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Chronic Carnitine Ingestion Does not Affect Carbohydrate Metabolism Determinatives and Aerobic Capacity

Short Running title
Glucose metabolism in relation to carnitine supplementation

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ABSTRACT

Background and objective: Carnitine (LC) plays an essential metabolic role in transferring the long chain fatty acids into the mitochondrial matrix for subsequent beta-oxidation, although the molecular mechanisms for this are less understood. In this study, we investigated the effect of chronic L-carnitine supplementation on glucose metabolism and aerobic capacity during submaximal cycling in none-trained boy students. **Method:** In a randomized, placebo-controlled, double-blind study, 34 none-trained healthy male students divided into experimental and control groups by randomly. All participants cycled for 20 min steady state cycling at 70%VO2max before and after 3 week (3g daily) L-carnitine (experimental) and placebo (control) supplementation. A venous blood sample was collected from all the subjects immediately followed up each exercise for measuring of glucose and Lactate concentration, lactate dehydrogenase activity (LDH). Statistical analysis was performed with the SPSS software version 15.0 using an independent paired t-test ($P<0.05$). **Result:** No significant change in plasma glucose, lactate and LDH activity were observed by L-carnitine supplementation in experimental group ($p\geq0.05$). Moreover, aerobic capacity and heart rate remained without changes in these subjects ($p\geq0.05$).All variables were unaffected in the control subjects ($p\geq0.05$). **Discussion:** Our data strongly suggest that chronic L-carnitine supplementation does not effect on carbohydrate metabolism or aerobic capacity in healthy subjects.
KEYWORD: Glucose Metabolism, Lactate, L-carnitine ingestion, cycling.

INTRODUCTION

Muscle and liver glycogen reserves can provide only 1200 to 2000 kcal of energy; while body fat reserves can generate an amount of energy approximately equivalent to 70,000 to 75,000 kcal [1]. At the beginning of exercise the major share of energy is generated through carbohydrate metabolism; and fat represents a smaller share than does carbohydrate. With prolonged activity, especially aerobic activities the share of fat in energy generation increases and the share of carbohydrate decreases [2].

Due to limited body carbohydrate reserves, if energy production, especially in prolonged exercises, is more reliant on carbohydrate reserves than fat, it leads to premature depletion of muscle and liver glycogen reserves and this would lead to early fatigue the outcome of which would be the halt of athletic activity [2]. On the other hand, the continuation of fat oxidation is dependent on maintaining glycogen reserves in order to keep the aerobic cycle [2]. Body fat reserves, however, are regarded as inexhaustible energy sources during exercise. Hence, many contemporary studies by biochemistry and physiology scholars focus on identification of suitable strategies to increase the share of energy produced by fats and reduce reliance on body on carbohydrate during exercise, especially during prolonged aerobic activity. Different ways, such as caffeine or carnitine supplementation or consumption of triglyceride-containing solutions have been studied for this purpose [3, 4].

Carnitine (**L-3-hydroxy-4-trimethylamino butyrate**) is an amino acid synthesized in mammals from lysine and methionine essential amino acids or is absorbed through the diet [5]. L-carnitine is the physiologically active form of it [6]. The role of carnitine is to transport free fatty acids into mitochondrial matrix for the purpose of Beta-oxidation process [7]. Hence, there is always the question whether L-carnitine supplementation leads to increase transport of fatty acids into the mitochondria or enhance athletic performance. Research results concerning the effect of L-carnitine on metabolism cycle and the level of energy generation of fat or Carbohydrate and athletic performance are contradictory.

In this area, the study of *Broad* (2005) showed that 4 weeks of L-carnitine supplementation (3g/day) would have no effect on endurance performance, substrate consumption and fat or Carbohydrate oxidation and their metabolites during sub-maximal exercise [8]. Findings by Lee (2007) and Erglu (2008) also indicate that lactate, glucose, respiratory exchange ratio, VO2max, oxygen pulse, lactate threshold remain unchanged by L-carnitine supplementation [9, 10]. But the studies of

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Abramowicz (2005), Mueller (2002) and Borghi (2006) refer to boosted fat oxidation and athletic performance and decreased lactate and heart rate and decreased glucose consumption by L-carnitine supplementation during sports activities [11, 12, 13]. A review of other research studies also indicates that the heterogeneity of results in this field. Hence, the aim of this study is to assess the impact of supplementation L-carnitine (3 g daily, 3 weeks) on certain determinatives of carbohydrate and lipid metabolism such as glucose and lactate and athletic performance in a group of Saveh University students.

MATERIALS AND METHODS

This randomized, placebo-controlled, double-blind study was approved by the Ethics Committee of Islamic Azad University, Saveh. The purpose of this study was to determine effect of chronic L-carnitine supplementation on some carbohydrate metabolism determinatives such as glucose, Lactate and lactate dehydrogenase activity and maximal oxygen consumption (VO2max) during cycling test on stationary leg ergometer in boy students. For this purpose, thirty four none-trained male students of Saveh University with an age range of 20±3 years and weight range of 77±11 kg participated in study by randomly. Then, these participants were divided into experimental (L-carnitine supplementation) and placebo (Lactose supplementation) groups. Each participant received written and verbal explanations about the nature of the study before signing an informed consent form. Subjects included individuals with no cardiovascular diseases, gastrointestinal diseases, kidney and liver disorders or diabetes. All subjects were non-smokers. The exclusion criteria were infections, renal diseases, hepatic disorders, use of alcohol. In addition, exclusion criteria included inability to exercise and supplementations that alter carbohydrate-fat metabolism.

In this study, at first, resting heart rate (HR) was measured after a 15-min rest in a sitting position and in a quiet environment. Then, all participants of experimental and control groups were completed a steady state cycling test according to Astrand protocol guideline (work load = 98 Watt) for 20 minutes on cycle ergometer (F90 Tuntury, Finland) [14]. Immediately after the test, venous blood samples were taken from subjects in order to measures the concentrations of lactate and glucose factors and lactate dehydrogenase activity (pretest). In exercise test, at first each subject pedaled for 2 minutes on a cycle ergometer without any load. Then the main phase of the test was carried out at the pedaling speed of 50 rpm and the work intensity of 98 watts. Work intensity rate and pedaling speed remained constant during the test. The duration of implementation of this test was considered 6 minutes for the purpose of calculating the maximum oxygen consumption. In order to activate the oxidative mechanism, the test was continued up to 20 minutes. Daily food records were kept for 48 h preceding each test session, and subjects were instructed to refrain from caffeine consumption and intense physical activity for 24 h before testing. No difference was observed in the subjects' diets 48 h before each trial.

In next stem, the participants of experimental group ingested oral L-carnitine (3g daily / 3 weeks) and control group ingested placebo (Lactose). All participants were asked to divide regular exercise in this period. After 3 weeks supplementation, all participants completed cycling protocol and blood samplings were immediately collected after cycling test (post-test). Maximum oxygen consumption was calculated by Astrand nomogram [17]. Measurement of serum glucose, by glucose oxidase enzymatic method was done using Pars Azmoon Test Kit made in Iran. Lactate and lactate dehydrogenase values were measured by Kobas Auto-analyzer with kits manufactured by Randox Company of England.

STATISTICAL ANALYSIS: Statistical analysis was performed with the SPSS software version 15.0. Comparisons of parameters between the two groups in first exercise test were made by independent sample T-test. Paired Student T-test was used to determine significance levels of changes in any of the variables in response to chronic supplementation compared to first blood sampling in two groups. P-value less than 0.05 were considered statistically significant.

RESULTS

Table 1 show the descriptive physiological and biochemical features in pre and post-test of the study groups. The finding of independent sample T-test showed no significant differences in all variables between experimental and control groups in pretest ($P<0.05$). Our finding also showed that Plasma glucose levels did not change by L-carnitine supplementation in experimental group ($P<0.05$). On the other hand, blood glucose did not change during cycling exercise after 3 weeks L-carnitine supplementation. In addition, Blood lactate concentration and lactate dehydrogenase activity did not change significantly during cycling test in experimental subjects ($P<0.05$). No differences in resting heart rate and VO2max were observed after supplementation compared to their respective baseline values ($P<0.05$). All variables remained without change in control subjects ($P<0.05$).

Table 1: Mean and standard deviation of physiological and biochemical variables of pre and post-test of two studied groups

Variable	Control		Experimental	
	Pretest	Post-test	Pretest	Post-test
Glucose (mg/dL)	99 ± 11	98 ± 13	97 ± 14	101 ± 9
Lactate (mmol/L)	5.68 ± 0.65	5.33 ± 0.39	5.44 ± 0.62	5.17 ± 0.48
LDL (U/L)	298 ± 58	323 ± 63	311 ± 38	298 ± 68
VO2max (ml/kg/min)	34 ± 7	35 ± 9	32 ± 7	33 ± 6
Rest heart rate (bpm)	**77 ± 11**	**75 ± 9**	**78 ± 14**	**75 ± 11**

DISCUSSION

In recent years the use of L-carnitine by athletes has widely increased. One reason for making use of carnitine is that its supplementation causes no side effects [15]. Reduction of blood lactate accumulation during certain sports activities signifies more dependence of energy generation on aerobic metabolism, especially lipid metabolism [2]. Some studies have shown that L-carnitine congestion leads to reduce lactate accumulation during exercise and consequently improves exercise performance [16, 17]. But in other studies, long-term L-carnitine supplementation did not cause any significant change in blood lactate concentration during exercise [18, 19]. It is also believed that L-carnitine supplementation decreases lactate dehydrogenase activity which returns lactate to pyruvate and consequently reduces lactate accumulation during exercise [20]. The findings of our study showed that L-carnitine supplementation for 21 days would cause no change in lactate concentration and lactate dehydrogenase activity during exercise test.

The studies of Stephen (2007) and Abramowicz (2005) note that the increase in muscle carnitine content by its supplementation in healthy individuals reduces carbohydrate oxidation and increases the body's glycogen reserves and blood glucose reserves [7,11]. Despite these findings, Broad study (2005) showed that L-carnitine supplementation for three months would bring about no change in glucose concentrations, carbohydrate oxidation and its substrates [8]. Also the study of Panjwani (2007) on rats indicates no effect of a 25-day L-carnitine supplementation on plasma glucose levels during exercise [21]. Our study findings also showed that L-carnitine supplementation would not lead to significant change in glucose concentration.

Increased VO2max or decreased resting/exercise heart rate due to prolonged exercise or supplementation with ergogenic aids represents enhanced athletic performance or aerobic fitness of the athlete, non-athletes or patients [1]. In this context, the studies of Borghi (2006) as well as some other studies indicate a significant reduction in heart rate by prolonged L-carnitine supplementation [13, 22]. Prolonged use of L-carnitine in the studies of Arenas (2002) and Marconi (1985) also led to a significant increase in VO2max [23, 24]. But the studies of Erglu (2008), Greek (1987) and Stowissie (2005) indicate no change in heart rate and VO2max due to L-carnitine supplementation [10, 25, 26]. Findings of Brass's et collegous (2001) showed that consumption of L-carnitine in renal patients, despite increasing plasma carnitine concentrations would have no effect on the levels of their VO2max [27]. Our study findings also showed that daily consumption of 3 g L-carnitine for 21 days would have no significant effect on VO2max and resting heart rate. On the other hand, some studies point out that the positive effects of this supplement are limited to athletes [28]

CONCLUSION

Some research studies suggest that carnitine supplementation is associated with increase of free fatty acid transport into mitochondria, reducing Carbohydrate oxidation and delayed onset of fatigue during exercise. Some modern studies have also suggested the ergogenic benefits of carnitine supplementation are limited to people with carnitine deficiency and such a supplementation in healthy individuals does not change the energy yield of fuel resources. This study too reports no effect of supplementation on parameters involved in carbohydrate metabolism and endurance performance and this supports the hypothesis that L-carnitine supplementation has no impact in healthy individuals. It is likely that the L-carnitine ergogenic advantages are limited to the later stages of prolonged activities that require further studies in this field.

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