Effect of Dietary Vitamin E Supplementation on the Integrity of Skeletal Muscle in Exercised Horses

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ABSTRACT: The effect of vitamin E intake on indicators of muscle integrity was studied in exercised horses. Nineteen horses were blocked by sex and then assigned to one of three diets: no supplemental vitamin E (BASAL), BASAL plus 80 IU of supplemental vitamin E/kg DM (80), or BASAL plus 300 IU of supplemental vitamin E/kg DM (300). The BASAL diet contained less than 44 IU of vitamin E/kg DM, but it was adequate in all other nutrients. During the 90-d treatment period, horses were exercised 5 d/wk; in addition, serum and middle gluteal muscle \( \alpha \)-tocopherol concentrations were measured at 0, 30, and 90 d. All horses performed a repeated submaximal exercise test (RSET) at the end of the 90-d period. The following were measured before and after the RSET: \( \alpha \)-tocopherol, thiobarbituric acid reactive substances (TBARS), conjugated diene (CD) concentrations of the middle gluteal muscle, and serum creatine kinase (CK) and aspartate aminotransferase (AST) activities. Serum \( \alpha \)-tocopherol concentrations of horses receiving the BASAL and 80 diets decreased \((P < .05\) and \(P < .06,\) respectively) during the 90-d treatment period but did not change in horses receiving the 300 diet. Serum and muscle \( \alpha \)-tocopherol concentrations were higher \((P < .05)\) at 30 and 90 d in horses receiving the 300 diet than in horses receiving the BASAL and 80 diets. Serum CK and AST activities increased \((P < .05)\) following RSET but were not affected by dietary vitamin E level. Muscle \( \alpha \)-tocopherol level did not affect muscle CD or TBARS.

Key Words: Horses, Vitamin E, Creatine Kinase, Muscles

Introduction

Decreased vitamin E status has been implicated, but not substantiated, in the etiology of exercise-induced muscle damage (EIMD) in horses (Lindholm, 1987; Turner, 1992; Snow and Valberg, 1994). Current recommendations suggest that horses at maintenance receive 50 IU of vitamin E/kg dietary DM and that working horses receive 80 IU of vitamin E/kg dietary DM (NRC, 1989). Recent studies with exercised horses have not shown any negative effects of vitamin E intakes below 80 IU/kg, but those studies used exercise tests that may not have been sufficient to adequately challenge the effect of vitamin E on skeletal muscle integrity (Petersson et al., 1991; McMeniman and Hintz, 1992).

Previously, we described an exercise test for horses that significantly elevated serum creatine kinase (CK) and aspartate aminotransferase (AST) activities, two commonly used markers of muscle damage in horses (Siciliano et al., 1995). The present study used that exercise test to assess the effect of feeding a diet without supplemental vitamin E and the same diet supplemented with either 80 or 300 IU of d,l-\( \alpha \)-tocopheryl acetate/kg DM on the integrity of skeletal muscle in exercised horses. In addition, the effect of dietary vitamin E intake on \( \alpha \)-tocopherol concentration in serum and skeletal muscle of horses was monitored during a 90-d exercise conditioning period.

Materials and Methods

Horses

Nineteen horses of Thoroughbred or Quarter Horse breeding were used. All procedures in this study involving horses were approved by the Institutional Animal Care and Use Committee at the University of Kentucky. Only 12 horses could be housed and trained...
at one time, so the study was divided into two replications (Rep. A and Rep. B). The first replication (Rep. A) used 10 Thoroughbred horses (ranging in age from 4 to 15 yr; eight geldings and two mares) and two Quarter Horses (3 yr of age; one gelding and one mare). The second replication (Rep. B) used seven Thoroughbred horses (ranging in age from 7 to 15 yr; five geldings and two mares). Average body weights of the horses were 532 ± 11 and 539 ± 9 kg in Rep. A and B, respectively. One horse had to be removed from Rep. A (Quarter Horse, mare) due to injury not related to the study.

Thirty days before the beginning of each replication, horses were placed in drylots and adapted to a diet consisting of a 50% commercial feed grade d,l-concentrate portion of the diet with a supplement containing 80% grass hay (15.4 ± 3.5 IU/kg DM vitamin E) and 20% grain mix concentrate containing no supplemental vitamin E (15 IU/kg DM vitamin E). The diet was fed at 2% of body weight. The aim of this initial 30-d period was to accustom the horses to the testing facility and to adjust body condition to the moderate range (Henneke et al., 1984).

### Treatment Period

Following the 30-d adjustment period, horses were blocked by sex and then randomly assigned within blocks to one of three dietary treatments for a period of 90 d. Horses were not blocked by age, but the mean age for each treatment was similar (7.5 to 8.5 yr). The three treatments were as follows: a basal diet (Table 1) containing no supplemental vitamin E (BASAL), BASAL supplemented with 80 IU vitamin E/kg DM (80), and BASAL supplemented with 300 IU vitamin E/kg DM (300).

The basal diet consisted of 60% grass hay (timothy or orchard grass) and 40% mixed grain concentrate. When fed at 2% of body weight (DM basis), the BASAL diet met all nutrient requirements for moderately exercised horses (NRC, 1989) except vitamin E. The vitamin E content of the BASAL diet was less than 44 IU/kg DM. The desired levels of vitamin E in the 80 and 300 diets were attained by top-dressing the concentrate portion of the diet with a supplement consisting of a 50% commercial feed grade dl-α-tocopheryl acetate (BASF Corp., Parsippany, NJ) diluted in ground corn to 40 IU/g. Horses were individually fed throughout the study.

Horses were exercised 5 d/wk during the 90-d treatment period. Exercise consisted of treadmill exercise or free lunging in a round pen. Duration of each exercise period ranged from 20 to 30 min at speeds of 4 to 8 m/s. The exercise periods lasted longer and higher speeds were attained as the horses became more physically conditioned. All horses received the same amount and type of exercise on a given day.

Venous blood and muscle samples were obtained at the beginning of the 90-d treatment period (d 0), d 30, and d 90 for the determination of serum and muscle α-tocopherol concentrations. Blood and muscle samples were taken at rest before feeding and exercise. Blood samples were obtained from the jugular vein. Muscle biopsy samples were taken from the middle gluteal muscle using the method described by Snow and Guy (1976). To obtain enough tissue for subsequent analyses, a total of four biopsy sites were used at each sampling, and at least four muscle samples were harvested from each site. Muscle samples were immediately frozen in liquid nitrogen and then stored at −80°C. Blood samples were refrigerated at 4°C for 24 h and then the sera were removed and stored at −80°C.

### Exercise Test

At the end of the 90-d treatment period the horses were subjected to a repeated submaximal exercise test (RSET). The RSET consisted of three consecutive 30-min exercise bouts at submaximal speeds and heart rates with a 10- to 15-min rest period between each bout. Each bout consisted of the following sequence: 5 min of trotting at 4 m/s, 10 min of cantering at 9 m/s, 5 min of trotting at 4 m/s, 10 min of cantering at 9 m/s. Horses were hand-walked during the rest periods. Heart rate was continuously measured using values guaranteed by the provider (McCauley Bros., Versailles, KY) for concentrate portion of the diet and values from NRC (1989) for the forage portion, with the exception of vitamin E, for which actual values were used for both concentrate and forage. The concentrate portion of the BASAL diet consisted of the following ingredients in the following proportions: cracked corn 71%, oats 10%, soybean meal (48% CP) 10%, molasses (cane) 7.2%, calcium carbonate 1%, NaCl .5%, vitamin (excluding vitamin E) and mineral premix .3%. Diets in Rep. A and Rep. B differed only in the type of hay fed. Rep. A used timothy hay (15.4 ± 3.5 IU/kg vitamin E) and Rep. B used orchard grass hay (62.8 ± 6.9 IU/kg vitamin E).

### Table 1. Nutrient composition of BASAL diet (concentrate + forage)

<table>
<thead>
<tr>
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<tr>
<td>DE, Mcal/kg</td>
<td>2.7</td>
<td>2.8</td>
</tr>
<tr>
<td>CP, %</td>
<td>10.5</td>
<td>13.0</td>
</tr>
<tr>
<td>Fat, %</td>
<td>2.8</td>
<td>2.4</td>
</tr>
<tr>
<td>ADF, %</td>
<td>26.3</td>
<td>22.5</td>
</tr>
<tr>
<td>Ca, %</td>
<td>0.47</td>
<td>0.35</td>
</tr>
<tr>
<td>P, %</td>
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<td>0.34</td>
</tr>
<tr>
<td>Mg, %</td>
<td>0.15</td>
<td>0.13</td>
</tr>
<tr>
<td>K, %</td>
<td>1.47</td>
<td>2.13</td>
</tr>
<tr>
<td>Na, %</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Cl, %</td>
<td>0.36</td>
<td>0.33</td>
</tr>
<tr>
<td>S, %</td>
<td>0.08</td>
<td>0.17</td>
</tr>
<tr>
<td>Fe, ppm</td>
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<td>101</td>
</tr>
<tr>
<td>Zn, ppm</td>
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<td>Cu, ppm</td>
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<td>21.4</td>
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<tr>
<td>Mn, ppm</td>
<td>59</td>
<td>119</td>
</tr>
<tr>
<td>I, ppm</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Se, ppm</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>Vitamin A, IU/kg</td>
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<td>2,378</td>
</tr>
<tr>
<td>Vitamin D, IU/kg</td>
<td>178</td>
<td>176</td>
</tr>
<tr>
<td>Vitamin E, IU/kg</td>
<td>15.2</td>
<td>43.6</td>
</tr>
</tbody>
</table>

*Nutrients are expressed on a 100% dry matter basis. Diets in both Rep. A and Rep. B were fed at 2% of body weight in dry matter in a concentrate:forage ratio of 40:60. Nutrient values were determined using values guaranteed by the provider (McCauley Bros., Versailles, KY) for concentrate portion of the diet and values from NRC (1989) for the forage portion, with the exception of vitamin E, for which actual values were used for both concentrate and forage."
monitored throughout the exercise bouts and rest periods. Horses were not allowed to begin the next bout until their resting heart rate decreased to at least 60 beats/min (bpm). Because of the length of the RSET, all horses were not tested on the same day. No more than three horses were tested each day and treatment order was balanced.

Blood samples were taken 30 min before exercise (preexercise), immediately following the RSET (0), and at 1, 3, 6, 24, 48, and 72 h after the end of RSET for determination of serum CK and AST activities. Muscle biopsies were taken from the middle gluteal muscle immediately before and immediately after the RSET for determination of \( \alpha \)-tocopherol, thiobarbituric acid reactive substances (TBARS), and conjugated dienes (CD). In all horses, postexercise muscle biopsies were obtained from the same middle gluteal muscle as the preexercise biopsies. In the last seven horses subjected to the RSET, postexercise samples were also obtained from the opposite middle gluteal muscle. This additional sampling was prompted by the observation that a few horses developed some local inflammation around the biopsy site.

Sample Analysis

Samples were analyzed for \( \alpha \)-tocopherol using HPLC with a fluorescence detector according to the method of Hidiroglou (1989) for serum samples and the method of Chan et al. (1994) for muscle samples. All samples were analyzed in duplicate. Coefficients of variation between injections and between extractions within a sample were less than 5% and 10% for serum and muscle, respectively. Muscle samples taken from each horse were freeze-dried, powdered, and combined within a horse within a sampling time before analysis. The analytical column used for \( \alpha \)-tocopherol measurement was a Nova-Pak C18 4 \( \mu \)m (3.9 mm x 150 mm) (Waters, Milford, MA). Coefficients of variation between duplicate samples for serum and muscle \( \alpha \)-tocopherol concentration were less than 5% and less than 10%, respectively.

Muscle samples collected before and after exercise were also analyzed for CD using the spectrophotometric method of Recknagel et al. (1984) and for TBARS using the spectrophotometric method of Sinnhuber and Yu (1958). Coefficients of variation between duplicate samples for middle gluteal muscle concentrations of TBARS and CD was less than 10%. Serum samples taken before exercise and following the RSET test were analyzed for CK and AST activity using commercially available reagents (Sigma Chemical, St. Louis, MO) with a programmed semiautomated spectrophotometer (Cobas Fara II, Roche Diagnostic Systems, Montclair, NJ). Coefficients of variation between duplicate samples were less than 5% for serum CK and AST activities.

Statistical Analyses

Variation in response variables (serum CK [U/L], serum AST [U/L], serum \( \alpha \)-tocopherol [\( \mu \)g/mL], muscle \( \alpha \)-tocopherol [\( \mu \)g/g of dry muscle], muscle CD [nmol/mg of dry muscle], and muscle TBARS [pmol/mg of dry muscle]) were partitioned using the GLM procedure of SAS for repeated measures design (SAS, 1985). The statistical model included level of vitamin E intake and replication. No differences (\( P > .05 \)) were found between Rep. A and Rep. B for any of the response variables. Therefore, both replications were combined and analyzed as one experiment. Differences between dietary treatments were analyzed using between-horse variation as the error term. Differences due to time (i.e., days after the start of supplementation before or vs after exercise) and time \( \times \) diet interaction were analyzed using within-horse variation. Treatment differences were considered significant at the \( P < .05 \) level. A least significant difference test was used for means separation. Within each treatment, contrasts between days (0 vs 30, and 0 vs 90) were used to determine when changes occurred when the effect of time was significant.

Results

Changes During the 90-Day Treatment Period

Mean serum \( \alpha \)-tocopherol concentrations in the three treatment groups during the 90-d treatment period are shown in Figure 1. There were no differences between supplemental treatment groups at
Figure 2. Mean muscle α-tocopherol concentrations in horses receiving the BASAL diet (n = 6; solid line), the diet containing 80 IU of supplemental vitamin E/kg DM (n = 5; dashed line), or the diet containing 300 IU of supplemental vitamin E/kg DM (n = 7; dotted line). Effect of diet (P < .05), 300 > 80 and BASAL at d 30 and 90 (P < .05). Pooled SEM = 2.

d 0 when horses were initially assigned to treatments; mean α-tocopherol concentration was 2.9 ± .2 μg/mL. However, during the subsequent 90-d period when horses were exercised 5 d/wk, there was an interaction between level of dietary vitamin E supplementation and days of supplementation (P < .05). Mean serum α-tocopherol concentrations decreased from d 0 through d 90 in the BASAL (P < .05) and 80 (P < .06) treatment groups. Mean serum α-tocopherol concentrations in the 300 group did not change over the 90-d treatment period. Mean serum α-tocopherol concentration was higher (P < .05) in the 300 treatment than in either BASAL or 80 at 30 and 90 d. Mean serum α-tocopherol concentration was not different (P > .05) between the BASAL and 80 treatments at any time.

Changes in mean middle gluteal muscle α-tocopherol concentrations are shown in Figure 2. Mean α-tocopherol concentration in biopsies taken from the middle gluteal muscle was 10.1 ± 1.1 μg/g on d 0 and was not different among treatment groups. At d 30 and 90, mean middle gluteal muscle α-tocopherol concentration was higher (P < .05) in horses receiving the 300 diet than in horses receiving the BASAL or the 80 diet. At d 30 and 90, mean middle gluteal muscle α-tocopherol concentration was not different (P < .05) in horses receiving the BASAL and 80 diets.

Response to the Repeated Submaximal Exercise Test

Following the 90-d treatment period, 17 horses completed the RSET. Mean serum CK and AST activities before and 0, 1, 3, 6, 24, 48, and 72 h after exercise are shown in Figure 3. Mean serum AST and CK activities were markedly increased in all treatments following the RSET (P < .01). However, dietary vitamin E supplementation had no effect (P > .05) on the magnitude of the postexercise increases in serum AST and CK activities. The larger numerical values for mean CK and AST activities after exercise in the BASAL group were due to the response of one horse.

The RSET did not affect (P > .05) mean concentrations of α-tocopherol in the serum or middle gluteal muscle (Figure 4). Mean middle gluteal muscle TBARS concentration was not affected by the RSET or level of dietary vitamin E supplementation (Figure 5). Middle gluteal muscle CD concentrations were not affected by level of vitamin E supplementation. Across treatments, postexercise mean middle gluteal muscle CD concentration, measured in the samples taken from the same muscle as the preexercise samples, was greater (P < .05) than preexercise concentrations (Figure 6). However, the mean CD concentrations of
Figure 4. Mean serum and muscle \( \alpha \)-tocopherol concentrations before (solid bars), immediately after (hatched bars), and 72 h after (dotted bars; serum only) the RSET in horses receiving diets containing no supplemental vitamin E (BASAL; \( n = 5 \)), 80 IU of supplemental vitamin E/kg DM (80; \( n = 5 \)), or 300 IU of supplemental vitamin E/kg DM (300; \( n = 7 \)). Serum \( \alpha \)-tocopherol concentrations were higher \( (P < .05) \) in horses receiving the 300 treatment than in horses receiving the 0 or 80 treatments. Pooled SEM = .2 and 1.4 for serum and muscle \( \alpha \)-tocopherol, respectively.

middle gluteal muscle samples taken from the muscle opposite that of the preexercise sample were not different \( (P > .05) \) from the preexercise CD concentrations (Figure 7). There was no difference between site of sampling for postexercise TBARS concentration of the middle gluteal muscle.

Discussion

Exercise-induced muscle damage is a common problem in performance horses (Lindholm, 1987; Turner, 1992). The production of potentially harmful free radicals during exercise has been proposed as a causative agent in EIMD (Sjodin et al., 1990), and, as a result, enhancement of antioxidant defense mechanisms has been investigated as a means of preventing EIMD (Goldfarb, 1993). Vitamin E, the major lipid-soluble antioxidant defense in cells, plays an important role in maintaining the integrity of cell membranes (Combs et al., 1975). Some researchers have suggested that free radicals produced during exercise increase vitamin E requirements (Quintanilha, 1984; Bendich, 1991), and vitamin E deficiency has been reported to exacerbate EIMD in rodents (Amelink et al., 1991).

In the present study, the decline in mean serum \( \alpha \)-tocopherol concentration in horses receiving the BASAL and 80 treatments was consistent with previous reports. Petersson et al. (1991) reported that plasma vitamin E was lower, over a 4-mo period, in exercised horses compared with nonexercised controls. Exercise also has been reported to decrease vitamin E status in humans (Meydani et al., 1993) and rodents (Bowles et al., 1991). The results observed in the horses receiving the 300 treatment are in agreement with the recent work of Saastamoinen and Juusela (1993), who found that approximately 150 to 250 IU of vitamin E/kg DM was necessary to keep serum vitamin E concentration from declining in horses receiving regular exercise.

Results of the present study suggest that a single bout of repeated submaximal exercise does not alter vitamin E status in the blood and skeletal muscle of horses. Similarly, McMeniman and Hintz (1992) found no difference in plasma or muscle \( \alpha \)-tocopherol concentrations in horses following moderate to intense exercise of relatively short duration. However, when

Figure 5. Mean concentrations of thiobarbituric acid reactive substances (TBARS) in muscle biopsies taken from the middle gluteal muscle before exercise (solid bars) and after exercise in the same muscle (hatched bars) in horses receiving diets containing no supplemental vitamin E (BASAL; \( n = 5 \)), 80 IU of supplemental vitamin E/kg DM (80; \( n = 5 \)), or 300 IU of supplemental vitamin E/kg DM (300; \( n = 7 \)). Pooled SEM = 27.
Figure 6. Mean concentrations of conjugated dienes in muscle biopsies taken from the middle gluteal muscle before exercise (solid bars) and immediately after exercise in the same muscle as the before sample (hatched bars) in horses receiving diets containing no supplemental vitamin E (BASAL; n = 5), 80 IU of supplemental vitamin E/kg DM (80; n = 5), or 300 IU of supplemental vitamin E/kg DM (300; n = 7). Mean conjugated diene concentration after exercise was greater ($P < .05$) than before exercise. Pooled SEM = .2.

Figure 7. Mean concentrations of conjugated dienes and thiobarbituric acid reactive substances (TBARS) in muscle biopsies taken from the last seven horses subjected to the repeated submaximal exercise test. Left panel: compared with the biopsies taken before exercise (solid bar), conjugated diene concentration was increased ($P < .05$) after exercise in the biopsies taken from the same muscle (checkered bar), but not in the biopsies taken from the muscle opposite the before-exercise samples (hatched bars). Pooled SEM = .2. Right panel: concentration of TBARS in muscle biopsies taken before exercise (solid bar) was not different ($P > .05$) from concentration after exercise in the biopsies taken from the same muscle (checkered bar) or in the biopsies taken from the muscle opposite the before-exercise samples (hatched bars). Pooled SEM = 21.

Horses are conditioned for several weeks, they may require a level of dietary vitamin E supplementation above 80 IU/kg DM, and potentially approaching 300 IU/kg DM to maintain vitamin E status in the blood and skeletal muscle.

In this study, peak serum activities of CK and AST were observed at 3 to 6 and 6 to 24 h after exercise, respectively. Although CK and AST activities increased in all treatment groups, level of vitamin E supplementation did not influence postexercise serum CK and AST activity. Although vitamin E deficiency exacerbates EIMD in rodents (Jackson et al., 1983; Amelink et al., 1991), supplementation of dietary vitamin E to humans and rodents consuming diets sufficient in vitamin E does not seem to entirely prevent the occurrence of EIMD (Cannon et al., 1990; Warren et al., 1992). All horses in this study were not likely vitamin E-deficient, because serum and muscle $\alpha$-tocopherol concentrations at all time points were within the typical range for clinically normal horses (1 to 9 $\mu$g/mL for serum and 2 to 12 $\mu$g/g wet muscle; Roneus et al., 1986; Craig et al., 1989; Petersson et al., 1991).

It has been argued that postexercise elevations in serum CK do not reflect significant myolysis unless they exceed 10,000 U/L (Volfinger et al., 1994). In addition, horses showing histological evidence of muscle damage following submaximal exercise have an earlier and more marked rise in serum CK and AST than those horses not having histological evidence of muscle damage (Valberg et al., 1993). At least one horse in each treatment group had a postexercise CK activity greater than 10,000 U/L and had a pattern of CK and AST response similar to that described by Valberg et al. (1993) for horses with histological signs of muscle damage. In addition, one horse in the BASAL group experienced clinical signs of exertional rhabdomyolysis (ER) near the end of the treatment period and one horse in the 300 treatment group had elevated preexercise CK and AST activities that may be indicative of a recent attack of subclinical ER (Valberg, 1993). Exertional rhabdomyolysis is one of the most serious forms of EIMD, and acutely affected horses often exhibit severe muscle cramping ("tying up"). The above results indicate that ER may have occurred in horses consuming diets supplemented with up to 300 IU of vitamin E/kg DM.
Free radical attack on membrane lipids leading to lipid oxidation and cell damage has been suggested as a mechanism by which EIMD occurs (Davies et al., 1982). However, a direct cause and effect relationship between lipid oxidation and EIMD has not been established, because lipid oxidation may be a consequence of EIMD rather than the cause (Alessio, 1993). The presence of TBARS is a commonly used measure of lipid peroxidation (Holley and Cheeseman, 1993). The concentration of TBARS in biopsies from the middle gluteal muscle was not affected by diet or exercise in this study, possibly because of the large amount of variation in the TBARS measurement. This variation may have been due to interfering substances other than malonaldehyde (Holley and Cheeseman, 1993) or to the effect of the muscle sampling technique on individual horses, because some horses appeared to have more local swelling than others after the biopsy procedure.

Lipid peroxides possess a conjugated diene structure having a characteristic UV absorbance around 233 nm and may be used as a measure of lipid peroxidation (Holley and Cheeseman, 1993). In the present study, middle gluteal muscle CD increased significantly from before to after exercise when both samples were taken from the same middle gluteal muscle. However, there was no difference between preexercise and postexercise middle gluteal muscle CD concentration when the samples were taken from opposing middle gluteal muscles in the last seven horses subjected to the RSET. Opposing middle gluteal muscle samples were only taken from a few horses in each treatment (BASAL, n = 2; 80, n = 2; 300, n = 3); thus, comparisons regarding treatment effects were not made. Nonetheless, these results suggest that the postexercise increase in middle gluteal muscle CD concentration may have been an artifact due to inflammation from repeated sampling of the same muscle and not exercise. The preexercise biopsy may have induced an inflammatory response in the middle gluteal muscle, resulting in free radical production and increased lipid peroxidation (Kehrer, 1993). The explanation as to why mean CD concentration, but not mean TBARS concentration, was influenced by repeated sampling of the same muscle in this study may be related to differences in sample preparation between the two assays. The concentration of TBARS was measured in whole muscle samples and may have included interfering substances (Holley and Cheeseman, 1993) that could cause the large variation reported for TBARS, whereas CD was measured in a lipid extract of the skeletal muscle.

Although the results of the present study suggest that lipid oxidation was not a factor in the development of EIMD, lipid oxidation may be important in the period following the onset of muscle damage. The results in this experiment suggest that trauma associated with the needle biopsy resulted in an increase in lipid oxidation in the 2.5 h that elapsed between the preexercise biopsy and the postexercise biopsy that was taken immediately following the cessation of exercise. Therefore, to detect an increase in lipid oxidation as a result of exercise, a later sampling period (i.e., 2 to 6 h after the end of exercise) might have been necessary. Lipid oxidation occurring secondary to EIMD could potentially exacerbate muscle damage in the absence of adequate vitamin E. Therefore, an area of interest for future studies may be the effect of vitamin E supplementation on recovery from EIMD.

Implications

The results of this study indicate that dietary levels of vitamin E greater than 80 international units per kilogram of dry matter, and potentially approaching 300 international units per kilogram of dry matter, are necessary to maintain blood and skeletal muscle concentrations in horses undergoing exercise conditioning. However, vitamin E status of the horses did not seem to affect the integrity of skeletal muscle following repeated submaximal exercise, as measured by changes in serum creatine kinase and aspartate aminotransferase activities.

Literature Cited


