Research Article

Effect of L-carnitine and Raspberry Ketones on Metabolic Parameters in Iraqi Obese Females, a Comparative Study.

Hamoudi Aliwi Mosah, Faris Abdul Kareem Khazaal, Hayder B Sahib*, Ahmed Shakir Hamdi
Al-Nahrain University, College of Medicine, Iraq.
***Corresponding author's E-mail:** haider_bahaa@yahoo.com

Accepted on: 07-02-2015; **Finalized on:** 31-03-2015.

ABSTRACT

The aim of this study was to compare the effect of raspberry ketones (RK) and L-carnitine on metabolic indices parameter in obese Iraqi women. Sixty obese women aged 20-40 with a BMI ≥ 30 were randomly divided to three groups twenty patients in each; group one received raspberry ketones 500 mg capsule, the second received L-carnitine 1000 mg hard capsule and the third was control without treatment. All patients were informed about diet advice (low calorie diet) and the physical activity needed. The treatment course was 12 weeks. For each group, biochemical parameters total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL), high density lipoprotein (HDL), fasting blood glucose (FBG), serum glutamate pyruvate transaminase (SGPT) and SGOT serum glutamate oxaloacetate-transaminase were measured at baseline and after 12 weeks. All lipid profile parameters were reduced significantly after intervention while HDL was elevated. However; L-carnitine was more effective than RK and RK more than control group. Whereas on FBG, RK more effective than L-carnitine and L-carnitine more than control group. AST, ALT, the weight and body mass index were improved in all patients in various degrees. According to the results presented in this study it is concluded that L-carnitine improve biochemical parameters of obesity especially lipid profile better than RK in improving dyslipidemia while RK is more effective in reducing FBG.

Keywords: Raspberry Ketones, L-carnitine, lipid profile.

INTRODUCTION

Obesity is a public health problem that has raised concern worldwide. STEPS survey (2006), reveal that two thirds (66.9%) of the Iraqi population aged (25-65) were found to be overweight or obese. The rate of overweight among females was higher than males (69.6 Vs 63.6), nearly one third of the respondents were obese.

Obesity was proportionately higher than overweight among female, whereas overweight supervened among male[1]. Numerous epidemiological studies have been conducted to show the relationship between excess weight, abdominal fatness and risk of a wide range of illnesses[2].

Metabolic syndrome (MetS) is a complex disorder defined as a cluster of interconnected factors that directly increase the risk of coronary heart disease (CHD), other forms of cardiovascular atherosclerotic diseases (CVD), and diabetes mellitus type 2 (DMT2). Of its main components are dyslipidemia (elevated triglycerides and apolipoprotein B (apoB)-containing lipoproteins, and low high-density lipoproteins (HDL), elevation of arterial blood pressure (BP) and dysregulated glucose homeostasis, while abdominal obesity and/or insulin resistance (IR) have gained increasing attention as the core manifestations of the syndrome[3].

A systematic review on medicinal plants useful in diabetes mellitus showed that some herbal plants possess anti hyperlipidemic effects and this property is statistically significant, in the treatment of obesity[4]. Some components affect body weight by changes in body fat metabolism and oxidation or increasing metabolic rate, which was shown in trials by Epigallocatechin-3-gallate of green tea and Capsinoids[5,6] causing a higher fat oxidation in human. These compounds act by activating lipid metabolism, acceleration of oxidation, and suppression of fatty acid synthesis and peroxisome proliferator-activated receptor (PPARc) agonistic activity[7].

PATIENTS AND METHODS

In this Prospective randomized single blind clinical study, sixty (60) obese women aged 20-40 with a BMI ≥ 30 who referred to (Obesity Research & Therapeutic Center in Alkindy college of medicine) were recruited. The study protocol was approved by Research Ethical Committee in Al Nahrain University \ College of medicine. All patients had informed consent and statement of confidentiality. Exclusion criteria: patients had hypertension, diabetes, smoking, ischemic heart disease, or any active inflammatory disease.

RESULTS

According to Table 2 that presented the results of control group, the mean and ± SD for baseline and after 3 months biochemical parameters. All lipid profile parameter were reduced after intervention where statistical analysis revealed high significant difference between pre and post intervention ($p < 0.001$). However, the AST differ significantly ($p<0.05$) and the p value of LDL were more than (0.05), and the percent of changes were TC (9.52 %), TG (12.80%), LDL (9.20%), HDL (-5.85 %), FBG (6.32%), AST (5.13%) and ALT (3.35%).

Table 1: The Mean and Standard deviation (±SD) of pretreatment anthropological parameters for all groups

Groups	Age (year)	Weight (Kg)	BMI Kg/m^2
Control (no=18)	32.72 ± 7.002	88.061 ± 9.383	34.833 ± 2.991
L-carnitine (no=18)	33.11 ± 6.533	86.68 ± 6.930	34.588 ± 2.774
Raspberry ketones (no=20)	31.75 ± 5.580	89.46 ± 9.014	35.415 ± 3.341
Control compared with L-carnitine	NS	NS	NS
Raspberry ketones	NS	NS	NS
L-carnitine compared with Raspberry ketones	NS	NS	NS

Body Mass Index (BMI). NS = Non significant (p > 0.05).

Table 2: The Mean ± SD and the percent of changes of biochemical parameter for control pre and after 12 weeks intervention.

Bioch. Parameter	Control (baseline) (n=18)	Control after 3months (n=18)	Percent of change
TC mg/dL	188.7 ± 24.97	172.5 ± 23.38 **	9.52
TG mg/dL	127.7 ± 33.77	113.7 ± 31.76 **	12.80
LDL mg/dL	116.8 ± 22.91	108.4 ± 21.92	9.20
HDL mg/dL	42.38 ± 4.44	39.83 ± 3.77 ↓ **	-5.85
FBG mg/dL	94.50 ± 9.53	88.94 ± 9.57 **	6.32
SGPT (ALT) (U/l)	17.77 ± 2.86	16.88 ± 2.47 **	5.13
SGOT (AST) (U/l)	15.66 ± 2.27	15.16 ± 2.09 *	3.35

TC= total cholesterol. TG= triglyceride. LDL= low density lipoprotein. HDL = high density lipoprotein. FBG= fasting blood glucose. Bioch. = biochemical. SGPT= serum glutamate pyruvate transaminase. SGOT= serum glutamate oxaloacetate-transaminase. * = Significant difference (P<0.05). ** = Highly Significant difference (P<0.001). ↓= decrease

The effect of L-carnitine on obese women were measured and presented on Table 3. This table shows the Mean and ±SD of the biochemical parameter and the percent of changes in pre and post intervention of this group. All lipid profiles were reduced except HDL which was increased to 45.94 mg/dL and the percent of increment was (12.37%). On the other hand the percent of reduction in TC (16.93%), TG (17.99%), LDL (31.53%), HDL (12.37%), FBG (6.50%), ALT (4.32%) and AST (2.63%). The statistical analysis showed the following results: there were highly significant differences in all parameter (p<0.001) from the baseline, except for ALT was different significantly (p<0.05). Although, there was no significant difference in AST parameter (p>0.05).

Table 3: The Mean ± SD and the percent of changes of biochemical parameter for L-carnitine group pre and after 12 weeks intervention.

Bioch. parameter	L-carnitine (n=18)	L-carnitine after 3months. (n=18)	Percent of Reduction
TC mg/dL	194.39 ± 20.05	167.5 ± 24.71**	16.93
TG mg/dL	143.33 ± 25.41	121.89 ± 23.08 **	17.99
LDL mg/dL	123.94 ± 19.04	96.88 ± 23.94**	31.53
HDL mg/dL	41.00 ± 4.08	45.94 ± 4.03 **	12.37 ↑
FBG mg/dL	97.55 ± 8.96	91.66 ± 8.35**	6.50
SGPT (ALT) (U/l)	18.83 ± 4.16	18.11 ± 4.18*	4.32
SGOT (AST) (U/l)	16.00 ± 2.93	15.55 ± 2.45	2.63

TC= total cholesterol. TG= triglyceride. LDL= low density lipoprotein. HDL = high density lipoprotein. FBG= fasting blood glucose. Bioch. = biochemical. SGPT= serum glutamate pyruvate transaminase. SGOT= serum glutamate oxaloacetate transaminase. * = Significant difference (P<0.05). ** = Highly Significant difference (P<0.001). ↑= increase

Moreover, the statistical differences between L-carnitin and control groups were presented in Table 4 and it revealed that there were highly significant difference (p<0.001) in HDL, while, only significant differences (p<0.05), in TC, TG and LDL. Although, the p values of FBG, ALT and AST were (p>0.05).

Table 4: Biochemical parameter difference between L-carnitine and control group after 3 months treatment

Biochemical Parameter	Control (n=18)	Percent of Reduction Control	L-Carnitine (n=18)	Percent of Reduction L-carnitine
TC mg/dL	172.5 ± 23.38	9.52%	167.5 ± 24.71	16.93% *
TG mg/dL	113.7 ± 31.76	12.80	121.89 ± 23.08	17.99 *
LDL mg/dL	108.4 ± 21.92	9.20	96.88 ± 23.94	31.53 *
HDL mg/dL	39.83 ± 3.77	-5.85	45.94 ± 4.03	12.37 ↑ **
FBG mg/dL	88.94 ± 9.57	6.32	91.66 ± 8.35	6.50
SGPT (ALT) (U/l)	16.88 ± 2.47	5.13	18.11 ±4.18	4.32
SGOT (AST) (U/l)	15.16 ± 2.09	3.35	15.5 5 ± 2.45	2.63

TC= total cholesterol. TG= triglyceride. LDL= low density lipoprotein. HDL = high density lipoprotein. FBG= fasting blood glucose. Bioch.= biochemical. SGPT= serum glutamate pyruvate transaminase. SGOT= serum glutamate oxaloacetate transaminase. * = Significant difference (P<0.05). ** = Highly Significant difference (P<0.001). ↑= increase

Table 5: The Mean ± SD and percent of changes of biochemical parameter for raspberry ketones (RK) group pre and after 12 weeks intervention.

Biochemical Parameter	RK (n=20)	RK after 3months (n=20)	Percent of change
TC mg/dL	190.90 ± 36.65	173.95 ± 31.02**	9.54
TG mg/dL	128.20 ± 40.07	114.55 ± 37.36**	12.51
LDL mg/dL	118.4 ± 33.80	101.55 ± 27.25**	16.19
HDL mg/dL	46.60 ± 7.37	48.35 ± 6.96**	4.01↑
FBG mg/dL	98.50 ± 9.28	92.35 ± 8.15**	6.64
SGPT (ALT) (U/l)	19.35 ± 5.97	18.70 ± 5.41*	3.12
SGOT (AST) (U/l)	16.35 ± 5.20	15.75 ± 4.30	2.99

TC= total cholesterol. TG= triglyceride. LDL= low density lipoprotein. HDL = high density lipoprotein. FBG= fasting blood glucose. SGPT= serum glutamate pyruvate transaminase. SGOT= serum glutamate oxaloacetate transaminase. * = Significant difference (P<0.05). ** = highly Significant difference (P<0.001). ↑= increase.

Table 6: The Mean ± SD and percent of changes biochemical parameter between control & Raspberry Ketones (RK) groups after 3 months treatment.

Biochemical parameter	Control (n=18)	Percent of change	RK (n=20)	Percent of Change
TC mg/dL	172.5 ± 23.38	9.52	173.95 ± 31.02	9.54
TG mg/dL	113.7 ± 31.76	12.80	114.55 ± 37.36	12.51
LDL mg/dL	108.4 ± 21.92	9.20	101.55 ± 27.25	16.19
HDL mg/dL	39.83 ± 3.77	-5.85	48.35 ± 6.96*	4.01↑ **
FBG mg/dL	88.94 ± 9.57	6.32	92.35 ± 8.15	6.64
SGPT (ALT) (U/l)	16.88 ± 2.47	5.13	18.70 ± 5.41	3.12
SGOT (AST) (U/l)	15.16 ± 2.09	3.35	15.75 ± 4.30	2.99

TC= total cholesterol. TG= triglyceride. LDL= low density lipoprotein. HDL = high density lipoprotein. FBG= fasting blood glucose. SGPT= serum glutamate-pyruvate transaminase. SGOT= serum glutamate-oxaloacetate transaminase. * = Significant difference (P<0.05). ** = Highly Significant difference (P<0.001). ↑= increase

The effect of Raspberry Ketones on obese women were measured and presented in Table 5. All lipid profiles were reduced except HDL was increased by (4.01%). On the other hand the percent of reduction were TC (9.54 %),TG

(12.51%), LDL (16.19%), HDL (4.01%), FBG (6.64%), ALT(3.12%) and AST(2.99%). The statistical analysis were high significant difference ($p<0.001$) in all parameter except in ALT was ($p<0.05$) and in AST there was no significant difference from baseline group. And although the Table 6 revealed the statistical difference in the mean of the control and RK groups but showed no significant difference in all biochemical parameter ($p>0.05$) except in HDL where there was a highly significant difference ($p<0.001$).

When we compared L-Carnitine to RK groups effect on biochemical parameter especially in the percent of reduction, the results revealed that there were a significant differences in lipid profile ($p< 0.05$) and there were no significant differences in FBG, ALT and AST ($p>0.05$) Table 7.

Table 7: The Mean ± SD and percent of changes biochemical parameter between RK & L-Carnitine groups after 3 months treatment.

biochemical Parameter	L-Carnitine (n=18)	Percent of changes L-Carnitine	RK (n=20)	Percent of changes RK
TC mg/dL	167.5 ± 24.71	16.93 %*	173.95 ± 31.02	9.54 %
TG mg/dL	121.89 ± 23.08	17.99*	114.55 ± 37.36	12.51 %
LDL mg/dL	96.88 ± 23.94	31.53*	101.55 ± 27.25	16.19%
HDL mg/dL	45.94 ± 4.03	12.37 ↑ *	48.35 ± 6.96	4.01 %
FBG mg/dL	91.66 ± 8.35	6.50	92.35 ± 8.15	6.64 %
SGPT (ALT) (U/l)	18.11 ± 4.18	4.32	18.70 ± 5.41	3.12%
SGOT (AST) (U/l)	15.5 5 ± 2.45	2.63	15.75 ± 4.30	2.99%

TC= total cholesterol. TG= triglyceride. LDL= low density lipoprotein. HDL = high density lipoprotein. FBG= fasting blood glucose. SGPT= serum glutamate-pyruvate transaminase. SGOT= serum glutamate-oxaloacetate transaminase.* = Significant difference (P<0.05). ** = highly Significant difference (P<0.001). RK=Raspberry ketones.

Table 8: The Mean ± SD and percent of changes of anthropological parameter of control, L-Carnitine and raspberry ketones groups after 12 weeks intervention.

Groups	Age (year)	Weight (Kg)	BMI Kg/m2
Control (no=18)	32.72 ± 7.002	88.061 ± 9.383	34.833 ± 2.991
Control after intervention	_____	84.844 ±8.943	33.622 ± 2.756
Percent of reduction		3.79 %	3.58 %
L-carnitine (no=18)	33.11 ± 6.533	86.68 ± 6.930	34.588 ± 2.774
L-carnitine after intervention	_____	80.74 ± 6.440	32.205 ± 2.503
Percent of reduction	_____	7.38 %	7.41 %
Raspberry ketones (no=20)	31.75 ± 5.580	89.46 ± 9.014	35.415 ± 3.341
Raspberry ketones after intervention	_____	85.2 ± 9.258	33.723 ± 3.371
Percent of reduction	_____	5.10 %	5.08 %

DISCUSSION

The present study demonstrated that 1 g/day oral L-carnitine supplementation in obese women was able to reduce lipid profile. It has been suggested that L-carnitine has a useful effect on several diabetic risk parameters, including plasma lipids and lipoprotein[8]. This alteration could decrease triglycerides synthesis, and increase mitochondrial B-oxidation of fatty acids, Casciani support this opinion and found that L-carnitine decreases serum cholesterol, triglycerides, and free fatty acids[9], in the current study a significant decrease in total cholesterol, LDL-C, and triglycerides in patients who received L-carnitine supplementation was observed compared to the pretreatment group Table 3. These results were in agreement with Alipour results[10]. It was also consistent with those of El-Metwally[11] and Gonzalez-Ortiz[12], who reported that oral administration of L-carnitine improves dyslipidemia and decreases diabetic parameters. Reduction of serum hyper triglyceridemia in diabetic patients who consumed L-carnitine resulted in decrease of triglycerides synthesis in the liver or inhibition of triglyceride release from the liver. Moreover, L-carnitine induced significant reduction in total serum cholesterol in skeletal muscles of obese patients[13].

These results are in consistent with the present results, were a significant reduction in both case and control groups was observed, but the reduction was stronger and clinically valuable in the therapy group, which shows the role of L-carnitine supplementation in this regard. Nevertheless, another study reported no statistically significant effect of carnitine on serum triglyceride [14]. Chronic exercise can affect the concentration of total cholesterol in the plasma and its distribution in LDL and HDL and regular aerobic exercise involving considerable weekly energy expenditure increases HDL[15], this change in LDL was in consistent with the present results but the HDL was decreased rather than increase as in Table 2. However, in control group the HDL level reduced about 2.55 mg/dL in contrast to its level in L-carnitine group that increased, this finding was in consistent with Leenen results[16], he suggested that HDL cholesterol was significantly reduced on a weight-stable low-fat, low-saturated-fat diet both in males and in females. In a diet with a decreased percent of energy provided by total and saturated fat without energy restriction by obese subjects resulted in reductions of all serum lipid levels, including HDL cholesterol[17]. It has been suggested that a reduction in total fat intake may lower HDL cholesterol levels[18]. The study of Al-Zahrani has proved the ability of exercise in decreasing weight, body mass index and altering the lipid profile (decreasing the total cholesterol, triglycerides, low density lipoproteins and increasing high density lipoproteins)[19], that consistent with our results except with HDL-C. Other study suggested that triglyceride level has decreased and high density lipoproteins have increased in the individuals with hypercholesterol as a result of aerobic exercise[20]. Although some studies reported reduction in TC levels only[21]. Still other studies showed no changes in lipid profile[22]. Since TG and HDL-C are very important parameters for the evaluation of metabolic syndrome. A possible reason for these discrepant results may be that the clinical studies cited above analyzed data from independent samples.

Alshammari, in his study also indicated a decreased lipogram levels after the physical activity program and another decrease in lipogram when added L-carnitine with exercise except the HDL which increased due to carnitine rather than physical activity alone[23]. In this study the L-carnitine group differ significantly from the control group which is consistent with the study mentioned above. It has been estimated that L-carnitine is necessary for mitochondrial transport metabolism of long chain fatty acids, thus for myocardial energetic metabolism. Fatty acids cross mitochondrial membranes as acyl carnitine derivatives to enter pathways for oxidation, acylation, chain shortening or chain elongation-desaturation. Therefore, L-carnitine dependent fatty acid transfer is central to lipid metabolism; dietary supplementation of L-carnitine improves the utilization of fat providing marked reduction in plasma levels of triglyceride[24]. Moreover, Amin and Nagy had been postulated that L-carnitine administrated to obese rats reduced significantly serum hypertriglyceridemia via decrease synthesis of triglycerides by the liver or by inhibition of triglyceride release from the liver[25]. In the current study, the average reduction of FBG in L-carnitine group between pre and post treatment which was highly significant ($P < 0.001$), was in consistent with Alipour[10], who suggested that the weight loss due to oral administration of L-carnitine is associated with decrease glucose level because of elevated insulin sensitivity, thus decreasing insulin resistance in obese patients is due to regulating the cell energy metabolism or reducing free fatty acids. Also these results are in agreement with those of Gonzalez-Ortiz.[12].The inflammatory effect of cytokine release during diabetes is one of the causative agents for the insulin resistance; L-carnitine may reduce this effect of cytokines[26]. However, there was no significant difference in our results between the L-carnitine and control groups ($P > 0.05$) which may suggest that weight reduction and or exercise are the main determinants. Asymptomatic elevations in aminotransferase concentrations are common, particularly in individuals with risk factors for diabetes including overweight or obesity, or elevations in insulin and glucose levels[27]. Serum ALT levels are often used to screen for liver disease, including nonalcoholic fatty liver and NASH. However, patients with normal ALT levels can have fatty liver and the presence of ALT elevations does not reliably separate patients with pure fatty liver from those with NASH with accompanying ongoing inflammation and necrosis[28]. The American Gastroenterological Association medical position statement concluded that there is no definitive therapy for NAFLD, Although weight loss is recommended[29]. In this study the liver enzymes reduced (ALT 4.32%, $p < 0.05$) while the reduction in AST was not significant in L-carnitine group. Although usually the elevations in ALT and aspartate aminotransferase (AST) are typically mild when present and are usually not greater than four times the upper limit of normal in obese[30]. The ratio of AST/ALT is usually less than 1 in patients who have either no or minimal fibrosis, although this ratio may be greater than 1 with the development of cirrhosis[31]. These results was in consistent with the other study which found that administration of L-carnitine produces a significant lowering effect in the activity of AST and ALT in obese rats[25], Supplementation of L-carnitine is considered safe for doses up to 15 g/d in healthy men[32]. Therefore, the possibility that changes in markers of hepatic function in Iraqi obese woman after daily ingestion of a dose of L-carnitine of 1000 mg/d for 12 weeks to show no abnormalities was expected. This was in agreement with other study[33].

Little can be found about RK from a scientific perspective studies, possibly this is the first study that assess the effectiveness of Raspberry ketones on metabolic parameters. Lipid profile in RK treatment group have been differ significantly ($P < 0.001$) from the baseline. There were a decrement in total cholesterol, triglyceride and Low density lipoprotein while an increment in high density lipoprotein. Lopez have been reported that neither placebo nor (METABO) which is a multi-ingredient dietary supplement that contains primarily raspberry ketone administration affected concentrations of blood lipids, including cholesterol, HDL, LDL, cholesterol/HDL ratio and TG, although there was a strong trend ($p < 0.07$) for TG concentrations to decrease more in the METABO group compared to the placebo group[34]. These results were in consistent with our results. In strategies to prevent obesity, one of the key steps is to inhibit the digestion and absorption of the dietary fat. Morimoto, explore this strategy, they studied the effects of RK on fat absorption. RK at a concentration of 5% reduced the elevation of plasma triacylglycerol after oral administration of a lipid emulsion containing corn oil in rats, although a lower concentration of RK 1% elicited no such effect[35]. Whereas the same concentrations exhibited no such inhibitory effect when the trioleoylglycerol was emulsified with gum Arabic instead of lecithin in the same system. This means that the site of the inhibitory action of RK on trioleoylglycerol hydrolysis may be the substrate rather than the enzyme. Anyway, these results suggested that RK suppresses the dietary fat absorption by inhibiting the trioleoylglycerol hydrolysis[36]. Significantly, the inhibitory effect of RK on fat absorption is not the main anti obesity mechanism, because the minimum RK dose required to exert these effects is much higher than that required to exert anti obese effects[35]. In our study the reduction in body weight were slightly more in RK group compared to the control group and there were no significant differences between these two. This may be due to the small dose of RK that had been used in this study. On the other hand the higher dose may trigger the side effects on the liver of patients, however, in our study the transaminase enzymes (AST and ALT) reduced; significant reduction ($p<0.05$) for ALT and not for AST. These results was in agreement with Wang[37]. ALT is more sensitive to liver cell damage than AST, whereas AST can reflect the extent of damage to liver cells more precisely

than ALT[38]. It has been showed that RK can reduce fatty deposition of liver cells and promote cytostasis to decrease the degeneration and extent of necrosis in liver cells[37]. In present study the level of FBG were decreased significantly in treated group with RK which was in agreement with the previous study, they postulated that glucose level were significantly reduced after treatment with RK[41]. The theory of NASH pathogenesis mainly expresses insulin resistance (IR). IR has significant correlation with distribution of body fat. Accompanying the increase in adipose cell population, the sensitivity of the population degrades gradually, which is an obstacle in the normal pancreatic island–adipose cell axis feedback mechanism to generate hyperinsulinemia, while hyperinsulinemia can further worsen the blood lipid metabolic disorder. Wang, in his experiment found that insulin content and the insulin resistance index (IRI) of the NASH rat model greatly increased, whereas the insulin-sensitive index (ISI) decreased significantly. After treatment with RK, INS serum content, blood glucose level, and IRI of the model control group decreased significantly, whereas the ISI increased, indicating that IR is drastically improved by RK[37]. In contrast in other study the level of blood glucose increased slightly without statistically significant differences ($p < 0.60$)[34]. While in the present study there were no statistically significant difference ($p > 0.05$) between RK and control groups in reduction of blood glucose. However the percent of reduction was 6.64% in RK group. The comparison between L-carnitine and RK effect on the lipid profile reveled the superiority of L-carntine over RK in these parameters. It has been suggested that L-carnitine has a useful effect on several diabetic risk parameters, including plasma lipids and lipoprotein[39]. This alteration could decrease triglycerides synthesis, and increase mitochondrial B-oxidation of fatty acids. Other biochemical parameter like FBG, ALT and AST were not significantly different.

Further, the effect of two substances on the results of biochemical test were ranking as follows; L-carnitine more effective than RK, and RK more effective than control group. Except for FBG, where RK more effective than L-carnitine and control group.

CONCLUSION

According to the results presented in this study it is concluded that L-carnitine improves biochemical parameters associate obesity especially lipid profile and correct liver function better than RK, while the latter reduce blood glucose more.

REFERENCES

1 Iraq Ministry of Health (MOH), Ministry of Planning and Development Cooperation in collaboration with World Health Organization. (Chronic Non-Communicable Diseases Risk Factors Survey In Iraq - A STEP wise Approach), 2006.

2 Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. BMC Public Health, 9, 2009, 88.

3 Kassi E, Pervanidou P, Kaltsas G and Chrousos G. Metabolic syndrome: definitions and controversies. BMC Medicine, 9, 2011, 48.

4 Hasani-Ranjbar S, Larijani B, Abdollahi M: A systematic review of Iranian medicinal plants useful in diabetes mellitus. Arch Med Sci. 4, 2008, 285–292.

5 Thielecke F, Rahn G, Böhnke J, Adams F, Birkenfeld AL, Jordan J, Boschmann M: Epigallocatechin-3-gallate and postprandial fat oxidation in overweight/obese male volunteers: a pilot study. Eur J Clin Nutr, 64, 2010, 704–713.

6 Snitker S, Fujishima Y, Shen H, Ott S, Pi-Sunyer X, Furuhata Y, Sato H, Takahashi M: Effects of novel capsinoid treatment on fatness and energy metabolism in humans: possible pharmacogenetic implications. Am J Clin Nutr, 89, 2009, 45–50.

7 Yun JW: Possible anti-obesity therapeutics from nature–a review. Phytochemistry, 71, 2010, 1625–1641.

8 Sirtori CR, Calabresi L, Ferrara S, Pazzucconi F, Bondioli A, Baldassarre D. L-carnitine reduces plasma lipoprotein(a) levels in patients with hyper Lp(a). Nutr Metab Cardiovasc Dis, 10(5), 2000, 247–251.

9 Casciani CU, Caruso U, Cravotto E, Corsi M, Pola P, Savi L. EFFECT of l-carnitine on lipid pattern in haemodialysis. Lancet, 316(8207), 1980, 1309–1310.

10 Alipour B, Barzegar A, Panahi F, Safaeian A, Es. Haghi M. Effect of L-Carnitine Supplementation on Metabolic Status in Obese Diabetic Women with Hypocaloric Diet. Health Scope, 2(4), 2014, e14615.

11 El-Metwally TH, Hamed EA, Ahmad AR, Mohamed NA. Dyslipidemia, oxidative stress and cardiac dysfunction in children with chronic renal failure: effects of L-carnitine supplementation. Ann Saudi Med. 23(5), 2003, 270–277.

12 Gonzalez-Ortiz M, Hernandez-Gonzalez SO, Hernandez-Salazar E, Martinez-Abundis E. Effect of oral L-carnitine administration on insulin sensitivity and lipid profile in type 2 diabetes mellitus patients. Ann Nutr Metab, 52(4), 2008, 335–338.

13 Rajasekar P, Anuradha CV. Effect of L-carnitine on skeletal muscle lipids and oxidative stress in rats fed high-fructose diet. Exp Diabetes Res, 2007, 2007, 72741.

14 Shakeri A, Tabibi H, Ossareh S, Neyestani T. Effects of L-Carnitine Supplementation on Serum Lipids and Apoproteins in Hemodialysis Patients with Lp(a) Hyperlipoproteinemia. Nutrition Sciences & Food Technology, 2(2), 2007, 1-14.

15 Mougios V. Exercise Biochemistry. Champaign, IL: Human Kinetics, 2006.

16 Leenen R, Kooy K, Meyboom S, Seidell JC, Deurenberg P, and Weststrates JA. Relative effects of weight loss and dietary fat modification on serum lipid levels in the dietary treatment of obesity. J. Lipid Res, 34, 1993, 2183-2191.

17 Erkelens D W. 1989. Cholesterol consensus in the Netherlands. Euz J. Clin. Nutr, 43, 1989, 89-96.

18 Grundy SM, Brewer HB Jr, Cleeman JI, Smith SC Jr, Lenfant C. Definition of Metabolic Syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association Conference on Scientific Issues Related to Definition. Circulation, 109, 2004, 433-438.

19 Al-Zahrani SS. Effect of diet control and exercise on the lipid profile of obese men. International Journal of Research in Medical Sciences Al-Zahrani SS. Int J Res Med Sci, 2(1), 2014, 95-99.

20 Haskell WL, Leon AS, Caspersen CJ, Froelicher VF, Hagberg JM, Harlan W, Holloszy JO, Regensteiner JG, Thompson PD, Washburn RA. Cardiovascular benefits and assessment of physical activity and physical fitness in adults. Medicine and science in sports and exercise, 24, 1992, S201-20.

21 Ginsberg H, Plutzky J & Sobel BE. A review of metabolic and cardiovascular effects of oral antidiabetic agents: beyond glucose level lowering. Journal of Cardiovascular Risk, 6, 1999, 337-346.

22 Nobili V, Manco M, Ciampalini P, Alisi A, Devito R, Bugianesi E. Metformin use in children with nonalcoholic fatty liver disease: an open-label, 24-month, observational pilot study. Clin Ther, 30(6), 2008, 1168-1176.

23 Alshammari N M. The Effect of L-Carnitine and Physical Activity on Adipocytokines and Lipid Profile in Obese Women. World Journal of Sport Sciences, 4(1), 2011, 21-23.

24 Ramsay RR. The carnitine acyltransferases: modulators of acyl-CoA-dependent reactions. Biochem Soc Trans, 28(2), 2000; 182-186.

25 Amin KA, Nagy MA. Effect of Carnitine and Herbal Mixture Extract on Obesity Induced by High Fat Diet in Rats. Diabetology & Metabolic Syndrome, 1(17), 2009, 1-14.

26 Rao NK, Nammi S. Antidiabetic and renoprotective effects of the chloroform extract of Terminalia chebula Retz. seeds in streptozotocin induced diabetic rats. BMC Complement Altern Med, 6, 2006, 17-25.

27 Clark JM, Brancati FL, Diehl AM. The prevalence and etiology of elevated aminotransferase levels in the United States. Am J Gastroenterol, 98, 2003, 960–967.

28 Mofrad P, Contos MJ, Haque M. Clinical and histologic spectrum of nonalcoholic fatty liver disease associated with normal ALT values. Hepatology, 37, 2003, 1286–1292.

29 American Gastroenterological Association medical position statement (AGA): nonalcoholic fatty liver disease. Gastroenterology, 123, 2002, 1702–1704.

30 Torres DM, Harrison SA. Diagnosis and therapy of nonalcoholic steatohepatitis. Gastroenterology, 134(6), 2008, 1682-1698.

31 Obika M and Noguchi H. Diagnosis and Evaluation of Nonalcoholic Fatty Liver Disease. Exp Diabetes Res, 27, 2012, 145-159.

32 Borzelleca J F, Munro I C and Thomas JA. "Evaluation of L-Carnitine for GRAS Status," Report for Lonza Inc. Switzerland, 2011.

33 Odo S, Tanabe K, Yamauchi M. A Pilot Clinical Trial on L-Carnitine Supplementation in Combination with Motivation Training: Effects on Weight Management in Healthy Volunteers. Food and Nutrition Sciences, 4, 2013, 222-231.

34 Lopez HL, Ziegenfuss TN, Hofheins JE, Habowski SM, Arent SM, Weir JP and Ferrando AA. Eight weeks of supplementation with a multi ingredient weight loss product enhances body composition, reduces hip and waist girth, and increases energy levels in overweight men and women. *J Int Soc Sports Nutr,* 10, 2013, 1–22.

35 Morimoto C, Satoh Y, Hara M, Inoue S, Tsujita T, Okuda H. Anti-obese action of raspberry ketone. Life Sci, 77, 2005, 194–204.

36 Tsujita T, Matsuura Y, Okuda H. Studies on the inhibition of pancreatic and carboxylester lipases by protamine. Journal of Lipid Research, 37, 1996, 1481–1487.

37 Wang L, Meng X, Zhang F. Raspberry ketone protects rats fed high-fat diets against nonalcoholic steatohepatitis. *J Med Food*. 2012.

38 Adams LA, Angulo P, Lindor KD. Nonalcoholic fatty liver disease. Can Med Assoc J, 173, 2005, 734–745.

39 Sirtori CR, Calabresi L, Ferrara S, Pazzucconi F, Bondioli A, Baldassarre D. L-carnitine reduces plasma lipoprotein(a) levels in patients with hyper Lp(a). Nutr Metab Cardiovasc Dis, 10(5), 2000, 247–251.

Source of Support: Nil, **Conflict of Interest:** None.