Effects of Amino Acids Supplement on Physiological Adaptations to Resistance Training

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ABSTRACT

KRAEMER, W. J., D. L. HATFIELD, J. S. VOLEK, M. S. FRAGALA, J. L. VINGREN, J. M. ANDERSON, B. A. SPIERING, G. A. THOMAS, J. Y. HO, E. E. QUANN, M. IZQUIERDO, K. HAKKINEN, and C. M. MARESH. Effects of Amino Acids Supplement on Physiological Adaptations to Resistance Training. Med. Sci. Sports Exerc., Vol. 41, No. 5, pp. 1111–1121, 2009. Introduction: Previous research has demonstrated that ingestion of essential amino acids and their metabolites induce anabolic effects with the potential to augment gains in lean body mass and strength after resistance exercise training. Purpose: The purpose of the present study was to examine the effects of an essential amino acid-based formula (Muscle ArmorTM (MA); Abbott Laboratories, Abbott Park, IL) containing β-hydroxy-β-methylbutyrate (HMB) on hormonal and muscle damage markers in response to 12 wk of resistance exercise. Methods: Seventeen healthy men (mean body mass: 77.9 ± 7.2 kg; mean height: 174.3 ± 12.4 cm; mean age: 22.9 ± 3.8 yr) were matched and randomized into two groups and performed 12 wk of periodized heavy resistance training while supplementing with either MA or an isocaloric, isonitrogenous placebo (CON). Every 2 wk during the 12-wk intervention, resting blood draws were obtained, and muscle strength and power were measured. In addition, blood draws were obtained before, during, and after a standardized resistance exercise challenge performed pre-, mid-, and posttraining. Results: Lean body mass, muscle strength, and muscle power significantly (P ≤ 0.05) increased in both groups after training; however, MA supplementation augmented these responses to a significantly greater extent when compared with the CON group. MA supplementation promoted increases in resting and exercise-induced testosterone and resting growth hormone concentrations. In addition, MA reduced preexercise cortisol concentrations. Throughout the training protocol, MA attenuated circulating creatine kinase and malondialdehyde compared with the CON group, suggesting that MA might have influenced a reduction in muscle damage. Conclusion: MA supplementation beneficially affected training-induced changes in lean body mass, muscle strength, and power, as well as hormonal responses and markers of muscle damage in response to 12 wk of resistance exercise training when compared with an isonitrogenous control. Key Words: ENDOCRINE, ERGOGENIC, BODY COMPOSITION, STRENGTH, HMB, AMINO ACIDS

Chronic resistance exercise promotes increases in muscle size and strength. Activation of muscle fibers to produce force leads to a subsequent cascade of events including hormone and immune responses, stimulation of muscle cell signaling pathways, activation of satellite cells, and, ultimately, increases in protein synthesis (13,16). In total, these responses promote muscle protein accretion and improve force production capabilities. Although resistance exercise in a fasted state increases protein synthesis, net protein balance remains negative unless nutrients are provided (32). In particular, it seems that provision of essential amino acids drives the net increase in muscle protein synthesis after resistance exercise (40).

Essential amino acids serve two critical functions for protein synthesis: 1) essential amino acids are substrates for protein synthesis because mRNA translation cannot progress unless the entire array of amino acids are immediately available, which highlights the importance of ingesting essential amino acids (i.e., those that cannot be synthesized de novo), and 2) essential amino acids are signals for protein synthesis. Several recent studies have highlighted the role of essential amino acids as independent signals for translation initiation (3,5). For instance, ingesting essential amino acids before and/or after resistance exercise...
potentiates the resistance exercise-induced activation of the 70-kDa ribosomal protein S6 kinase (p70S6K) (3), an important enzyme for increasing translational efficiency and protein synthesis after exercise (2). Further, it seems that leucine has a primary and non-insulin-dependent role in stimulating this pathway. Amino acid supplementation also reduces circulating markers of muscle damage (e.g., creatine kinase [CK] and lactate dehydrogenase) after exercise (9). Specific amino acids such as arginine have been shown to acutely increase circulating levels of growth hormone (GH) (7,30), whereas glutamine is purported to have beneficial effects on the immune system and glucose regulation during periods of intense training (1). Further, L-arginine in particular has been shown to positively influence muscular strength and power with training (6). The mechanisms by which L-arginine may affect athletic performance have yet to be elucidated; however, it might be via changes in growth hormone and testosterone, both of which impinge upon transcription and translation processes, triggering enhanced muscle protein synthesis (7). Therefore, it seems that amino acid supplementation can potentiate responses and adaptations to resistance exercise via several mechanisms (e.g., promoting protein synthesis, attenuating muscle damage).

Because of the critical role of amino acids, recent studies have investigated the efficacy of β-hydroxy-β-methylbutyrate (HMB) supplementation for promoting resistance exercise adaptations. β-hydroxy-β-methylbutyrate is a metabolite of the amino acid leucine, a potent stimulus of translation initiation and protein synthesis (3). In accord with the beneficial effects of amino acid supplementation, HMB supplementation decreases circulating markers of muscle damage (e.g., CK) after resistance exercise and may have anticytolytic properties (11,31). With regard to chronic adaptations, several studies of HMB supplementation have reported positive effects on muscle mass, body composition, and strength in men and women (28,29,31,38). These results may be due to HMB’s ability to attenuate protein degradation and stimulate protein synthesis through multiple mechanisms (10,39). Recent evidence published by Eley et al. (10) in 2007 showed that HMB increased phosphorylation activity of the mammalian inhibitor target of rapamycin (mTOR) pathway and mTOR initiation factors in the muscle of cachectic mice (10). In humans, HMB has been shown to interfere with the ubiquitin–proteosome proteolysis pathway in human cancer patients, thus preserving lean body mass (34).

The metabolic research supporting anabolic actions of amino acids and HMB led us to hypothesize that an amino acid supplement complemented with HMB would result in increased lean body mass and strength compared with ingestion of an isonitrogenous placebo. Here, we show for the first time that daily supplementation with Muscle Armor™ (Abbott Laboratories, Abbott Park, IL; an amino acid supplement containing HMB) enhanced lean body mass and performance gains from 12 wk of heavy resistance training. We further elucidate potential mechanisms by showing that a supplement containing HMB alters anabolic and catabolic hormonal responses as well as markers of membrane disruption and oxidative stress.

METHODS
Experimental Approach to the Problem

A randomized, double-blind, placebo-controlled design was used to determine the effects of adding Muscle Armor™ (Abbott Laboratories) to a resistance exercise training program. Subjects ingested Muscle Armor™ (MA) or an isonitrogenous control (CON) twice daily during a 12-wk resistance training protocol. Bench press strength, squat strength, lower-body power, body composition, resting hormonal concentrations, and markers of muscle damage were measured pretraining (V annotations) (V1), after the first six sets of 10 repetition maximums (RM) of an acute resistance exercise protocol (AREP) or V2, week 2 (V3), week 4 (V4), week 6 (V5), week 6 for the second AREP (V6), week 8 (V7), week 12 (V8), and the last AREP of the 12-wk intervention (V9). Again, subjects performed a standardized bout of acute resistance exercise at pre- (V2), mid- (V6), and postraining (V9) to determine resistance exercise-induced hormonal and muscle damage responses. All subjects were verified to be hydrated before all tests using a handheld refractometer to measure urine specific gravity.
Subjects

Seventeen healthy men who were recreationally active (Mean ± SD body mass: 77.9 ± 7.2 kg; mean height: 174.3 ± 12.4 cm; mean age: 22.9 ± 3.8 yr) volunteered to participate in the study. Study participation required that all subjects were not currently had not been resistance training within the previous 6 months. Each subject was screened by a physician to exclude those with orthopedic limitations or medical conditions that would prevent them from safely participating in the study. In addition, dietetic screening was performed by registered dieticians to ensure that subjects were 1) on a diet consisting of 15–20% protein, 45–55% carbohydrate, and 25–30% fat; 2) not taking creatine or HMB supplements; 3) not smoking; 4) not taking protein or amino acid supplements; 5) not taking anabolic or catabolic hormones; and 6) not taking medication or supplements known to influence any of the variables measured in the study. Subjects were carefully matched by age, body mass, vertical jump height, and resistance training and physical activity background, then randomly placed into either the MA (n = 8) or the CON (n = 9) group. After an explanation of the procedures and associated risks, all volunteers provided written informed consent. All procedures were approved by the University of Connecticut’s Institutional Review Board for use of human subjects.

Procedures

Diet assessment and counseling. Each subject was screened for dietary habits before inclusion into the study. Subjects completed 5-d diet records pretraining, mid-training, and during the last week of the training period. After the first 5-d diet record, but before the onset of the training period, participants received dietary counseling by a registered dietitian to ensure that the participants maintained their habitual dietary habits and ingested the supplement twice daily. The registered dietitian met with the subjects each week to review a 1-day diet record and assure adherence to all supplement and food instructions. Food diaries were analyzed for energy and macro/micronutrient content with NUTRITIONIST PRO™ (Version 4.2.0, Axxya Systems, Stafford, TX).

Supplementation. Volunteers either received an MA supplement (1.5 g of calcium HMB, 7 g of arginine, 7 g of glutamine, 3 g of taurine, and 5.824 g of dextrose) or an isocaloric, isonitrogenous control containing nonessential amino acids (10 g of glycine, 11.5 g of alanine, 1.5 g of glutamic acid, and 1.5 g of serine) as well as calcium citrate (200 mg of calcium). The treatments were matched in sensory attributes of flavor, sweetness, and acidity as well as delivering an equivalent amount of calcium (200 mg). Mass and calorie balance were achieved by decreasing dextrose in the placebo. Each volunteer received the supplements in plain, white blinded packets, given in two equal daily doses. Supplements were consumed twice per day (once with breakfast, once with dinner) throughout the 12-wk training period. To ensure compliance, participants completed supplement logs, met with the registered dietitian, handed in empty supplement packets, and received phone call “reminders” and “checks” at all workout sessions.

Resistance exercise training protocol. Subjects were familiarized with all testing and training procedures before the onset of the study to minimize the influence of learning effects on dependent measures. The resistance exercise training protocol consisted of a 12-wk (36 sessions) nonlinear periodized program. The resistance exercise program stressed all major muscle groups and included the following exercises (or variations of) in each session: bench presses, squats, lunges, shoulder presses, arm curls, stiff-leg dead lifts, lat pull down, seated rows, calf raises, and sit-ups. Exercise volume and intensity progressed during the training program according to previous recommendations (18,21). We used a planned nonlinear periodization resistance training program with different workouts during the week. Briefly, training days were split into “light,” “moderate,” and “heavy” days in a nonlinear periodized manner. Repetition maximum (RM) zones were used to progress intensity. “Light” days consisted of a 12- to 14-RM zone (three sets), “moderate” days consisted of an 8- to 10-RM zone (three sets), and “heavy” days consisted of a 3- to 5-RM zone (three sets but five sets for squat and bench press exercises). Weight was increased systematically if the prescribed amount of repetitions were completed. The planned training sequence is shown in Table 1. Subjects also performed supplemental endurance exercise (two to three sessions per week) in addition to their resistance training program. Endurance exercise was included in the program because these subjects were recreationally active and were encouraged to maintain their normal activities in addition to the resistance training they

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Training days were split into “light,” “moderate,” and “heavy” days in a nonlinear periodized manner. Repetition maximum (RM) zones were used to progress intensity: “light” days consisted of a 12- to 14-RM zone; “moderate” days consisted of an 8- to 10-RM zone, and “heavy” days consisted of a 3- to 5-RM zone. Weight was increased systematically if the prescribed amount of repetitions were completed. H, heavy; L, light; M, moderate.
Resting blood draws were obtained for 15 min at 4°C. Venous blood samples were obtained via venipuncture by a trained phlebotomist at pretraining (V1) and at week 2 (V3), week 4 (V4), week 6 (V5), week 8 (V7), and week 12 (V8) of the 12-wk intervention. Whole blood was collected and transferred into appropriate tubes for obtaining serum and plasma and centrifuged at 1500g for 15 min at 4°C. Resulting serum and plasma was aliquoted and stored at −80°C until subsequent analyses.

Strength testing. One repetition maximum (1-RM) strength was assessed in the free-weight bench press and free-weight squat exercises using previously described methods (20). Briefly, subjects performed a warm-up on a cycle ergometer followed by light dynamic stretching. Then, subjects performed 8–10 repetitions at ~50% of estimated 1-RM, followed by another set of 3–5 repetitions at ~85% of 1-RM. Three to four maximal trials separated by 2–3 min of rest were used to determine individual 1-RM for each resistance exercise. 1-RM testing was performed at pretraining (V1) and at week 2 (V3), week 4 (V4), week 6 (V5), week 8 (V7), and week 12 (V8) of the 12-wk intervention.

Power testing. Countermovement vertical jump power was assessed using a force plate and associate software (Accupower; Advanced Mechanical Technologies, Inc., Watertown, MA). After familiarization, subjects were asked to place their hands on their hips and jump as high as they could for three subsequent repetitions. The highest power for the set was recorded.

Acute resistance exercise protocol. Subjects performed a standardized acute resistance exercise protocol (AREP) in a 8 hr fasted state at pre- (V2), mid- (V6), and posttraining (V9) to determine exercise-induced hormonal and muscle damage responses in the blood. The AREP consisted of six sets of 10 maximal repetitions in the squat exercise with 2-min rest between sets. The initial load was 80% of individual 1-RM. If a subject was unable to complete all 10 repetitions during a given set, then spotters provided assistance until all 10 repetitions were completed; the resistance was then decreased for subsequent sets.

Before commencing the AREP, a trained phlebotomist inserted an indwelling Teflon cannula into a superficial forearm vein of the subject. The cannula was kept patent with a 10% heparin–saline solution. Venous blood samples were obtained at preexercise (pre), after the third set (mid), immediately postexercise (post), and at 5 min (+5), 15 min (+15), and 30 min (+30) postexercise. Before each blood draw, 3 mL of blood was drawn and discarded to avoid inadvertent dilution of the blood sample. Subsequently, whole blood was collected and processed as previously described.

Measures of body composition, body circumferences, and tendon size. Whole body composition was assessed by dual-energy x-ray absorptiometry (DXA) (Prodigy™; Lunar Corporation, Madison, WI) before (V1), at the midpoint (V5), and after (V8) the training period. Analyses were performed by the same blinded technician using commercial software (enCORE version 6.00.270, GE Lunar Corporation, Madison, WI). Coefficients of variation for lean body mass, fat mass, and bone mineral content on repeat scans with repositioning on a group of men/women were 0.4%, 1.4%, and 0.6%, respectively. Body circumferences were assessed at the neck, chest, upper arm, waist, thigh, and calf by the same tester. The cross-sectional area of the patella tendon was assessed using ultrasonography to determine connective tissue adaptations during the 12-wk resistance exercise protocol.

Biochemical Analyses

Samples were thawed one time and analyzed in duplicate for each analyte. All AREP days were scheduled at the same time of day to negate confounding influences of diurnal hormonal variations.

Plasma glucose and lactate concentrations were measured in duplicate using the STAT 2300 (Yellow Springs, Inc., Yellow Springs, OH). Serum total testosterone, GH, and insulin-like growth factor 1 (IGF-1), insulin, and cortisol were assayed via ELISA kits obtained from Diagnostic Systems Laboratories (Webster, TX). All hormones were measured in the same assay on the same day to avoid compounded interassay variance. Intra-assay variance was less than 3% for all analytes. Serum creatine kinase (CK) was measured using colorimetric procedures at 340 nm (Diagnostics Chemicals, Oxford, CT). Plasma malondialdehyde (MDA) was determined using colorimetric procedures at 532 nm on the basis of the formation of thiobarbituric acid reactive substances.

Statistical Analyses

A linear model with a two-way mixed factorial ANOVA (i.e., groups × time) was used with repeated measures for time. Linear assumptions were tested for, and sphericity correction was made if needed with the Huynh–Feldt correction because of higher variance in the hormones. Any variables that did not meet the requirements were logarithmically corrected and tested again. All pairwise comparisons were evaluated with Bonferroni corrections (or LSD-equivalent and to a no Type I error rate adjustment) for all of the comparisons. Using the nQuery Advisor® software (Statistical Solutions, Saugus, MA), the statistical
power for the n size used ranged from 0.76 to 0.87. The power is based on a variety of probability equations by Cohen (8) and represents the needed number of subjects to defend the 0.05 level of significance fourfold and allow detection of a 5% to 10% treatment effect. The test–retest reliability of the tests used in our laboratory have had intraclass R values ranging from 0.90 to 0.99, and this allows for the assessment of such treatment effects with the n size of 8–10 in a group. Significance was set at P ≤ 0.05.

RESULTS

Body mass, body composition, and circumferences. Body mass increased above baseline (V1) values at posttraining (V8) in both groups; however, the MA group had significantly greater gains than the CON group (Fig. 2A). Changes in lean body mass (Fig. 2B) closely reflected changes in total body mass, indicating that increased muscle mass occurred after the periodized resistance exercise training protocol. The MA group had greater increases in lean body mass (Fig. 1B) as well as lower percent body fat (Fig. 2C).

After 12 wk of resistance exercise training, both groups showed increased circumferences of the biceps, thigh, and chest; however, no change in waist circumference occurred (Fig. 3A). Subjects in the MA group had greater thigh and chest circumference than the CON group after training (at V8). Although there was a significant increase in patella tendon thickness in both groups, there was no difference between groups (Fig. 3B).

Exercise performance. The MA group made significantly greater percentage gains in 1-RM strength in the squat and bench press during the first 6 wk and posttraining compared with the CON group (Figs. 4, A and B). Maximal power (W) in the countermovement vertical jump also improved at a faster rate in the MA group than that in the CON group, yielding significantly higher percentage gains at mid- and posttraining time points (Fig. 4C).

Hormones. The resistance exercise training protocol significantly increased resting testosterone concentrations above baseline (V1) values at V3 through V8 in the MA group, but only at V4 and V7 in the CON group. The AREP increased testosterone concentrations above preexercise values at post, +5, and +15 in all three AREP trials. The resistance exercise-induced increases in testosterone were
greater in the MA group than that in the CON group at various postexercise time points during V6 and V9. Figure 5 displays testosterone values.

No change in resting cortisol concentrations occurred during the training protocol. However, MA supplementation reduced preexercise cortisol values (V2, PRE) compared with baseline values. Although the AREP increased circulating cortisol values, there were no differences between the MA and CON groups in resistance exercise-induced cortisol responses at any time point. Figure 6 displays the cortisol values.

Increased resting GH concentrations were apparent at V8 and V9 in the MA group only. Moreover, these values were significantly greater than corresponding CON values. Resistance exercise increased GH concentrations at all postexercise time points; however, there were no differences between groups. Figure 7 displays the GH values.

Resistance exercise training had no effect on resting IGF-1 values. The AREP increased IGF-1 concentration at mid- and postexercise in all trials. However, there were no differences between groups for any resting or postexercise time point. Figure 8 displays the IGF-1 values.

There was no effect of resistance exercise training on resting insulin concentrations. The AREP increased insulin at +5, +15, and +30 in V2 and V9; however, this was only significant during V9. There were no differences between groups for any resting or postexercise time point. Figure 9 displays the insulin values.

**Markers of muscle damage.** Differences between trials in circulating CK began to appear after the first AREP. At V3, CK was dramatically elevated in the CON group only, and this was significantly greater than values for the MA group. CK values for the CON group remained above baseline values at V4, V8, and at preexercise during

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**FIGURE 4—Squat 1-RM (panel A), bench 1-RM (panel B), and vertical jump power (panel C) values (mean ± SE) before training (V1), after week 6 of resistance exercise training (V5), and after 12 wk of resistance exercise training (V8). CON, control group; MA, Muscle Armor group. †Significant difference from corresponding V1 value, P ≤ 0.05; ‡significant difference from corresponding control value, P ≤ 0.05.**

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**FIGURE 5—Testosterone responses (mean ± SE) to the 12-wk resistance exercise intervention. Bars represent resting values; lines represent responses to the AREP. *Significant difference from corresponding preexercise value, P ≤ 0.05; †significant difference from corresponding V1 value, P ≤ 0.05; ‡significant difference from corresponding control value, P ≤ 0.05.**

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**FIGURE 5—Testosterone responses (mean ± SE) to the 12-wk resistance exercise intervention. Bars represent resting values; lines represent responses to the AREP. *Significant difference from corresponding preexercise value, P ≤ 0.05; †significant difference from corresponding V1 value, P ≤ 0.05; ‡significant difference from corresponding control value, P ≤ 0.05.**
V9. Alternately, CK values for the MA group were below baseline values at V5 and at preexercise during V6. During the third AREP, CK values for the MA group were significantly lower than the CON group at all time points. Figure 10 displays the CK values.

MDA values increased during the first AREP, yet returned to baseline and remained stable throughout V6. Beginning at V7, MDA values for the MA group were less than baseline values and corresponding values for the CON group. Figure 11 displays the MDA values.

**DISCUSSION**

The prominent findings of the present investigation were that 1) Muscle ArmorTM supplementation potentiated gains in lean body mass and muscle strength after 12 wk of
periodized resistance training; and 2) these adaptations were potentially mediated, in part, by accentuated hormonal responses and/or attenuated muscle damage. Overall, these results indicate that supplementation with a blend of HMB and conditionally essential amino acids (arginine, glutamine) and taurine promote adaptations to resistance exercise training.

Resistance exercise training increased total and lean body mass and decreased percent body fat; these improvements in body composition were potentiated by MA supplementation. This may be explained by a greater availability of extracellular amino acids in the MA group, which would promote protein synthesis (4,5). Essential amino acids, particularly leucine, stimulate muscle anabolism by activating the mammalian target of rapamycin (mTOR) and subsequently p70^S6K^ (3,16,27), and this has been shown in model systems for HMB as well (10,39). Alternately,
MA might have improved body composition because of the anticasabatic properties of its ingredients: exercise-induced proteolysis is reduced by HMB (28,34), arginine (26), and glutamine (1,9). Gains in lean body mass were concomitant with muscle strength improvements. Bench press and squat 1-RM were increased in both groups after 12 wk of training. However, the increases in 1-RM bench press and squat were significantly greater ($P \leq 0.05$) in the MA group when compared with the CON group. Our findings agree with several previous studies that demonstrated HMB supplementation, in conjunction with resistance exercise, improved muscle mass, body composition % fat and strength, and vertical jump power (28,29,31,38). Little is known concerning the adaptations of tendon in response to heavy resistance training. The findings of this study indicate that the patella tendon thickness does increase after heavy, periodized resistance training, although there were no between-group differences. Currently, only one other study

![Graph showing creatine kinase responses](image)

**FIGURE 10**—CK responses (mean ± SE) to the 12-wk resistance exercise intervention. **Bars** represent resting values; **lines** represent responses to the AREP. *Significant difference from corresponding preexercise value, $P \leq 0.05$; †significant difference from corresponding V1 value, $P \leq 0.05$; #significant difference from corresponding control value, $P \leq 0.05$.

![Graph showing malondialdehyde responses](image)

**FIGURE 11**—Malondialdehyde responses (mean ± SE) to the 12-wk resistance exercise intervention. **Bars** represent resting values; **lines** represent responses to the AREP. *Significant difference from corresponding preexercise value, $P \leq 0.05$; †significant difference from corresponding V1 value, $P \leq 0.05$; #significant difference from corresponding control value, $P \leq 0.05$. 

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has directly reported patella tendon hypertrophy after heavy resistance exercise (17).

Acute resistance exercise transiently increases circulating concentrations of testosterone, cortisol, into hormone sequence and GH (14), and nutritional consumption modulates these responses (23). The influence of chronic resistance exercise training on resting and exercise-induced responses is less clear. In the present study, resting testosterone was elevated after training commencement in both groups. However, although the CON group had increased resting testosterone only at V4 and V7, the MA group showed increased resting testosterone at all time points after V3. MA supplementation also potentiated resistance exercise-induced testosterone responses during the AREP at mid- and posttraining when compared with the CON group. The importance of circulating testosterone for promoting resistance exercise-induced adaptations as a primary anabolic stimulus is clear. When endogenous testosterone release is blocked (via a gonadotropin-releasing hormone analog), strength and lean mass adaptations to training are severely attenuated (24). Therefore, potentiation of circulating testosterone might be an important mechanism by which MA supplementation promoted resistance exercise adaptations.

Resting and exercise-induced cortisol concentrations remained stable throughout the 12-wk intervention. Interestingly, though, preexercise cortisol concentrations were reduced in the MA group immediately before the AREP at mid- and posttraining. The mechanism for this response is not entirely clear. Cortisol displays an anticipatory response to impending intense exercise and competition (19,35), and our laboratory previously demonstrated that this response was attenuated using a specifically designed herbal supplement (19). Because supplementation with amino acids increases the circulating amino acid concentrations (26), possibly less cortisol was required to degrade proteins into amino acids for gluconeogenesis before exercise. MA supplementation also increased resting GH concentrations at V8 and V9 above baseline and corresponding CON values. Most studies have shown no change in resting GH concentrations with chronic training (15,22). However, the present study is the first to use an HMB, arginine, glutamine, and taurine supplement in conjunction with resistance training. Reduced cortisol and increased GH at rest improves the anabolic-to-catabolic hormone ratio, which, in theory, would improve muscle tissue protein balance.

Resting CK values peaked at V2 and decreased with progression of training; this was consistent with previous studies showing that long-term resistance training reduced CK concentrations after exercise (12,36). A possible mechanism is that repeated bouts of resistance exercise impart a protective effect on skeletal muscle, and therefore, skeletal muscle is less susceptible to damage (25). Resting CK values were significantly lower (P ≤ 0.05) in the MA group than that in CON group during training (V2 to V9). Previous studies have shown that reduced protein degradation is associated with a lower CK value (28,31) and that supplementation with HMB (28) or branched chain amino acids (9) can partially attenuate exercise-induced proteolysis and/or muscle damage. Therefore, the findings of the present study suggest that HMB supplementation attenuated muscle damage during training and reduced protein degradation.

Consistent with prior research (37), the AREP increased plasma MDA concentrations at pretraining. Yet, this exercise challenge did not promote significant changes in plasma MDA for either group at mid- and posttraining. At posttraining, the MA group had significantly lower (P ≤ 0.05) plasma MDA concentrations at preexercise and at 5 and 15 min into the recovery as compared with pretraining and CON values. Because plasma MDA is a marker of free radical formation and lipid peroxidation (27), which are partly responsible for membrane disruption associated with exercise, we conclude that MA was effective in attenuating muscle tissue disruption via reduced free radical formation at rest and in response to the AREP. Prior research has similarly shown that supplementation with l-carnitine l-tartrate was effective in attenuating the plasma MDA response to exercise (37).

In conclusion, 12 wk of periodized resistance training increased lean body mass and muscle strength. In accord with previous research, we demonstrated that concomitant supplementation with HMB and conditionally essential amino acids and taurine (the ingredients of Muscle Armor™) potentiated adaptations to resistance exercise training. Muscle Armor™ supplementation also improved anabolic hormonal responses, reduced preexercise catabolic hormonal concentrations, and attenuated markers of muscle damage. These results provide compelling evidence that long-term supplementation with Muscle Armor™ promotes physical and physiological adaptations to resistance exercise.

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