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Effects of L-Carnitine L-Tartrate Supplementation on Muscle Oxygenation Responses to Resistance Exercise

Barry A. Spiering,[1] William J. Kraemer,[1] Disa L. Hatfield,[1] Jakob L. Vingren,[1] Maren S. Fragala,[1] Jen-Yu Ho,[1] Gwendolyn A. Thomas,[1] Keijo Häkkinen,[2] and Jeff S. Volek[1]

[1]*Human Performance Laboratory, Department of Kinesiology, University of Connecticut, Storrs, Connecticut;* [2]*Department of Biology of Physical Activity, University of Jyväskylä, Jyväskylä, Finland*

Abstract

Spiering, BA, Kraemer, WJ, Hatfield, DL, Vingren, JL, Fragala, MS, Ho, J-Y, Thomas, GA, Häkkinen, K, and Volek, JS. Effects of L-carnitine L-tartrate supplementation on muscle oxygenation responses to resistance exercise. *J Strength Cond Res* 22: 1130–1135, 2008–Previous research has shown that L-carnitine L-tartrate (LCLT) supplementation beneficially affects markers of hypoxic stress following resistance exercise. However, the mechanism of this response is unclear. Therefore, the primary purpose of this study was to determine the effects of LCLT supplementation on muscle tissue oxygenation during and after multiple sets of squat exercise. Nine healthy, previously resistance-trained men (25.2 ± 6 years, 91.2 ± 10.2 kg, 180.2 ± 6.3 cm) ingested LCLT (equivalent to 2g L-Carnitine/day) or an identical placebo for 23 days using a randomized, balanced, crossover, double-blind, placebo-controlled, repeated-measures study design. On day 21, forearm muscle oxygenation was measured during and after an upper arm occlusion protocol using near infrared spectroscopy (NIRS), which measures the balance of oxygen delivery in relation to oxygen consumption. On day 22, subjects performed 5 sets of 15 to 20 repetitions of squat exercise with corresponding measures of thigh muscle oxygenation, via NIRS, and serial blood draws. Compared to the placebo trial, muscle oxygenation was reduced in the LCLT trial during upper arm occlusion and following each set of resistance exercise. Despite reduced oxygenation, plasma malondealdehyde, a marker of membrane damage, was attenuated during the LCLT trial. There were no differences between trials in the vasoactive substance prostacyclin. In conclusion, because oxygen delivery was occluded during the forearm protocol, it is proposed that enhanced oxygen consumption mediated the reduced muscle oxygenation during the LCLT trial. Enhanced oxygen consumption would explain why hypoxic stress was attenuated with LCLT supplementation.

Key Words ergogenic aids, metabolic stress, performance, recovery, supplements

Introduction

Traditional resistance exercise is characterized by repetitive concentric and eccentric muscle actions that cause transient muscle hypoxia. Muscle hypoxia subsequently triggers a biochemical cascade of purine catabolism, intracellular Ca^{++} accumulation, free radical formation, and membrane disruption (5,6,16). Increased circulating concentrations of hypoxanthine, xanthine oxidase, malondealdehyde (MDA), and myoglobin reflect the degree of hypoxic damage and membrane disruption (20). More importantly to the athlete, though, this hypoxic cascade results in muscle damage, muscle soreness, and reduced performance.

Carnitine supplementation represents a promising intervention for mediating hypoxia-induced muscle damage. Hypoxia in endothelial cells stimulates carnitine release, increases oxidative stress, and compromises blood flow regulation (4,7). However, hypoxia-induced carnitine release and the subsequent dysregulation of blood flow are attenuated by intravascular carnitine administration (4,7). This beneficial effect prompted the authors to investigate the efficacy of carnitine supplementation for mediating hypoxia-induced muscle damage and enhancing recovery following an acute bout of resistance exercise. Based on previous research (4,7), the authors generated a paradigm indicating that carnitine supplementation protects against carnitine deficiency in vascular endothelial cells and thereby improves blood flow regulation and delivery of oxygen to muscle tissue during and after exercise (10). Enhanced oxygen delivery would theoretically reduce the magnitude of exercise-induced hypoxia and thus attenuate the cascade of events leading to purine catabolism, free radical formation, membrane disruption, and muscle soreness.

Address correspondence to Dr. William J. Kraemer, william.kraemer@uconn.edu.

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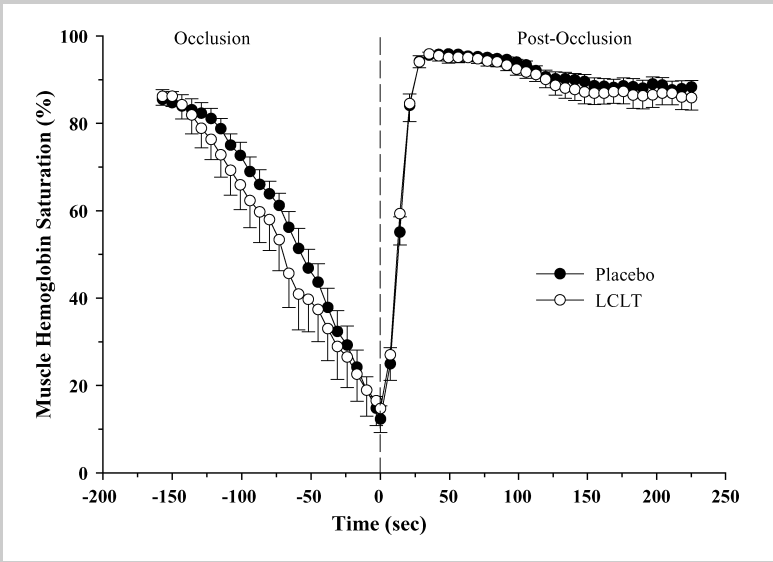

Figure 1. Forearm muscle tissue oxygenation responses (mean ± *SE*) during and following upper arm occlusion. A significant time × trial interaction existed during the occlusion portion of the protocol and indicated significantly less hemoglobin saturation from −129 seconds to −31 seconds during the l-carnitine l-tartrate trial compared to the placebo trial.

The authors' initial investigation demonstrated that, in fact, carnitine supplementation attenuated the hypoxic stress response to resistance exercise, as measured by several biomarkers of oxidative stress (e.g., hypoxanthine and xanthine oxidase) and membrane damage (e.g., MDA and myoglobin) (20). Although these results supported the proposition that carnitine supplementation mediates hypoxia-induced muscle damage, further research was required to confirm the hypothesis that carnitine supplementation improves blood flow regulation and delivery of oxygen to muscle tissue during and after exercise. Therefore, the primary purpose of the current investigation was to determine the effects of 3 weeks of carnitine supplementation on muscle tissue oxygenation during and after multiple sets of squat exercise with a reduction in MDA. Because in vitro research showed that prostocyclin synthesis mediates carnitine-induced vasodilation (1), the secondary purpose of the current investigation was to further explore the effects on carnitine supplementation on a circulating vasoactive marker (i.e., prostacyclin) following resistance exercise. It was hypothesized that carnitine supplementation would improve muscle tissue oxygenation during and after squatting, corresponding with enhanced circulating prostacyclin.

Methods

Experimental Approach to the Problem

A randomized, balanced, crossover, double-blind, placebo-controlled, repeated-measures design was used. Nine men with at least 1 year of resistance exercise experience were randomly assigned to receive either L-carnitine L-tartrate (LCLT) (equivalent to 2 g L-carnitine·d^{-1}) or a placebo for 23 days. On day 21 of the supplementation period, a fasting blood sample was obtained, and subjects performed an upper arm occlusion protocol with corresponding measurements of forearm muscle tissue oxygenation. On the following day (i.e., day 22), thigh muscle tissue oxygenation was assessed while subjects performed 5 sets of squat exercise; serial blood samples were obtained before exercise, for 3 hours after exercise, and 24 hours after exercise (i.e., on day 23). Following a 7-day washout, subjects began another 23-day supplementation period using the alternative supplement (i.e., LCLT or placebo), and all experimental procedures were repeated.

Subjects

The subjects were recreationally resistance-trained and had experience with the squat exercise. Subject characteristics were as follows: 25.2 ± 6.years, 91.2 ± 10.2 kg, and 180.2 ± 6.3 cm. Each signed an informed consent document approved by the institutional review board of the university after having had all of the risks of the study explained to them. Previous resistance training experience was thought to be important as to limit the amount of exercise-induced muscle tissue damage in order to optimize the role of LCLT in vascular beds, allowing for less hypoxia and more oxygen availability (3). Recreational training consisted of 2 or 3 times per week by using multiple sets and periodized heavy resistance exercise routines.

Procedures

Supplementation Protocol. A within-subject design in which subjects were their own controls was used. Subjects were provided with capsules of either L-Carnipure (Lonza, Inc., Fair Lawn, NJ), which contains 736 mg LCLT (500 mg L-carnitine and 236 mg L-tartrate), or an identical-looking placebo of powdered cellulose. Subjects consumed 2 capsules with breakfast and 2 capsules with lunch, for a total dose of 2 g L-carnitine·d^{-1}, for 23 days. Compliance was entirely based on interview, phone follow-ups, and returned bottles and supplement log sheets.

Upper Arm Occlusion Protocol. An upper arm occlusion protocol was performed to test the effects of carnitine on forearm muscle tissue oxygenation in response to local hypoxia. The upper arm was occluded with a blood pressure cuff set to approximately 50 mm Hg above the systolic blood pressure for 150 seconds and then rapidly released. Muscle tissue oxygenation of the forearm was continuously recorded with near infrared spectroscopy (NIRS) (InSpectra; Hutchinson Technology, Inc., Hutchinson, MN) for 30 seconds prior to occlusion, during 150 seconds of occlusion, and for

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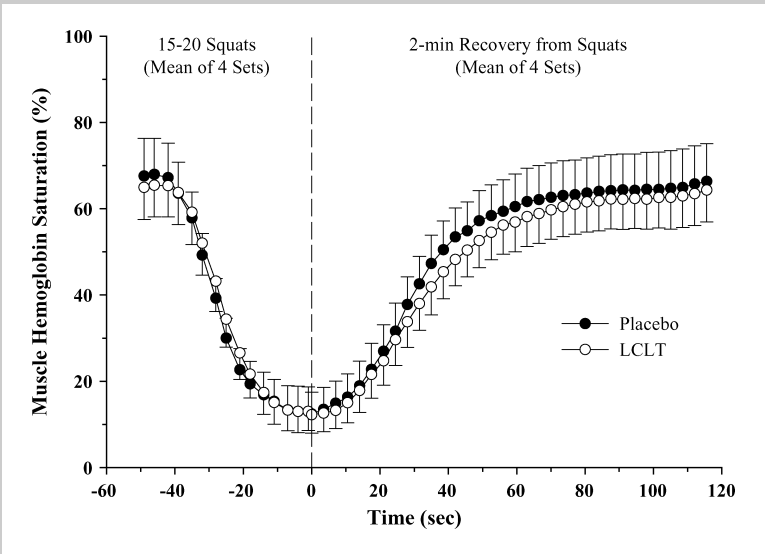

Figure 2. Thigh muscle tissue oxygenation responses (mean ± *SE*) during and following 15 to 20 repetitions of back squat exercise. Hemoglobin saturation responses to the first 4 sets of the 5-set protocol were pooled. A significant time × trial interaction existed during the recovery period and indicated significantly less hemoglobin saturation at 30 to 70 seconds of recovery during the L-carnitine L-tartrate trial compared to the placebo trial.

4 minutes after occlusion. The technique is noninvasive and measures the percentage of hemoglobin saturation in tissue based on spectrophotometric principles (i.e., the absorption spectra of oxygenated and deoxygenated hemoglobin).

Squat Exercise Protocol. The same exercise testing protocol used in previous studies examining LCLT (8,9,18,20) was also used in the current investigation. Subjects arrived to the laboratory in the afternoon, 3 hours after lunch and their last daily dose of LCLT. Subjects performed a 5-minute warm-up on a cycle ergometer followed by light stretching. They then performed 5 sets of 15 to 20 repetitions of back squats with 2 minutes of rest between sets. The starting load for the squat exercise protocol was 50% of a previously determined 1 repetition maximum. Subjects were instructed to complete as many repetitions as possible, up to 20 repetitions. If a subject was unable to perform at least 15 repetitions on their own, assistance was provided until 15 repetitions were achieved and the load was reduced for subsequent sets. Squats were performed with the Plyometric Power System (Lismore, Australia). This system allows only vertical movement of the bar along 2 steel shafts with minimal friction (i.e., typical of a Smith machine). The authors have previously used this experimental approach to induce muscle tissue hypoxia without inducing excessive muscle damage; this is an important consideration, as excessive muscle damage might affect capillary integrity and subsequently mitigate the effects of LCLT on muscle blood flow.

Blood Draws. A resting blood sample was obtained via venipuncture before the upper arm occlusion protocol and 24 hours prior to the squat exercise protocol (–24 hours). On the next day, subjects rested in a comfortable chair and a flexible Teflon cannula was inserted into a superficial arm vein prior to the squat exercise protocol. Blood samples were then collected before exercise; 0, +15, +30, +60, +90, +120, +150, and +180 minutes postexercise; and 24 hours postexercise (+24 hours).

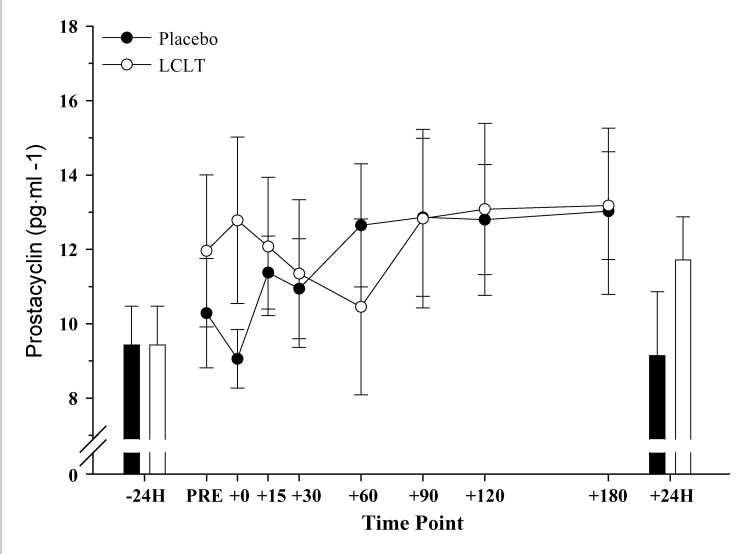

Figure 3. Prostacyclin responses (mean ± *SE*) before and after 5 sets of 15 to 20 repetitions of back squat exercise.

Biochemical Analyses. All samples were assayed in duplicate. Malondialdehyde was analyzed by using methods previously described (12). Prostacyclin was analyzed by using a competitive enzyme immunoassay (Cayman Chemical, Ann Arbor, MI) with a detection limit of 100 $pg \cdot mL^{-1}$ using a VersaMax microplate reader (Molecular Devices, Sunnyvale, CA) Intra-assay variances for all assays were less than 5%.

Statistical Analyses

Measures of central tendency and variation were calculated for all variables; obvious outliers (i.e., >2 *SD*) were removed from the data set. Data were analyzed by using a trial × time repeated-measures analysis of variance. In the event of a significant *F* score, the

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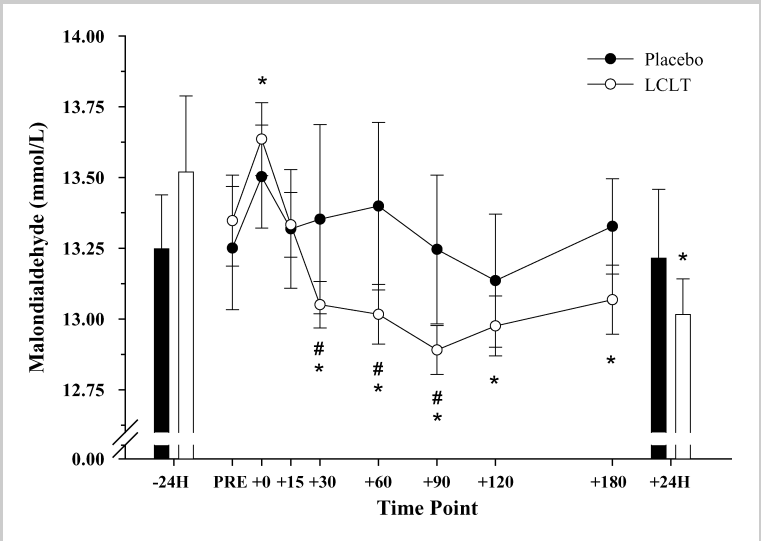

Figure 4. Malondealdehyde responses (mean ± *SE*) before and after 5 sets of 15 to 20 repetitions of back squat exercise.

Fisher least significant difference post hoc test was used to determine pairwise differences. The criterion for significance was set at $p \leq 0.05$.

Results

Forearm Muscle Tissue Oxygenation Responses to Upper Arm Occlusion

As expected, upper arm occlusion resulted in desaturation ($p \leq 0.05$) of hemoglobin in the forearm muscle tissue (Figure 1). Hemoglobin saturation rapidly (i.e., within 20 seconds) returned to resting values once the blood pressure cuff was released. A significant time × trial interaction existed during the occlusion portion of the protocol and indicated significantly less hemoglobin saturation from –129 seconds to –31 seconds during the LCLT trial compared to the placebo trial. There were no differences between trials in hemoglobin resaturation kinetics following occlusion.

Thigh Muscle Tissue Oxygenation Responses to the Squat Exercise Protocol

Due to memory storage limitations, hemoglobin saturation could not be recorded during set 5 of the 5 sets of 15 to 20 repetitions of the back squat protocol. The desaturation and resaturation responses to the first 4 sets of the back squat protocol were similar. Therefore, muscle oxygenation responses to the first 4 sets were pooled (Figure 2). Thigh muscle tissue oxygenation decreased during each set of squats and gradually returned to pre-exercise values during the 2-minute rest interval between sets. Although there were no differences between trials in muscle oxygenation during squatting, there was a significant time × trial interaction during the recovery period. This interaction indicated significantly less hemoglobin saturation from 30 to 70 seconds of recovery during the LCLT trial compared to the placebo trial.

Prostacyclin and Malondealdehyde Responses to Resistance Exercise

Circulating prostacyclin concentrations did not change following resistance exercise, nor were there significant differences between trials (Figure 3). Large intrasubject and intersubject variation potentially masked any potential differences between trials.

Malondealdehyde concentrations were measured to confirm the beneficial effects of LCLT supplementation on markers of membrane damage, as previously reported (20). Resistance exercise increased MDA concentrations at +0 in both trials (Figure 4). During the LCLT trial, MDA concentrations fell below pre-exercise values at +30 through +24 hours. Moreover, LCLT supplementation suppressed MDA concentrations compared to placebo values at +30, +60, and +90. In general, the current MDA responses were quite similar to those in the authors' previous investigation (20).

Discussion

The salient findings of the current investigation were that muscle tissue oxygenation was reduced in the LCLT trial during occlusion and during recovery from resistance exercise, that LCLT supplementation had no significant affect on circulating vasoactive prostacyclin, and that LCLT supplementation favorably affected markers of membrane damage (i.e., MDA). Although the tissue oxygenation responses were contrary to the original hypothesis, the inherent technical constraints of NIRS might rectify the seemingly contradictory finding that LCLT attenuated markers of muscle damage despite reduced tissue oxygenation.

The authors' working paradigm proposed that carnitine supplementation improves blood flow regulation (10). However, NIRS measures the ratio of oxyhemoglobin to deoxyhemoglobin to calculate the percentage of hemoglobin saturation. Because of these fundamental technical principles, NIRS can measure neither oxygen delivery nor oxygen consumption; it merely reflects the balance of oxygen delivery in relation to oxygen consumption (13). During the upper arm protocol, oxygen delivery to the forearm muscle was occluded and consequently equated between trials. Based on the principles of NIRS, this would indicate that any difference between trials was due to differences in oxygen consumption. Therefore, it is plausible that lower forearm muscle tissue oxygenation during the LCLT trial was due to enhanced muscle oxygen consumption.

Because carnitine is essential for the transport of long-chain fatty acids (i.e., carbon chain length ≥10) across the outer and inner mitochondrial membranes (i.e., carnitine

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palmitoyltransferanse I and II, respectively), early studies investigated the effects of carnitine focused on fatty acid oxidation and whole-body $\dot{V}o_2$. A preponderance of studies showed no effect of carnitine supplementation on fat metabolism during exercise (2,11,14,17,21). However, several studies reported that carnitine supplementation increased whole-body $\dot{V}o_2$max during exercise (3,11,19). These results support the contention that carnitine supplementation may have increased muscle tissue oxygen consumption.

During the squat exercise protocol, there were no differences between trials in oxygen saturation kinetics during exercise. However, during recovery, there was significantly less muscle oxygenation during the LCLT trial. As previously stated, reduced oxygenation during recovery reflects reduced oxygen delivery, enhanced oxygen consumption, or both. The contention that LCLT increased muscle tissue oxygen consumption during the forearm protocol, along with previous reports of increased $\dot{V}o_2$max following carnitine supplementation (3,11,19), provides evidence that enhanced oxygen consumption mediated the differences between trials in reoxygenation during recovery from squatting. Furthermore, enhanced tissue-level oxygen consumption during recovery may be the mechanism of LCLT-induced attenuation of hypoxic stress markers reported herein (i.e., MDA) and in previous studies (i.e., hypoxanthine, xanthine oxidase, uric acid, and MDA) (18,20).

Contrary to the hypothesis, there were no differences between trials in the vasoactive substance prostacyclin. Previous in vitro research has shown that prostacyclin mediates the beneficial effect of carnitine as an endothelium-dependent vasodilator (1). In the current investigation, large intrasubject and intersubject variation masked any potential differences between trials. Future research should further evaluate this observation, as in vivo variations in subjects' response patterns and temporal kinetics remain unknown.

In conclusion, muscle oxygenation was reduced during occlusion and following resistance exercise in response to LCLT supplementation, indicating that the balance of oxygen delivery in relation to oxygen consumption was altered. The authors contend that the reduction in hemoglobin saturation was due to LCLT-induced increases in muscle oxygen consumption, providing a potential mechanism for reduced hypoxic stress following resistance exercise reported herein and previously (18,20). Due to the technical constraints of NIRS, the effects of LCLT supplementation on blood flow delivery per se could not be determined. Therefore, future studies should consider alternate techniques (e.g., Doppler imaging, plethysmography, and functional magnetic resonance imaging) to further elucidate the mechanism or mechanisms of LCLT-induced attenuation of muscle hypoxia.

Practical Applications

Many coaches and athletes seek to improve muscle recovery following resistance exercise training sessions. Previous studies have shown that 1 to 2 $g \cdot d^{-1}$ of LCLT supplementation is safe (15) and effective (8–10,18,20) in reducing metabolic stress, minimizing muscle damage, improving hormonal receptor content, and promoting recovery following resistance exercise. Although the exact mechanism or mechanisms are unclear, LCLT supplementation likely improves muscle recovery by enhancing muscle oxygen consumption, oxygen delivery, or both. It may be considered an important compound in the array of functional foods being developed in nutrition today.

Acknowledgments

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