The effect of chronic L-carnitine L-tartarate supplementation on glucose and lactate concentration and aerobic capacity

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Abstract

Many athletes adopt nutritional manipulations to improve their performance. Among the substances generally consumed, carnitine (L-trimethyl-3-hydroxy-ammoniobutanoate) has been used by athletes as an ergogenic aid because of its role in the transport of long-chain fatty acids across mitochondrial membranes. **Aim:** This study was performed with purpose to determine the effect of chronic carnitine supplementation on aerobic performance and fat-carbohydrate metabolism enzymes and cardiovascular factors. **Method:** In a randomized, placebo-controlled, double-blind crossover design, Thirty healthy untrained males cycled for 20 min at 70% maximal O2 uptake (VO2max) in two separate stages (baseline and while ingested oral L-carnitine (Int) or lactose (Con), 3 g daily for 3 Week). Blood samples were drawn for the purpose of calculation plasma glucose and Lactate concentration, lactate dehydrogenase activity (LDH), heart rate and VO2max. A two-way repeated measure ANOVA was used to determine significant differences between the two groups. Statically significant was accepted at (P<0.05). **Result:** The finding of our study showed that L-carnitine supplementation had no influence on plasma glucose and lactate concentration. Also rest and submaximal heart rate, VO2max and LDH activity was equal in pre and posttest (P<0.05). All variables unaffected in the placebo trial. **Discussion:** Our finding indicated that L-carnitine L-tartarate ingestion, 3g for 3 week could not affect mentioned variables and aerobic capacity. Additional investigation is required to directly identify these supplementations on the substrate utilization and fat-carbohydrate metabolism and exercise performance.

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1. Introduction

It is well known that athletes supplement their diets not only with single but also with multiple nutritional factors. The endurance Professional and nonprofessional athletes seek nutritional supplements that will enhance exercise performance. These substances theoretically improve exercise capacity by enhancing lipid oxidation and slowing rates of muscle glycogen depletion, therefore reducing fatigue (10). Among the substances generally consumed, carnitine has been used by athletes as an ergogenic aid because of its role in the transport of long-chain fatty acids across mitochondrial membranes (5, 6). Carnitine (L-3-hydroxytrimethylamminobutanoate) is a naturally occurring compound that can be synthesized in mammals from the essential amino acids lysine and methionine or ingested through diet. Primary sources of dietary carnitine are red meat and dairy products; however, commercially produced supplements also are available and have been shown to be safe in humans. Carnitine is stored primarily in skeletal muscle, with lower concentrations in plasma (13). Carnitine supplementation enhances fatty acid oxidation during exercise and, hence, spares glycogen (9). The increases in fatty acid oxidation and plasma free fatty acid concentration suggested a glycogen-sparing effect of carnitine supplementation (7). Conflicting results characterized the research focused on L-carnitine supplementation’s ability to enhance endurance performance. In this area, Matera (2003) stated that L-carnitine supplementation led to reduction of lactate production during exercise (14). But, results of study by Eroğlu (2008) show that L-carnitine intake one hour prior to the exercise has no effect on the metabolic and blood lactate values of badminton players (8). Four weeks L-carnitine supplementation in Broad study (2005) also had no effect on substrate utilization or endurance performance (3).

Despite this strong foundation and 20 years of research, no compelling evidence exists that carnitine supplementation can improve physical performance in healthy subjects. Therefore, this study was performed with purpose to determine the effect of chronic carnitine supplementation on aerobic capacity, rest and submaximal heart rate, plasma Lactate and glucose concentration during submaximal ergometry cycling.

2. Subjects and Method

Thirty untrained healthy male subjects with an age of 18–24 years volunteered for this study. The subjects were randomly divided into experimental and placebo groups. This study performed by physical education department of Saveh Azad University in Iran. All subjects were fully informed about the study’s purpose and its possible risks prior to giving their written consent. In this randomized, placebo-controlled, double-blind crossover design, the subject ingested during 3 wk supplementation periods, with either 3g L-Carnitine L-tartrate (n=15) or 3g Lactose (n=15) daily in experimental and placebo groups. Before and after of supplementation periods, the all subjects performed, ergometery cycling test according to Astrand submaximal protocol on cycle for twenty minute (2). Blood samples were drawn immediately followed up exercise. The blood samples were immediately centrifuged and serum was stored at −80°C. Following blood collection, regional fat pads samples were collected, frozen in liquid nitrogen, and stored at −80°C. The samples were assayed for the concentrations of lactate, glucose and lactate dehydrogenase according to the established procedures. Rest and submaximal heart rate monitored by polar telemetry. Maximal oxygen consumption calculated by the formula of Astrand protocol. Data are reported as means ± standard deviation. A two-way repeated measure ANOVA was used to determine significant differences between the two groups. A value of P<0.05 was considered to be significant.

Result: The finding of our study showed that L-carnitine supplementation had no influence on plasma glucose concentration (figure 1). Also plasma Lactate concentration remained unchanged after chronic L-carnitine supplementation (figure 2). In addition, rest and submaximal heart rate (figure 3), VO2max (figure 4) and LDH activity was equal in pre and posttest (P<0.05). All variables unaffected with the placebo trial (P<0.05). The mean and standard deviation of all variables to be visible in table number one.
3. Discussion:

In the overnight-fasted state, during the resting state, and during exercise of low to moderate intensity, long-chain fatty acids represent up to 80% of the energy sources (15). Oral ingestion of carnitine would result in an increase of the total carnitine concentration in muscle. This increase in muscle carnitine would result in an increased rate of oxidation of intramuscular fatty acids and triacylglycerols during exercise, thereby reducing muscle glycogen breakdown and postponing fatigue (11). The best described function of L-carnitine is in its role as a cofactor of carnitine, acyltransferases transporting long-chain fatty acids across the mitochondrial inner membrane. In the absence of L-carnitine, the inner mitochondrial membrane would be impermeable to long-chain fatty acids and fatty
acyl-CoA esters. Once inside the mitochondria, these compounds can be degraded to acetyl-CoA through a process known as β-oxidation (15).

Although Carnitine is famous for its fat-burning properties, growing evidence indicates that it is also important in carbohydrate metabolism (4). Carnitine can act as anti-catabolic agent because of its 'Glycogen Sparing' effect to improve energy production from fats and effectively reduces the need to burn glycogen (4). The study of Panjwani (2007) showed that L-Carnitine supplementation had no effect on plasma glucose levels during exercise (16). Our study also showed that chronic L-carnitine supplementation had not affect plasma glucose concentration during exercise protocol.

Maximal oxygen consumption (VO2max) provides an assessment of maximal exercise capacity and is an accepted index of the functional limit of the cardiovascular system (18). As reported in the majority of studies, an increase in maximal oxygen consumption and a lowering of the respiratory quotient indicate that dietary carnitine has the potential to stimulate lipid metabolism (12). Treatment with L-carnitine also has been shown to induce a significant postexercise decrease in plasma lactate, which is formed and used continuously under fully aerobic conditions (12). Eroglu (2008) and Abramowitz (2005) showed that acute L-carnitine supplementation has no effect on the metabolic and VO2max, energy consumption, heart rate, respiratory exchange ratio, minute ventilation, oxygen pulse and blood lactate and CHO oxidation during exercise(1,8). In addition, Stuessi study (2005) indicated that L-carnitine supplementation had not influence on lactate concentration (17). Our finding indicated that L-carnitine L-tartarate ingestion, 3g for 3 week could not affect mentioned variables and aerobic capacity. Additional investigation is required to directly identify these supplementations on the substrate utilization and fat-carbohydrate metabolism and exercise performance.

References