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Effect of vitamin E supplementation on delayed onset muscle soreness in young men

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Abstract

Introduction: It has been hypothesized that markers of delayed onset muscle soreness (DOMS) induced by eccentric training could be decreased by supplementing subjects with vitamin E. Hence, this study was carried out to investigate the effect of vitamin E supplementation on DOMS indexes.

Material & Methods: Twenty healthy male age 19 to 27 years participated as subjects in this study. The subjects were assigned to either a supplemental (400 IU of vitamin E per day for one month; n=10 and 22.4 $\pm$ 2.5 years of age) or a placebo group (n=10 and 22.7 $\pm$ 2.7 years of age) using a double-blind research design. peak power (PP) of Lower body, perceived pain, serum activity of the enzyme creatine kinase (CK) and C reactive protein (CRP) were taken before, immediately and 48 hours after the eccentric exercise.

Results: The results indicated that perceived pain and serum levels of CK and CRP increased and PP of Lower body decreased significantly immediately after eccentric exercise in

the both groups and these changes to be continued until 48 h after the intervention. No significant differences were observed between supplemental and placebo group during the study.

Conclusion: In conclusion, vitamin E supplementation had no effect in ameliorating markers of DOMS induced by eccentric exercise. Further studies are needed to examine the effects of vitamin E supplementation on DOMS induced by eccentric exercise.

Keywords: Delayed onset muscle soreness, Creatine kinase, C reactive protein, Vitamin E, Peak power of lower body

1. Introduction

Athletic performance and preparation are typically impaired when an athlete is sore or injured. Thus, any practice that limits the extent of damage or hastens recovery would be of interest and practical value to the coach, trainer, or therapist (1). Exercise-induced muscle damage is a tear of muscle fibers and connective tissues. It occurs when a person receives harmful physical, chemical and biological stimuli and also occurs as a result of physical trauma during exercise and physical activity (2). Muscle injury can be categorized into three major types in clinical presentation: Type I injury is characterized by muscle soreness that occurs 24 to 48 hours after unaccustomed exercise, and is known as delayed onset muscle soreness (DOMS); Type II injury is characterized by an acute disabling pain from a muscle tear of a few fibers with fascia remaining intact to a complete tear of the muscle and fascia; in type III injury, muscle soreness or cramping occurs during or immediately after exercise (2,3).

Exercise-induced muscle damage and its clinical corollary DOMS often result from unfamiliar predominantly eccentric exercise, such as downhill running. Furthermore, the degree of injury or damage is often a function of the trained state of the muscle. The injury itself is a mechanical disruption to sarcomeres (4) that proliferates secondary to an inflammatory response (5). Eccentric exercise results in injury to the cell

membrane, setting off an inflammatory response that leads to prostaglandin E2 and leukotriene synthesis. Prostaglandin E2 directly causes the sensation of pain by sensitizing type III and IV pain afferents to the effects of chemical stimuli, whereas leukotrienes increase vascular permeability and attract neutrophils to the site of damage. The ''respiratory burst'' of the neutrophils generates free radicals, which can exacerbate damage to the cell membrane. Swelling results from the movement of cells and fluid from the bloodstream into the interstitial spaces with inflammation and can contribute to the sensation of pain. The injury pattern is relatively sporadic throughout the muscle (6), and the tenderness that occurs appears to vary regionally within the muscle belly itself (7). Sarcomere disruption does not extend the length of a myofibril and usually does not extend across a whole muscle fiber (1,6).

Vitamins are organic compounds found in small amounts in food and classified based on their solubility in water or fat. Since most of the vitamins cannot be synthesized or manufactured in the body they should be acquired from the diet, as they are essential for body functions and are required to support health and well-being (8). Vitamins are important for antioxidant protection. The fat-soluble vitamin E plays an important role in membrane stabilization and synthesis of connective tissues (9). The effect of Vitamin E supplementation on DOMS indexes is not well known. Silva et al. (2008) reported that Vitamin E supplementation significantly decreases muscular and oxidative damage but not inflammatory response induced by eccentric contraction (10). Mohammed et al. (2015) also indicated that vitamin C and E were not effective in ameliorating markers of muscle damage and oxidative stress induced by weightlifting training (9). Hence, this study has been carried out to examine the possible effects of Vitamin E supplementation on eccentric exercise-induced DOMS indexes.

2. Material & Methods

Subjects

Twenty healthy male with a mean (±SD) age of 22.5 ± 2.3 year, volunteered to participate in this study. The exclusion criteria included history of muscle injury, non-compliance with research intervention, and

those who had consumed dietary or vitamin supplements or exogenous anabolic–androgenic steroids or other drugs, as stated in the anti-doping regulations, within the 6 weeks that preceded the study. Subjects were interviewed personally to ensure that they did not meet any obvious exclusion criteria. The subjects were informed about the experimental design and protocol and possible risks before signing the informed written consent form. The study was approved by the Marvdasht branch, Islamic Azad University Ethics Committee.

The subjects were assigned to either an experimental (n=10) or a placebo group (n=10) using a single-blind research design. The subjects in the experimental group were given one tablet of vitamin E 400 IU for one month and the subjects in the placebo group were given capsules containing maltodextrine, noncaloric filled in empty capsules during the intervention. Both groups were advised to take the supplements and/or placebo capsules on a daily basis and after dinner. To improve the compliance, subjects were contacted more than three times a week to ensure that they had taken the supplements and/or placebo capsules which were provided on the weekly basis.

Eccentric exercise

The schematic of eccentric exercise is shown in the figure 1. Subjects stepped up on a bench set at 110% of their lower leg length. Exercise was continued for 10 minutes at a rate of one step per second. The order of steps was right leg up, left leg up, left leg down, right leg down. This regimen causes greatest soreness in the right thigh and left calf. If the subject failed to complete the exercise test, this was recorded.

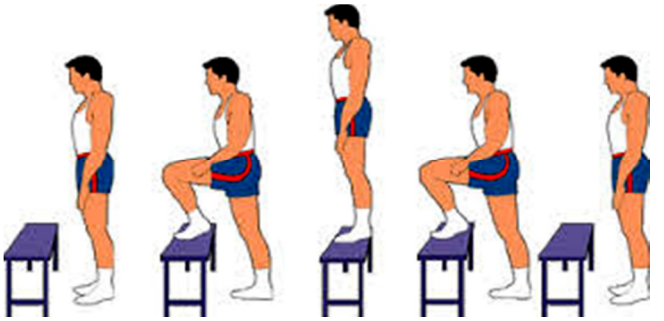

Figure1. Schematic of eccentric exercise

Nutrition

Before the beginning of the study, each subject was supervised to continue his normal sport nutrition program. On the testing day the subjects were supervised not to use any sport or dietary supplements. They were supervised also to keep food diaries for seven days in the 2-week period for what they were provided with specific verbal and written instructions and procedures for reporting detailed dietary intake, including how to record portions by using household measures, exact brand names and preparation techniques.

Blood sampling

Blood samples were taken from an antecubital vein in the sitting position. Five milliliters blood from a vein was taken pre exercise, immediately after the exercise, and at 48 hours after the eccentric exercise. Serum was separated and frozen at -20^{0C} prior to analysis. CK and CRP levels were measured using an enzyme-linked immunosorbent assay (ELISA) kits.

Score of muscle soreness and pain

After the bench stepping exercise, each subject was given an outcome form, which they were asked to complete at specified times, immediately and 48 hours after exercise. The outcome form consisted of a Likert scale as described by High et al (Figure 2; 11). The outcome was the mean score of soreness and pain over the tree period.

Please tick the sentence below that best describes your level of muscle soreness over the past 12 hours.
[ ] $_0$ A complete absence of soreness
[ ] $_1$ A light pain felt only when touched/a vague ache
[ ] $_2$ A moderate pain felt only when touched/a slight persistent pain
[ ] $_3$ A light pain when walking up or down stairs
[ ] $_4$ A light pain when walking on a flat surface/painful
[ ] $_5$ A moderate pain, stiffness or weakness when walking/very painful
[ ] $_6$ A severe pain that limits my ability to move

Figure 2. Likert scale of muscle soreness (taken from High et al. 1989).

Peak power measurement of Lower body

Peak power (PP) of lower body was measured by Sargent jump test (SJT) and Harman formula. In order to assess the SJT performance, according to the protocol of Harman et al. (1991) (12), the volunteers had their fingers on the right hand marked with orange chalk. While standing flatfooted next to a wall on their right side, and right arm extended above the head, the volunteer would mark on the wall the highest point that could be reached. At the moment preceding the jump, the volunteers could freely flex the lower limbs, as well as preparing the upper limbs for a sudden upward thrust, in effort to promote the highest vertical jump possible. At the highest point of the jump, the volunteers should extend the right hand against the wall as to mark the maximum height jumped. The jump height was the difference between the two points marked on the wall. All of the volunteers jumped three times, with a minimum interval of 45 seconds between the jumps and only the highest jump was considered. PP of lower body was measured by the following formula (12):

$$\text{Peak power(W)} = (61.9 \times \text{jump height(cm)}) + (36 \times \text{body weight(kg)}) + 1822$$

Statistical analysis

Results were expressed as the mean ± SD and distributions of all variables were assessed for normality. 2×3 repeated measures of ANOVA test was used to evaluate time-course change in variables. Post hoc analyses (Bonferroni) were then performed when warranted. The level of significance in all statistical analyses was set at $P \leq 0.05$. Data analysis was performed using SPSS software for windows (version 17, SPSS, Inc., Chicago, IL).

3. Results

In the present study, based on these results, anthropometrical measurements showed no significant differences in any of variables among the two groups. Anthropometric characteristics of the subjects are presented in Table 1.

Table 1. Anthropometric characteristics (mean ± SD) of the subjects

	Placebo	Experimental
Age (yr)	22.7 ± 2.2	22.4 ± 2.5
Body weight (kg)	76.6 ± 12.1	82.4 ± 19.0
Waist circumstance (cm)	84.6 ± 11.2	90.0 ± 15.3
Hip circumstance (cm)	96.1 ± 6.8	99.7 ± 14.0
WHR	0.88 ± 0.01	0.9 ± 0.02

The values of pain score, PP of lower body, serum CRP and serum CK of the subjects at baseline, immediately and 48h after the eccentric exercise are presented in the Figure 3. The results indicated that pain score increased significantly after the eccentric exercise and the increase continued until 48h later in the both groups ($P<0.05$). No significant differences were observed between two groups in any stages of blood sampling (Figure 3-a).

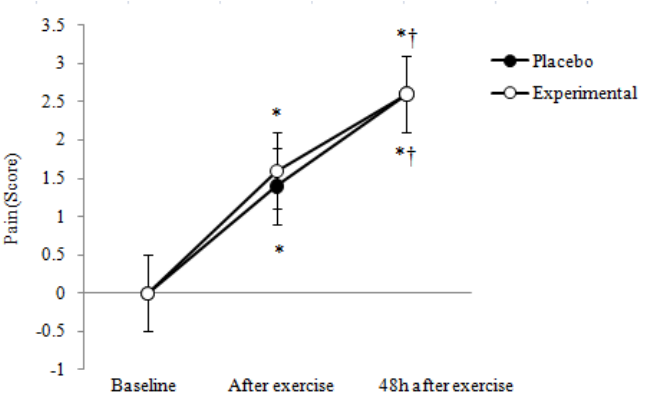

Figure 3-a. Changes of pain score during the intervention

* $P<0.05$, Baseline *vs.* immediately and 48h after eccentric exercise values
† $P<0.05$, Immediately *vs.* 48h after eccentric exercise values

Figure 3. Changes of pain score, PP of lower body, serum CRP and serum CK of the subjects during the intervention.

As shown in the Figure 3-b, PP of lower body was reduced significantly 48h after the eccentric exercise in the experimental and placebo groups in compare to the baseline ($P<0.05$), however no significant differences were observed between two groups.

The data on CRP levels are presented in the Figure 3-c. The results demonstrated that serum CRP had not significant changes during the intervention in the experimental and placebo groups. At the end, the data indicated that serum CK increased significantly after the eccentric exercise and the increase continued until 48h later in the both groups ($P<0.05$). No significant differences were observed between two groups in any stages of blood sampling (Figure 3-d).

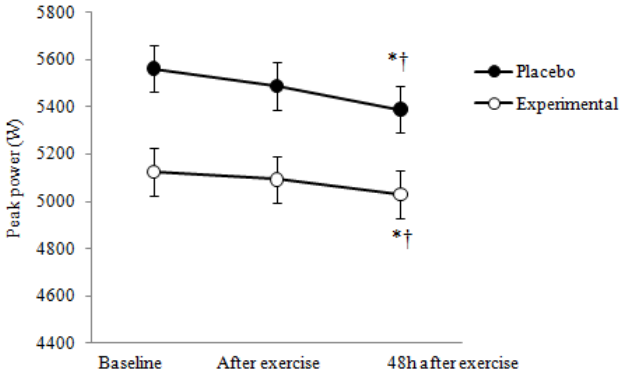

Figure 3. (Continued.) 3-b. Changes of PP of lower body during the intervention

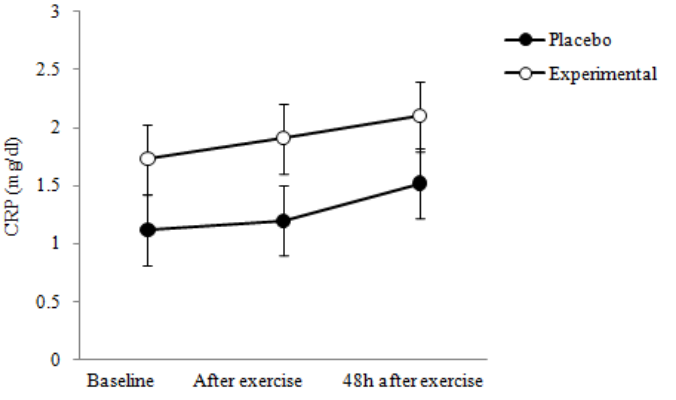

Figure 3. (Continued.) 3-c. Changes of serum CRP during the intervention

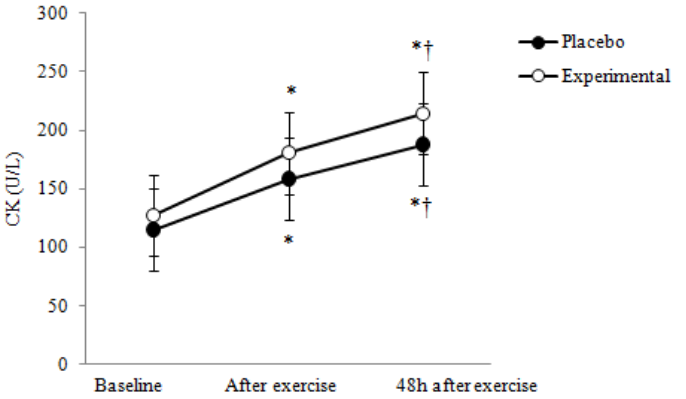

Figure 3. (Continued.) 3-d. Changes of serum CRP during the intervention

4. Discussion

Exercise that results in the development of soreness is associated with the rapid destruction of muscle tissue (13). DOMS is a familiar experience for the elite or novice athlete. Eccentric activities induce micro-injury at a greater frequency and severity than other types of muscle actions (14). Vitamin E has been reported to protect cellular velum and other fatty cellular parts by gifting electrons to the free radicals, and in this way it helps to reduce muscle damage (15,16). We had hypothesized that vitamin E supplementation would increase the structural integrity of muscle cell and thereby reduce the symptoms of the DOMS.

Our results indicate that vitamin E supplementation does not affect eccentric exercise-induced muscle pain, PP of lower body and serum levels of CK and CRP. Eccentric exercise is an active contraction of a muscle occurring simultaneously with lengthening of the muscle and induces severe muscle damage characterised by decreased muscle force production (17,18), increased serum CK activity (17,18), and inflammation response (19-21). Specifically, eccentric exercise induces damage to skeletal muscle in a fiber specific manner (22).

The results indicated that serum CRP had not significant changes during the intervention in the experimental and placebo groups and serum CK increased significantly after the eccentric exercise and the increase continued until 48h later in the both groups ($P<0.05$). No

significant differences were observed between two groups in any stages of blood sampling. In prior studies, the effect of vitamin E supplementation on CK and CRP responses to exercise have been inconsistent with reductions (23,24), no effect (25,26), or even increases (27) being reported. For example, Silva et al. (2010) investigated the effects of vitamin E (800 IU) supplementation on muscular and oxidative damage, as well as the inflammatory response induced by eccentric exercise on 21 participants. Fourteen days after starting the supplementation, the subjects performed eccentric exercise. Blood samples were obtained on days 0, 2, 4, and 7 after eccentric exercise. Both groups showed significantly increased TNF-alpha on the second day and IL-10 concentration on the fourth and seventh days after eccentric exercise. The results suggested that vitamin E supplementation represented an important factor in the defense against oxidative stress and muscle damage, but not against the inflammatory response in humans (10). Raphael et al. (2007) examined the effect of antioxidant supplementation and repeated bouts of moderate intensity endurance exercise on markers of muscle damage (CK) and systemic inflammation (CRP) in 20 healthy, young, sedentary men. They revealed that antioxidant supplementation might reduce muscle damage if caused by prolonged moderate intensity endurance exercises, but had no effect on the systemic inflammatory response (28).

Some researchers reported different results. Mastaloudis et al. (2006) examined the effect of vitamin supplements E (300 RRR-a-tocopheryl acetate IU) and C (1000 mg) in 22, over 50 km ultra-marathon runners. Blood samples were obtained before supplementation (baseline), 24 hours pre-, 12 hours pre-, and one hour pre-race; midrace, and for post-race, two hours post-race and six hours post-race. They showed that antioxidants appeared to have no effect on exercise-induced increases in muscle damage or recovery, but important sex differences were observed (29). Mohammed et al. (2015) also indicated that vitamin C and E were not effective in ameliorating markers of muscle damage and oxidative stress induced by weightlifting training (9). Gaeini et al. (2006) investigated the effect of vitamin E supplements on oxidative markers in 20 male student athletes. The supplement group received 450 mg of alpha-tocopherol daily for eight weeks. They then evaluated the CK,

MDA, protein carbonyl levels, and endurance performance. The results showed that E supplementation had no significant effect on these variables (30). Dowson et al. (2002) investigated whether four weeks of daily supplementation with 500 or 1000 mg of Vitamin C and 500 or 1000 IU of Vitamin E could modify the biochemical and ultrastructural indices of muscle damage following a 21 km run. They examined the indicators of creatine kinase, myoglobin, and malondialdehyde in the male runners after the endurance exercises. In both groups, there was a significant increase in creatine kinase and myoglobin, but the study did not report any significant changes in these three variables (25).

Several factors may explain the inconsistencies between these studies. First, investigations have used both animals and human subjects, and there is disagreement as to the comparability between models. Second, both the muscle group damaged and the mode of exercise used to do so vary widely. Third, dosages varying in both concentration and duration have been used. Finally, not all investigators report on the same indices of muscle damage and recovery.

5. Conclusion

Our findings indicate that vitamin E supplementation (400 IU/day for one month) had no effect on perceived muscle soreness, membrane disruption (assessed by CK levels), inflammatory responses (assessed by CRP), or PP of lower body following a bout of eccentric exercise.

In summary, our results do not support the use of vitamin E supplementation as a method to enhance recovery from a single bout of eccentric exercise in young healthy men. These findings do not rule out the possibility that vitamin E supplementation could have benefit for other individuals.

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