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Effect of Oral Supplementation with $_{L}$-Carnitine on Performance Time in a 5000 m Race and Responses of Free Fatty Acid and Carnitine Concentrations in Trained-Endurance Athletes

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Abstract

This study was designed to determine the effect of oral supplementation with $_{L}$-carnitine on the performance time in a 5000 m race. In addition, free fatty acid, blood carnitine, lactate, and glucose responses to the race following the supplementation period were measured. Twenty male trained-endurance athletes were randomly divided into two groups ($_{L}$-carnitine, n = 10 (22.13 ± 2.66 yrs) or placebo, n = 10 (21.63 ± 2.23 yrs)). The study was performed with a randomized, double-blind, placebo-controlled parallel-group, in which participants ingested an $_{L}$-carnitine supplement or a placebo 2 × 1.5 g/day for 3 weeks. Athletes completed a 5000 m race before and after the supplementation period. Blood samples were collected from each athlete before and after the race, pre- and post-supplementation to measure the physiological responses. Data showed that there were no differences in performance time before (p=0.624) and after (p=0.407) supplementation period between groups and within a group (p>0.05). No differences existed in physiological responses between groups after supplementation before beginning the race (p>0.05), except for the blood carnitine level, which was significantly higher in the $_{L}$-carnitine than the placebo (P=0.001) group. After the finish of the race, however, data showed better physiological responses in response to $_{L}$-carnitine supplementation compared to the placebo group (p<0.05). In conclusion, although $_{L}$-carnitine supplementation increases blood carnitine concentration, it has no beneficial effect on performance time of 5000 m race probably due to the short duration of the race; it might also have no ergogenic effect.

Keywords: *β-oxidation, mitochondria, neuropathies, nitric oxide, running economy*

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Introduction

Despite the obvious cardiopulmonary benefits associated with distance running in a physically active population, several potential liming factors have been observed in endurance athletes (Abbias & Laursen, 2005). Metabolic factors seem to play a central role in fatigue during prolonged endurance exercise

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in elite runners (Petersen, Hansen, Aagaard, & Madsen, 2007). Muscle and liver glycogen depletion is a major cause of fatigue in prolonged exercise events (Abbias & Laursen, 2005; Hanon, Thépaut-Mathieu, & Vandewalle, 2005). Elite runners require from 5,600 to over 40,000 kcal of energy during competitive endurance events (Brouns et al., 1989; Tarnopolsky, 2004). Carbohydrates and fats represent the predominant fuel sources during prolonged endurance exercise (Belcastro, Albisser, & Litteljohn, 1996; Tarnopolsky, 2004). However, muscle and liver glycogen is the primary source of energy during the beginning and the late stages of an endurance race (Petersen et al., 2007). Higher glycogen stores can maintain muscle contractile properties, excitation-contraction coupling, and delay muscular fatigue (Abbias & Laursen, 2005; Gandevia, 2001; Petersen et al., 2007). Several factors can improve running economy by using more fatty acids and sparing glycogen, including L-carnitine.

L-Carnitine (3-hydroxy-4-N-trimethylaminobutirate) is a hydrophilic (Bene, Csiky, Komlosi, Sulyok, & Melegh, 2011; Miklos et al., 2016) amino acid derivative presented throughout the central and peripheral nervous system (Bavari, Tabandeh, Varzi, & Bahramzadeh, 2016) and is predominantly found in cardiac and skeletal muscle (Broad, Bolger, & Galloway, 2006). L-Carnitine is biosynthesized from the amino acids lysine and methionine (Bavari et al., 2016; Mojtaba, Laleh, Mohsen, & Zohreh, 2011) in the kidney, liver, and brain (Delaš, Dražić, Čačić-Hribjan, & Sanković, 2008), in a process that requires the vitamins B_6, B_3, C, niacin, and iron (Broad et al., 2006; Delaš et al., 2008). The total estimated carnitine content is approximately 1.2 μmol/kg body mass (Rebouche, 1992). Red meat, fish and dairy products are reported to be the primary exogenous carnitine, which is estimated to be 2–12 μmol/kg body mass daily (Demarquory et al., 2004) or 20–300 mg per day (Delaš et al., 2008).

L-Carnitine plays a primary physiological role in mitochondrial β-oxidation (Bene et al., 2011; Zhang et al., 2012; Miklos et al., 2016) by the transportation of long-chain fatty acids from cytosole into the mitochondria (Bene et al., 2011; Siddiqui, Mughal, Siddiqui, & Hayat, 2015; Zhang et al., 2012). This process is mediated by the carnitine-palmitoyltransferase (CPT) enzymatic system (CPT I, carnitine-acylcarnitinetranslocase and CPT II) (Brass, & Hiatt, 1998; Delaš et al., 2008). In addition, L-carnitine modulates the ratio of acyl coenzyme A (CoA):CoA (Stumpf, Parker, & Angelini, 1985), serves as an energy source in the form of acetyl carnitine (Bene et al., 2011), and acts as an antioxidant (Pekala et al., 2011). All together, L-carnitine has a potential role in reducing intramuscular metabolic stress. Of relevance, L-carnitine decreases cytosolic iron concentration (Pekala et al., 2011), thereby reducing reactive oxygen species (ROS) production (Bavari et al., 2016).

Oral supplementation with L-carnitine is used by endurance athletes to increase its content in skeletal muscle, increase fatty acid oxidation during exercise (Brass, & Hiatt, 1998), decrease toxic acyl groups (Peters et al., 2015; Stumpf et al., 1985), maintain the activity of pyruvate dehydrogenase (Brass, & Hiatt, 1998), preserve muscle glycogen, and delay muscular fatigue (Brass, & Hiatt, 1998; Smith, Fry, Tschume, & Bloomer, 2008; Wall et al., 2013). The increased use of fatty acids for energy production during prolonged exercise is beneficial to runners because it reduces muscle glycogen and thus increases aerobic capacity.

To the best knowledge of the author of the present paper, several studies have studied the effect of L-carnitine with acute ingestion (Eizadi, Pourvaghar, Nazem, Eghdami, & Khorshidi, 2009; Kashef & Saei, 2017; Mojtaba et al., 2011; Vecchiet et al., 1990) and different supplementation periods (Greig et al., 1987; Smith et al., 2008; Wächter, Vogt, & Kreis, 2002); most of those studies focused on maximal oxygen consumption (VO_{2max}) measurements on a cycle ergometer and conducted on healthy untrained subjects. However, no study has investigated the concentration of carnitine following a middle-distance race, such as 5000 m in endurance athletes. Consequently, the present study aimed to determine the effect of L-carnitine supplementation on performance time of 5000 m race. In addition, free fatty acid, blood carnitine, lactate and glucose responses to the race following L-carnitine supplementation period were measured. This study hypothesized that L-carnitine may enhance performance by using more fatty acids that produce more than 100 adenosine triphosphate (ATP) in each molecule and reducing glycogen utilization.

Methods

Participants

The participants were 20 male trained-endurance athletes who used no medical drugs, dietary supplements, or doping. Demographic data of the participants are shown in Table 1. All participants trained once a day (approximately 90 min), three times per week. The participants were informed about the potential risks and benefits involved in participation. Each participant voluntarily provided written informed consent before participation in the present study. This study was approved in advance by the local scientific research committee (protocol SS 100-2020).

Study design

In a randomized, double-blind, and parallel-group approach, the participants were divided into two groups: L-carnitine (n=10) and placebo-control (n=10). Participants ingested

Table 1. Participants' demographic data

Variables	LC	Pla	*P* value
Age (years)	22.13± 2.66	21.63± 2.23	0.522
Height (cm)	176.30 ± 3.86	174.12 ± 4.06	0.278
Mass (kg)	66.07 ± 4.30	64.90 ± 3.30	0.141
BMI (kg/m^2)	21.89± 0.57	22.19± 0.47	0.304
resting HR (bpm)	64.70 ± 3.52	66.42± 4.22	0.232
Training volume (min/week)	460.00± 26.42	400.00± 37.35	0.095
Training experience (years)	3.80 ± 1.89	4.32 ± 2.16	0.615

Note. All data are expressed as mean ± SD. No differences existed between groups for any demographic variable, ensuring homogeneity between groups. Significance level was set at P < 0.05. LC: L-Carnitine, Pla: Placebo.

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no coffee, energy drink, or other substances that could affect the results 24 hours prior to the beginning of the trials. Each trial in both groups consisted of a 5000 m race following a 10 min warm-up (jogging, joint mobilization, and stretching).

Study protocol

All athletes in both groups completed the 5000 m race on a track before supplementation of L-carnitine or placebo. On the following day, athletes were instructed to ingest L-carnitine in the L-carnitine group and maltodextrin in the placebo group for three weeks. After one day of the completion of supplementation, the race was performed in the same order for all athletes in both groups. Both pre- and post-supplementation trials for both groups were performed at the same time of the day (08.20 AM) to control for the circadian rhythm effect. All athletes were instructed to fast three hours prior to the trial, except for 500 ml water (21 ˚C) taken 90 min prior to the race to avoid possible dehydration. Randomization was equalized by speed and experience of training to ensure the homogeneity between groups. The homogeneity of the demographic variables of the participants between groups was equal (P > 0.05) (see Table 1).

Control of pre-experimental status

Athletes were asked to maintain their routine training sessions and were instructed to refrain from strenuous exercise 48 hours prior to each trial. None of the athletes had ever consumed L-carnitine supplement before this study. They were not permitted to ingest any nutritional supplement throughout the supplementation period. They were also requested to maintain their normal diet throughout the supplementation period. Athletes wore the same attire for each test; a T-shirt, shorts, and shoes that they normally train in.

Supplementation protocol

For three weeks, 2 × 1.5 g of L-carnitine capsule (L-carnitine, Capsule, Arazo Nutrition, USA) and maltodextrin per day were provided to experimental and placebo participants, respectively. The selected dose of L-carnitine was used depending on the literature that stated that the normal dosage of L-carnitine is 1–5 g/day (Wall et al., 2013).

Blood sample analysis

Blood samples were collected before and immediately after the race pre- and post-supplementation in both groups to measure blood carnitine, lactate, glucose, and free fatty acid. Venepuncture was used from the median vein to obtain blood samples; 2 ml of blood was dispensed into a plain tube containing clot activator to measure blood glucose using (Integral 400, Switzerland); 2 ml was dispensed into an anticoagulant EDTA tube to measure plasma lactate and free fatty acids, and 6 ml was frozen (-4 ˚C) for 1 hour to measure carnitine. The blood tubes were centrifuged at 5000 rpm for 5 min. The plain blood tube was centrifuged at -4 ˚C and 3500 rev/min for 5 min to allow for the extraction of serum, which was used to measure carnitine with a spectrometry device (Molecular Analysis, Spectrometry, Iceland). Plasma-free fatty acid was analysed with an Elecsys device (RIA, 2010, Switzerland), and plasma lactate was similarly analysed (Integral 400, Switzerland). The reference ranges of variables were as follows: 33.0–71.0 μmol/L for carnitine, 0.63– 2.44 mmol/L for lactate, < 0.7 for free fatty acid, 3.9–6.2 for blood glucose.

Statistical analysis

Because the normal distribution was verified (p>0.05) using Shapiro-Wilk test, a paired sample t-test was used to analyse the possible differences in carnitine, free fatty acid, glucose, and lactate within a group (between before and after the race, and between before and after supplementation). An independent t-test was utilized to analyse the differences in these variables between groups. Two-way ANOVA with repeated measures on (pre vs post) was used to determine if any significant main effects were present for the performance times of the 5000 m race between groups (experimental and placebo). Statistical analyses were carried out using SPSS version 23.0. All data are presented as mean ± SD. The level of statistical significance was set at P < 0.05.

Results

Figure 1 illustrates the performance times for the 5000 m race after the supplementation period in both groups. There was no significant difference in 5000 m times between groups before (p=0.624) and after (p=0.407) the supplementation period. In addition, data also revealed that the time of the race was not changed from pre- (14.76±3.01 min) to post- (14.66±1.21 min) L-carnitine supplementation, and from pre- (14.81±2.05 min) to post- (14.72±1.05 min) placebo supplementation (p=0.694; p=0.701, respectively).

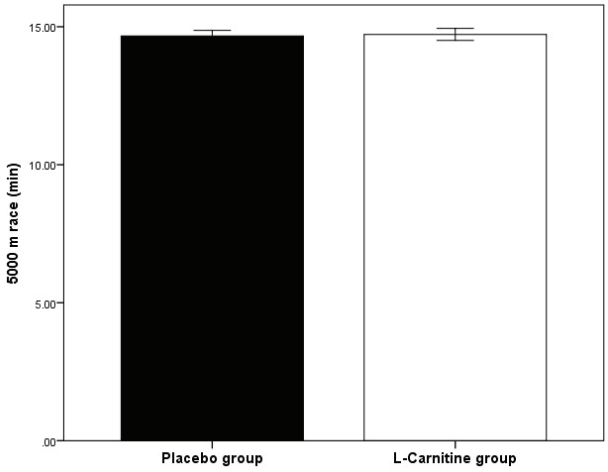

Figure 1. Time of 5000 m race. Mean values ± standard deviation (SD) are shown in the figure. No differences existed between groups (p=0.407). Significance level was set at p<0.05.

At baseline, there were no differences in the physiological parameters between the pre- and post-supplementation periods within a group (p>0.05), except for blood carnitine, which was significantly higher after than before L-carnitine supplementation (Table 2). All parameters were at similar levels between the L-carnitine and placebo groups, except carnitine was higher in the L-carnitine group than in the placebo group (Table 3).

Table 2. Physiological parameters before start of the race within a group before and after supplementation period

Parameters	LC group			Pla group		
	Pre-suppl (Pre-race)	Post-suppl (Pre-race)	*P* value	Pre-suppl (Pre-race)	Post-suppl (Pre-race)	*P* value
Carnitine (μmol/L)	51.81±4.36	62.31±4.12	0.002*	51.64±3.17	51.97±2.47	0.346
Free fatty acid (mmol/L)	0.57±0.03	0.56±0.06	0.053	0.58±0.04	0.58±0.05	0.162
Blood glucose (mmol/L)	5.67±1.30	5.72±2.27	0.181	5.68±0.96	5.66±1.33	0.529
Blood lactate (mmol/L)	2.11±1.22	2.01±2.86	0.631	2.05±1.67	1.98±2.05	0.425

Note. All data are expressed as mean ± SD. *Significant differences between before and after supplementation (before starting the race) within a group. Significance level was set at p<0.05. LC: L-Carnitine, Pla: Placebo, Pre: before, Post: after, suppl: supplementation.

Table 3. Physiological parameters before start of the race between groups before and after supplementation period

Parameters	Pre-suppl			Post-suppl		
	LC (Pre-race)	Pla (Pre-race)	*P* value	LC (Pre-race)	Pla (Pre-race)	*P* value
Carnitine (μmol/L)	51.81±4.36	51.64±3.17	0.637	62.31±4.12	51.97±2.47	0.001*
Free fatty acid (mmol/L)	0.57±0.03	0.58±0.04	0.067	0.56±0.06	0.58±0.05	0.052
Blood glucose (mmol/L)	5.67±1.30	5.68±0.96	0.468	5.72±2.27	5.66±1.33	0.318
Blood lactate (mmol/L)	2.11±1.22	2.05±1.67	0.492	2.01±2.86	1.98±2.05	0.546

Note. All data are expressed as mean ± SD. *Significant differences between before and after supplementation (before starting the race) between groups. Significance level was set at p<0.05. LC: L-Carnitine, Pla: Placebo, Pre: before, Post: after, suppl: supplementation

After completion of the race, the data revealed significantly (p<0.05) physiological changes between pre- and post-L-carnitine supplementation but there were no differences noted in the placebo group (Table 4). Additionally, the data indicated better responses after L-carnitine supplementation compared to the placebo group (Table 5).

Table 4. Physiological responses to the race within a group before and after supplementation

Parameters	LC group			Pla group		
	Pre-suppl (Post-race)	Post-suppl (Post-race)	P value	Pre-suppl (Post-race)	Post-suppl (Post-race)	P value
Carnitine (μmol/L)	49.88±4.36	57.61±3.44	0.001*	50.91±3.11	50.87±3.12	0.846
Free fatty acid (mmol/L)	0.59±0.05	0.49±0.07	0.003*	0.59±0.03	0.60±0.03	0.102
Blood glucose (mmol/L)	4.72±2.24	5.34±1.41	0.031*	4.85±2.36	4.69±0.21	0.711
Blood lactate (mmol/L)	6.85±1.92	4.66±0.92	0.002*	6.94±2.18	7.21±0.74	0.335

Note. All data are expressed as mean ± SD. *Significant differences between before and after supplementation, immediately after the race, within a group. Significance level was set at p<0.05. LC: L-Carnitine, Pla: Placebo, Pre: before, Post: after, suppl: supplementation.

Table 5. Physiological responses to the race between groups before and after supplementation

Parameters	Pre-suppl			Post-suppl		
	LC (Post-race)	Pla (Post-race)	*P* value	LC (Post-race)	Pla (Post-race)	*P* value
Carnitine (μmol/L)	49.88±4.36	50.91±3.11	0.362	57.61±3.44	50.87±3.12	0.002*
Free fatty acid (mmol/L)	0.59±0.05	0.59±0.03	0.335	0.49±0.07	0.60±0.03	0.001*
Blood glucose (mmol/L)	4.72±2.24	4.85±2.36	0.462	5.34±1.41	4.69±0.21	0.002*
Blood lactate (mmol/L)	6.85±1.92	6.94±2.18	0.277	4.66±0.92	7.21±0.74	0.001*

Note. All data are expressed as mean ± SD. *Significant differences between before and after supplementation between groups. Significance level was set at p<0.05. LC: L-Carnitine, Pla: Placebo, Pre: before, Post: after, suppl: supplementation

Discussion

This study was designed to allow the assessment of physiological parameters during a 5000 m race and performance time following L-carnitine supplementation in endurance-trained athletes.

The main finding was that athletes completed the 5000 m race in approximately similar times regardless of the supplementation (p=0.701). This finding was associated with the ele-

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vated blood carnitine in the L-carnitine group compared to the placebo group. It might be suggested that L-carnitine should not be classified as anergogenic aid (Mojtaba et al., 2011). In addition, this finding might be attributed to the short distance of the race, leading to the inability of athletes in the L-carnitine group to complete the race in a shorter time. Thus, we suggest a further protocol, such as an incremental exercise protocol on (time until volitional fatigue) or a 10,000 m race, which determine the real potential effect of L-carnitine supplementation. Greig et al. (1987) showed no significant changes in VO_{2max} between L-carnitine supplementation (2 g/day for 2–4 weeks) and placebo in healthy untrained subjects. Smith et al. (2008) showed no differences between or within groups in muscle (Vastus lateralis) carnitine content, time to fatigue, and anaerobic power in untrained men and women after eight weeks of L-carnitine supplementation (1 g/day, 3 g/day, or placebo). Kashef & Saei (2017) observed an increased VO_{2max} during testing to exhaustion (Bruce incremental exercise) following acute ingestion of 3 g of L-carnitine 90 min prior to testing compared to the placebo group in students. Vecchiet et al. (1990) demonstrated enhanced incremental cycle ergometer performance following a single dose of L-carnitine (2 g) 1 h prior to exercise compared to placebo in collegiate students. Eizadi et al. (2009) demonstrated that a single dose of 3 g of L-carnitine did not improve exercise performance during submaximal cycle ergometer in healthy people. Wächter et al. (2002) reported that long-term supplementation with L-carnitine (4 g/day for 3 months) had no positive effect on VO_{2max}. The failure of long term L-carnitine supplementation to improve performance might be because of the greater excretion of L-carnitine with prolonged supplementation (Eizadi et al., 2009).

A normal plasma concentration range of total carnitine (TC) is 30–90 μmol, free carnitine (FC) 26–52 μmol, and acyl-canrnitne esters (AC) 2–10 μmol (Rebouche, 1992). L-Carnitine deficiency occurs when its concentrations below 20 μmol or when the AC:FC ratio is higher than 0.4 (Lennon, Shrago, Madden, Nagle, & Hanson, 1986). Usually, the range of daily urinary carnitine excretion is 22–291 μmol (Broad et al., 2006). The reduction in FC could result in reduced performance, particularly endurance exercise. Broad et al., (2006) investigated the concentration of plasma carnitine and urinary carnitine excretion following a seven-day weighed food program (3 meals/day of carbohydrate, fats and proteins) in 14 non-vegetarian endurance-trained adult males. They found that total plasma carnitine was 44 μmol/L, urinary carnitine excretion was 437 μmol/day, and the acyl free carnitine was 0.28, indicating athletes were not at risk of carnitine deficiency. Bene et al. (2011) showed that L-carnitine supplementation (1 g intravenously for 12 weeks) increased acylcarnitine to 1.6–4.8-fold in haemodialysis patients aged 39–85 years, and decreased by 11–74% three months after the cessation of supplementation. They suggested that the elevation of carnitine or acylcarnitine return to its normal range following carnitine discontinuation.

L-Carnitine supplementation has a beneficial effect in the L-carnitine group in the present study. For instance, the physiological parameters were better in response to L-carnitine supplementation.

Although the plasma FFA value measured for all athletes in both groups did not exceed reference range values by means, data analysis revealed a significant decrease in its value three weeks after L-carnitine supplementation compared to the Placebo group. This might indicate an increased cellular uptake of fatty acids from blood and subsequent enhanced transportation of fatty acids into mitochondria for β-oxidation (Kashef & Saei, 2017). Delaš et al. (2008) demonstrated that L-carnitine supplementation (2 g/day for 2 weeks) only in sedentary healthy subjects did not induce changes in blood glucose, triacylglycerols, total cholesterol, high-density lipoprotein (HDL), creatine kinase; however, the FFA level was decreased from 0.439 mmol/dry muscle to 0.279 mmol/dry muscle. Mojtaba et al. (2011) demonstrated that FFA, VO_{2max}, HR, HDL, and low-density lipoprotein (LDL) were similar either after a single dose of 3 g of L-carnitine-L-tartrate or placebo in healthy non-active subjects. Eizadi et al. (2009) reported no differences in FFA levels between pre (0.69 ± 0.24 mg/dl) and post (0.72 ± 0.14 mg/dl) supplementation. Thus, athletes can perform an exercise with high energy expenditure following L-carnitine supplementation when the time or distance of exercise is not determined.

It has been reported that elevation in plasma fatty acid availability prior to exercise could reduce utilization of muscle glycogen (Brass & Hiatt, 1998; Smith et al., 2008), which promotes an important pathway to spare glycogen stores (Wall et al., 2013; Brass & Hiatt, 1998; Kashef & Saei, 2017) and subsequent improvement in endurance capacity. However, the fatty acid oxidation ratio is decreased when an exercise intensity exceeds 70% VO_{2max} (Wall et al., 2013), and glycolysis is increased (Peters et al., 2015; Wall al., 2013). This results in depletion of glycogen stores and subsequent exercise cessation. In-line, L-carnitine is an indispensable compound for mitochondrial energy source by transporting long-chain fatty acids across the mitochondrial inner membrane as acyl-carnitine esters (Peters et al., 2015; Siddiqui et al., 2015), by regulating COA homeostasis (Miklos et al., 2016), by buffering toxic acyl-COA (Broad et al., 2006; Peters et al., 2015), by serving as a source of acetyl-L-carnitine, acetylcholine, and L-glutamate, in which they contribute to energy-producing reactions (Zhang et al., 2012), and by stimulating nitric oxide production (Miklos et al., 2016), in which it enhances pulmonary gas exchange and acts as a vasodilator and therefore increased blood flow (Verges, Flore, Favri-Juvin, Lévy, & Wuyam, 2005).

Our findings showed higher blood glucose with L-carnitine supplementation than with maltodextrin in the placebo group, which might indicate the role of L-carnitine supplementation in the utilization of FFAs for a fuel source instead of glycogen. This supported by our finding that showed lower blood lactate concentration in the L-carnitine group than the placebo group. The explanation of this result might be attributed to the lesser glucose utilization as an energy source during the race following L-carnitine supplementation and therefore decreased glucose cellular uptake. Another explanation by which L-carnitine supplementation elevated glucose levels and decreased lactate concentrations in the working skeletal muscle is that L-carnitine can reduce lactic acid accumulation by decreasing the utilization of liver and muscle glycogen (Siddiqui et al. 2015). Blood lactate in the study of Greig et al. (1987) was higher in placebo (11.7 ± 2.1 mmol/L) than L-carnitine (10.1 ± 2.6 mmol/L) after an incremental cycle ergometer, although the difference was not statistically significant.

Furthermore, slow-twitch fibres are the predominant working fibres during endurance running, such as the 5000 m race, ensuring increased ATP production by beta-oxidation.

In an animal study, Siddiqui et al. (2015) demonstrated that the rabbit soleus muscle exhibited a decline in muscle contraction force of about 57% in an L-carnitine group (5 mg/day for 2 weeks) with lactate levels of 23 mmol/kg compared with about 70% in a control group with lactate levels of 23 mmol/kg. They suggested that L-carnitine supplementation could delay the onset of fatigue, specifically in Type II muscle fibres.

Conclusion

The finding of this study revealed that 2 x 1.5 g/day of L-carnitine for three weeks had no effect on the performance time of 5000 m in endurance athletes, although blood carnitine was significantly higher following the supplementation period compared to the placebo group. L-carnitine supplementation decreased free fatty acids and blood lactate and maintained blood glucose levels following the endurance trial.

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References

Abbias, C.R., & Laursen, P.B. (2005). Models to explain fatigue during prolonged endurance cycling. *Sports Medicine*, 35(10), 865-898. doi: 10.2165/00007256-200535100-00004

Bavari, M., Tabandeh, M.R., Varzi, H.N., & Bahramzadeh, S. (2016). Neuroprotective, antiapoptotic and antioxidant effects of L-carnitine against caffeine-induced neurotoxicity in SH-SY5Y neuroblastoma cell line. *Drug and Chemical Toxicology*, 39(2), 157-166. doi: 10.3109/01480545.2015.1063062

Belcastro, A.N., Albisser, T.A., & Litteljohn, B. (1996). Role of calcium-activated neural protease (calpain) with diet and exercise. *Canadian Journal of Applied Physiology*, 21, 328-346. doi: 10.1139/h96-029

Bene, J., Csiky, B., Komlosi, K., Sulyok, E., & Melegh, B. (2011). Dynamic adaptive changes of the serum carnitine esters during and after L-carnitine supplementation in patients with maintenance haemodialysis. *Scandinavian Journal of Clinical & Laboratory Investigation*, 71, 280-286. doi: 10.3109/00365513.2011.560674

Brass, E.P., & Hiatt, W.R. (1998). The role of carnitine and carnitine supplementation during exercise in man and in individuals with special needs. *Journal of American College of Nutrition*, 17(3), 207-215. doi: 10.1080/07315724.1998.10718750

Broad, E., Bolger, C., & Galloway, S. (2006). Dietary carnitine intake and carnitine status in endurance-trained males. *Nutrition & Dietetics*, 63, 148-154. doi: 10.1111/j.1747-0080.2006.00068.x

Brouns, F., Saris, W.H., Beckers, E., Aldercreutz, H., van der Vusse, G.J., Keizer, H.A., Kuipers, H., Menheere, P., Wagenmakers, A.J., & ten Hoor, F. (1989). Metabolic changes induced by sustained exhaustive cycling and diet manipulation. *International Journal of Sports Medicine*, 10 (suppl 1), S49-62. doi: 10.1055/s-2007-1024954

Delaš, I., Dražić, T., Čačić-Hribjan, M., &Sanković, K. (2008). Effect of L-carnitine supplementation on some biochemical parameters in blood serum of sedentary population. *Croatica Chemica Acta Ccacaa*, 81(1), 163-168.

Demarquory, J., Geofges, B., Rigault, C., Royer, M., Clairet, A., Soty, M., Lekounoungou, S., & Le Borgne, F. (2004). Radioisotopic determination of L-carnitine content in foods commonly eaten in Western countries. *Food Chemistry*, 86, 137-142. doi: 10.1016/j.foodchem.2003.09.023

Eizadi, M., Pourvaghar, A.K., Nazem, F., Eghdami, A., & Khorshidi, D. (2009). The determination of acute oral L-carnitine ingestion on physiological and biochemical parameters related with lipids in endurance exercise. *Journal of Babol University of Medical Sciences*, 11(5), 45-51.

Gandevia, S.C. (2001). Spinal and supraspinal factors in human muscle fatigue. *Physiological Reviews*, 81(4), 1725-1789. doi: 10.1152/physrev.2001.81.4.1725

Greig, C., Finch, K.M., Jones, D.A., Coopr, M., Sargeant, A.J., & Forte, C.A. (1987). The effect of oral supplementation with L-carnitine on maximum and submaximum exercise capacity. *European Journal of Applied Physiology*, 56, 475-460. doi: 10.1007/BF00417775

Hanon, C., Thépaut-Mathieu, C., & Vandewalle, H. (2005). Determination of muscular fatigue in elite runners. *European Journal of Applied Physiology*, 94, 118-125. doi: 10.1007/s00421-004-1276-1

Kashef, M., & Saei, M.A. (2017). Acute effect of L-carnitine supplementation on lactate, glucose, saturated oxygen and VO2max variations in young males. *International Journal of Basic Science in Medicine*, 2(1), 46-51. doi: 10.15171/ijbsm.2017.10

Lennon, D.L.F., Shrago, E.R., Madden, M., Nagle, F.J., & Hanson, P. (1986). dietary carnitine intake related to skeletal muscle and plasma carnitine concentrations in adult men and women. *Food Chemistry*, 86, 137-142. doi: 10.1093/ajcn/43.2.234

Miklos, A., Ciulea, L., Vari, C.E., Imre, S., Ősz, BE.,& Tero-Vescan, A. (2016). The efficiency and safety of L-carnitine and cafeeine after short-and long-term administration. *Palestrica of the third millenium-Civlization and Sport*, 17(3), 229-232.

Mojtaba, E., Laleh, B., Mohsen, S., & Zohreh, A. (2011). Fat metabolism and aerobic capacity does not affect by acute L-carnitine-L-tartrate supplementation. *Journal of Applied Environmental and Biological Sciences,* 1(12), 695-699.

Pekala, J., Patkowska-Sokola, B., Bodkoeski, R., Jamroz, D., Nowakowski, P., Lochynski, S., & Librowski, T. (2011). L-Carnitine: metabolic functions and meaning in humans life. *Current Drug Metabolism*, 12(7), 667-678. doi: 10.2174/138920011796504536

Peters, L.W.E., Smiet, E., de Sain-van der Velden, M.G.M., & van der Kolk, J.H. (2015). Acylcarnitine ester utilization by the hindlimb of warm blood horses at rest and following low intensity exercise and carnitine supplementation. *Veterinary Quarterly*, 35(2), 76-81. doi: 10.1080/01652176.2015.1027039

Petersen, K., Hansen, C.B., Aagaard, P., & Madsen, K. (2007). Muscle mechanical characteristics in fatigue and recovery from a marathon race in highly trained runners. *European Journal of Applied Physiology*, 101, 385-396. doi: 10.1007/s00421-007-0504-x

Rebouche, C.J. (1992). Carnitine function and requirements during the life cycle. *FASEB*, 6, 3379-3386. doi: 10.1096/fasebj.6.15.1464372

 DOI 10.26773/mjssm.210901

Siddiqui, M.K., Mughal, S.A., Siddiqui, M.S., & Hayat, A.S. (2015). Effects of L-carnitine; on skeletal muscle of rabbit. *Professional Medical Journal*, 22(8), 1001-1005.

Smith, W.A., Fry, A.C., Tschume, L.C., & Bloomer, R.J. (2008). Effect of glycine propionyl-L-carnitine on aerobic and anaerobic exercise performance. *International Journal of Sport Nutrition and Exercise Metabolism*, 18, 19-36. doi: 10.1123/ijsnem.18.1.19

Stumpf, D.A., Parker, W.D., & Angelini, C. (1985). Carnitine deficiency, organic acidemias, and Reye's syndrome. *Neurology*, 35(7), 1041-1045.

Tarnopolsky, M. (2004). Protein requirements for endurance athletes. *European Journal of Sport Science*, 4(1), 1-16. doi: 10.1080/17461390400074102

Vecchiet, L., Di Lisa, F., Pieralisi, G., Ripari, P., Menabo, R., Giamberardino, M.A., & Siliprandi, N. (1990). Influence of L-carnitine administration on maximal physical exercise. *European Journal of Applied Physiology*, 61, 486-490. doi: 10.1007/BF00236072

Verges, S., Flore, P., Favri-Juvin, A., Lévy, P., & Wuyam, B. (2005). Exhaled nitric oxide during normoxic and hypoxic exercise in endurance athletes. *Acta Physiologica*, 185, 123-131. doi: 10.1111/j.1365-201X.2005.01475.x

Wächter, S., Vogt, M., & Kreis, R. (2002). Long-term administration of L-carnitine to humans: effect on skeletal muscle carnitine content and physical performance. *Clinica Chimica Acta*, 318(1), 51-61. doi: 10.1016/S0009-8981(01)00804-X

Wall, B.T., Stphens, F.B., Van Loon, L.J.C., Constantin-Teodosiu, D., Macdonald, I.A., & Greenhaff, P.L. (2013). Reduced fat oxidation during high intensity, submaximal exercise: is the availability of carnitine important? *European Journal of Sport Science*, 13(2), 191-199. doi: 10.1080/17461391.2011.630103

Zhang, R., Zhang, H., Zhang, Z., Wang, T., Niu, J., Cui, D., & Xu, S. (2012). Neuroprotective effects of pre-treatment with L-carnitine and acetyl-L-carnitine on ischemic injury in vivo and in vitro. *International Journal of Molecular Sciences, 13*(2), 2078-2090. doi: https://doi.org/10.3390/ijms13022078