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Effect of L-Carnitine Supplementation on Some Biochemical Parameters in Blood Serum of Sedentary Population

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Because of its role in the transport of fatty acids from cytosol into mitochondrion, the consumption of L-carnitine became popular among athletes, and/or as a mass loss supplement. In an attempt to obtain more data on the effect of L-carnitine supplementation on some biochemical parameters in blood serum, a double-blind, placebo-controlled study was carried. Healthy volunteers with declared sedentary activities received 2 g/day of either L-carnitine or placebo for 2 weeks. L-carnitine administration did not induce statistically significant changes in blood serum concentrations of glucose, triacylglycerols, total cholesterol, HDL cholesterol and creatinine, nor affected the activity of analysed enzymes (AST, ALT, LDH, and CK). The only observed effect was a decrease in the concentration of free fatty acids in the serum from 0.439 mmol dm^{-3} at the beginning to 0.279 mmol dm^{-3} at the end of the experiment. Body mass reduction was not achieved. We conclude that L-carnitine supplementation cannot be used for body mass reduction *per se*, but might be involved in energy utilisation.

Keywords
L-carnitine
body mass reduction
sedentary population
free fatty acids

INTRODUCTION

L-Carnitine (3-hydroxy-4-*N*-trimethylaminobutirate) plays an important role in lipid metabolism. In order to be used as a source of energy, long chain fatty acids have to be transferred into the mitochondria for oxidation. This transport, mediated by the carnitine-palmitoyltransferase (CPT) enzymatic system (CPT I, carnitine-acylcarnitine translocase and CPT II), is considered to be the rate-limiting step in β-oxidation.[1] L-Carnitine can be synthesized from the amino acids lysine and methionine in liver, kidney and brain, with iron and vitamins B_3, B_6 and C as co-factors,[2] or it can be ingested with food. Diet intake is highly variable and the major sources are red meat, fish and dairy products. For healthy omnivores the intake has been estimated to be from 20 to 300 mg per day,[3–5] while for strict vegetarians it is in the range of 1–3 mg per day.[6] Therefore, in non-vegetarians approximately 75 % of carnitine sources come from the diet, while in vegetarians more than 90 % of the requirements have to be obtained from endogenous synthesis.[7] Carnitine is taken primarily by cardiac and skeletal muscle tissues, which contain ap-

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proximately 98 % of the body stores. Since human body can synthesize L-carnitine, it is not considered to be an essential compound, although insufficient data exist on the carnitine biosynthetic capacities in humans. Low tissue levels typical in neonates fed with diets low in carnitine (*i.e.* soy-based formulas) indicate that they may have limited capacities for the synthesis of carnitine.[8] Primary carnitine deficiency is usually the result of innate errors in metabolism characterized by the lack or impaired endogenous synthesis, or of a defect that results from the absence of carnitine transporters.[9–12] Tissue depletion of carnitine has also been reported as the result of severe liver disease, renal disease (hemodialysis), malabsorption, drug therapy such as valproate and others.[13–15] Many of them are often associated with cardiomyopathy.[16]

The importance of carnitine in lipid metabolism, and the fact that fat oxidation can be increased when the availability of fatty acids is increased, raised the hypothesis that increasing the carnitine content in skeletal muscle may improve fatty acid oxidation during exercise. Using fatty acids as energy source during prolonged exercise may be of benefit to endurance of the athletes by sparing muscle glycogen and thus increasing exercise capacity. Furthermore, based on these facts, it has been suggested that carnitine could act as »fat burner« by increasing the supplementation of fatty acids as a substrate for β-oxidation, thus reducing its availability for storage. For these reasons, carnitine became one of the most popular dietary supplements, being used by athletes and/or as a mass loss supplement, but with equivocal results.

Recently, a few good reviews on this topic have been published, showing that most of the researches have been performed on carnitine effects under specific conditions, *i.e.* endurance training, reduced energy diet, metabolic disorder *etc*.[17–19] Since carnitine supplementation is freely used even by those who are looking for a »magic pill« to reduce the body mass, unwilling to make any additional effort by taking up physical activity or by reducing ingested food, we have undertaken this research in order to check the effects of additional carnitine intake on some biochemical parameters in blood serum of healthy non-vegetarian individuals with average nutritional intake and everyday routine. Blood serum concentrations of some metabolites, mainly lipids, were measured, along with the serum activities of certain enzymes, indicators of the liver and muscle tissue condition, in order to see whether carnitine supplementation *per se* causes any recordable changes in otherwise healthy individuals.

EXPERIMENTAL

Subjects

A double-blind, placebo-controlled study was carried out on 30 healthy volunteers with declared sedentary activities. The study group consisted of 18 females and 12 males, aged from 18 to 32, who were on no medications and had no current illness. At the beginning of the study, all subjects completed a general health questionnaire, and gave their written consent. The study protocol was approved by the Medical School Ethics Commeetee, Zagreb, Croatia.

Procedure

Volunteers were divided randomly into two groups: experimental group received 2 x 1 g of L-carnitine (as L-carnitine-L-tartarate, Carni-X, Scitec Nutrition, Orlando, USA) per day, administered in the morning before the first meal, and in the afternoon, during two weeks. Carnitine was partially donated by Sport Line Ladanyi plc., Zagreb, Croatia. Placebo group was treated in the same way but with a nutritional starch packed into identically appearing capsules, the gift of Razvitak plc., Ludbreg, Croatia. The height and the mass of the subjects were measured at the beginning and at the end of the study. Fasting morning blood samples were collected on four occasions during the experiment: at the beginning (day 0) and again on days 5, 10 and 15. Throughout the experiment the subjects practiced their usual eating pattern and kept daily routine which they declared to be sedentary *i.e.* without regular physical activity or exercise. They were asked to make note of all the food taken during the day for two weeks of the experiment.

Sample Collection and Analysis

Samples of 5 mL volume of venous blood were drawn after a 12-hour overnight fast. Serum was removed after centrifugation at 3500 *g* for 30 min and stored at 4 °C or frozen at –80 °C until evaluation. The serum concentrations of glucose (GLC), triacylglycerols (TG), total cholesterol ($CHOL_{tot}$), HDL cholesterol (HDL-CHOL), and free fatty acids (FFA) were measured enzymatically using commercial kits (Herbos Dijagnostika plc., Sisak, Croatia, for GLC-PAP, $CHOL_{tot}$ and HDL-CHOL, and Roche Diagnostics GmbH, Penzberg, Germany for FFA). Activities of the enzymes alanine aminotransferase (ALT), aspartate aminotransferase (AST), creatine kinase (CK) and lactate dehydrogenase (LDH), as well as creatinine concentration were determined on Olympus AU 2700. All the analyses were performed in duplicate.

Statistical Analyses

All data are reported as mean ± SD. For statistical analysis Student's *t*-test was used to compare the data for blood parameters between the first and the last measurement in each group, and between the control and the treated group for each parameter. The resultes were also analysed by Two-Way ANOVA test for treatment and time. Statistical significance was declared at $P<0.05$.

RESULTS AND DISCUSION

Thirty-six subjects without noticeable disease took part in the study. Six volunteers failed to complete the study

TABLE I. Data on age, height, body mass, body mass index (BMI) and daily energy intake of the individuals treated with placebo and L-carnitin [a]

	Placebo	Carnitine	Total
Subjects (male)	12 (4)	18 (8)	30 (12)
Age / year	21.3 ± 2.6	23.1 ± 3.5	22.5 ± 3.3
Height / m	1.72 ± 0.08	1.75 ± 0.08	1.74 ± 0.08
Initial body mass / kg	71.2 ± 18.3	69.7 ± 10.2	70.4 ± 14.0
Initial BMI / kg m^{-2}	23.8 ± 5.2	22.7 ± 2.7	23.2 ± 3.9
Final body mass / kg	70.7 ± 19.2	69.9 ± 10.7	70.2 ± 14.5
Final BMI / kg m^{-2}	23.9 ± 5.5	22.7 ± 2.9	23.2 ± 4.1
Energy intake / kJ d^{-1}	8580 ± 1400	8850 ± 3070	8670 ± 2360

[a]All values are expressed as mean ± SD.

due to: acute illness (3), family problems (2) and one did not follow through because of gastrointestinal discomfort. Data on age, gender, height, mass and body mass index (BMI) of thirty individuals who completed the study are given in Table I.

There are many dietary recommendations for mass loss ranging from low fat or even fat-free diets to very high fat, ketogenic, diets. Exercise is regularly recommended not only as a method for body mass reduction, but also as every day requirement. Unfortunately, to our knowledge, the most frequently desired and used methods are those prescribing »magic« food supplements and doing nothing. Along with the increased prevalence of obesity and accompanying disorders like diabetes, coronary heart disease *etc.*, an increase in commercially available food supplements declared as body mass reducers is obtained. The review of such formulas prepared by Saper *et al.*[20] revealed the fact that most of them are of disputable results and their influence on metabolism unclear, and therefore should be used with caution.

This study evaluated the effects of L-carnitine supplementation, with no other interventions, on body mass reduction/maintenance, and more importantly, on some biochemical parameters in blood serum as indicators of metabolic state. As shown in Table I there was no significant loss of body mass in either placebo- or carnitine-treated group and consequently no changes in BMI were calculated. Other authors also failed to show additional body mass loss with carnitine supplementation.[21] Aoki *et al.*[22] and Brandsch and Eder[23] showed that energy-reduced diet and exercise, but not carnitine supplementation, resulted in marked mass reduction.

Because of its role in intracellular fatty acid transport, L-carnitine was most frequently investigated in the context of lipid metabolism. Our results did not reveal statistically significant differences in blood serum levels of triacylglycerols (Figure 1), total cholesterol (Figure 2) and HDL cholesterol (Figure 3) between subjects from

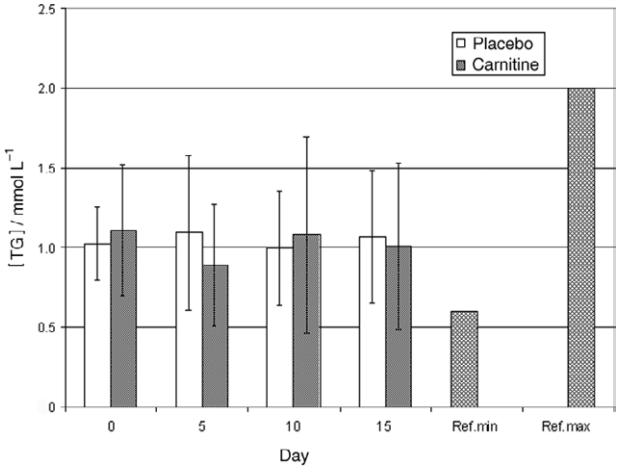

Figure 1. Concentration of triacylglycerols (TG) in blood serum of the subjects treated with placebo and L-carnitine (2 g/d) compared to reference values. All values are expressed as mean ± SD; no significant differences were found.

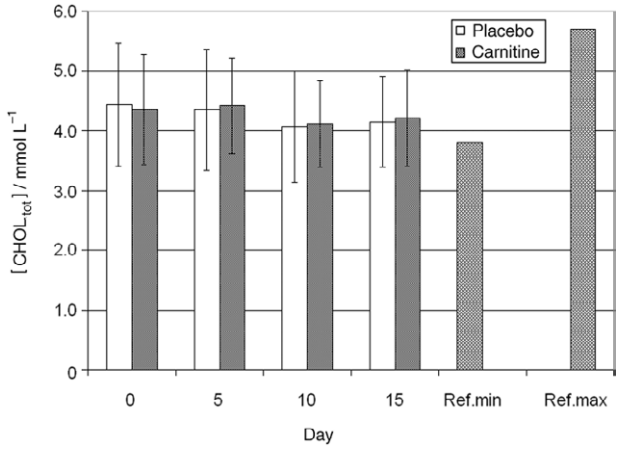

Figure 2. Concentration of total cholesterol ($CHOL_{tot}$) in blood serum of the subjects treated with placebo and L-carnitine (2 g/d) compared to reference values. All values are expressed as mean ± SD; no significant differences were found.

TABLE II. Activity of AST, ALT, LDH and CK (expressed in U L^{-1})[a] and the concentration of glucose (expressed in mmol L^{-1}) and creatinine (expressed in μmol L^{-1}) in serum of the individuals treated with placebo (P) and L-carnitine (C)

Variable	Treatment	Activities or concentrations[b]				Reference values
		Day				
		0	5	10	15	
AST	P	19.0 ± 3.1	18.3 ± 2.1	22.1 ± 4.5	19.6 ± 3.0	M 11–38
	C	18.7 ± 3.3	21.2 ± 1.7	23.7 ± 6.7	18.4 ± 4.4	F 8–30
ALT	P	12.1 ± 4.4	13.8 ± 4.1	15.8 ± 7.2	15.3 ± 4.9	M 12–48
	C	13.2 ± 3.5	14.5 ± 7.9	16.8 ± 8.0	15.4 ± 8.5	F 10–36
LDH	P	151.0 ± 23.9	138.5 ± 23.3	169.6 ± 23.8	141.0 ± 18.7	<460
	C	158.9 ± 14.4	152.4 ± 46.6	187.2 ± 37.9	137.0 ± 16.4	
CK	P	90.6 ± 30.2	97.0 ± 35.5	98.0 ± 34.6	125.4 ± 73.4	M<205
	C	98.6 ± 39.8	98.6 ± 47.1	105.7 ± 44.8	107.8 ± 50.8	F<175
Glucose	P	4.73 ± 0.55	4.49 ± 0.42	4.76 ± 0.58	4.31 ± 0.65	3.8–5.9
	C	4.58 ± 0.49	4.33 ± 0.39	4.46 ± 0.61	4.57 ± 0.59	
Creatinine	P	78.5 ± 9.9	90.1 ± 12.2	99.9 ± 10.9	93.4 ± 14.4	35–115
	C	79.1 ± 10.3	90.3 ± 13.0	102.5 ± 16.0	89.5 ± 11.9	

[a] One unit presents one μmol min^{-1}. [b]All values are expressed as mean ± SD.

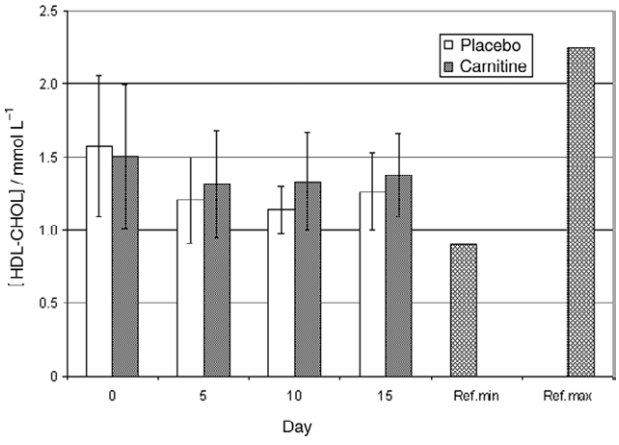

Figure 3. Concentration of HDL cholesterol (HDL-CHOL) in blood serum of the subjects treated with placebo and L-carnitine (2 g/d) compared to reference values. All values are expressed as mean ± SD; no significant differences were found.

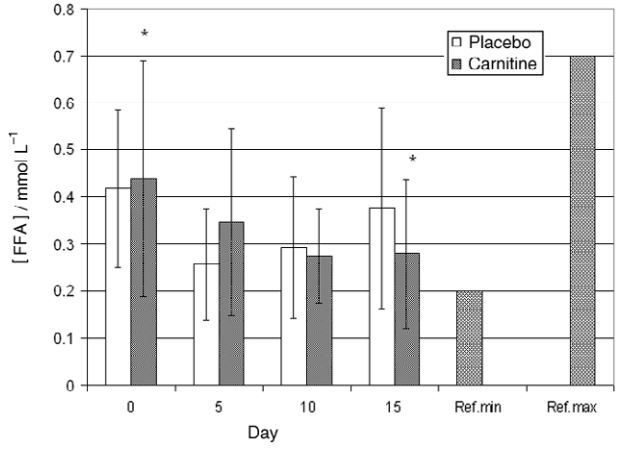

Figure 4. Concentration of free fatty acids (FFA) in blood serum of the subjects treated with placebo and L-carnitine (2 g/d) compared to reference values. All values are expressed as mean ± SD; asterisk (*) denotes $P<0.05$.

the placebo and the carnitine group. The same results were obtained in our previous experiment conducted in the same way but with aerobically trained athletes.[24] According to some other studies, exogenous carnitine administration reduces plasma lipids in hyperlipidaemic rat and rabbits.[25–28] However, in normolipidaemic rats and humans this was not the result, not even as a cumulative effect of exercise or a hypocaloric diet.[22,23,29,30] In this study, the only statistically significant alteration caused by L-carnitine supplementation was the decrease in free fatty acids (FFA) concentration (Figure 4). Although the values measured for all individuals from both placebo and carnitine group did not exceed reference values by any means, statistical analysis revealed significant decrease after 2 weeks of L-carnitine supplementation compared to the initial value. This might indicate an increased cellular uptake of fatty acids from blood circulation and their transport into mi tochondria for β-oxidation. However, in our experiment with athletes, L-carnitine supplementation resulted in an increased serum concentration of FFA, assuming impaired fatty acid utilization. Nevertheless, having in mind the main regulatory mechanisms in mitochondrial metabo-

lism, including β-oxidation, Krebs' cycle and respiratory chain, it is hard to believe that these processes are activated simply by carnitine supplementation and without increased energy consumption. Furthermore, free, nonesterified fatty acids present in blood circulation originate mostly from adipose tissue as a result of increased triacylglycerol hydrolysis, catalyzed by hormone sensitive lipase. Therefore, more reasonable seems the hypothesis that the decrease in FFA concentration is the result of increased esterification with available L-carnitine instead of improved FA utilization. As shown in Figure 4, the concentration of FFA in the control group decreased on day 5 even more than for treated group after two weeks, but statistical analyses did not show the significance at the level of $P<0.05$. However, further analysis was performed by Two-Way ANOVA test for treatment and time. This test confirmed the differences inside the treated group at the beginning and at the end of the experiment, but failed to confirm differences between the control and the treated group. Therefore we concluded that the observed effect can not be explained exclusively by carnitine supplementation, and that other impact factors like diet should be considered.

These results are also in agreement with other studies, which failed to demonstrate improved lipid metabolism. Some authors offered increased glucose oxidation as an explanation for the obtained positive effects of carnitine supplementation.[31]

Commercially available preparations of carnitine differ in dosage and purity. Formulas with racemic mixtures contain, besides naturally occurring L-carnitine, the same amount of D-carnitine. In human body only L-carnitine is utilized, while D-form is not simply unused, but can induce toxic effects.[32] Most of the consumers are not informed about these risks and quite a number of professionals responsible for administering such information is not aware of the hazards. Since these formulas are usually cheaper, additional efforts should be done to improve education and to eliminate such preparations. Although the preparation used in this research was declared as L-carnitine, we decided to measure the activity of some enzymes in blood serum in order to screen for possible side effects. We measured the activities of alanin aminotransferase (ALT), aspartate aminotransferase (AST), lactate dehydrogenase (LDH) and creatine kinase (CK). Concentrations of glucose and creatinine were also measured. The obtained results are shown in Table II, along with reference values, without statistically significant differences found between the two groups.

These results are encouraging for those patients who are dependent on carnitine supplementation. In clinical studies, positive effects of carnitine therapy are unquestionable in the treatment of patients with metabolic disorders like carnitine uptake defect,[33] cardiac patients[34,35] and in hemodialysis patients.[36] More recent research has provided evidence of links between L-carnitine and antioxidant status. The potential antioxidant role of carnitine supplementation is thought to work through reduced lipid peroxidation, reduced ammonia toxicity, increased vitamin C availability or other mechanisms.[37,38] Furthermore, some authors suggest the involvement of carnitine in the process of apoptosis by regulation of caspase activity.[39] However, detailed mechanisms of carnitine activity are still to be established.

CONCLUSIONS

The results of this study show that carnitine supplementation fails to reduce the body mass in healthy subjects without additional interventions like exercise or reduced energy intake. Intake of 2 g of L-carnitine per day does not affect blood serum concentrations of triacylglycerols, total cholesterol, HDL cholesterol, glucose and creatinine. The applied preparation was well tolerated with no changes in the activity of the enzymes characteristic for liver and muscle metabolism, but subsequent metabolic consequences are still a matter for discussion.

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SAŽETAK

Utjecaj L-karnitina na neke biokemijske parametre u serumu osoba sjedilačkog načina života

Ivančica Delaš, Tonko Dražić, Melita Čačić-Hribljan i Krešimir Sanković

Zbog svoje uloge u transportu masnih kiselina iz citosola u mitohondrij, korištenje L-karnitina postalo je popularno među sportašima i/ili kao sredstvo za mršavljenje. Kako bismo utvrdili učinak L-karnitina na neke biokemijske parametre u serumu, proveli smo dvostruko slijepu studiju uz kontrolu placebom. Zdravi dobrovoljci sjedilačkog načina života primali su dnevno 2 g L-karnitina ili placebo kroz dva tjedna. Primjena karnitina nije uzrokovala promjene u koncentraciji glukoze, triacilglicerola, ukupnog kolesterola, HDL-kolesterola i kreatinina u serumu ispitanika, kao ni promjene aktivnosti ispitivanih enzima (AST, ALT, LDH, CK). Jedini značajni učinak karnitina bilo je smanjenje koncentracije slobodnih masnih kiselina s 0.439 mmol dm^{-1} na početku na 0.279 mmol dm^{-1} na kraju eksperimenta. Smanjenje tjelesne mase nije postignuto. Temeljem rezultata zaključeno je da se karnitin ne može samostalno koristiti kao sredstvo za smanjenje tjelesne mase, ali ima utjecaja na iskorištavanje energije u organizmu.