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The Effects of L-Carnitine L-Tartrate Supplementation on Hormonal Responses to Resistance Exercise and Recovery

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ABSTRACT

The purpose of this investigation was to examine the influence of L-carnitine L-tartrate (LCLT) supplementation using a balanced, cross-over, placebo-controlled research design on the anabolic hormone response (i.e., testosterone [T], insulin-like growth factor-I, insulin-like growth factor-binding protein-3 [IGFBP-3], and immunofunctional and immunoreactive growth hormone [GHif and GHir]) to acute resistance exercise. Ten healthy, recreationally weight-trained men (mean ± *SD* age 23.7 ± 2.3 years, weight 78.7 ± 8.5 kg, and height 179.2 ± 4.6 cm) volunteered and were matched, and after 3 weeks of supplementation (2 g LCLT per day), fasting morning blood samples were obtained on six consecutive days (D1–D6). Subjects performed a squat protocol (5 sets of 15–20 repetitions) on D2. During the squat protocol, blood samples were obtained before exercise and 0, 15, 30, 120, and 180 minutes postexercise. After a 1-week washout period, subjects consumed the other supplement for a 3-week period, and the same experimental protocol was repeated using the exact same procedures. Expected exercise-induced increases in all of the hormones were observed for GHir, GHif, IGFBP-3, and T. Over the recovery period, LCLT reduced the amount of exercise-induced muscle tissue damage, which was assessed via magnetic resonance imaging scans of the thigh. LCLT supplementation significantly ($p < 0.05$) increased IGFBP-3 concentrations prior to and at 30, 120, and 180 minutes after acute exercise. No other direct effects of LCLT supplementation were observed on the absolute concentrations of the hormones examined, but with more undamaged tissue, a greater number of intact receptors would be available for hormonal interactions. These data support the use of LCLT as a recovery supplement for hypoxic exercise and lend further insights into the hormonal mechanisms that may help to mediate quicker recovery.

Key Words: hypoxic stress, growth hormone, muscle damage, testosterone, growth factors, IGFBP-3 binding protein

Reference Data: Kraemer, W.J., J.S. Volek, D.N. French, M.R. Rubin, M.J. Sharman, A.L. Gómez, N.A. Ratamess, R.U. Newton, B. Jemiolo, B.W. Craig, and K. Häkkinen. The effects of L-carnitine L-tartrate supplementation on hormonal responses to resistance excercise and recovery. *J. Strength Cond. Res. 17(3):455–462. 2003.*

Introduction

Endogenous carnitine is a necessary component of fat oxidation in cells. Specifically, carnitine acts in a transport system at the level of the mitochondria that facilitates the transport of fatty acids across the mitochondrial matrix for the subsequent metabolism and energy production via beta-oxidation. As a result of carnitine's known role in fat oxidation, dietary supplementation with the amino acid-like nutrient L-carnitine was examined extensively in the context of exercise in the early to mid-1990s as a potential tool to enhance fatty acid delivery to the mitochondria, thus enhancing fat utilization during exercise (2, 4, 5, 12, 26, 28). Unfortunately, despite a logical theoretical rational for its use, carnitine supplementation proved to be largely ineffective in improving exercise performance and in enhancing fatty acid oxidation during exercise, most likely because of the fact that supplemental carnitine in the diet has not proven to increase the skeletal muscle content of carnitine in humans (28), compared with rats (1).

More recent research on L-carnitine L-tartrate

(LCLT) supplementation in the context of exercise has focused on a different hypothesis separate from the previously examined role of L-carnitine in fat metabolism (27). Recent work from our laboratory involves the examination of L-carnitine as a potential protective mechanism to attenuate hypoxic-free radical tissue damage during exercise and into recovery (27). L-carnitine, by accumulating in the capillary endothelial cells, may enhance oxygen delivery to exercising muscles via a vasodilatory effect on the capillary, thereby reducing local muscular hypoxia normally observed during high-intensity exercise and a cascade of events leading to free radical formation and chemical-induced tissue disruption and damage. In addition, blood flow to other important tissues and glands in the body may be enhanced and provide additional mechanisms beyond the circulatory flow to muscle to enhance recovery (e.g., cells of the pituitary gland, liver, and testis all have complex capillary systems that provide oxygen and other nutrients to these endocrine glands and provide a route for hormone transport into the circulation). In general, the results from our prior study provided the first data to support this hypothesis (27). We found a favorable effect of LCLT supplementation 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. In addition, Giamberardino et al. (9) had also indicated that supplementation with L-carnitine (3 g per day for 3 weeks) can attenuate the exercise-induced delayed onset of muscle soreness and the accumulation of creatine kinase following eccentric effort of the quadriceps muscle, but this study focused more on its impact because of high levels of mechanical damage in which optimal blood flow would not be present. Thus, LCLT supplementation may provide benefit in the recovery process from exercise stress, yet more research is needed to elucidate the potential-mediating mechanisms of this process (7, 27).

The biochemical events that occur consequent to intense exercise are numerous and complex and include catabolism of purines, generation of reactive oxygen species, and disruption of cell membranes and cytoskeleton (26, 27). To promote recovery from individual exercise bouts, optimal conditions are necessary. Recovery involves the coordinated functioning of several physiological processes that are heavily influenced by the availability and actions of specific hormones (6, 10, 13–15). Of particular importance to the recovery following exercise-induced muscle damage and regeneration of cellular structure are the anabolic hormones and growth factors, including testosterone, growth hormone (GH), and insulin-like growth factor-I (IGF-I) (6, 16–20). Interactions between these hormones and their muscle cell receptors during the recovery phase from exercise have the effect of stimulating repair and promoting structural remodeling through processes of protein synthesis (14, 15, 20). Enhanced protein turnover favors the repair and growth of damaged muscle tissue and is acutely regulated by the homeostatic interactions of these anabolic hormones. Strenuous exercise clearly disrupts or damages the structure of muscle fibers, which later during recovery must undergo a remodeling process. It may therefore be reasonable to consider a link between circulating concentrations of anabolic hormones and increased rates of protein synthesis leading to improved regeneration and recovery.

The importance of the anabolic hormones during the recovery process has been highlighted by MacDougal et al. (20), who observed elevated protein synthesis in trained muscle up to 36 hours after the completion of resistance exercise. Many studies have shown that the circulating concentrations of anabolic hormones are acutely increased following resistance exercise (16–19). In addition, studies using supraphysiological doses of testosterone have further documented its anabolic effect on muscle tissue (3, 15). Though previous research has indicated L-carnitine may have a protective mechanism during exercise and promotes increased recovery following exercise (7, 27), no studies have examined the effects of L-carnitine supplementation on anabolic hormones and growth factors following resistance exercise. It therefore remains unknown whether the improved recovery status reported in previous studies (7, 9, 27) is the consequence of changes in circulating concentrations of anabolic hormones and their ability to augment cell-tissue repair and regeneration. Therefore, the purpose of this investigation was to examine the influence of LCLT supplementation on the anabolic hormone response to acute resistance exercise.

Methods

Experimental Approach to the Problem

We wanted to further extend our understanding about the recovery benefits and possible mechanisms related to LCLT supplementation and hormonal factors mediating such anabolic repair have not been elucidated. This study involved a balanced, cross-over, placebo-controlled research design that examined the effects of LCLT on markers of anabolic hormonal response 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 weeks of supplementation (2 g LCLT per day), fasting morning blood samples were obtained on 6 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 before

exercise and 0, 15, 30, 120, and 180 minutes after exercise. After a 1-week washout period, subjects consumed the other supplement for a 3-week period, and the same experimental protocol was repeated using the exact same procedures.

Subjects

This study extended our work from our prior study using the same 10 healthy, recreationally weight-trained men with a mean ± *SD* age 23.7 ± 2.3 years, weight 78.7 ± 8.5 kg, and height 179.2 ± 4.6 cm who served as subjects. All subjects were required to be engaged in a strength-training program that included the squat exercise for 1 year 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 the institutional review board. A registered dietitian was utilized to screen the potential subjects for dietary behaviors or confounding supplement use. Dietary and activity logs were used to standardize the behaviors under each experimental condition to remove the potential for confounding nutritional and behavioral influences.

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 2 capsules with breakfast and lunch for a total dose of 2 g L-carnitine per day. Supplementation commenced 3 weeks 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 (11, 25). Blood concentrations acted as the internal marker of adherence to the protocol. This dose has also been shown to be safe (24).

Exercise Protocol

The squat exercise protocol was performed on a Plyometric Power System (PPS, Lismore, Australia) 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 2 steel shafts with a minimum of friction. We determined each subject's 1 repetition maximum (1RM) in the squat exercise 1 week 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 hours after lunch (reproduced by each subject during both exercise days) and 3 hours after the last dose of carnitine on D2. After a standardized warm-up (5 minutes of cycling), subjects performed 5 sets of 15–20 repetitions of squat with a load equal to 50% of their previously determined 1RM squat. There was a 2-minute recovery between each set. The load was decreased if <15 repetitions were performed.

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 by Dudley et al. (8) in detail. 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 (Apple Computer, Inc., Cupertino, CA), and a scanner (ArtixScan 1100, Microtek Laboratories, Inc., Redondo Beach, CA). 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 6 consecutive days at the same time of the morning after a 12-hour 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 (~18–20 hours earlier). Subjects reported to the laboratory between 7:00 and 9:00 AM and rested quietly for 10 minutes in the supine position, and ~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 into a forearm vein, which was kept patent with a constant saline drip (60 ml·h^{-1}). Before all 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 2 10-ml tubes with a clot activator and 1 10-ml tube containing EDTA. Within 15 minutes, whole blood was centrifuged (1,200*g* for 15 minutes at 10° C), and the resultant serum-plasma was divided into aliquots and stored frozen at −80° C. Subjects rested quietly in a seated position during the 3-hour postexercise recovery period.

Biochemical Analyses

Circulating immunoreactive plasma GH was measured via the Nichols immunoradiometric assay

(IRMA) (Nichols Institute Diagnostics, San Juan Capistrano, CA). This commercially available assay uses two monoclonal antibodies of high affinity and specificity for GH. Each antibody detects a different epitope on the GH molecule. One antibody is labeled with ^{125}I for detection and the other is coupled to biotin. The sensitivity for this assay is 0.04 dn·ml. Intra-assay variances were less than 5%.

Immunofunctional plasma GH was determined in an assay using an amplified 2-step sandwich-type assay requiring an anti-GH monoclonal antibody and biotinylated recombinant GH binding protein (GHBP) that bind, respectively, to GH receptor-binding site 2 and site 1 present on all ''biologically active'' GH molecules (Diagnostics Systems Laboratories, Inc. [DSL], Webster, TX). Plasma samples were incubated sequentially (with intervening wash steps) with (a) an immobilized anti-GH antibody, (b) biotinylated GHBP followed by streptavidin-labeled with horseradish peroxidase, and finally (c) tetramethylbenzidine substrate. After addition of an acidic stop solution, enzyme turnover was determined by dual-wavelength absorbency measurements at 450 and 630 nm. The absorbancy measured is directly proportional to the concentration of biologically active ''intact'' GH that possesses both GH receptor-binding sites. The sensitivity for this assay, when the $B_0 \pm 2$ *SD* method was used, was 0.20 ng·ml^{-1}. Intra-assay variances for low, medium, and high GH concentrations were less than 9%.

Total serum IGF-I was determined via 2-site IRMA (DSL). This assay includes a simple ethanol extraction procedure in which IGF-I is separated from its binding protein in the serum sample, a step considered to be essential for accurate IGF-I measurement. This is a noncompetitive, sandwich-type assay in which the analyte is sandwiched between 2 antibodies, 1 immobilized to the wall of the tube and the other radiolabeled with ^{125}I for detection. The sensitivity of this assay is 0.03 ng·ml^{-1}. Intra-assay variances were less than 5%.

Measurement of circulating IGFBP-3 was performed using a 2-site coated-tube IRMA (DSL). This IRMA is a noncompetitive assay in which the analyte to be measured is sandwiched between 2 antibodies. The first antibody is immobilized on the inside walls of the tube, and the other is radiolabeled for detection. The sensitivity of this assay is 0.5 ng·ml^{-1}. Intra-assay variances were less than 5%.

Total serum testosterone was assayed in duplicate using a sold-phase ^{125}I-labeled radioimmunoassay with a sensitivity of 0.278 nmol·L^{-1} (DSL). Intra-assay variances were less than 5%.

Statistical Analyses

A 2-way repeated-measures analysis of variance (ANOVA) was used to evaluate changes over time and between the L-carnitine and placebo conditions. Trapezoidal analysis over the data collection time points was used to determine area under the curve values. Tests for normality of distribution (Kolmogorov-Smirnov chi-square test) and homogeneity of variance (Levene's test) were used on all data sets prior to ANOVAs. Post hoc comparisons were accomplished via a Fisher's least significant difference (LSD) test or Tukey test when appropriate. Statistical power was determined to be >0.80 for all measures for the sample size used at the 0.05 alpha level (nQuery Advisor software, Statistical Solutions, Saugus, MA). All statistical significance in this study was set at $p \leq 0.05$.

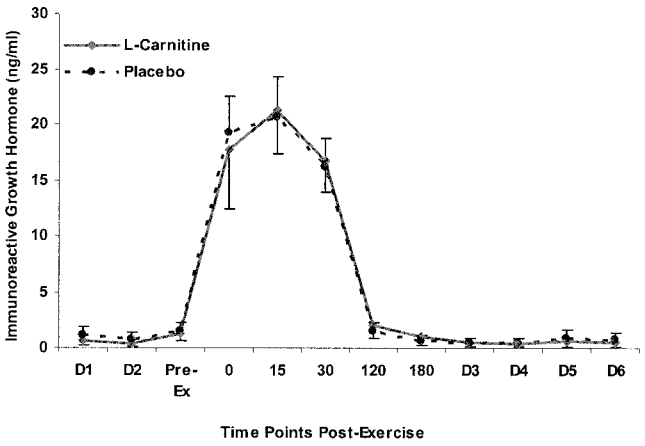

Figure 1. Responses of immunoreactive growth hormone to the squat exercise protocol. Significant increases ($p < 0.05$) above rest at 0, 15, and 30 minutes postexercise with no differences between placebo (dashed line) and L-carnitine L-tartrate (solid line).

Results

The responses of immunoreactive GH (Figure 1) and immunofunctional GH (Figure 2) demonstrated identical responses with significant increases above rest at 0, 15, and 30 minutes postexercise. The magnitude of the concentrations of immunoreactive GH was all greater than the immunofunctional GH values. No differences were observed between the LCLT and placebo conditions.

The responses of IGF-I to the squat exercise protocol (Figure 3) demonstrated no significant exercise-induced changes or any differences between the placebo and LCLT treatment conditions.

The responses of IGFBP-3 to the squat exercise protocol (Figure 4) demonstrated a significant increase above rest at 0 minutes after exercise. There were significant differences between the placebo and LCLT treatment conditions on D1 and D2 as well as at 30, 120, and 180 minutes after the exercise protocol with the LCLT treatment responding with higher binding protein. In addition, the LCLT treatment demonstrated a significantly higher area under the curve concentrations than the placebo treatment.

The responses of testosterone to the squat exercise

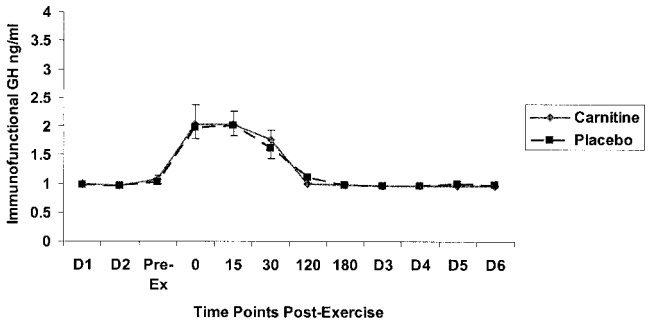

Figure 2. Responses of immunofunctional growth hormone to the squat exercise protocol. Significant increases ($p < 0.05$) above rest at 0, 15, and 30 minutes postexercise with no differences between placebo (dashed line) and L-carnitine L-tartrate (solid line).

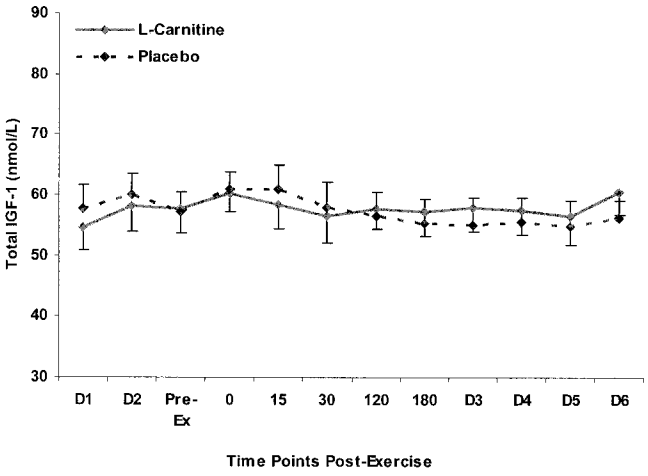

Figure 3. Responses of total insulin-like growth factor-I to the squat exercise protocol. No significant ($p > 0.05$) changes with exercise and no differences between placebo (dashed line) and L-carnitine L-tartrate (solid line).

protocol (Figure 5) demonstrated significant increases above rest at 15 minutes postexercise. There were no differences between the placebo and LCLT treatment conditions.

Using a control resting MRI cross-sectional scan of the midthigh as the baseline for comparison with postexercise changes to determine the effects of the resistance exercise stress, the percentage of muscle tissue disruption-damage assessed at D3 and D6 were 23 ± 8 and 16 ± 5% for LCLT and 39 ± 5 and 29 ± 6% for placebo, respectively. The percent muscle tissue disruption was significantly greater during placebo treatment condition. The delta damage from D3 to D6 was significantly less in the LCLT treatment condition.

Discussion

Accumulation of LCLT in the capillary endothelial cells appears to have enhanced the oxygen delivery to exercising muscles via a vasodilatory effect on the capillary, resulting in a reduced local muscular hypoxia

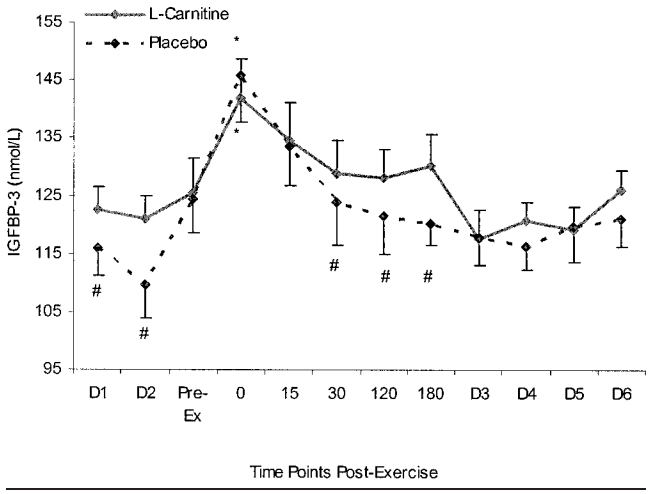

Figure 4. Responses of insulin-like growth factor-binding protein-3 to the squat exercise protocol. Significant increases ($p < 0.05$) above rest at 0 minutes postexercise with significant differences between placebo (dashed line) and L-carnitine L-tartrate (solid line) at D1 and D2 and at 30, 120, and 180 minutes after exercise.

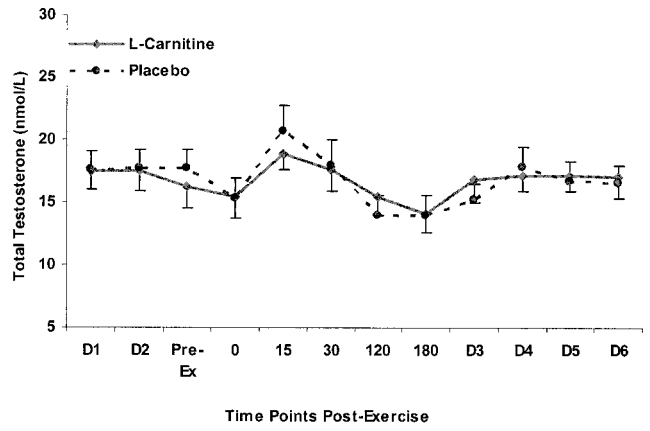

Figure 5. Responses of testosterone to the squat exercise protocol. Significant increases ($p < 0.05$) above rest at 15 minutes postexercise with no significant differences between placebo (dashed line) and L-carnitine L-tartrate (solid line) treatment groups.

typically observed during exercise stress. This appears to have reduced the magnitude of damage related to prevented to free radical formation and chemical-induced tissue disruption and damage (27). Important to this investigation was the finding that LCLT supplementation significantly reduced the amount of damage by 7–10% of the area allowing for greater numbers of stabilized cellular receptors available for binding interactions and cellular signals for increased protein synthesis or anticatabolic effects at the receptor level. This finding may provide key insights into the mechanisms of action and the response patterns of the anabolic hormones in circulation. Thus, the greater amount of undamaged tissue may have preserved the ability for hormonal interactions with muscle tissue

and help to explain the reduced progression of chemical tissue damage that occurred from D3 to D6, compared with the placebo treatment conditions.

Discovered in 1957, the evolution of the study of the IGF-I system has been shown to be an important hormonal interface for metabolic, mitogenic, and anabolic cellular dynamics in response to exercise stress (23). With the use of 3 weeks of LCLT supplementation, increases in IGFP-3 were observed on D1 and D2 and from 30 to 180 minutes during the recovery period following the acute squat exercise protocol. Concomitantly no increases or supplement effects were observed with IGF-I concentrations. The interpretation of these findings needs to be examined in unison because of the intimate interrelationships of their cybernetic function.

IGFBP-3 is the predominant IGF-binding protein in adult serum and more than 75% of IGF-I circulates in a 150 kD ternary complex comprised of IGF-I–IGFBP-3 and an acid labile subunit. Smaller proportions of IGF-I are bound to smaller-molecular-weight–binding proteins or in free form. It appears that IGFBP-3 is independent of GH regulation and inhibits IGF-I actions implying that the IGFBP-3 can modulate IGF-I binding to its receptor and thereby affect and titrate biological actions. This essentially allows the sequestering of IGF-I away from its cellular receptor when increases of the binding protein occur. It is the free and nonternary complex that has the acceptable molecular size to pass through the capillary fenestrations and into the extracellular fluid to interact with the cellular membrane to exert its effects. The IGFBP-3 can increase and extend the half-life of the IGF-I–IGFBP-3 complex by 1–2 hours before degradation of the ternary complex occurs. Thus, this binding protein helps to provide a mechanism for a longer period of time in which IGF-I concentrations can be biologically dosed to enhance the cellular interactive environment with free IGF-I. This may be one of the hormonal mechanisms that provides for more optimal trophic interactions that would positively influence protein metabolism and enhance muscle tissue repair. Thus, although no changes in the IGF-I concentrations were observed, the use of LCLT supplementation apparently enhances viability, half-life, and biocompartment kinetics of IGF-I. Such findings extend the supplementation construct of LCLT to affecting the partitioning of the IGF-I system in a manner that can be enhance cellular interactions.

The lack of an exercise-induced increase in IGF-I concentrations in response to acute exercise stress may well be related to the already highly conditioned nature of our subjects in this study. This is supported by findings that resistance training has been shown to have the ability to increase resting serum concentrations of IGF-I (21). In prior studies by our laboratory groups, we have observed increases or no changes in response to a resistance exercise protocol. Although many factors related to composition of the exercise stress might be put forth as to possible reasons for a lack of change with acute exercise, one important observation is the fact that the starting concentrations of IGF-I may be the most important factor related to the potential to observe an exercise-induced increase in the circulatory blood (16, 18, 19). Higher starting concentrations may inhibit the ability to stimulate a further increase in IGF-I with exercise stress because of negative feedback mechanisms or at least mask the small changes that may occur within a very narrow biological window of the maximal endogenous concentrations possible with exercise stress. The concentrations found in the current study were similar to our group's prior study in which no increases in response to acute resistance exercise stress were observed (16, 19) and were much higher than earlier research demonstrating increases (17, 18).

No changes were observed in immunoreactive GH or immunofunctional GH in response to LCLT supplementation. Although only a few studies have examined resistance exercise and immunofunctional GH responses concomitant with immunoreactive GH concentrations, expected increases with acute exercise stress were observed and the lower magnitude of concentrations of immunofunctional GH, compared with immunoreactive GH were consistent with the findings in our prior research team's reports (13, 22). The increase in GH is important for the somatogenic and metabolic actions following resistance exercise and has been thought to be one of the major influences of anabolic actions including influencing the IGF system and testosterone. The immunofunctional assay used in this investigation was designed on the basis that this assay detects only those intact molecules that possess both sites 1 and 2, which are required to induce signal transduction. Conversely, the Nichols IRMA used to determine immunoreactive GH concentrations utilizes monoclonal antibodies that interact with epitopes of unknown specificity.

Interestingly, the immunofunctional concentrations were almost 10-fold lower in response to this specific resistance exercise protocol, compared with our prior work using 10RM at a minimal of 75% of 1RM. This may support the importance of load to anabolic hormonal signals. However, as we have previously theorized the biological importance of other GH variants not able to dimerize receptors cannot be dismissed as these forms may have important inhibitory and modulatory roles in the regulation cellular events (22). The reproducibility of the hormonal fingerprints of GH as well as the other hormones in this study again show that one can examine circulatory hormones with great sensitivity (19). The molar concentrations of GH hormone(s) had a greater amount of undamaged area of muscle tissues to interact with during the LCLT treat-

ment condition, potentially leading to a greater anabolic effect (6). This may be a vital factor for the optimal muscle tissue interactions of both biologically potent immunofunctional GH and potentially potent immunoreactive GH. How aggregates as measured by other bioassays (13) (i.e., higher-molecular-weight isoforms) of such GH monomers respond to such LCLT supplementation remains a missing perspective in the study of the pituitary function and exercise responses.

Different from peptide hormone interfaces with the cell myonuclei, testosterone as a steroid hormone has a more direct interaction on the DNA regulatory element in skeletal muscle. Increases in serum testosterone were observed with exercise stress under both the LCLT and placebo treatment conditions at 15 minutes of recovery, demonstrating as with immunofunctional GH a reduced magnitude of the response pattern most likely caused by the loading used in the study design (16, 17, 19). No absolute effect of LCLT supplementation was observed, but again more undamaged muscle tissue was available for hormonal interactions during the LCLT treatment condition. The importance of this fact lies in the greater number of receptors that are stabilized leading to greater transcription and ultimately to greater protein synthesis (15). Thus, the importance of optimizing tissue interfaces for hormonal interactions may play an important role in optimizing the recovery patterns of muscle tissue as demonstrated with LCLT supplementation in this study and our prior work.

In summary, the anabolic hormones examined in this study showed amazing reproducibility over the course of the investigation. The LCLT supplementation had been previously shown to reduce the hypoxic stress and chemical damage related to such exercise and in addition had reduced soreness levels most likely caused by less tissue damage. IGFBP-3 concentrations were most dramatic in their response patterns to the LCLT supplementation with our volunteers acting as their own controls. The net result appears to be a greater preservation of IGF-I concentrations in recovery as well as the days leading up to the exercise stress. The time course for the increase in IGFBP-3 with LCLT supplementation after an exercise stress remains speculative and in need of further research. Each of the hormones would seem to benefit from reduced muscle tissue damage allowing greater numbers of intact and viable receptors for both direct (steroid) and indirect (peptide hormones) interactions with the DNA regulatory elements related to protein synthesis.

Practical Applications

LCLT supplementation was demonstrated to reduce the amount of muscle tissue damage most likely mediated though a reduction in hypoxia-related damage from free radicals resulting in the typical chemical damage days after the mechanical events associated with the resistance exercise protocol. Hormonal mechanisms appear to be enhanced because of the greater viability of receptors to successfully interact with the anabolic hormonal signals. The concentrations used in the current study have been shown to be safe, and these data provide additional support for positive effects of LCLT use as a recovery supplement for hypoxic exercise stress.

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