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ARTICLE

Effects of taurine supplementation following eccentric exercise in young adults

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Abstract: The purpose of the present study was to investigate the effects of taurine supplementation on muscle performance, oxidative stress, and inflammation response after eccentric exercise (EE) in males. Twenty-one participants (mean age, 21 ± 6 years; weight, 78.2 ± 5 kg; height, 176 ± 7 cm) were selected and randomly divided into two groups: placebo ($n = 10$) and taurine ($n = 11$). Fourteen days after starting supplementation, subjects performed EE (3 sets until exhaustion, with EE of the elbow flexors on the Scott bench, 80% 1 repetition maximum (RM)). Blood samples were collected and muscle performance was measured on days 1, 14, 16, 18, and 21 after starting the supplements. Then, performance, muscle damage, oxidative stress, and inflammatory markers were analyzed. The taurine supplementation resulted in increased strength levels and thiol total content and decreased muscle soreness, lactate dehydrogenase level, creatine kinase activity, and oxidative damage (xylenol and protein carbonyl). Antioxidant enzymes (superoxide dismutase, catalase, and gluthatione peroxidase) and inflammatory markers (tumor necrosis factor, interleukin-1β (IL-1β), and interleukin-10 (IL-10)) were not altered during the recovery period compared with the placebo group. The results suggest that taurine supplementation represents an important factor in improving performance and decreasing muscle damage and oxidative stress but does not decrease the inflammatory response after EE.

Key words: taurine, eccentric exercise, physical performance, oxidative stress, inflammation, supplementation.

Résumé : Cette étude se propose d'examiner les effets de la supplémentation en taurine sur la performance musculaire, le stress oxydatif et la réponse inflammatoire à la suite d'un exercice pliométrique chez des hommes. On répartit aléatoirement une sélection de 21 hommes (21 ± 6 ans, 78,2 ± 5 kg, 176 ± 7 cm) dans deux groupes : placebo ($n = 10$) et taurine ($n = 11$). Quatorze jours après le début de la supplémentation, les sujets effectuent sur un banc Scott trois séries d'exercices pliométriques des fléchisseurs du coude (80 % 1 RM) jusqu'à épuisement. On prélève des échantillons sanguins et on évalue la performance musculaire aux jours 1, 14, 16, 18 et 21 suivant le début de la supplémentation. Puis on analyse la performance, les lésions musculaires, le stress oxydatif et les marqueurs de l'inflammation. La supplémentation en taurine suscite une augmentation de la force et du contenu total en thiol ainsi qu'une diminution de la douleur musculaire, de la concentration de lactate déshydrogénase, de l'activité de la créatine kinase et des lésions oxydatives (xylénol et protéine carbonylée). Pendant la période de récupération, on n'observe dans les groupes supplémentés aucune modification de l'activité des enzymes antioxydantes (superoxyde dismutase, catalase et glutathion peroxydase) et des marqueurs de l'inflammation (facteur de nécrose tumorale, interleukine-1β (IL-1β) et interleukine-10 (IL-10)) comparativement au groupe placebo. D'après ces observations, la supplémentation en taurine constitue un moyen important pour améliorer la performance et diminuer les lésions musculaires et le stress oxydatif, mais elle ne diminue pas la réponse inflammatoire consécutive à un exercice pliométrique. [Traduit par la Rédaction]

Mots-clés : taurine, exercice pliométrique, performance physique, stress oxydatif, inflammation, supplémentation.

Introduction

Eccentric actions are characterized by a loading profile that combines high force and low fiber recruitment (i.e., high force per fiber ratio), which places a substantial mechanical stress on the associated structures (Enoka 1996; Armstrong et al. 1991). The mechanical stress that causes the disruption of the sarcomeres and failure within the excitation contraction coupling system is theoretically believed to be the primary factor initiating injury (Chiang et al. 2009), with a host of biochemical changes within the affected area such as increased inflammatory cytokines and reactive oxygen species (ROS) that may exacerbate the injury (Urso and Clarkson 2003; Tidball 2005; Mahoney et al. 2008).

The degree of muscle injury may be assessed through a variety of direct and indirect measures, which include indirect markers such as muscle soreness and reduced muscle force, as well as changes in oxidative stress and inflammatory biomarkers. Impaired performance, most notably a reduction in maximum isometric voluntary contraction force, has been suggested to be the best marker in assessing muscle injury (Falvo and Bloomer 2009).

Prior studies have reported varying degrees of protection against muscle soreness and (or) strength loss (Kaminsky and Boal
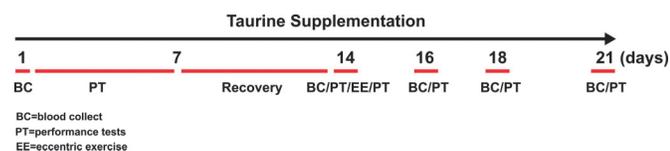
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Fig. 1. Study design of young university students conducted over 21 days and comparing a control group (placebo) with a dietary-supplement group.

Table 1. Dietary analysis in young university students provided taurine supplement or placebo.

Groups	Energy (Kcal)	Protein (%)	Fat (%)	Carbohydrate (%)
Placebo	2667±228	16.5±4.4	32.5±3.9	42.9±7
Taurine	2692±364	15.4±2.2	30.4±3.5	53.7±4.5

Note: Protein, fat, and carbohydrate were quantified as available energy (%). There were no significant differences between groups ($p > 0.05$). The values are presented as means ± SEM.

1992; Bloomer and Goldfarb 2004; Shafat et al. 2004; Connolly et al. 2006), inflammation (Phillips et al. 2003), and oxidative stress (Goldfarb et al. 2005; Silva et al. 2010) following antioxidant supplementation. Many other investigations report little or no benefit of antioxidant supplementation in relation to muscle injury or oxidative stress (Childs et al. 2001; Beaton et al. 2002; Avery et al. 2003; Bloomer et al. 2005; Silva et al. 2008). However, this probably depends on which component of the endogenous antioxidant defense system is favorably altered by exercise training.

Taurine (2-aminoethanesulfonic acid) is an amino acid derived from cysteine metabolism and is involved in cell volume regulation, detoxification, and osmoregulation, membrane stabilization, intracellular calcium flux regulation, and antioxidant production due to its molecular structure (Huxtable 1992; Zembron-Lacny et al. 2007; Miyamoto et al. 2009; Silva et al. 2011). It has been reported that taurine can protect against oxidative stress under various conditions (Zhang et al. 2004; Redmond et al. 1998; Qi et al. 1995; Silva et al. 2011) and act as an anti-inflammatory agent (Huxtable 1992; Zembron-Lacny et al. 2007; Miyamoto et al. 2009; Silva et al. 2011). Furthermore, several studies reported the beneficial effects of taurine administration not only in rats, but also in humans (Baum and Weiss 2001; Dawson et al. 2002; Zhang et al. 2004; Silva et al. 2011). Taurine has also been reported to reduce oxidative damage to DNA (Messina and Dawson 2000). Dawson et al. (2002) found that 3% taurine in drinking water could improve the running performance in adult male rats. Zhang et al. (2004) showed that taurine supplementation could prevent exercise-induced oxidative stress in healthy young men. Mechanistically, during the inflammatory process, taurine interacts with hypochorous acid (HOCl), the product of the myeloperoxidase–halide system of neutrophils, generating a less toxic product (taurine chloramine). It has been shown in vitro that taurine chloramine exerts anti-inflammatory effects (Walczewska and Marcinkiewicz 2011). Another possibility suggested by Joo et al. (2009) is that the anti-inflammatory effect of taurine is attributed to the inhibition of the interleukin-1β (IL-1β) mediated NFκB activation.

While the potential effect of taurine supplementation on oxidative stress and inflammation during the recovery period after eccentric contraction in humans has not been investigated, it is reasonable to hypothesize that taurine supplementation may exert beneficial effects on those variables and contribute to improving muscle performance.

Materials and methods

Study design

This double-blind randomized study was conducted over 21 days and compared a control group (placebo) with a dietary-supplement group (Fig. 1). The subjects received taurine supplementation or placebo for 21 consecutive days. On day 14, an eccentric exercise (EE) session was performed. Blood samples were collected and performance tests were conducted at baseline and before and immediately after EE and aerobic exercise sessions, as shown below.

Subjects

Twenty-one male volunteers, students at the Universidade do Extremo Sul Catarinense (Unesc) (Criciuma, Santa Catarina, Brazil), of a mean age of 21 ± 6 years, body mass of 78.2 ± 5 kg, and height of 176 ± 7 cm participated in this randomized, double-blind, placebo-controlled study. The subjects were randomly divided into two groups: taurine ($n = 11$) and placebo ($n = 10$). Personal characteristics did not differ between the two groups (Table 1). Once the purpose and risks of the study had been explained to the subjects, oral and written consent was obtained from them. The protocol was approved by the local Research Ethics Committee (84/2010/CEP/UNESC).

Exclusion criteria

All subjects were nonsmokers, were not taking taurine or any other antioxidant or related supplements, had not participated in resistance training or any other form of structured exercise for at least 6 months, did not have a history of muscular lesions, and were not carriers of any disease that might compromise the results or be aggravated by physical exercise. During the postexercise period, all subjects were advised not to use NSAIDS, massage, ice, or other interventions to help speed up recovery and decrease the symptoms of muscle injury.

Supplementation

The supplementation consisted of taurine capsules containing 50 mg·kg body mass^{-1}·day^{-1} according to Mero et al. (2008), whereas the placebo consisted of a similar capsule containing starch. Volunteers received one capsule per day for a total of 21 days, beginning 14 days before the eccentric protocol and continuing throughout the 7-day postexercise period.

Blood sampling

A 10 mL sample of blood was drawn from an antecubital vein on days 1 and 14 (before EE) and on days 16, 18, and 21. The blood was collected in vacutainers without additives and centrifuged at 1500 r·min^{-1} for 10 min at 4 °C. Aliquots of washed (lysed) red blood cells and plasma samples were stored at –70 °C until biochemical assays were performed.

Dietary records

All subjects were instructed to maintain their normal diet during the study period and complete daily food records for the 2 days before testing, the day of testing, and the day after testing. Records were analyzed for total kilocalories, proteins, carbohydrates, and fats using commercially available software (Diet Analysis Plus; ESHA Research, Salem, Oregon).

One repetition maximum (1 RM) test and concentric strength

The subject's one repetition maximum (1 RM) level was assessed (Bompa 2001) by elbow flexors and extensors. Before testing, a standardized warm-up consisting of a 10 min ride on a bicycle ergometer was carried out. The weights lifted in the 1 RM test for each group are described in Table 1. The 1 RM test was done at baseline and then repeated 14 days later before the EE protocol and during the recovery period. It was quantified as the percentage of subjects that succeeded in executing the movement (1 RM).

It was considered that 100% of the subjects could perform 1 RM before EE.

Isometric strength and muscular fatigue

Isometric strength was evaluated by a load transducer (model 100; Takei Scientific Instrument Co. Ltd., Tokyo, Japan) connected to a digital recorder (F360A; Unipulse Corp., Saitama, Japan) and a computer (Macintosh Performa 5410; Apple Computer, Inc., Cupertino, California). The maximal isometric strength (MIF) was measured twice (1 min between the measurements) at an elbow flexion joint of 90° for 4 s. The peak force of each 4 s value was determined, and the mean of the two measurements was used for analysis (MaTon 1981).

Perceived effort

The perception of effort was performed using a numerical scale. The subjects indicated their own perception of effort after the fatigue test on a scale of 0 to 10. This scale was adapted from the original scale that had scores from 0 to 20.

Relaxed elbow joint angle

A universal goniometer (Lafayette Instrument Co. Inc., Lafayette, Indiana) was used for the angle measures according to Johnson and Nelson (1979). Flexed elbow joint angle was performed when the subject while standing was asked to fully flex the elbow joint by leading the palm to the shoulder while keeping the elbow joint at the side of the body. The angle was measured three times and the mean of the three measurements was used.

Analyses of performance

The analyses of performance (concentric and isometric strength and muscular fatigue) were performed on day 1, before and after EE, and on days 16, 18, and 21. After 1 RM testing, the participants were instructed to follow their normal dietary patterns and to refrain from strenuous physical exercise during the 21 days of supplementation.

Familiarization of eccentric contraction protocol and analyses of performance

The subjects were seated with elbows resting on a Scott bench (equipment used during muscular exercise) with an inclination of 45°. The angle of the elbow joint at the starting position was 90° and the final angle was 170°.

All volunteers were familiarized with the Scott bench exercise with 2 kg between days 1 to 7 after starting supplementation (3 sets, 15 repeats with 2 min rest between the sets).

Eccentric exercise (EE) protocol

EE was performed with elbow flexion and extension on the Scott bench at an intensity of 80% of 1 RM (Silva et al. 2010). The concentric phase (flexion) of the exercise was performed with manual assistance from the instructor. The eccentric phase (extension) was performed for a duration of 6–8 s. Three sets of exercises were performed with 2 min rest intervals and repeats, with average number of repeats between 11 and 15.

Muscle soreness

The visual analogue method has been established as a reliable method for assessing soreness (Revill et al. 1976). The intensity of the perceived soreness of the biceps muscle was assessed using a 10 cm visual analog scale (VAS), the left and right extremes of which refer to "no muscular soreness" and "maximum muscular soreness," respectively. The VAS is easily and quickly administered and has been used as a reliable measurement for determining the intensity of human pain. Thus, subjects were asked to draw a vertical line toward the amount of soreness that they perceived, which was quantified as the distance (with a precision of 0.1 cm) between the left extreme of the line and the vertical line drawn by the subject. All of the subjects had been previously familiarized with the VAS.

Muscular damage

Creatine kinase (CK) enzyme activity was used as a marker of muscular damage. A specific kit, supplied by Labtest Diagnóstica SA (Lagoa Santa – Minas Gerais, Brazil) was used to determine levels using an enzymatic system with a final point reaction in serum samples. A 0.1 mL sample was added to 0.2 mL of standard reagent, and the mixture was homogenized and incubated for 5 min. After transfer to bucket at 37 °C, there was a wait for 30 s. The initial absorbance (0 min) and final absorbance (5 min) were read at 340 nm using an Ultraspec 2000 spectrophotometer (Pharmacia Biotech, Uppsala, Sweden). The results are expressed in units per litre ($U \cdot L^{-1}$). The intra- and inter-assay coefficients of variation (CVs) were 5.2% and 6.5%, respectively.

Oxidative damage

Oxidation xylenol orange (xylenol)

This method detects hydroperoxides (ROOHs), which are products of lipoperoxidation (Jiang et al. 1991). The xylenol orange assay is based on the oxidation of ferrous ions to ferric ions by ROOHs under acidic conditions. Samples were homogenized (30 mg·mL^{-1}), and aliquots (90 μL) were transferred to microcentrifuge vials (1.0 mL). Ten microlitres of 10 mmol·L^{-1} thymidine triphosphate (TPP) in methanol were added to the vials to reduce ROOHs. All the vials were then vortexed and incubated at room temperature for 30 min prior to the addition of 900 mL of rotor (Hettich Rotanta/RP centrifuge; Hettich-Zentrifugen, Tuttlingen, Germany). Absorbance of the supernatant was measured at 560 nm using an Ultraspec 2000 spectrophotometer (Pharmacia Biotech). The intra- and inter-assay CVs were 4.3% and 5.4%, respectively.

Protein carbonylation

This method detects the determination of the carbonyl groups based on the reaction with dinitrophenylhydrazine (DNPH) (Levine et al. 1990). Proteins were precipitated by the addition of 20% trichloroacetic acid and reacted with DNPH. The samples were then redissolved in 6 mol·L^{-1} guanidine hydrochloride, and the carbonyl contents were determined through absorbance at 370 nm using a molar absorption coefficient of 22.000 M^{-1}. The intra- and inter-assay CVs were 3.6% and 4.8%, respectively.

Total thiol (TT) content

The TT content was determined using the 5,5'-dithioibis (2-nitrobenzoic acid) (DTNB; Sigma-Aldrich, St. Louis, Missouri) method. The reaction was started by the addition of 30 μL of 10 mmol·L^{-1} DTNB stock solution to phosphate-buffered saline (PBS). Control samples did not include DTNB. After 30 min of incubation at room temperature, the absorbance at 412 nm was measured, and the amounts of trinitrobenzene (TNB) formed were calculated (equivalent to the amount of sulfhydryl (SH) groups) using the Aksenov technique (Aksenov and Markesbery 2001). The intra- and inter-assay CVs were 3.3% and 3.9%, respectively.

Enzyme antioxidant activities

Superoxide dismutase (SOD)

The SOD activity was determined in erythrocytes according to the method suggested by Bannister and Calabrese (1987). Specific activity was expressed as units per milligram ($U \cdot mg^{-1}$) of protein. One unit is estimated by 50% inhibition of adrenaline autooxidation read at 480 nm. The intra- and inter-assay CVs were 7.7% and 8.2%, respectively.

Catalase (CAT)

The SOD activity was determined in erythrocytes according to the method suggested by Aebi (1984). The samples were sonicated in 50 mmol·L^{-1} phosphate buffer, and the resulting suspension

Table 2. Perceived effort, relaxed flexed elbow joint angles, motor unit recruitment in concentric strength (RMS), and lactate dehydrogenase (LDH) activity in young university students provided taurine supplement (50 mg·kg body mass^{-1}·day^{-1}) or placebo before and after muscle damage induced by eccentric exercise (Pre-EE and Post-EE, respectively).

Parameter	Group	Pre-EE		Post-EE			
		Day 1	Day 14	Day 14	Day 16	Day 18	Day 21
Perceived effort (cm)	Placebo	3.6±0.6	3.4±0.5	9.8±0.3*	8±0.3*	7.5±0.6*	4.2±0.8
	Taurine	3.5±0.5	3.8±0.4	9±0.4*	7.6±0.7*	6.2±0.7*	3.6±0.5
Relaxed flexed elbow joint angles (°)	Placebo	139±2	141±0.6	114±2*	128±2*	132±1*	139±1
	Taurine	140±1	139±2	118±3*	130±2*	136±2*	140±3
RMS	Placebo	475±73	629±65	1192±113*	716±42	565±45	536±61
	Taurine	578±50	610±84	1167±165*	877±86*#	763±69#	694±57#
LDH activity (U·L^{-1})	Placebo	24±5.3	29±3.5		82±2*	96±13*	39±4
	Taurine	25±4	20±3		44±4#	56±9#	24±6

Note: The differences in relation to pre-EE (*) and placebo group (#) were significant at $p < 0.05$. Values are presented as means ± SEM.

was centrifuged at 3000*g* for 10 min. The supernatant was used for the enzyme assay. CAT activity was measured by the rate of decrease in hydrogen peroxide (10 mmol·L^{-1}) absorbance at 240 nm. The intra- and inter-assay CVs were 6.1% and 6.9%, respectively.

Glutatione peroxidase (GPx)

The GPx activity was measured in erythrocytes by monitoring the oxidation of nicotinamide adenine dinucleotide phosphate (NADPH) at 340 nm in the presence of H_2O_2 (Flohé and Günzler 1984). Enzyme activity was expressed as units per milligram (U·mg^{-1}) of protein. The intra- and inter-assay CVs were 3.3% and 4.4%, respectively.

Interleukins

Tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β), and interleukin-10 (IL-10) were determined by ELISA with commercially available kits (R&D Systems, Minneapolis, Minnesota). The CVs for these assays were 4.8% and 5.7%, respectively.

Protein determination

The quantity of proteins in xylenol orange, carbonyl, TT content, enzyme antioxidant, and interleukin assays was measured using the technique of Lowry et al. (1951). When the Folin phenol was added, the reagent bound to the protein. The bound reagent was slowly reduced and changed in color from yellow to blue. The absorbance was read at 700 nm.

Statistical treatment

Data are expressed as means ± standard errors of the mean (SEM). The Kolmogorov–Smirnov test was used to confirm normality. Data were analyzed using a 2 (group) × 6 (time) repeated-measures ANOVA, followed by the Bonferroni post-hoc test. The χ^2 test for nonparametric analyses was also used. The level of significance established for the test was $p < 0.05$. SPSS version 16.0 (IBM SPSS Software, Armonk, New York) was used.

Results

Participant characteristics

Body composition (placebo, 22.5 ± 2 kg·m^{-2}; taurine, 19.4 ± 3 kg·m^{-2}), strength level 1 RM (placebo, 36.2 ± 3 kg; taurine, 39.8 ± 4 kg), and repetition mean in each series (placebo, 10 ± 2; taurine, 11 ± 4) of EE were taken as average characteristics. There was no significant difference ($p > 0.05$) between groups in relation to body composition, 1 RM test, and average number of repetitions.

Dietary analysis

A 1-week dietary analysis (excluding supplementation) revealed the total energy, proteins, fats, and carbohydrate intake in both groups throughout the study (Table 1). There were no significant differences ($p > 0.05$) between groups.

Muscle performance assessment

Perceived effort, relaxed flexed elbow joint angles, motor unit recruitment in concentric strength, and lactate dehydrogenase (LDH) activity

As shown in Table 2, the perceived effort significantly increased in both groups immediately after EE ($p < 0.001$), on day 16 ($p < 0.01$), and on day 18 ($p < 0.05$) compared with the pre-exercise period. Relaxed flexed elbow joint angles (FANG) were significantly decreased in both groups immediately after EE ($p < 0.001$), on day 16 ($p < 0.05$), and day 18 ($p < 0.05$) compared with the pre-exercise period. Values of the motor unit recruitment for concentric strength during the recovery period in the taurine group were larger than those in the placebo group on day 16 ($p < 0.05$), day 18 ($p < 0.05$), and day 21 ($p < 0.05$). LDH activity was significantly increased in both subject groups on day 16 ($p < 0.05$) and day 18 ($p < 0.01$) after the EE in relation to the pre-exercise period. Values from the supplement group were lower than those from the placebo group on day 16 ($p < 0.01$) and day 18 ($p < 0.05$).

Concentric strength

Immediately after the EE, a significant decrease in concentric strength was observed in both groups. No differences were detected in relation to both groups. During the recovery period, values of the taurine group were higher than those of the placebo group on day 16 ($p < 0.05$), day 18 ($p < 0.01$), and day 21 ($p < 0.05$) (Fig. 2).

Isometric strength

Immediately after the EE, a significant decrease in the isometric strength was observed in both groups. During the recovery period, values of the taurine group were higher than those of the placebo group on day 16 ($p < 0.05$) and day 18 ($p < 0.05$) (Fig. 3).

Muscle soreness

We observed a significant increase in muscle soreness (MS) in both groups immediately after EE and on days 16 and 18 compared with the pre-exercise period. The values of the supplemented group were lower than those of the placebo group on day 16 ($p < 0.05$) and day 18 ($p < 0.01$) (Fig. 4).

Muscle damage

CK activity showed a significant increase in the placebo group on day 16 ($p < 0.001$), day 18 ($p < 0.001$), and day 21 ($p < 0.05$) compared with the pre-exercise period. The values of the supplemented group were lower than those of the placebo group on day 16 ($p < 0.001$) and day 18 ($p < 0.0001$) (Fig. 5).

Oxidative damage

As shown in Table 3, xylenol levels in the placebo group increased on day 16 ($p < 0.05$), day 18 ($p < 0.001$), and day 21 ($p < 0.01$) compared with the pre-exercise period. Values of the supplemented

Fig. 2. Concentric strength of young university students before eccentric exercise (Pre-EE), after EE (Post-EE), and on days 16, 18, and 21 after EE for subjects provided taurine supplement or placebo. The values are presented as means ± SEM, and the results are expressed in percentage (percentage of subjects that succeeded in performing the movement – 1 repetition maximum (RM)). Asterisk (*) indicates that the difference in relation to placebo group was significant at $p < 0.05$.
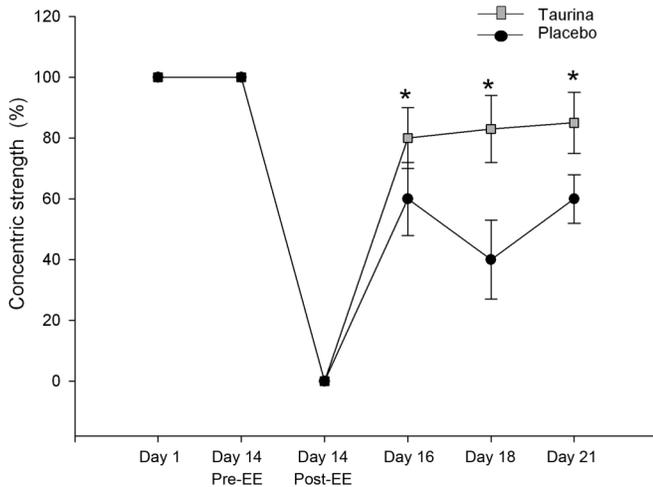

Fig. 3. Isometric strength of young university students before eccentric exercise (Pre-EE), after EE (Post-EE), and on days 16, 18, and 21 after EE for subjects provided taurine supplement or placebo. The values are presented as means ± SEM, and the results are expressed in kilograms. Asterisk (*) indicates that the difference in relation to placebo group was significant at $p < 0.05$.
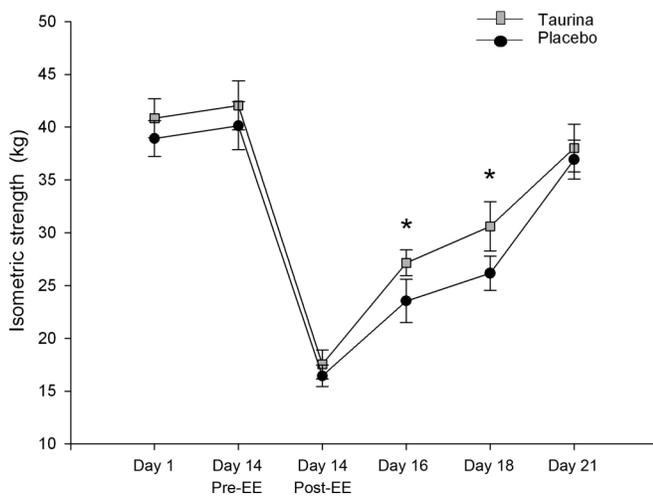
group were lower than those of the placebo group on day 16 ($p < 0.01$), day 18 ($p < 0.05$), and day 21 ($p < 0.05$). Carbonyl levels significantly increased in the placebo group on day 16 ($p < 0.01$), day 18 ($p < 0.01$), and day 21 ($p < 0.001$) compared with the pre-exercise period. Values of the supplemented group were lower than those of the placebo group on day 16 ($p < 0.05$), day 18 ($p < 0.05$), and day 21 ($p < 0.01$). The TT content significantly decreased in the placebo group on day 16 ($p < 0.01$), day 18 ($p < 0.001$), and day 21 ($p < 0.001$) compared with the pre-exercise period. Values of the supplemented group were higher than those of the placebo group on day 16 ($p < 0.01$), day 18 ($p < 0.05$), and day 21 ($p < 0.05$).

Fig. 4. Muscle soreness of young university students before eccentric exercise (Pre-EE), after EE (Post-EE), and on days 16, 18, and 21 after EE for subjects provided taurine supplement or placebo. The values are presented as means ± SEM, and the results are expressed in centimetres. Asterisk (*) indicates that the difference in relation to placebo group was significant at $p < 0.05$.
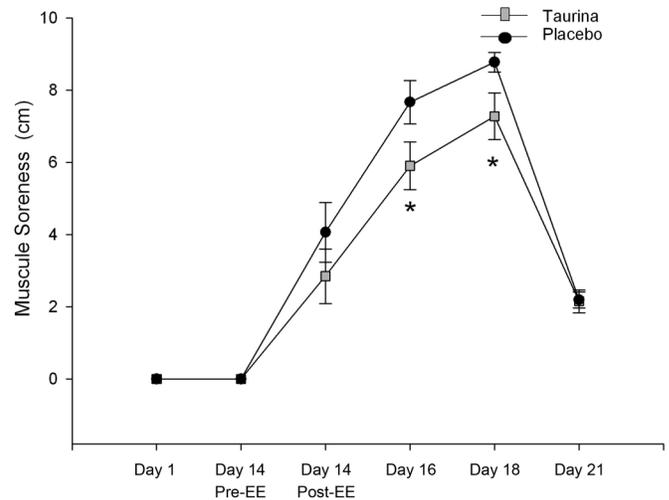

Fig. 5. Creatine kinase (CK) activity of young university students before eccentric exercise (Pre-EE), after EE (Post-EE), and on days 16, 18, and 21 after EE for subjects provided taurine supplement or placebo. The values are presented as means ± SEM, and the results are expressed in units per litre (U/L). Asterisk (*) indicates that the difference in relation to placebo group was significant at $p < 0.05$.
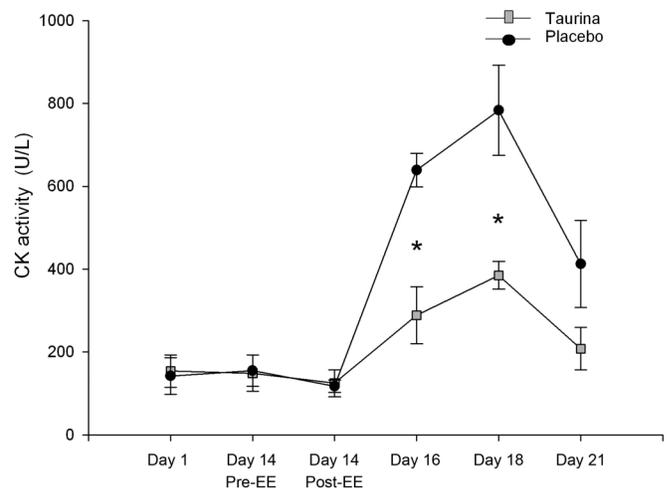
Antioxidant enzymes

As shown in Table 3, erythrocyte SOD activities increased in both groups on day 16 ($p < 0.05$), day 18 ($p < 0.01$), and day 21 ($p < 0.01$) compared with the pre-exercise period ($p < 0.05$). No differences were detected in relation to both groups. Erythrocyte CAT activity showed a significant increase in both groups on day 16 ($p < 0.001$) compared with the pre-exercise period. No differences were detected in relation to both groups. Erythrocyte GPx activity showed a significant increase in both groups on day 18 ($p < 0.001$) and day 21 ($p < 0.001$) compared with the pre-exercise period. No differences were detected in relation to both groups.

Interleukins

To quantify the inflammatory response, we measured TNF-α, IL-1β, and IL-10. The groups showed significantly increased TNF-α

Table 3. Oxidative stress parameters and inflammation in university students provided taurine supplement (50 mg·kg body mass^{-1}·day^{-1}) or placebo before and after muscle lesion induced by eccentric exercise (Pre-EE and Post-EE, respectively).

Parameter	Group	Pre-EE		Post-EE		
		Day 1	Day 14	Day 16	Day 18	Day 21
Xylenol orange (nmol·mg protein^{-1})	Placebo	0.24±0.08	0.34±0.04	2±0.4*	5.7±1.1*	3±0.2*
	Taurine	0.21±0.06	0.49±0.09	1±0.1#	3.7±0.9#	1.7±0.3#
Protein carbonylation (nmol·mg protein^{-1})	Placebo	0.97±0.12	0.73±0.14	3.63±0.43*	6.14±0.53*	10.2±0.73*
	Taurine	0.76±0.25	0.63±0.19	2.05±0.12#	3.79±0.68#	6.5±0.4#
Total thiol content (nmol·mg protein^{-1})	Placebo	31.3±2.2	30.9±1.6	14.4±0.7*	5.4±0.4*	3.8±0.6*
	Taurine	29.3±2.8	29.9±2	22.6±1.4*#	9.2±0.7#	6.5±0.5#
Superoxide dismutase (U·mg protein^{-1})	Placebo	1.24±0.09	1.29±0.16	2.3±0.25*	2.9±0.13*	3.1±0.13*
	Taurine	1.26±0.06	1.21±0.09	2.8±0.29*	3.17±0.15*	3.3±0.23*
Catalase (U·mg protein^{-1})	Placebo	2.43±0.41	2.3±0.21	6.1±0.3*	1.4±0.4	1.3±0.2
	Taurine	2.33±0.3	2.4±0.17	5±0.4*	1.5±0.3	1.2±0.5
Glutathione peroxidase (U·mg protein^{-1})	Placebo	0.23±0.01	0.27±0.03	0.11±0.02	0.6±0.07*	1.1±0.1*
	Taurine	0.26±0.02	0.23±0.02	0.14±0.03	0.67±0.03*	1.2±0.1*
Tumor necrosis factor-α (pg·mL^{-1})	Placebo	0.72±0.09	0.86±0.01	1.69±0.06*	1.61±0.087*	0.87±0.06
	Taurine	0.87±0.02	0.87±0.04	1.72±0.08*	1.57±0.06*	1.02±0.04
Interleukin-1β (pg·mL^{-1})	Placebo	0.41±0.01	0.29±0.03	0.64±0.05*	0.81±0.07*	0.11±0.02
	Taurine	0.32±0.02	0.22±0.05	0.79±0.03*	0.75±0.07*	0.22±0.03
Interleukin-10 (pg·mL^{-1})	Placebo	0.29±0.03	0.18±0.02	0.56±0.05*	0.6±0.07*	0.56±0.08*
	Taurine	0.23±0.01	0.22±0.01	0.55±0.06*	0.7±0.06*	0.72±0.07*

Note: The differences in relation to pre-EE (*) and placebo group (#) were significant at $p < 0.05$. The values are presented as means ± SEM.

and IL-1β on days 16 and 18 ($p < 0.01$) and significantly increased IL-10 concentrations on days 16, 18, and 21 (Table 3) compared with the pre-exercise period ($p < 0.01$). There were no differences between the groups.

Discussion

We investigated the effects of taurine supplementation on muscle performance, oxidative stress, and inflammatory parameters after EE. In this study, taurine supplementation was shown to increase concentric and isometric strength, muscle damage (soreness and CK), and oxidative stress (xylenol, PC, TT), but it altered neither antioxidant enzymes (SOD, CAT, and GPx) nor inflammatory response (TNF-α, IL-1β, and IL-10) induced by EE during the recovery period.

Evidence shows that taurine is essential for the normal function of skeletal muscles (Bakker and Berg 2002; Warskulat et al. 2004). In the present study, taurine supplementation increased the concentric and isometric strengths during the recovery period after exercise. Taurine has been shown to increase force production in skinned skeletal muscle fibers by increasing Ca^{2+} release from the sarcoplasmic reticulum (SR) and increasing the sensitivity of the contractile filaments to Ca^{2+} (Bakker and Berg 2002; Hamilton et al. 2006). It is possible that the improved strength during the recovery period is due to some taurine-induced factors such as increase in the mechanical threshold of skeletal muscle contractions (De Luca et al. 1996), intracellular membrane stabilization (Huxtable and Bressler 1973), and increased membrane polarization (Gruener et al. 1975; Conte-Camerino et al. 1987) induced by taurine.

EE generally induces the strength development of fatigue, which has detrimental effects on performance (Michaut et al. 2002; Hody et al. 2011). One of the main mechanisms thought to be responsible for fatigue is a decrease in the release of Ca^{2+} from the SR. This is likely to be due to adenosine triphosphate (ATP) utilization and accumulation of the metabolic by-products of ATP hydrolysis (Fryer et al. 1995; Westerblad and Allen 2002; Steele and Duke 2003). According to Bakker and Berg (2002), taurine may increase the size of depolarization-induced force responses by augmenting SR Ca^{2+} accumulation and release.

The generation of ROS during and after eccentric contractions has been attributed mainly to ischemia reperfusion and excessive calcium accumulation (Childs et al. 2001; Bloomer and Goldfarb 2004), which commonly produce muscle pain and injury (Shafat

et al. 2004; Bloomer and Goldfarb 2004; Goldfarb et al. 2005; Silva et al. 2008, 2010) for several days after exercise. Our results showed that taurine supplementation decreased muscle soreness and damage during the recovery period. According to Terada and Hara (2011), taurine supplementation attenuated different models of neuropathic pain, and these effects seem to be mediated by the activation of glycinergic neurotransmission. This may help explain the decrease in pain in the supplemented group. Calcium overload induced by EE during reperfusion (Bagchi et al. 1997; Lemasters et al. 1997) induces alterations in the mitochondrial function, enzyme release, and disruption of the cell membrane (Akizuki et al. 1985; Lemasters et al. 1997). It is possible that this effect is associated with the capacity of taurine to mediate cell protection through its membrane-stabilizing property (Zhang et al. 2004; Gupta 2006; Albrecht and Schousboe 2005), reducing the amount of CK.

Although several studies have reported increased oxidative stress following EE (Bryer and Goldfarb 2001; Childs et al. 2001; Lee et al. 2002; Bloomer et al. 2007; Silva et al. 2008, 2010), our results also show that taurine supplementation partly prevents any increase in lipoperoxidation (xylenol orange) and carbonyl protein levels and decrease in TT content compared with the placebo. Increases in xylenol orange and carbonyl protein and decreases in TT content after EE may be attributed to invasion of phagocytic cells into the damaged tissue, which typically occurs on postexercise days and can generate a substantial amount of ROS (Aksenov and Markesbery 2001; Goldfarb et al. 2005; Silva et al. 2011). Taurine is an effective free radical scavenger and normally is concentrated in cells and tissues that possess a considerable potential for producing oxidants (Huxtable 1992; Redmond et al. 1998; Oz et al. 1999; Miyamoto et al. 2009). Previous studies in our laboratory showed that taurine affects skeletal muscle contractions by decreasing oxidative stress, as well as decreasing the production of superoxide radicals (Silva et al. 2011).

In fact, antioxidant defense mechanisms are responsible for scavenging ROS to prevent oxidative stress. SOD, CAT, and GPx are the key antioxidant molecules in the defense against oxidative stress. SOD detoxifies superoxide anions to the less toxic hydrogen peroxide, which is further reduced by CAT and GPx (Halliwell and Gutteridge 2007). Previous studies showed either a decrease or increase or no change in the activities of these enzymes in the blood after several physical exercises (Kerksick et al. 2008; Zembron-Lacny et al. 2006; Vesovic et al. 2004). Our results

showed increased SOD, CAT, and GPx in both groups after EE. In fact, EE increases ROS production (Close et al. 2004), especially the production of the superoxide anion (Silva et al. 2011), which causes a cascade effect that activates the antioxidant enzymes CAT and GPx. Increased CAT and GPx activities may be dependent on scavenger cell migration into the damaged muscle tissue (Miyamoto et al. 2003). Macrophage migration into damaged muscle fibers has been observed following exercise (Uchiyama et al. 2006). The changes in the activities of these enzymes probably occurred due, in part, to an increase in mRNA levels (Lambertucci et al. 2007). According to Franco et al. (1999), the induction of antioxidant enzyme mRNA levels coincides with increases in oxidative damage of proteins, supporting the postulated relationship between oxidative stress and antioxidant enzyme mRNA expression. It is possible that the EE increases H_2O_2 production (after 2 days), thus exceeding the ability of GPx to neutralize it. Therefore, CAT production would be expected to increase in response to EE as a mechanism for compensating for the inability of GPx to scavenge H_2O_2 in times of higher demand.

The cytokine response to EE has been examined in several studies (Nosaka and Clarkson 1996; Cannon et al. 1991; Beaton et al. 2002; Phillips et al. 2003; Mahoney et al. 2008; Afroundeh et al. 2010). Cytokines play a central role in the control of the immune response, acute inflammatory response, and tissue repair process (Rhind et al. 1995; Petersen and Pedersen 2005; Chiang et al. 2009). Our results showed an increase in IL-1β, TNF, and IL-10 during the recovery period after EE in both groups (7 days). It has been reported that the pro-inflammatory cytokines IL-1β and TNF-α are detected in skeletal muscle after exercise (Cannon and St Pierre 1998; Fielding et al. 1993; Hamada et al. 2005) and that they play a role in initiating the breakdown of damaged muscle tissues (Cannon and St Pierre 1998). Previous studies in our laboratory (Silva et al. 2008, 2010) showed increased TNF-α and IL-10 after EE. IL-10 is a primary anti-inflammatory cytokine that inhibits pro-inflammatory cytokine production by activating monocytes and macrophages. In fact, it is rather likely that there are multiple stimuli for cytokine production during exercise (Afroundeh et al. 2010). However, taurine supplementation did not alter the results. It has been suggested that taurine is involved in several molecular mechanisms of muscle damage repair and oxidative stress: (i) inhibition of ischemia-induced activation of p53 (Takahashi et al. 2003); (ii) elevation of expression of the anti-apoptotic protein and decrease in the pro-apoptotic protein (Takahashi et al. 2003); and (iii) inhibition of caspase-3 activation (Takatani et al. 2004a, 2004b). We believe that these mechanisms may help explain the decreased muscle damage and oxidative stress, irrespective of nonreduction of inflammation.

In conclusion, we have demonstrated that taurine supplementation improved performance, muscle damage, and oxidative stress, but not inflammation, during the recovery period after EE.

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