounsaturated fatty acid and short chain polyunsaturated fatty acid and long chain polyunsaturated were beneficial for diabetic disease. Tree nut oil extracts contained phospholipids, sphingolipids, sterols and tocopherols and phenolic compounds (Miraliakbari, and Shahidi, 2008). These phenolic compounds may inhibit lipid oxidation by scavenging free radicals, chelating metals, activating antioxidant enzymes, reducing tocopherol radicals and inhibiting enzymes that cause oxidation reactions. Takeoka and Dao, (2003), reported that chlorogenic acid and cryptochlorogenic acid are the main phenolic compounds in almond oil. In addition the antioxidant activity of phenolic compounds of almond oil is mainly due to their redox properties.

J. Effect of almond oil, vitamin-E and L-carnitine on MDA

Results of this study show that, the level of MDA was increased significantly in induced diabetic rats. Many research studies have recommended the same results concluded that diabetes associated with high level of MDA due to free radical production (Pritchard *et al*., 1986; Katyal *et al*., 2009; Bughdadi, 2013).

Oxidative stress plays a main role in cellular damage due to hyperglycemia, and high glucose level can stimulate free radical production (Tiwari *et al*., 2013). Lipid peroxidation is an achief biological result of oxidative cellular damage in patients with DM (Lapolla *et al.*, 2005). Hydroperoxides have toxic effects on cells both directly and through degradation to highly toxic hydroxyl radicals. They may also react with transition metals like iron or copper to form stable aldehydes, such as MDA, that damage cell membranes (Halliwell and Chirico, 1993).

In the present study diabetic rat group treated with Vitamin-E, L-carnitine, (L-carnitine + Vitamine-E), and almond oil the level of serum MDA reduced significantly.

The protective mechanism of vitamin-E is probably through its capacity to scavenge lipid peroxyl radicals. Furthermore, vitamin-E can also normalize the level of glutathione, which is an important for intracellular free radical scavenging system, thus reducing the degree of oxidative damage. Vitamin-E blocks lipid peroxidation of polyunsaturated fatty acids in membranes. It efficiently protects against lipid peroxidation through its chain-breaking antioxidant activity (Serbecic and Beutelspacher, 2005

The significant protective role of L-carnitine against lipid peroxidation has been demonstrated by improvement in the levels of serum MDA. It is known that the role of L-carnitine the reduction in lipid peroxidation is due to the iron-chelating property of L- carnitine (Uysal *et al*., 2005).

In addition the treatment of rats with L-carnitine+Vitamin-E improves serum MDA reduction due to their anti peroxidative and scavenger effects of both.

Almond oil contains antioxidant and antiradical activity, may be helpful in preventing or slowing the progress of various oxidative stress-related diseases. However, to use the extracts of these phenolic compounds as an antioxidant in foods (Isfahan et al., 2010), also low level of serum MDA in almond oil treating rat group refers to potentially of almonds content of anti oxidant these results documented with previous study (Jia et al., 2011).

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