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Effects of Vitamin E Supplementation on Recovery From Repeated Bouts of Resistance Exercise

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ABSTRACT

The purpose of this study was to examine the effects of vitamin E (VE) supplementation (1200 IU/day) on recovery responses to repeated bouts of resistance exercise. Non–resistance trained men were assigned to supplement with VE ($n = 9$) or placebo (PL; $n = 9$) for 3 weeks and then perform 3 resistance exercise sessions separated by 3 days of recovery (EX-1, EX-2, and EX-3). Performance was assessed at EX-1, EX-2, and EX-3. Fasting morning blood samples and perceived muscle soreness were obtained before EX-1 and for 10 consecutive days. Muscle soreness peaked after EX-1 and gradually returned to baseline values by day 6. Lower and upper body maximal strength and explosive power were significantly ($p \leq 0.05$) decreased at EX-2 and EX-3 (~10%). Plasma malondialdehyde (MDA) was significantly elevated on days 7 and 8. There were no significant differences between VE and PL in muscle soreness, performance measures, or plasma MDA. Creatine kinase (CK) area under the curve from day 1 to day 10 was significantly greater for VE because of a nearly 2-fold greater increase in CK after EX-1 in VE, compared with PL (404 ± 146 and 214 ± 179 U/L, respectively). VE supplementation was not effective at attenuating putative markers of membrane damage, oxidative stress, and performance decrements after repeated bouts of whole-body concentric/eccentric resistance exercise.

Key Words: lipid peroxidation, malondialdehyde, creatine kinase, delayed-onset muscle soreness, free radicals, antioxidant, muscle damage, ergogenic aid

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Introduction

Strenuous physical exercise has been shown to increase free radical production in skeletal muscle, as assessed by increased levels of malondialdehyde (MDA) (8, 10, 22, 27). Peroxidation of muscle fiber lipids, as well as lipid membranes of other tissue cells, leads to cellular disruption. This disturbance in cellular homeostasis can result in muscle fatigue or injury, possibly implicating free radical formation as a major cause of delayed-onset muscle soreness (DOMS) and the associated decrements in physical performance (4, 24, 30). The increase in generation of reactive oxygen species with intense physical exercise can exceed the capacity of the antioxidant defense systems in the body (13–15). Therefore, the ingestion of antioxidant vitamins has been proposed to attenuate this increase in reactive oxygen species. Vitamin E is the main lipid-soluble, chain-breaking antioxidant (13). Vitamin E accumulates in the phospholipid bilayer of cell membranes and limits lipid peroxidation within the membrane (35).

Vitamin E supplementation has been shown to significantly decrease the amount of lipid peroxidation and membrane damage associated with single bouts of low- and high-intensity submaximal exercise (18) and resistance exercise (22, 23). The impact of reducing the amount of lipid peroxidation and membrane damage after resistance exercise on muscular performance during subsequent training sessions has not been examined. Therefore, the purpose of this study was to examine the influence of vitamin E supplementation on lipid peroxidation (MDA), markers of membrane damage (CK), DOMS, and muscular performance after repeated days of whole-body resistance exercise.

We hypothesized that vitamin E supplementation would lead to tissue resistance from the damaging effects of lipid peroxidation and shear stress on the muscle membrane resulting from resistance exercise. This was predicted to attenuate delayed onset muscle soreness and CK release, speed recovery, and enhance per-

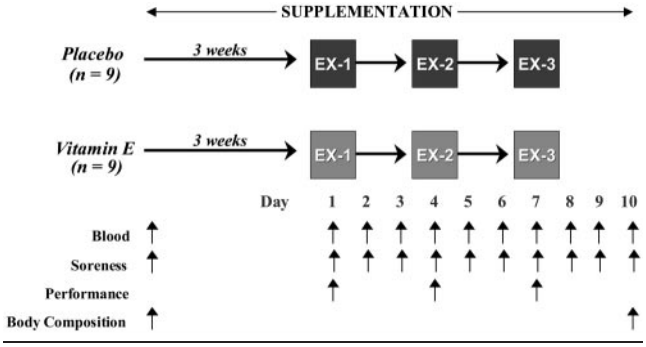

Figure 1. Experimental design. Subjects supplemented with either vitamin E or placebo for 3 weeks prior to performing three whole-body resistance exercise workouts on days 1 (EX-1), 4 (EX-2), and 7 (EX-3). Muscle soreness and fasting blood samples were obtained daily from days 1 to 10. Body composition was assessed before and after supplementation.

formance during subsequent resistance exercise sessions.

Methods

Experimental Approach to the Problem

A two-group, double-blind, placebo-controlled design with repeated measures was used in this study (Figure 1). Subjects were matched according to physical characteristics and training history and then randomly assigned to receive either vitamin E or placebo for a period of 31 days. After 21 days of supplementation, subjects performed 3 whole-body resistance exercise sessions (EX-1, EX-2, and EX-3) with 3 days of recovery between sessions. Fasting blood samples and perceived ratings of muscle soreness were obtained for 10 consecutive days in the morning after the initial 21 days of supplementation. Assessment of maximal strength, explosive power, and muscular endurance were determined prior to the exercise sessions on days 1, 4, and 7. Total and regional body composition was assessed before and after 31 days of supplementation using dual-energy X-ray absorptiometry.

Subjects

Eighteen healthy men volunteered to participate in this investigation. After randomly assigning subjects to a supplement group, there were no significant differences in any physical characteristics between the vitamin E ($n = 9$; age 22.7 ± 4.1 years, height 183.3 ± 4.4 cm, body mass 81.9 ± 7.8 kg, body fat 17.8 ± 4.2%) and placebo ($n = 9$; age 22.3 ± 3.6 years, height 178.9 ± 5.8 cm, body mass 81.3 ± 5.6 kg, body fat 19.0 ± 8.5) groups. Subjects were normally active participating in endurance and sports activities but not resistance training to ensure the whole-body resistance exercise protocol resulted in detectable muscle soreness and muscle disruption. The subjects had not lost or gained weight in the previous year, were not adhering to special diets, and were not regular consumers of nutritional supplements including vitamin E. All subjects were nonsmokers, and not currently taking any medication known to affect any of the dependent variables in this study. All subjects were informed of the purpose and possible risks of this investigation prior to signing an informed consent document approved by the Institutional Review Board.

Supplementation Protocol

Subjects received instructions to consume either vitamin E capsules [992 mg·day^{-1} or 1,200 IU (800 tocopherol equivalents)] in the form of RRR-d-alpha-tocopherol succinate (Twin Laboratories, Inc., Ronkonkoma, NY) or placebo capsules (microcrystalline cellulose). The entire dose was consumed with a meal, and supplementation continued for the entire experimental period lasting 31 days. Subjects recorded the time of day each dose was consumed on log sheets for the first 21 days. For the remainder of the experimental period, we observed consumption of the supplement during the morning visits to the laboratory for blood sampling. Compliance to the supplement protocol was 100% according to supplement log sheets that were signed and returned by each subject at the end of the study.

Body Composition

To determine whether changes in body composition occurred over the month period of supplementation, whole-body composition was assessed using dual-energy X-ray absorptiometry (DXA) with a total body scanner (Prodigy Lunar Corp., Madison, WI). Percent body fat from the DXA testing was calculated as fat tissue mass divided by the total soft tissue mass plus the estimated bone mineral content. All analyses were performed by the same technician using computer algorithms (software version 2.17.008). Coefficients of variation for lean body mass and fat mass on repeat scans with repositioning on a group of men and women in our laboratory were 0.4% and 1.4%, respectively.

Resistance Exercise Protocol

All resistance exercise sessions and performance testing were performed on a Ballistic measuring system (BMS; Norsearch Limited, Lismore, Australia). The BMS allows traditional barbell weight training movements such as bench press and squat to be done in a dynamic, ballistic manner (39). The machine allows only vertical movement of the bar, and metal stops limit the lower travel of the bar with an accuracy of 0.01 m. Linear bearings attached to either end of the bar allow it to slide up and down two steel shafts with minimal friction. The system was calibrated prior to all testing by counting the total number of pulses produced as the bar moved through its full vertical range of 2.8 m. The BMS was interfaced to a computer that

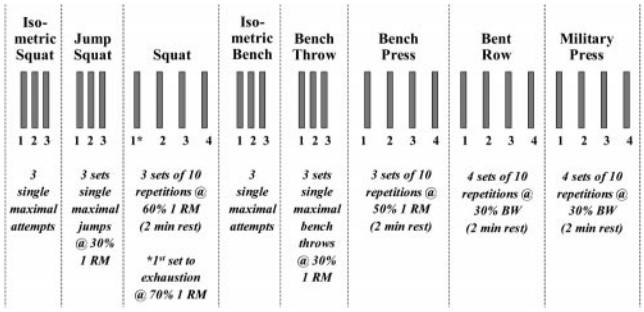

Figure 2. Whole-body resistance exercise protocol. Subjects performed 4 sets of 10 repetitions of squat, bench press, bent row, and shoulder press with 2 minutes of rest between sets and exercises. In addition, maximal strength was assessed in an isometric squat and bench press, explosive power was assessed in a maximal jump squat and bench throw, and muscular endurance was assessed by performing repetitions to exhaustion during the first set of squat. 1 RM = one repetition maximum, BW = body weight.

provided detailed information concerning the kinematics and kinetics of the performance.

The workout was designed to stimulate a large amount of muscle tissue previously shown by our laboratory group to result in elevated CK (19). Performance measures were incorporated into the protocol to prevent additional muscle disruption or damage during the recovery period between workouts, which would confound interpretation of the data. The first whole-body resistance protocol was performed after 21 days of supplementation. In sequence, the protocol involved maximal isometric squats, explosive jump squats, 4 sets of squat, maximal isometric bench press, explosive bench throws, 4 sets of bench press, 4 sets of bent row, and 4 sets of shoulder press (Figure 2). There was a 2-minute recovery between sets and exercises.

Performance Testing

Subjects were familiarized with all exercise protocols prior to testing. Test-retest reliability has shown intraclass correlations greater than $r = 0.98$ for the testing protocols. Performance criteria included assessment of maximal strength, explosive power, and muscular endurance. Isometric maximal strength of the lower and upper body was determined using an isometric squat and bench press. Warm-up trials at 30–70% of maximal voluntary contraction were performed prior to completing each test. A knee angle of 90° was used for the isometric squat, and the bar was positioned 5 cm away from the chest for the isometric bench press. Adequate rest was allowed between trials (3–5 minutes). The peak force was converted to kilograms, and then the subject' mass (kilograms) was subtracted to obtain their isometric 1 repetition maximum (1RM). This was done to obtain a reliable estimate of their dynamic 1RM without exposing the subject to further muscle disruption outside of the testing protocol.

Explosive power of the lower and upper body was determined by having subjects perform jump squats and bench throws with a load equal to 30% of their isometric 1RM. It has been shown that the maximal mechanical power output occurs using loads within 30–60% of a subject's 1RM (17, 25). The jump squat involved descending with the bar to a point at which the knee angle was approximately 70° (marked by an audible cue and adjustable stoppers) and explosively jumping upward as fast as possible with feet leaving the floor. The bench throw involved the subject lowering the bar to their chest and then explosively throwing the bar upward while releasing their grip. Two minutes of rest were allowed between the squat jumps and bench throws. Vertical ground reaction forces were recorded using a uniaxial force plate (Kistler Quattro Jump, model 9290AD, Winterthur, Switzerland) positioned within the BMS. A position transducer (Celesco, model PT 9510, Canoga Park, CA) was attached to the bar to measure bar displacement. Standard biomechanical analyses were performed using BMS software (BMS, Innervations, Muncie, IN).

To assess muscular endurance, subjects performed a squat endurance test using a load equal to 70% of their isometric squat 1RM. The subject lowered the bar to a knee angle of 90° and immediately moved the weight upward in a controlled manner to the starting position. No pause was allowed between repetitions and the subjects completed the maximum number of repetitions possible. The test was terminated when the subject could no longer perform the squat to the required depth or cadence.

Perceived Muscle Soreness

Perceived muscle soreness of the shoulders, chest, quadriceps, and hamstrings was evaluated using visual analog scales. Subjects marked their subjective rating of muscle soreness on a 10-cm line that corresponded from ''no pain'' to ''extreme pain'' after performing shoulder abduction, shoulder horizontal adduction, and hip flexion (unloaded squat). The visual analogue method has been established as a reliable method for assessing soreness (31).

Blood Collection and Analyses

Blood samples were collected prior to supplementation and each day beginning with the first exercise session. All blood samples were performed after a 10-hour overnight fast and abstinence from alcohol for 24 hours. Subjects reported to the laboratory between 0700 and 0900 hours, rested quietly for 10 minutes in the supine position, and blood was obtained from an antecubital vein using a 20-gauge needle and vacutainer. Whole blood was allowed to clot (serum tubes only) and then centrifuged at 1,200*g* for 15 minutes at

10° C and the resultant serum or plasma divided into aliquots and immediately stored frozen at −80° C. Samples were thawed one time at a later date for determination of CK activity and thiobarbituric acid reactive substances expressed as MDA. Serum CK (Sigma Diagnostics, St. Louis, MO) was determined at 340 nm on a spectrophotometer (Spectronic 601, Milton Roy Co., Rochester, NY). Intra-assay variance was 6.4%. Plasma MDA was determined using the methods described by Wong et al. (40) and modified as described by McBride et al. (23). A phosphoric acid solution (0.44 mol·L^{-1}) and a thiobarbituric acid solution (42.0 mol·L^{-1}) were added to plasma samples and placed in a water bath (0° C) until analysis. A methanol-NaOH solution was added to the boiled samples prior to being centrifuged to precipitate the plasma proteins. The protein-free plasma was extracted and the absorbance read at 532 nm on a spectrophotometer (Spectronic 601, Milton Roy Co.). Intra-assay variance was 11.5%.

Statistical Analyses

Dependent variables were analyzed using a two-way analysis of variance with supplement group (vitamin E versus placebo) and time as main effects. Creatine kinase and MDA values were not normally distributed and were logarithmically transformed before statistical analysis. Significant main effects or interactions were further analyzed using a Fisher's least significant differences post hoc test. The total area under curve (AUC) over the 10-day protocol was calculated for CK and MDA values using the trapezoidal method. The alpha level for significance was set at 0.05.

Results

Body Composition and Muscle Soreness

Body mass did not significantly change in the vitamin E (81.9 ± 7.8 to 82.8 ± 7.5) and placebo (81.3 ± 5.6 to 81.7 ± 6.1 kg) group. Likewise, body fat did not significantly change in the vitamin E (17.8 ± 4.2 to 17.4 ± 5.6%) and placebo (19.0 ± 8.5 to 18.9 ± 9.4%) group. There were significant main time effects for perceived muscle soreness in all regions. Muscle soreness of the upper body and lower body peaked the day after the first bout of resistance exercise (day 2) and remained significantly elevated above baseline until day 5 (Figures 3 and 4). Soreness was greater in the lower than the upper body. There were no significant interaction effects indicating a similar response in vitamin E and placebo groups.

Performance Measures

There were significant main time effects for maximal isometric strength of the squat and bench press (Figures 5 and 6). Maximal squat strength was significantly decreased at EX-2 (−11%) and remained decreased at EX-3 (−8%). Likewise, maximal bench press strength was significantly decreased at EX-2 (−13%) and remained lower at EX-3 (−11%). There were significant main time effects for explosive jump squat and bench throw power. Explosive jump squat power was significantly decreased at EX-2 (−8%) and remained lower at EX-3 (−6%). Explosive bench throw power was decreased at EX-2 (−7%), which reached statistical significance at EX-3 (−11%). There were no significant effects of time for repetitions to exhaustion during the squat endurance test (EX-1 to EX-2 to EX-3) for the placebo (12.3 ± 4.4, 11.4 ± 9.3, and 12.1 ± 4.5, respectively) or vitamin E (12.4 ± 4.4, 10.3 ± 6.3, and 15.2 ± 7.9, respectively) group. There were no significant interaction effects for any performance measure indicating a similar response in vitamin E and placebo groups.

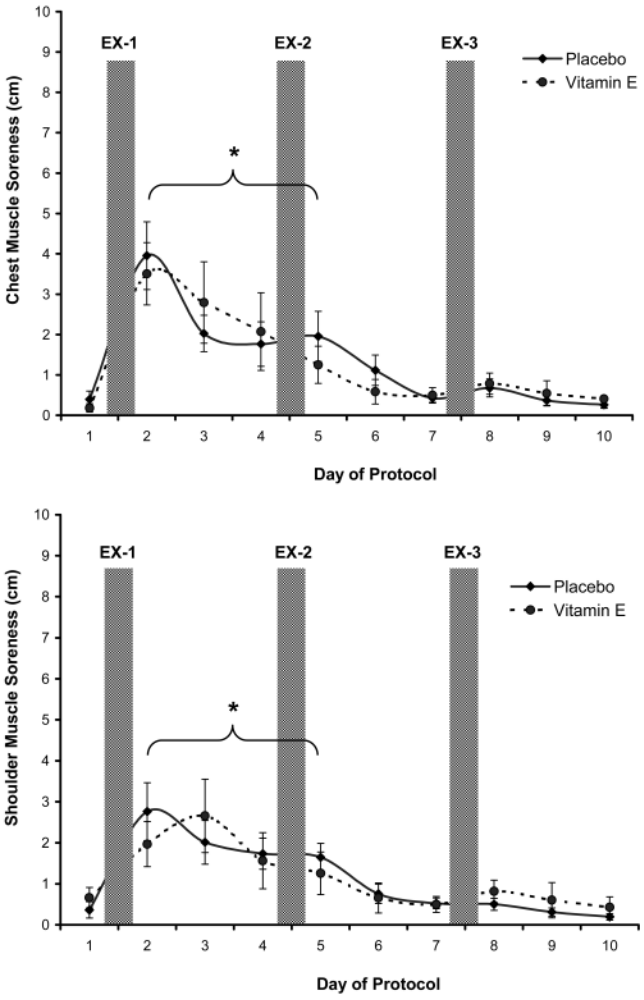

Figure 3. Perceived muscle soreness of the chest (upper graph) and shoulders (lower graph) after performing voluntary shoulder abduction and shoulder horizontal adduction, respectively. Subjects marked their subjective rating of muscle soreness on a 10 cm line. 0 cm = no pain; and 10 cm = extreme pain. Values are mean ± *SE*. * Significant ($p \leq 0.05$) main time effect vs. day 1 value.

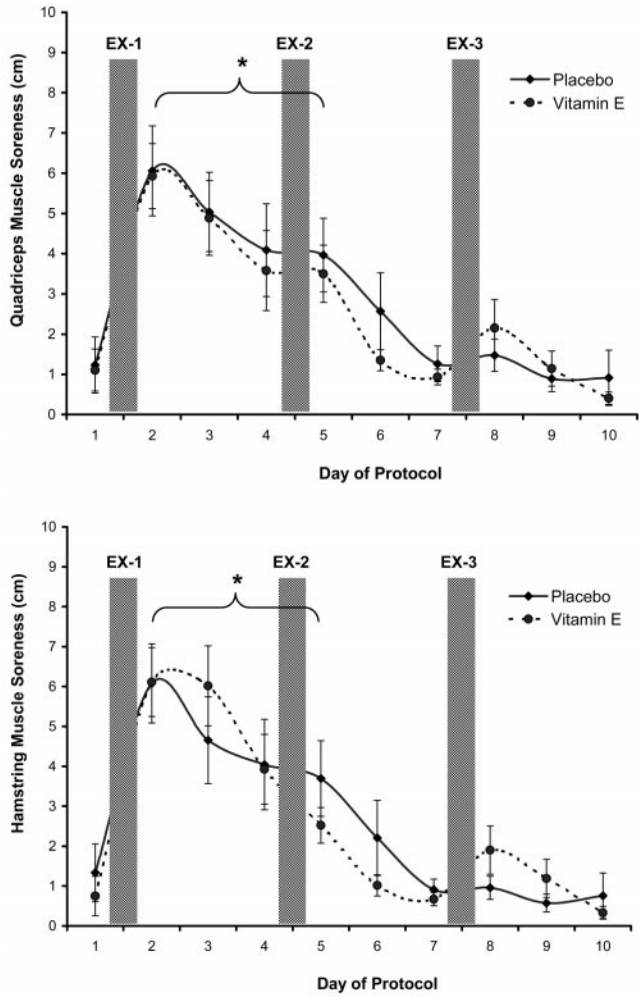

Figure 4. Perceived muscle soreness of the quadriceps (upper graph) and hamstrings (lower graph) after performing an unloaded squat movement. Subjects marked their subjective rating of muscle soreness on a 10 cm line. 0 cm = no pain; and 10 cm = extreme pain. Values are mean ± *SE*. * Significant ($p \leq 0.05$) main time effect vs. day 1 value.

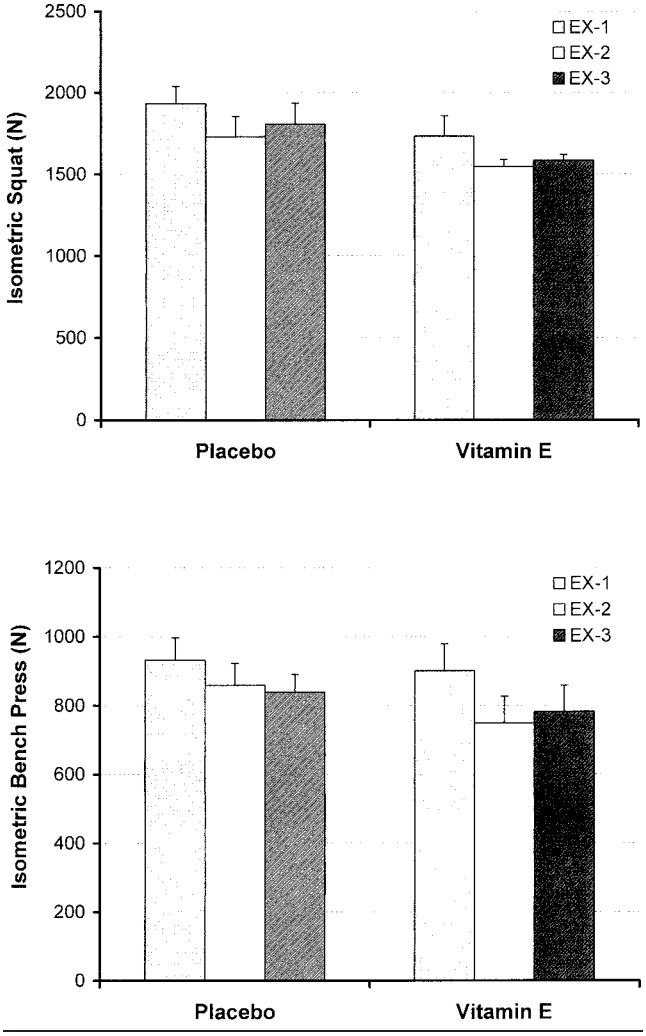

Figure 5. Maximal isometric squat (upper graph) and bench press (lower graph) performance at three exercise sessions (EX-1, EX-2, and EX-3) with 3 days of recovery between sessions. Values are mean ± *SE*. Significant main time effect ($p \leq 0.05$) EX-1 > EX-2 and EX-3.

Creatine Kinase and Malondialdehyde Responses

There were significant main group and time effects for CK but not an interaction effect (Figure 7). Plasma CK generally peaked on day 2 and gradually declined thereafter. However, creatine kinase remained significantly above baseline at day 10. The AUC for CK was significantly higher in the vitamin E (2088 ± 627 U/L × 10 days), compared with the placebo (1292 ± 878 U/L × 10 days) group. There was a significant main time effect for MDA (Figure 7). Plasma MDA was significantly higher on days 7 and 8. There were no significant differences in the AUC for MDA between groups.

Discussion

We had hypothesized that vitamin E supplementation would increase the structural integrity of muscle cell and thereby increase resistance to peroxidation and the mechanical shear forces encountered during high force resistance exercise. This in turn would result in improved maintenance of force generating capacity of muscle. Our results indicate that vitamin E supplementation does not affect muscle soreness, circulating levels of MDA, and measures of maximal strength and explosive power after a bout of whole-body concentric and eccentric resistance exercise. Furthermore, vitamin E did not affect these measures when the same bout of resistance exercise was performed 3 and 6 days later. In fact, we observed a significant increase in the CK response to the first bout of exercise in the vitamin E group.

Resistance exercise results in ultrastructural damage to certain muscle cell membranes (34), resulting in the release of intracellular contents such as CK (23). Although the CK response to the first exercise bout

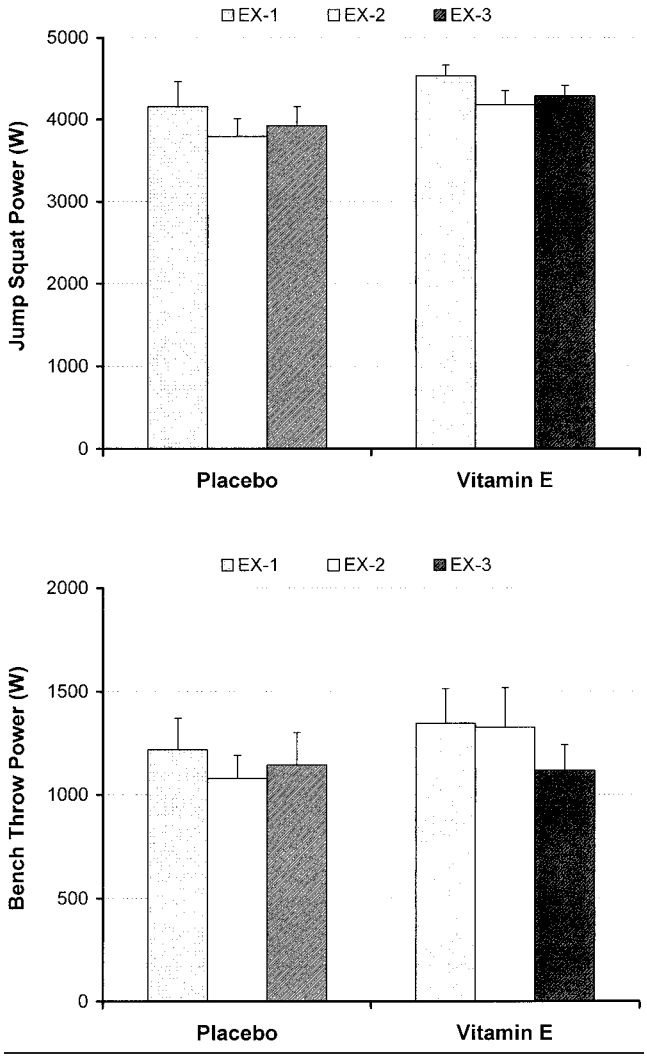

Figure 6. Maximal explosive jump squat (upper graph) and bench throw (lower graph) power performance at three exercise sessions (EX-1, EX-2, and EX-3) with 3 days of recovery between sessions. Values are mean ± *SE*. Significant main time effect ($p \leq 0.05$) EX-1 and EX-2 > EX-3.

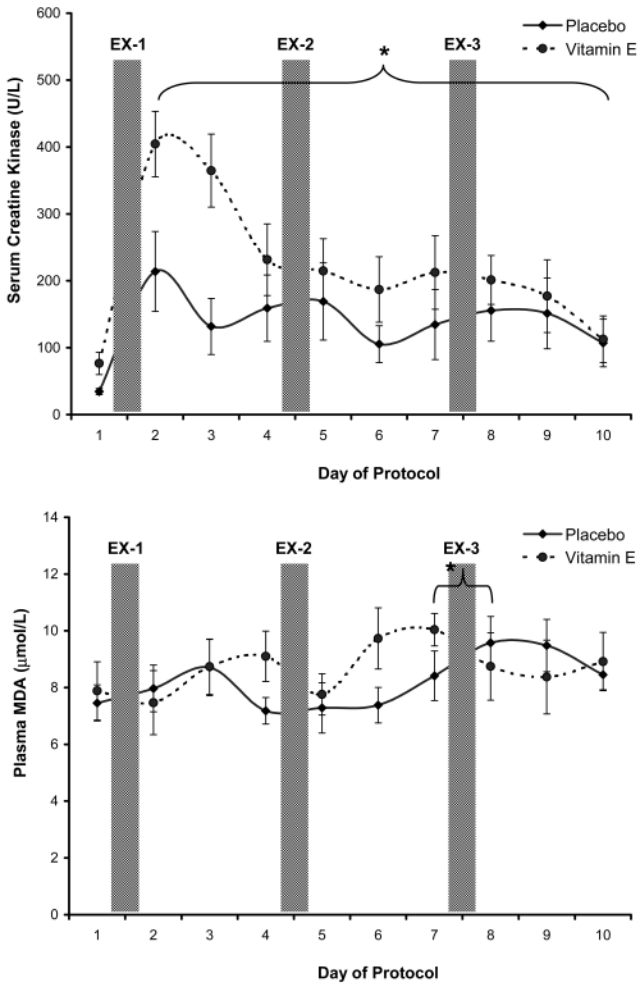

Figure 7. Serum creatine kinase activity (upper graph) and plasma malondialdehyde (MDA) (lower graph) responses to three exercise sessions (EX-1, EX-2, and EX-3) with 3 days of recovery between sessions. All blood samples are resting and obtained in the morning. Values are mean ± *SE*. * Significant ($p \leq 0.05$) main time effect vs. day 1 value. Creatine kinase area under the curve was significantly greater in the vitamin E group ($p \leq 0.05$; independent *t*-test).

was greater with vitamin E supplementation, recovery was rapid, and not different from placebo by the start of the second exercise bout. Thus, the transient increase in CK is probably not of practical significance. In prior studies, the effect of vitamin E supplementation on CK responses to exercise have been inconsistent with reductions (2, 11, 23, 32, 33), no effect (9, 12, 16, 29, 38), or even increases (5) being reported. We have no mechanistic explanation for the higher initial CK response with vitamin E supplementation other than vitamin E may have enhanced membrane fluidity and thereby promoted enzyme release. We postulate that the small sample size and high degree of variability among individual subjects, which has been reported in prior studies (2), or variability in starting levels of membrane vitamin E content (which was not measured) accounted for this unexpected finding. The CK values in this study were of similar magnitude to those in prior studies in our laboratory that used whole body concentric/eccentric resistance exercise (19, 23). Although the relative pattern of CK response over 10 days was somewhat similar to prior studies that used a similar design but an eccentric only exercise model (6, 26), the peak absolute values were approximately 10-fold lower (400+ vs. 4000+ $U \cdot L^{-1}$) indicating much less structural damage to the cell membrane.

We measured plasma MDA as a putative marker of free radical interaction with cell membranes, which has previously been shown to increase 24 hours after resistance exercise in a recent publication from our laboratory group (23). The lack of a clear increase in MDA after a single bout of resistance exercise in this study was unexpected. Because we did not measure time

points immediately after exercise, it is possible that transient increases occurred acutely after exercise, which was the case in a recent study by our laboratory examining high volume squats (37). Resistance exercise did result in a small elevation in resting morning MDA levels several days after the first bout of exercise but vitamin E had no effect on this small increase on days 7 and 8. Several mechanisms could contribute to production of reactive oxygen species evident after resistance exercise. Transient hypoxia, ischemic reperfusion, calcium overload, and/or mechanical shear forces evident during high-force resistance exercise could lead to inhibition of the respiratory chain and production of reactive oxygen species that result in loss of mitochondria membrane integrity (15, 22). Vitamin E supplementation has been shown to reduce lipid peroxidation after moderate to heavy exercise (18) but had no effect after marathon running (16). Thus, the effects of vitamin E supplementation may depend on the type of exercise and oxygen demands.

Perceived muscle soreness was highest the day after EX-1 and returned to values near baseline by EX-2. Despite this, strength and power decrements persisted. This loss in maximal force generation can be attributed to cellular disruption and ultrastructural damage of the muscle fiber contractile and cytoskeletal components (3). Maximal isometric strength decreased ~11–13% after 3 days and ~8–11% after 6 days. Decrements in explosive power followed a similar pattern as maximal isometric strength. We are not aware of any other studies that have examined maximal strength and power 3 days after a bout of whole-body resistance exercise for comparison purposes. Our results suggest that 3 days does not provide adequate time for complete recovery of maximal force-generating potential in non–resistance-trained men. The ~10% decrement in strength is moderate but nonetheless does indicate sustained impairment in the maximal force generating capacity of muscle during a third bout of exercise performed 6 days later. The force decrements could be attributed to ultrastructural disruption of the contractile apparatus (e.g., Z-line streaming, membrane damage), malfunction in excitation-contraction coupling, or neuromuscular deficits (3).

Several studies have shown that strenuous exercise performed during the early phases of recovery of damaged muscle does not exacerbate damage or hinder the repair process (6, 26). Our results concur with this hypothesis as the second and third bouts of exercise did not accentuate CK and MDA responses or result in further decrements in force and power output (except explosive upper body power). Two other studies (7, 26), both involving eccentric exercise of the elbow flexors, examined the effects of repeated exercise bouts on recovery responses. Similar to this study, an initial exercise bout (EX-1) was followed by an identical bout 3 days (EX-2) and 6 days (EX-3) later. Both studies reported that muscle soreness peaked after EX-1 and gradually declined thereafter. Muscle strength decreased the most at EX-2 (−25% to −35%) and started to recover at EX-3 (−15% to −25%). Plasma CK responses peaked at EX-2 with no further increase at EX-3. In agreement with findings from other studies (6, 28), these data indicate that performance of eccentric muscle actions several days after a damaging bout of eccentric exercise does not alter the normal recovery process (e.g., muscle soreness, membrane damage, oxidative stress, immune responses, or performance). The whole-body concentric and eccentric resistance exercise protocol in the present study resulted in much less damage and decrements in maximal strength, but the pattern in recovery is similar.

There has been little evidence to date that indicates an improvement in exercise performance in humans following vitamin E supplementation. Swimmers supplemented with vitamin E (900 IU daily for 6 months) exhibited no difference in 500-m swimming speed (20). There was no effect on endurance performance following supplementation with a mixture of vitamin E (200 IU), inosine (100 mg), cytochrome C (500 mg), and coenzyme Q10 (100 mg) (36). Cycling time at 75% of maximal oxygen consumption was not affected after 4 weeks of vitamin E supplementation (21). More recently, Akova et al. (1) reported no effect of vitamin E supplementation on performance of 3 bouts of exhaustive knee isokinetic exercise in women.

Practical Applications

Our findings indicate that vitamin E supplementation (1200 IU/day for 3 weeks) had no effect on perceived muscle soreness, membrane disruption (assessed by CK levels), free radical generation (assessed by MDA), or exercise performance following a bout of whole body concentric and eccentric resistance exercise. Furthermore, there was no effect of vitamin E after the same bout of resistance exercise performed after 3 and 6 days of recovery. This study was unique because a typical resistance exercise workout was used as opposed to an eccentric only model, which has limited practical application for coaches, athletes, or trainers because eccentric-only exercise programs are not a component of typical resistance training regimens. Irrespective of vitamin E supplementation, these data indicate that there are small but statistically significant reductions in maximal strength and explosive power (approximately 10%) that persist up to 6 days after exercise in subjects not accustomed to resistance exercise. Although the initial exercise protocol in this study was relatively intense for a non–resistance-trained person, trainers should be conscious of sustained reductions in muscular performance during the early phase of a training program and adjust the exercise intensities appropriately. In summary, our re-

sults do not support the use of vitamin E supplementation as a method to enhance recovery from a single or repeated bout of resistance exercise in non–resistance-trained men. These findings do not rule out the possibility that vitamin E supplementation could have benefit for trained individuals or under different exercise configurations.

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