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## L-Carnitine L-tartrate supplementation favorably affects markers of recovery from exercise stress

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**Volek, Jeff S., William J. Kraemer, Martyn R. Rubin, Ana L. Gómez, Nicholas A. Ratamess, and Paula Gaynor.** L-Carnitine L-tartrate supplementation favorably affects markers of recovery from exercise stress. *Am J Physiol Endocrinol Metab* 282: E474–E482, 2002; 10.1152/ajpendo.00277.2001.—We examined the influence of L-carnitine L-tartrate (LCLT) on markers of purine catabolism, free radical formation, and muscle tissue disruption after squat exercise. With the use of a balanced, crossover design (1 wk washout), 10 resistance-trained men consumed a placebo or LCLT supplement (2 g L-carnitine/day) for 3 wk before obtaining blood samples on six consecutive days (D1 to D6). Blood was also sampled before and after a squat protocol (5 sets, 15–20 repetitions) on D2. Muscle tissue disruption at the midthigh was assessed using magnetic resonance imaging (MRI) before exercise and on D3 and D6. Exercise-induced increases in plasma markers of purine catabolism (hypoxanthine, xanthine oxidase, and serum uric acid) and circulating cytosolic proteins (myoglobin, fatty acid-binding protein, and creatine kinase) were significantly ( $P \leq 0.05$ ) attenuated by LCLT. Exercise-induced increases in plasma malondialdehyde returned to resting values sooner during LCLT compared with placebo. The amount of muscle disruption from MRI scans during LCLT was 41–45% of the placebo area. These data indicate that LCLT supplementation is effective in assisting recovery from high-repetition squat exercise.

hypoxia; ergogenic aid; resistance exercise; muscle damage

GIVEN THE OBLIGATORY ROLE of the carnitine system in transporting long-chain fatty acids to the mitochondrial matrix for oxidation (16), it is not surprising that there has been a great deal of research examining the potential of carnitine supplementation to enhance lipid oxidation, spare muscle glycogen, and improve exercise performance (3, 10). Despite the theoretical basis for usage of carnitine, the scientific literature has provided few “metabolic” benefits of carnitine supplementation for skeletal muscle during exercise, perhaps because of the difficulty of increasing carnitine concentrations in muscle with oral supplementation (2, 25). Using a different approach to study the effects of carnitine supplementation, Giamberardino et al. (7) demon-

strated that eccentric exercise-induced delayed-onset muscle soreness and accumulation of creatine kinase during recovery was attenuated in subjects that supplemented with L-carnitine (3 g/day for 3 wk). Their findings indicate that carnitine supplementation may have a favorable effect on recovery from exercise. The study by Giamberardino et al., however, provided no data regarding potential mechanisms to explain the beneficial effect of carnitine supplementation.

This study was designed to further examine the role of carnitine supplementation in acute exercise stress and its influence on biochemical events during recovery. Carnitine has been shown to stimulate fatty acid oxidation in vascular endothelial cells (14). Ischemia in endothelial cells results in release of carnitine, increased oxidative stress, and compromised blood flow regulation, which can be overcome by intravascular carnitine administration (5, 15). Our working hypothesis was that carnitine supplementation could protect against carnitine deficiency in vascular endothelial cells and thereby improve blood flow regulation and delivery of oxygen to muscle tissue during and after exercise (18). The enhanced oxygen delivery was hypothesized to reduce the magnitude of exercise-induced hypoxia and thus attenuate the cascade of events that lead to purine catabolism, free radical formation, membrane disruption, and muscle soreness.

The biochemical events that occur with intense exercise are multiple and complex and involve catabolism of purines, generation of reactive oxygen species, and disruption of cell membranes. Performance of exercise with a strong eccentric component results in transient episodes of hypoxia, breakdown of ATP, accumulation of ADP within the smooth muscle of the precapillary sphincter, and activation of the enzyme adenylate kinase (24). Adenylate kinase catalyzes the formation of ATP and AMP from two molecules of ADP. Accumulation of AMP leads to the formation of hypoxanthine that diffuses out of the capillary endothelial cell (24). Hypoxia induced by exercise above maximal oxygen consumption also causes a mismatch between ATP supply and demand, resulting in the malfunction of

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ATP-dependent calcium pumps and intracellular accumulation of calcium (24). The increase in cellular calcium activates calcium-dependent proteases that lead to the proteolytic cleavage of a portion of xanthine dehydrogenase, converting it to xanthine oxidase (22). Recent work by Hellsten et al. (11) provides evidence for an increase in xanthine oxidase in human vascular cells of skeletal muscle after exercise. Xanthine oxidase then catalyzes the formation of xanthine from hypoxanthine and converts it to uric acid during intense exercise (12). These reactions use molecular oxygen as an electron acceptor and form the superoxide radical. The superoxide radical can combine with iron and form reactive hydroxy radicals that attack polyunsaturated fatty acids in cell membranes and initiate a chain of lipid peroxidation reactions that are the basis for part of the membrane disruption associated with exercise. Lipid peroxidation results in the formation of numerous aldehydes of different chain lengths, such as the 3-carbon product malondialdehyde (MDA), which has been shown to increase with dynamic resistance exercise (19, 20). The disruption to the cell membrane results in leakage of cytosolic proteins into the circulation, such as creatine kinase (19), myoglobin, and fatty acid-binding protein (FABP; see Ref. 26).

The primary purpose of this study was to examine the effects of L-carnitine L-tartrate (LCLT) supplementation on the magnitude of skeletal muscle disruption associated with the performance of moderate-intensity concentric/eccentric squat exercise. We measured several intermediate and end-point biochemical markers to assess the validity of our working hypothesis that carnitine, acting via an endothelial-mediated blood flow regulatory mechanism, could reduce catabolism of purines, free radical formation, sarcolemma disruption, and perceived soreness.

## METHODS

**Experimental design.** This study involved a balanced, crossover, placebo-controlled research design that examined the effects of LCLT on markers of recovery after concentric/eccentric squat exercise. Subjects were matched for pretesting clinical values, activity background, nutritional patterns, and body size and then randomly assigned to either an LCLT or placebo group in a double-blind fashion. After 3 wk of supplementation (2 g L-carnitine/day), fasting morning blood samples were obtained on six consecutive days (D1-D6). Subjects performed a concentric/eccentric squat protocol (5 sets of 15–20 repetitions) on D2. During the squat protocol, blood samples were obtained preexercise and 0, 15, 30, 120, and 180 min postexercise. After a 1-wk washout period, subjects consumed the other supplement for a 3-wk period and performed the exact same procedures.

**Subjects.** Ten healthy, recreationally weight-trained men with a mean  $\pm$  SD age  $23.7 \pm 2.3$  yr, weight  $78.7 \pm 8.5$  kg, and height  $179.2 \pm 4.6$  cm served as subjects. All subjects were required to be engaged in a weight-training program that included the squat exercise for 1 yr before the study to exclude individuals that would experience a high degree of damage to the quadriceps in response to squatting for the first time. Subjects continued their normal training with the exception of the time period between D1 and D6 during which they were not allowed to train. All subjects were informed of

the purpose and possible risks of this investigation before signing an informed consent document approved by an institutional review board.

**Supplementation protocol.** Subjects were provided with capsules of either L-CARNIPURE LCLT (Lonza, Fair Lawn, NJ) containing 736 mg LCLT (500 mg L-carnitine and 236 mg L-tartrate) or an identical-looking placebo (powdered cellulose) with written instructions to consume two capsules with breakfast and lunch for a total dose of 2 g L-carnitine/day. Supplementation commenced 3 wk before the squat protocol and continued through recovery. This dose of carnitine was chosen to maximize plasma carnitine concentrations without exceeding the renal threshold for carnitine (8, 23).

**Exercise protocol.** The squat exercise protocol was performed on a Plyometric Power System (PPS; Lismore) previously described in detail (29). Briefly, the PPS allows only vertical movement of the bar. Linear bearings attached to either end of the bar allow it to slide up and down two steel shafts with a minimum of friction. We determined each subject's one-repetition maximum (1-RM) in the squat exercise 1 wk before any supplementation using standard procedures in our laboratory (17). Pilot studies involving different exercise loads and magnetic resonance imaging (MRI) scans were performed to determine an exercise intensity that would elicit muscle tissue disruption but not severe damage to maximize the potential for LCLT to reduce hypoxia-mediated biochemical responses to exercise stress. The exercise protocol was performed in the afternoon 3 h after lunch (reproduced by each subject during both exercise days) and 3 h after the last dose of carnitine on D2. After a standardized warm-up (5 min of cycling), subjects performed five sets of 15–20 repetitions of squat with a load equal to 50% of their previously determined 1-RM squat. There was a 2-min recovery between each set. The load was decreased if  $<15$  repetitions were performed.

**Perceived muscle soreness.** Pain was assessed using a 10-cm linear visual analog scale with labels that corresponded to "no pain" and "extreme pain" on either end. Subjects marked their level of subjective pain along the continuum, and the distance was reported as the raw score. The visual analog method has been established as a reliable method for assessing pain (21).

**Muscle tissue disruption.** Direct assessment of muscle tissue disruption and repair was evaluated using MRI cross sections and spin-spin relaxation time of the thigh muscles before the exercise test and 1 and 4 days postexercise using methods previously described in detail (6). The same investigator did all of the measurements with a reliability of  $R = 0.99$ . Scans were collected using a 0.3-Tesla open MRI magnet (AIRIS II; Hitachi Medical Systems America, Twinsburg, OH), and areas were measured with the National Institutes of Health (NIH) Macintosh computer program, NIH Image 1.55b 20, a MacIntosh Quadra 800 computer, and a scanner (Microtek Scanner III). NIH Image 1.55b 20 uses pixels of light to determine the area of skeletal muscle where damage occurs.

**Blood collections.** Blood samples were obtained on six consecutive days at the same time of the morning after a 12-h overnight fast and abstinence from alcohol and strenuous exercise. The last dose of carnitine was consumed during lunch the day before each morning blood draw ( $\sim 18$ – $20$  h earlier). Subjects reported to the laboratory between 7:00 and 9:00 AM and rested quietly for 10 min in the supine position, and  $\sim 30$  ml of blood were obtained from an antecubital vein with a 20-gauge needle and Vacutainers. On exercise days, a flexible catheter was inserted in a forearm vein, which was kept patent with a constant saline drip (60 ml/h). Before all

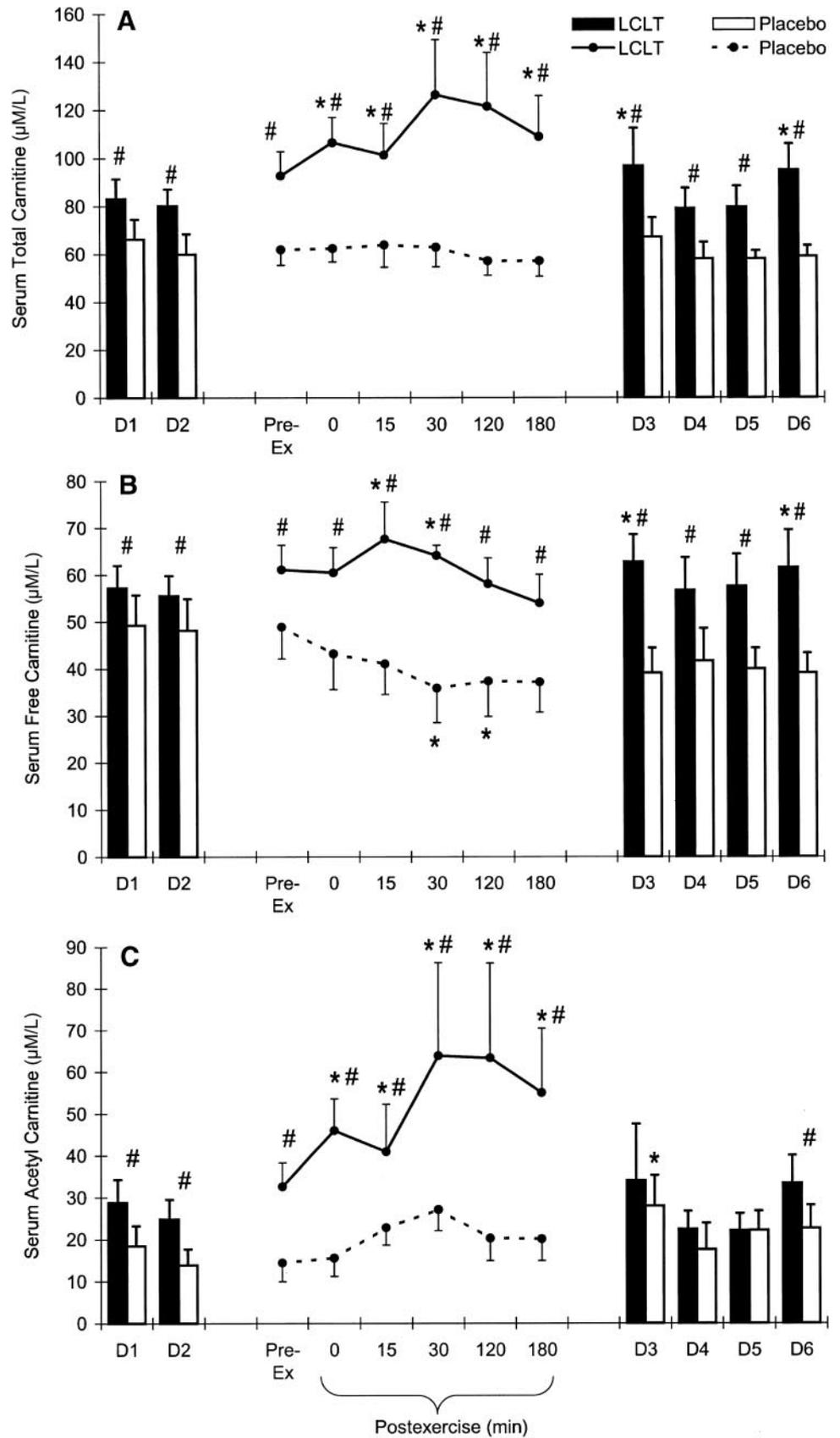


Fig. 1. Serum total carnitine (A), free carnitine (B), and acetylcarnitine (C) responses after 3 wk of either L-carnitine L-tartrate (LCLT) supplementation or placebo. Fasting morning blood samples were obtained in the morning for six consecutive days (D1-D6). Blood samples were also obtained preexercise and 0, 15, 30, 120, and 180 min after 5 sets of 15–20 repetitions of squat exercise on D2. \* $P \leq 0.05$  from corresponding preexercise value. # $P \leq 0.05$  between corresponding LCLT and placebo values.

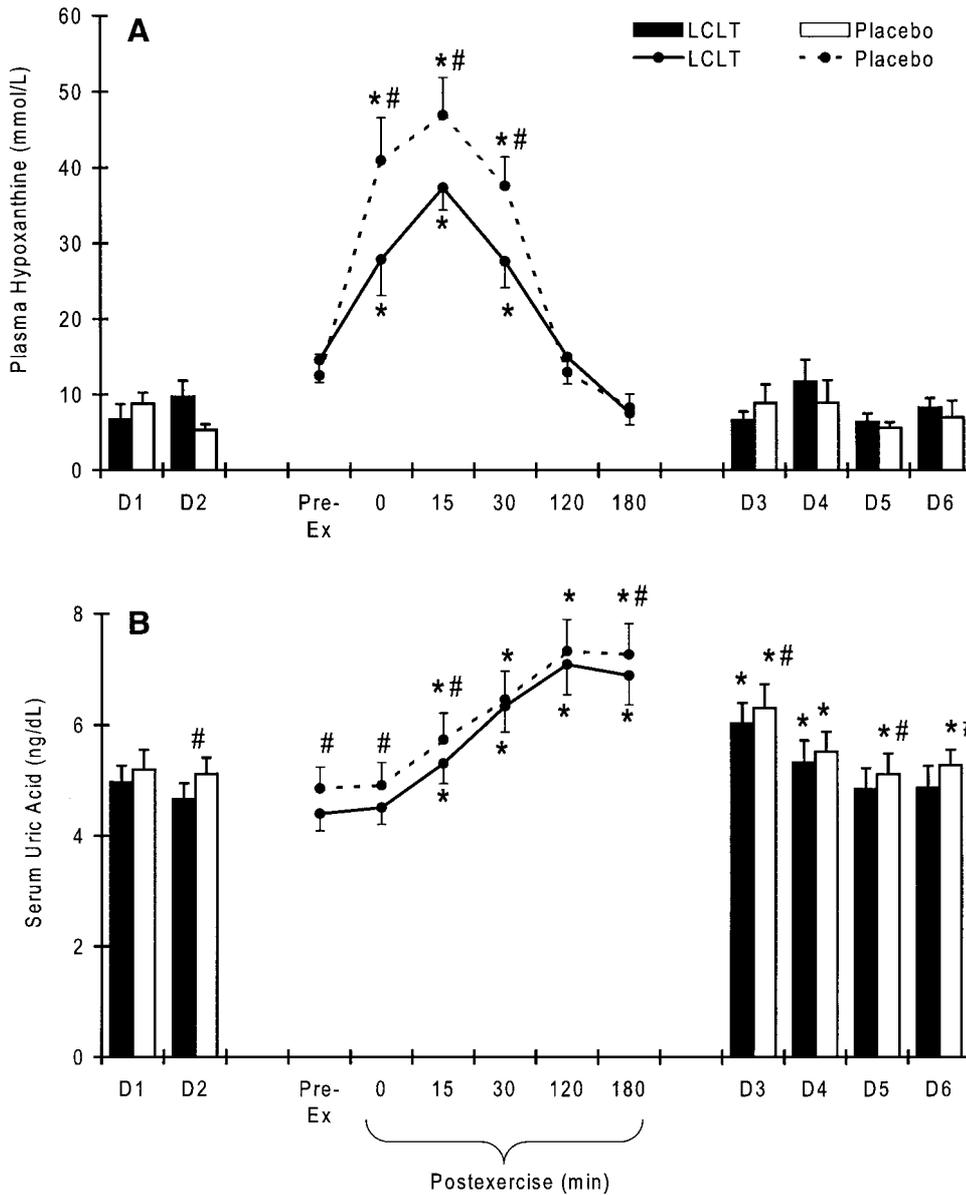


Fig. 2. Plasma hypoxanthine (A) and serum uric acid (B) responses after 3 wk of either LCLT supplementation or placebo. See Fig. 1 legend for description. \* $P \leq 0.05$  from corresponding pre-exercise value. # $P \leq 0.05$  between corresponding LCLT and placebo values.

blood collections, 3 ml of blood were withdrawn and discarded to avoid dilution of the sample, and ~30 ml were subsequently withdrawn and placed in two 10-ml tubes with a clot activator and one 10-ml tube containing EDTA. A 0.5-ml aliquot of whole blood was mixed with 1 ml of 0.8 M perchloric acid on ice for determination of plasma lactate. Within 15 min, whole blood was centrifuged (1,200 g for 15 min at 10°C), and the resultant serum/plasma was divided into aliquots and stored frozen at -80°C. Blood samples were collected preexercise and 0, 15, 30, 120, and 180 min postexercise. Subjects rested quietly in a seated position during the 3-h postexercise recovery period.

**Blood analyses.** Myoglobin was assayed in duplicate using a solid-phase <sup>125</sup>I-labeled RIA with a sensitivity of 10 ng/ml (American Laboratory Products, Windham, NH). Serum carnitine was assessed in the presence of acetyl-CoA by measuring the CoASH set free during acetyl transfer to carnitine by the enzyme carnitine acetyltransferase. The CoASH was trapped with 5,5'-dithiobis-(2-nitrobenzoic acid) and measured spectrophotometrically at 412 nm (28). Acetylcarnitine

was calculated by taking the difference between total carnitine and free carnitine. Plasma lactate and serum creatine kinase (Sigma Diagnostics, St. Louis, MO) were determined at 340 nm on a spectrophotometer (Spectronic 601; Milton Roy, Rochester, NY). Serum hypoxanthine and xanthine oxidase were analyzed in duplicate using the Amplex Red reagent-based assay (A-22182; Molecular Probes, Eugene, OR). After a 30-min incubation at 37°C, absorbances were read on a microplate reader (Wallac Victor; EG&G, Gaithersburg, MD) at 560 nm. The absorbance of the blank (zero standard) was subtracted from each data point to standardize for background absorbance. FABP was determined in plasma using a solid-phase enzyme-linked immunoabsorbent assay of the sandwich type (Hbt HK402; Cell Sciences, Norwood, MA). Plasma MDA was determined using the methods described by Wong et al. (30) and modified as described by McBride et al. (19). A phosphoric acid solution (0.44 mol/l) and a thiobarbituric acid solution (42 mol/l) were added to plasma samples and placed in a water bath (0°C) until analysis. A methanol-NaOH solution was added to the boiled

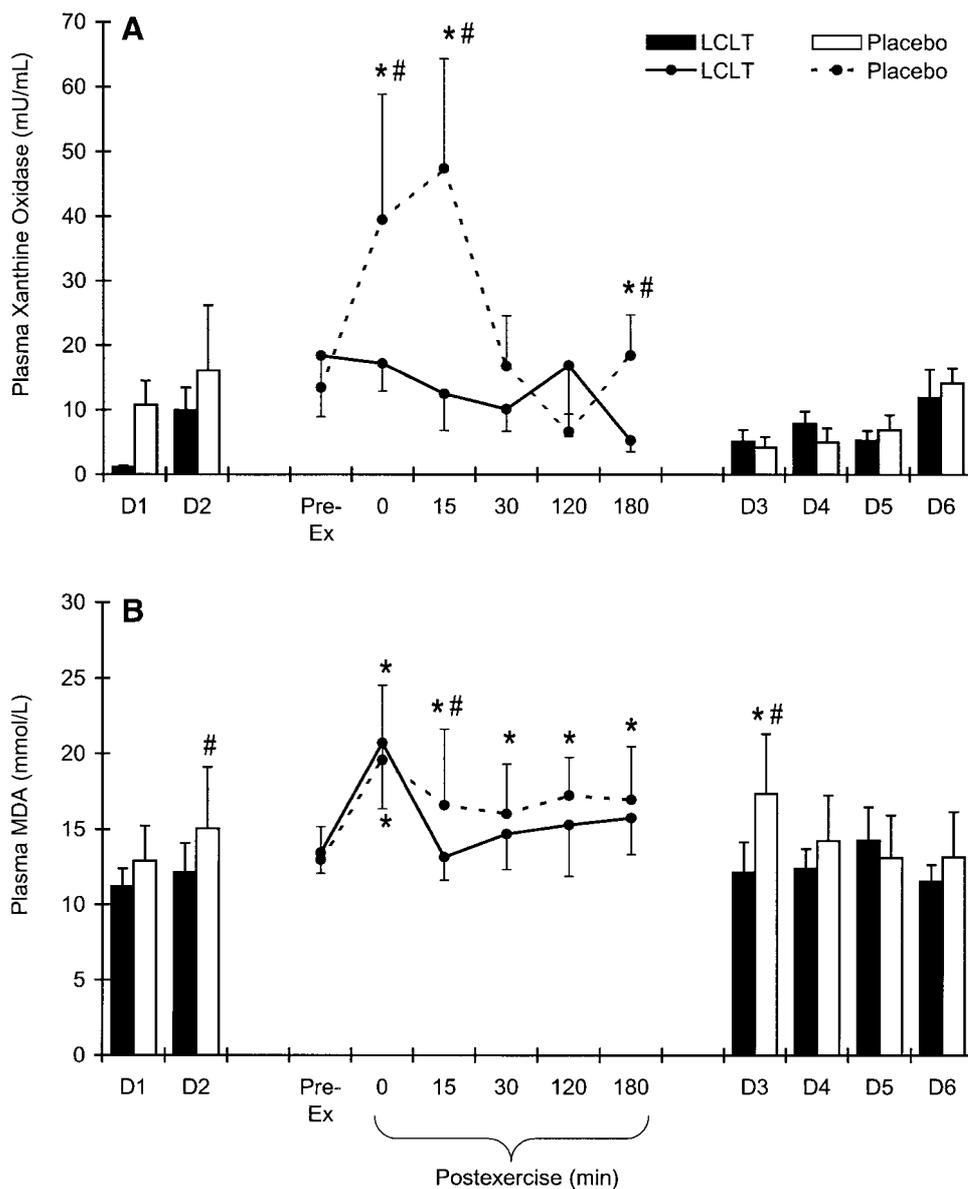


Fig. 3. Plasma xanthine oxidase (A) and plasma malondialdehyde (MDA; B) responses after 3 wk of either LCLT supplementation or placebo. See Fig. 1 legend for description.  $*P \leq 0.05$  from corresponding preexercise value.  $\#P \leq 0.05$  between corresponding LCLT and placebo values.

samples before being centrifuged to precipitate the plasma proteins. The protein-free plasma was extracted, and the absorbance was read at 532 nm on a spectrophotometer. All samples were thawed only one time, and all intra-assay and interassay coefficients of variance for each analyte were  $<10\%$ .

**Statistical analyses.** A two-way repeated-measures ANOVA was used to evaluate changes over time in the LCLT and placebo conditions. When a significant  $F$ -value was achieved, the Tukey's post hoc tests were used to locate the pairwise differences between means. The total area (serum concentration  $\times$  time) under the line connecting pre- and postexercise biochemical concentrations was calculated using the trapezoidal method. The level of significance was set at  $P \leq 0.05$ .

## RESULTS

**Serum carnitine.** Compared with placebo, serum total, free, and acetylcarnitine concentrations were significantly higher during LCLT at all time points measured with the exception of acetylcarnitine values

during D3, D4, and D5. Free carnitine was significantly reduced at 30 and 120 min postexercise during placebo, whereas free carnitine was significantly increased at 15 and 30 min postexercise during LCLT. The acetylcarnitine response to exercise was unchanged during placebo, whereas acetylcarnitine was significantly increased from 0 to 180 min postexercise during LCLT (Fig. 1).

**Lactate response.** There were significant increases in plasma lactate that peaked immediately after exercise ( $>10$  mmol/l) and returned to preexercise concentrations by 120 min postexercise. The lactate responses were not significantly different between LCLT and placebo conditions.

**Purine catabolism.** Plasma hypoxanthine concentrations were significantly elevated above preexercise values up to 30 min postexercise during LCLT and placebo. The exercise-induced responses were significantly greater during placebo. Preexercise serum uric

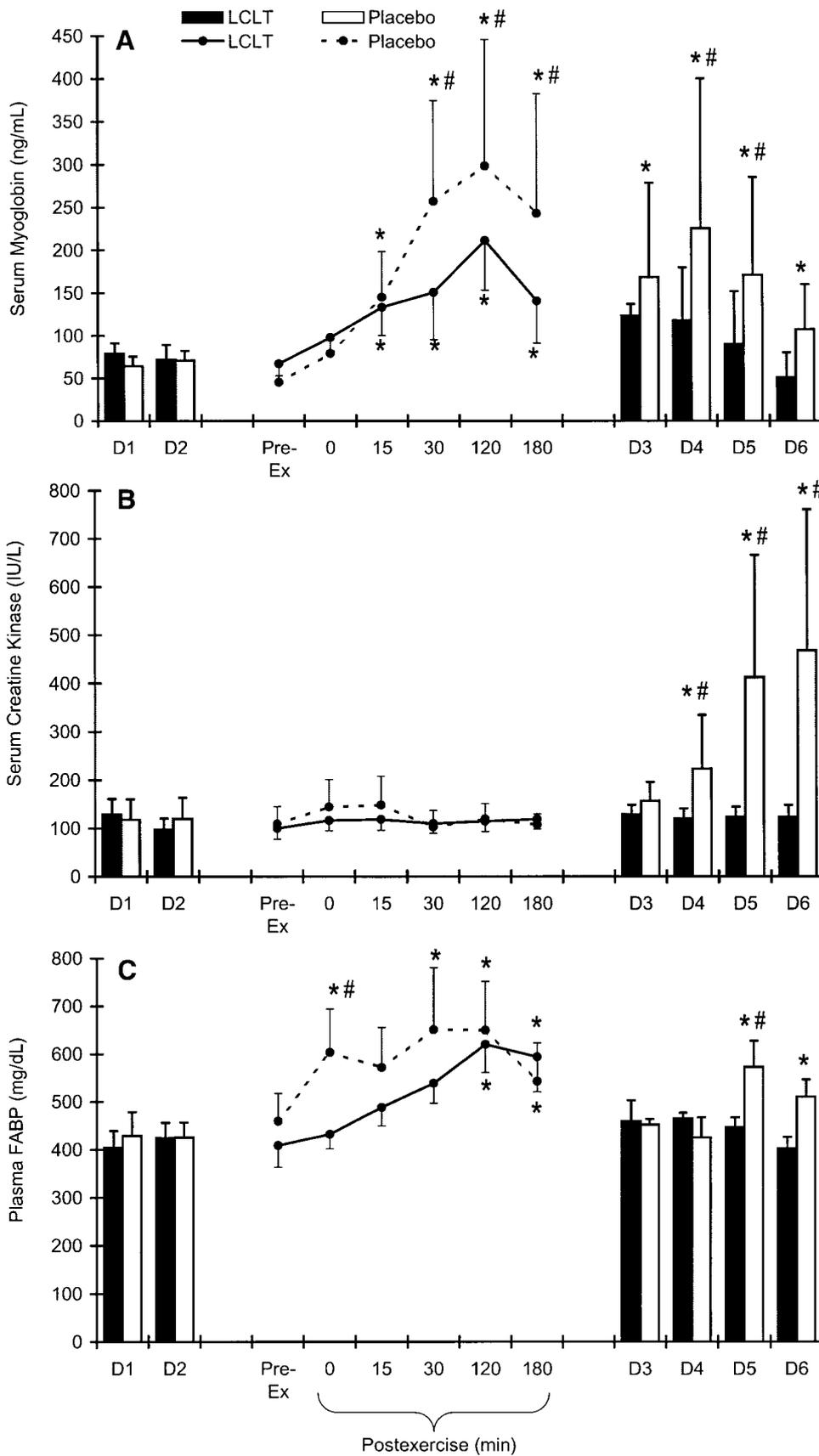


Fig. 4. Serum myoglobin (A), serum creatine kinase (B), and plasma fatty acid-binding protein (FABP; C) responses after 3 wk of either LCLT supplementation or placebo. See Fig. 1 legend for description. \* $P \leq 0.05$  from corresponding preexercise value. # $P \leq 0.05$  between corresponding LCLT and placebo values.

acid concentrations were significantly higher during the placebo vs. the LCLT condition. There were significant increases in uric concentrations that peaked ~120–180 min after exercise. Uric acid concentrations remained higher during placebo throughout the 4-day recovery period (D3–D6; Fig. 2).

**Free radical generation.** Plasma xanthine oxidase concentrations were not significantly different at any time point during LCLT but significantly increased above preexercise values at 0, 15, and 180 min postexercise during placebo. Plasma MDA responses peaked immediately after exercise during both LCLT and placebo. Plasma MDA returned to resting values by 15 min postexercise during LCLT, whereas MDA remained significantly elevated above preexercise throughout 24 h of recovery during placebo (Fig. 3).

**Cytosolic proteins.** Serum myoglobin concentrations peaked at 120 min postexercise, and the response was significantly greater during placebo. Serum myoglobin returned to preexercise concentrations the day after exercise during LCLT but remained significantly elevated throughout the 4-day recovery period during placebo. Serum creatine kinase activity was not significantly increased at any time point during LCLT. However, creatine kinase began to accumulate above baseline levels 2 days after exercise (D4) and continued to increase further 3 (D5) and 4 (D6) days after exercise during placebo. Plasma FABP responses to exercise were variable, increasing immediately after exercise during placebo and at 120 min postexercise during LCLT. Compared with LCLT, corresponding FABP values in the placebo group were significantly higher immediately after exercise and on the third day of recovery (D5; Fig. 4).

**Muscle disruption and perceived soreness.** The percentage of muscle tissue disruption assessed from MRI cross-sectional scans of the mid thigh at D3 and D6 were  $23 \pm 8$  and  $16 \pm 5\%$  for LCLT and  $39 \pm 5$  and  $29 \pm 6\%$  for placebo, respectively. The percent muscle tissue disruption was significantly greater during placebo. Perceived muscle soreness peaked the day after exercise and continued to decline throughout the 4 days of recovery. Compared with corresponding values for LCLT, soreness ratings were significantly higher 1, 2, and 4 days after exercise during placebo (Table 1).

## DISCUSSION

The mechanical and biochemical stress responses to exercise are complex and involve activation of the adenylate kinase system, generation of reactive oxygen species, inflammatory responses, and disruption to the contractile apparatus and cell membranes that ultimately contribute to fatigue by adversely impacting the metabolic, structural, and functional integrity of muscle. Similar to ischemia-reperfusion, intense exercise creates a scenario for certain active muscle fibers that can lead to transient or prolonged episodes of hypoxia followed by reoxygenation at the cessation of exercise. In addition, high rates of ATP turnover contribute to a mismatch between ATP supply and de-

Table 1. *Ratings of perceived muscle soreness*

	LCLT	Placebo
Preexercise (baseline)	$0.6 \pm 1.1$	$0.1 \pm 0.2$
Postexercise		
Day 1	$4.2 \pm 1.6^*$	$5.8 \pm 1.8^{*\dagger}$
Day 2	$4.9 \pm 1.8^*$	$5.5 \pm 1.7^{*\dagger}$
Day 3	$3.1 \pm 1.2^*$	$2.1 \pm 1.3^*$
Day 4	$1.0 \pm 1.2$	$1.8 \pm 0.6^{*\dagger}$

Values are means  $\pm$  SD. Pain was assessed using a 10-cm linear visual analog scale (1 = no pain; 10 = extreme pain). LCLT, L-carnitine L-tartrate. \* $P \leq 0.05$  from corresponding preexercise value.  $\dagger P \leq 0.05$  from corresponding LCLT value.

mand. To examine the potential for LCLT supplementation to impact recovery from exercise, we measured indicators of adenine nucleotide degradation (hypoxanthine and uric acid), free radical formation (MDA and xanthine oxidase), cell membrane damage (creatine kinase, FABP, and myoglobin), muscle disruption/damage, and perceived muscle soreness. A major assumption of our working hypothesis was that LCLT supplementation would lead to an accumulation of carnitine in endothelial cells (or prevent carnitine deficiency) and thereby increase blood flow, oxygen delivery, and regeneration of ATP. In general, the results support our hypothesis that LCLT would attenuate the biochemical and structural stress responses to high-repetition squat exercise.

Serum total carnitine concentrations were significantly higher during all time points of the study during LCLT supplementation, indicating good compliance with the supplementation protocol. During placebo, there was no change in the total carnitine response to exercise; however, free carnitine was significantly reduced at 30 and 120 min postexercise, and there was a slight increase in acetylcarnitine. A decrease in serum free carnitine and an increase in acetylcarnitine after intense exercise is in agreement with other studies that have examined carnitine responses to exercise (1, 9). Although there are proportional changes in free and acetylcarnitine in muscle during exercise, it is generally accepted that the total muscle carnitine concentration is not altered during exercise and does not interact significantly with carnitine metabolism in the circulation (10).

Plasma lactate responses to squat exercise were significantly increased ( $>10$  mmol/l) to a similar extent during LCLT and placebo, indicating a similar glycolytic and acid-base stress between conditions. High glycolytic rates are associated with accumulation of ADP and  $H^+$ , which favor activation of adenylate kinase and the formation of ATP and AMP from two molecules of ADP. In muscle, AMP is oxidized to hypoxanthine. Indeed, plasma hypoxanthine was significantly elevated above preexercise values during the early postexercise period, and the response was attenuated by LCLT supplementation, indicating a better match between ATP supply and demand.

During strenuous exercise, the inadequate generation of ATP can cause malfunctioning of calcium

ATPase pumps and an increase in intracellular calcium, which in turn activates calcium-dependent proteases. These proteases cleave a portion of xanthine dehydrogenase, converting it into xanthine oxidase, which utilizes molecular oxygen as an electron acceptor instead of NADH. Xanthine oxidase results in the generation of superoxide radicals during exercise (13), which can initiate lipid peroxidation and cell disruption/damage (4). In this study, plasma xanthine oxidase was significantly elevated above preexercise values during the 0 and 15 min postexercise time points during the placebo condition, whereas the exercise-induced response was eliminated during LCLT. The conversion of xanthine dehydrogenase to xanthine oxidase occurs via calcium-dependent proteases in the cytosol (22). The significantly lower xanthine oxidase after exercise during LCLT indirectly suggests that either calcium pumps were operating more efficiently (perhaps because of better ATP supply) and/or there was less disruption to the muscle/sarcoplasmic reticulum membrane that would cause ionic disturbances resulting in accumulation of calcium in the cytosol.

Inhibition of xanthine oxidase with allopurinol during exercise has been shown to result in significantly less generation of reactive oxygen species, less accumulation of cytosolic enzymes (creatine kinase and lactate dehydrogenase), and less tissue damage after exhaustive exercise (4, 13, 27). In this study, the lower xanthine oxidase response during LCLT was also associated with less accumulation of reactive oxygen species (as measured by MDA), less accumulation of cytosolic proteins (as measured by circulating creatine kinase activity, myoglobin, and FABP), less tissue damage (as measured by MRI), and less subjective muscle soreness after squat exercise.

Generation of the superoxide radical during intense exercise (presumably via endothelial xanthine oxidase) could initiate a series of tissue-destructive reactions, including lipid peroxidation. We used MDA as a quantitative marker for free radical interaction with cell membranes. There was a significant increase in plasma MDA to the squat exercise protocol that peaked immediately after exercise. We previously demonstrated that a whole body resistance exercise protocol resulted in significant increases in plasma MDA immediately postexercise (19). Plasma MDA returned to preexercise values by 15 min postexercise during LCLT but remained significantly above preexercise values for >180 min postexercise during placebo. The overall lower MDA response to the squat protocol indicates that LCLT resulted in less total exposure of cell membranes to the damaging effects of reactive oxygen species. Increased generation of reactive oxygen species would be predicted to cause greater disruption/damage to the sarcolemma and greater leakage of cytosolic proteins into the circulation. The extent of muscle damage measured using MRI and the accumulation of cytosolic proteins in the circulation were significantly reduced by LCLT, providing further evidence for a protective effect of LCLT on muscle disruption/damage.

It has been proposed that FABP and myoglobin represent more useful markers for the early detection of exercise-induced muscle damage because of their relatively rapid response, whereas the creatine kinase response to exercise occurs at a slower rate (26). In the acute 3-h recovery period after exercise, we observed significantly lower postexercise myoglobin and FABP responses during LCLT. Creatine kinase activity was not increased during LCLT or placebo during the 3-h recovery period but progressively increased above baseline beginning 2 days after exercise during placebo. The lack of a creatine kinase response to exercise during LCLT is indicative of less disruption to the sarcolemma. Similar to our findings, Giamberardino et al. (7) demonstrated that the creatine kinase responses to a 20-min eccentric step test were attenuated in subjects that consumed 3 g carnitine/day for 3 wk before exercise.

Our data provide indirect evidence of a favorable effect of LCLT supplementation (2 g L-carnitine/day for 3 wk) on endothelial blood flow regulation during and after moderate-intensity squat exercise, as evidenced by significantly less accumulation of markers of purine degradation, free radical formation, tissue damage, and muscle soreness. Neither endothelial carnitine concentrations nor local blood flow was measured in this study to add more direct support to our hypothesis. However, the data do favor a significant effect of LCLT on recovery stress responses acting via a cell/tissue-related mechanism. We favor the vascular compartment as the target for the beneficial effect of carnitine on exercise recovery responses.

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