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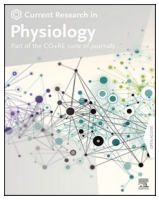

Effects of aerobic exercise during recovery from eccentric contraction on muscular performance, oxidative stress and inflammation

Luciano A. da Silva [a,b,d], Daniel Boeira [d], Ramiro Doeynart [d], Willians C. Longen [b,e], Luis Felipe Marqueze [c], Paulo C.L. Silveira [b], Anand Thirupathi [a,e,*], Yaodong Gu [a,e], Ricardo A. Pinho [c,e]

[a] Research Academy of Medicine Combining Sports, Ningbo No 2 Hospital, Ningbo, China
[b] Laboratory of Exercise Biochemistry and Physiology, Graduate Programme in Health Sciences, Health Sciences Unit, Universidade do Extremo Sul Catarinense, Criciúma, Brazil
[c] Graduate Program in Health Sciences, School of Medicine and Life Sciences, Pontifícia Universidade Católica do Paraná, Curitiba, Brazil
[d] Laboratory of Exercise Psychophysiology, Advanced Aquatic Exercise Research Group/Extremo Sul Catarinense, Criciúma, Brazil
[e] Faculty of Sports Sciences, Ningbo University, Ningbo, China

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ABSTRACT

This study investigated the effects of aerobic exercise during recovery from eccentric contraction (EC) on muscular performance, oxidative stress, and inflammation. Nineteen male subjects between 18 and 29 years were divided into unexercised (control, $n = 9$) and exercised ($n = 10$) groups. Initially, the subjects performed EC as 3 sets until exhaustion with elbow flexion and extension on the Scott bench at 80% in 1RM, followed by four aerobic exercise sessions. The results obtained indicated ($p > 0.05$) that aerobic physical exercise during the recovery period does not improve muscle performance (isometric strength and muscular fatigue), oxidative stress parameters (lipid peroxidation, protein oxidation and antioxidant enzyme activity), and inflammatory cytokines (IL-1β, TNF-α, IL-10). In conclusion, the aerobic exercise during the recovery period does not alter the parameters of performance, oxidative stress and inflammation induced by the EC.

1. Introduction

Eccentric muscle contractions result in myofibrillar disruption, proteolytic breakdown, mitochondrial apoptosis, inflammation (Silva et al., 2014; Theodorou et al., 2011), and increase in reactive oxygen species (ROS) production in the blood and muscles (Childs et al., 2001). Consequently, eccentric contractions (EC) induce redox status alterations and muscle function impairment, resulting in decreased performance, oxidative stress, and inflammation (Silva et al., 2010, 2014; Theodorou et al., 2011). Our previous studies have shown that reduced muscle performance, increased oxidative damage, and inflammation during the recovery period up to seven days after muscle injury caused by eccentric exercises (Silva et al., 2010, 2014). In contrast, other studies have reported that aerobic exercise not preceded by EC decreases oxidative stress and inflammation in several settings (Petersen and Pedersen, 2005; Jessen and Goodyear, 2005).

Rapid muscle recovery in the sports environment has been the topic of numerous studies (Bieuzen et al., 2012; Ispirlidis et al., 2008). Skeletal muscle of the athletes has the remarkable capacity to regenerate after injury. In response to injuries caused by EC, satellite cells are activated by inflammatory responses, proliferating within the muscle fibers, forming new myofibrils or repairing damaged ones through the process of cell differentiation (Koulmann et al., 2017).".

Some studies suggest that running exercises immediately after a muscle injury can improve metabolic parameters, accelerating the recovery process (Koulmann et al., 2017; Nikolaidis et al., 2007). In fact, low-intensity aerobic exercise training can activate the mitochondrial biogenesis, antioxidant system, and anti-inflammatory cytokines, helping to speed up the recovery process.

Recovery modalities have largely been investigated with regard to their ability to reduce the severity and duration of eccentric exercise-induced muscle injury and delayed onset muscle soreness (DOMS) (Barnett, 2006). DOMS involves an acute inflammatory response with edema formation (Chen et al., 2005). It is known that the

* Corresponding author. Faculty of Sports Science, Ningbo University, China.
E-mail address: ananthzeal@gmail.com (A. Thirupathi).

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anti-inflammatory and oxidative defense response is systemic, and it is produced in various organs and tissues and migrates to the injured muscle tissue to repair it.

However, the utility of aerobic exercise as a therapeutic tool to normalize biochemical and functional changes induced by EC, has not been adequately addressed. Thus, the aim of the present study was to determine the effect of aerobic exercise on muscle performance, oxidative stress, and inflammation during the recovery period after EC. We hypothesize that aerobic exercise during the recovery period after EC can accelerate the recovery process by reducing oxidative stress and inflammation and consequently improving performance.

2. Materials and methods

2.1. Study design

This randomized study was conducted for a period of 21 days and involved a comparison between an unexercised group (control) and an exercised group. Four sessions of aerobic exercise were carried out after the subjects performed EC as 3 sets until exhaustion (Fig. 1). Blood collection and performance tests were performed at the beginning (1st day) of the experiment, as well as on the day (14th day) immediately before and after EC, and aerobic exercise sessions (14th, 15th, 17th, 20th day) as shown below. Muscular performance, oxidative stress, and inflammation were examined as lesion indicators in the EC (Silva et al., 2010, 2014).

2.2. Subjects

Nineteen male volunteers, students at the Universidade do Extremo Sul Catarinense (Unesc), Criciúma, Santa Catarina, Brazil, took part in this randomized study and were assigned to either unexercised group (control, $n = 9$) or exercised group ($n = 10$) group. The tool (IPAP) was used to check the aptitude level of the suggestions and verify that they were sedentary (Matsudo et al., 2001). The subject's characteristics can be observed in Table 1. After the purpose and risks of the protocol had been explained to each subject, oral and written informed consent was obtained from them. The study was approved by the Ethics Committee of the Universidad do Extreme Sul Catarina's (CAAE: 8254248422.8.000.0229) and carried out in accordance with Resolution 466/12 of the National Health Council (CNS).

2.3. Exclusion criteria

All subjects were non-smokers, were not taking antioxidants 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 muscle lesions and were not carriers of any disease that might compromise the results or be aggravated by physical exercise.

Table 1
Basal characteristics of subjects.

Variables	Groups	
	Unexercised	Exercised
Age (yr)	24 (18–29)	23 (19–27)
Weight (kg)	74.2 (69.3–86.6)	77.8 (70.4–88.2)
Height (cm)	177.2 (165–185.3)	176.4 (167.1–189.3)
Body Mass Index (kg/m^2)	22.5 (19.7–26.1)	21.4 (18.9–24.8)
1 Repetition Maximum (kg)	33 (28–36)	35.5 (31–38)
Repetitions (average number)	10.3 (8–13)	11.5 (9–13)
Basal Heart Rate (bpm)	65.2 (61–71)	68.3 (59–68)
Basal Blood Pressure (Systolic, mm Hg)	127.6 (122-135	128.3 (126–138)
Basal Blood Pressure (diastolic, mm Hg)	82.1 (77–84)	78.6 (74–81)

Legends: All values are means ± SEMs. There were no significant differences between the Unexercised and exercise groups (The x^2 test for nonparametric analyses was also used).

2.4. Blood collection

Eight millilitre samples of blood were drawn from the antecubital vein on the 1st, 14th (before EE), 16th, 18th and 21st days (after EE) of the experiment. Blood was collected in vacutainers without additives and centrifuged at 1500 rpm for 10 min at 4 °C. Aliquots of washed/lysed red blood cells and serum samples were stored at −70 °C until biochemical assays were performed.

2.5. One-repetition maximum test

The performance test required flexion and extension of elbows supported by the Scott bench (3 sets with 5 min rest between each set). One-repetition maximum (1 RM) of each subject was assessed (Bompa, 2001). Before testing, a standardized warm-up consisting of a 10-min ride on a bicycle ergometer was employed, and 20 repetitions of elbow flexion and extension on the Scott bench, without load, using moderate cadence (2 s). The weights lifted in the 1 RM test for each group are described in Table 1. After 1 RM testing, all participants were instructed to follow their normal eating pattern and to refrain from strenuous physical exercise for 14 days before and 7 days after EC.

2.6. Eccentric contractions protocol

Eccentric contractions was performed with elbow flexion and extension on the Scott bench (equipment used in the muscle activity assessment) at an intensity of 80% of 1RM (Silva et al., 2014). The concentric phase of the exercise was performed with the manual assistance of the instructor. The eccentric phase was performed for 6–8 s. Exhaustion was defined by the participant's inability to perform the EC

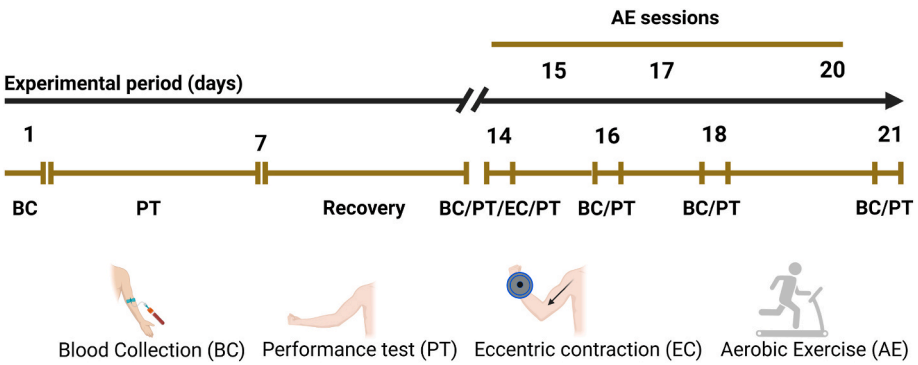

Fig. 1. *Study design*: Legends: The black lines indicate the total time of the study and the red lines indicate the interventions. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

within the pre-established time (6 s). Three exercise sets were performed with 2-min intervals until exhaustion. Subjects were familiarised and adapted to the Scott bench (flexion and extension movements of elbows) 7 days before EE (3 sets, 15 repetitions with 2 min rest between sets) with a 2-kg load.

2.7. Aerobic physical exercise

This exercise was conducted as a run drive with no inclination for 30 min (Barnett, 2006). Four sessions of aerobic exercises were performed after EC (on day 14, immediately after EE, and on days 15, 17 and 20). Heart rate and blood pressure of subjects were measured, and the aerobic exercise was carried out in low intensity (50—60% reserve heart rate) (date not shown). All individuals ran at a speed between 6 km/h and 9 km/h.

2.8. 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 4s.

The muscle fatigue was measured according to (Maton, 1981) using the duration of the period during which an individual could sustain 70% of the maximum isometric load at an angle between 80° and 100°. The exhaustion was determined by the volunteer's own reference or by verifying the volunteer's impossibility to maintain the load stipulated inside of the variation of ±10%.

2.9. Muscle soreness and damage

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 analogue scale (VAS), the left and right extremes of which refer to "no muscle soreness'' and "maximum muscle soreness,'' respectively.

Creatine kinase (CK) activity was used as a marker of muscle damage. A kit, supplied by Labtest Diagnóstica SA, Brazil, was used to determine MM-CK levels using coupled enzyme reaction in serum samples. The intra- and inter-assay coefficients of variation (CVs) were 5.9% and 7.1%, respectively. All test samples were made in duplicates.

2.10. Oxidative damage

This method detects hydroperoxides (ROOHs) which are products of lipid peroxidation (Jiang et al., 1991). The xylenol orange assay is based on the oxidation of ferrous ions to ferric ions by ROOHs under acidic conditions. Absorbance of the supernatant was measured at 560 nm using an Ultraspec 2000 spectrophotometer (Pharmacia Biotech, Uppsala, Sweden). The intra- and inter-assay CVs were 5.5% and 6.5%, respectively. All test samples were made in duplicates.

This method detects carbonyl groups based on the reaction with 2,4-dinitrophenylhydrazin (Levine et al., 1990). Proteins were precipitated by adding 20% trichloroacetic acid and incubated with 2,4-dinitrophenylhydrazine. The intra- and inter-assay CVs were 3.1% and 4.2%, respectively. All test samples were made in duplicates.

The total thiol content was determined using the 5,5′ -dithiobis-(2-nitrobenzoic acid) method (DTNB) (Ellman, 1959) (Sigma). After 30 min of incubation at room temperature, absorbance at 412 nm was measured and the amount of TNB formed (equivalent to the amount of SH groups) was calculated. The intra- and inter-assay CVs were 4.4% and 5.4%, respectively. All test samples were made in duplicates.

2.11. Antioxidant enzymes activities

Superoxide dismutase (SOD) activity was determined according to the method described by (McCord and Fridovich, 1969). Enzymatic activity was estimated by adrenaline auto-oxidation inhibition, read at 480 nm in a spectrophotometer. Enzyme activity was expressed as $U{\cdot}mg^{-1}$ protein. The intra- and inter-assay CVs were 8.1% and 9.3%, respectively. All test samples were made in duplicates.

The activity of CAT was determined in erythrocytes according to the method of (Aebi, 1984). CAT activity was measured by the rate of decrease in hydrogen peroxide (10 mM) absorbance at 240 nm and expressed as $U{\cdot}mg^{-1}$ protein. The intra- and inter-assay CVs were 5.5% and 7.3%, respectively. All test samples were made in duplicates.

Glutathione peroxidase (GPx) activity was measured by monitoring the oxidation of NADPH at 340 nm in the presence of H_2O_2 (Flohé and Günzler, 1984). Enzyme activity was expressed as $U{\cdot}mg^{-1}$ protein. The intra- and inter-assay CVs were 4.6% and 5.5%, respectively. All test samples were made in duplicates.

2.12. Cytokines

Tumour necrosis factor-α (TNF-α), interleukin 1β (IL-1β) and interleukin 10 (IL-10) were determined in by ELISA (R&D Systems, Minneapolis, MN, USA) using Cytokines kits (ml035385, ml059998, and ml135299, respectively; mlbio, BRAZIL). The procedures were performed according to the manufacturer's instructions. The intra- and inter-assay CVs were 4.3% and 4.9%, respectively. All test samples were made in duplicates.

2.13. Protein determination

The total protein quantity in xylenol orange, carbonyl, total thiol, antioxidant enzymes 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.

2.14. Statistical treatment

Data are expressed as the mean ± standard error of the mean (SEM). The Kolmogorov-Smirnov test was used to confirm normal distribution of values of analyzed parameters. Data were analyzed using a 2 (group) × 6 (time) repeated measures ANOVA, followed by the Bonferroni post-hoc test. The x^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.

3. Results

3.1. Isometric strength and muscular fatigue

With regards to isometric strength (Fig. 2A), a significant decrease was observed immediately after EE (19.5 ± 2.2 kgf) and on days 16 (28.6 ± 2.1 kgf), 18 (29.5 ± 2.5 kgf) and 21 (30.2 ± 1.9 kgf) comparison to that observed in the pre-exercise period (34.1 ± 2.3 Kgf) ($P < 0.05$). Significantly higher muscle fatigue was observed on days 16 (1063 ± 149 rms) and 18 (1409 ± 13 rms) ($P < 0.01$) than that observed in pre-exercise (718.4 ± 73 rms) conditions (Fig. 2B). No differences in any variables were observed after aerobic exercise sessions ($P > 0.05$).

3.2. Muscle soreness and damage

We observed a significant increase in muscle soreness (MS) in both groups immediately after EE (3 ± 0.8 cm; 3 ± 0.9 cm) ($P < 0.05$) and on days 16 (5.7±1 cm; 5 ± 0.6 cm) ($P < 0.001$) and 18 (4±1 cm; 4.2 ± 0.7

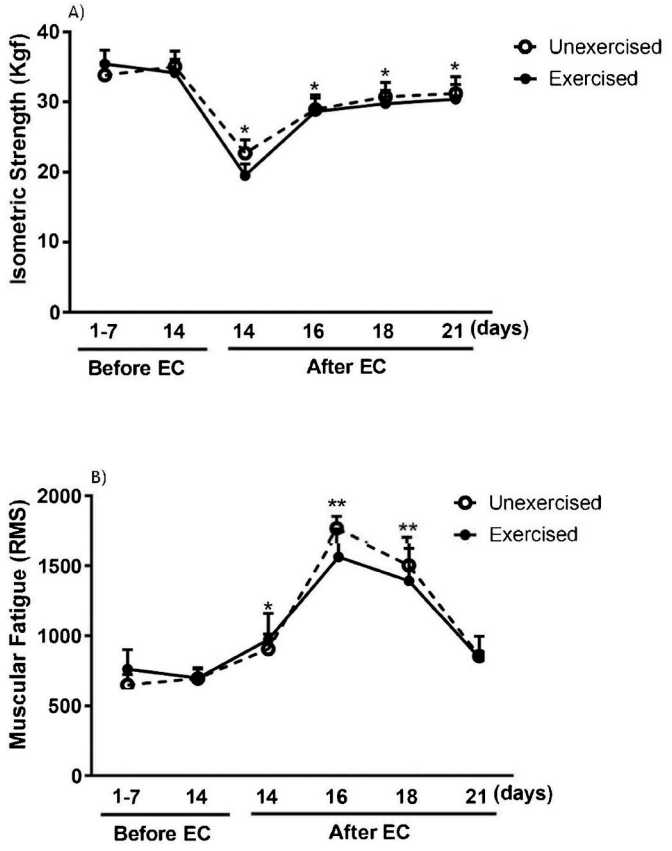

Fig. 2. (A–B): Muscle performance assessment of young university students before-EE, after-EC, 16th, 18th, and 21st days after the EC for subjects provided exercise ($n = 10$) or unexercised ($n = 9$). The values are presented as mean ± SEM. *P < 0.05 or **P < 0.01 were considered to establish a statistically significant difference between groups from ANOVA, Bonferroni's post hoc test. The x^2 test for nonparametric analyses was also used.

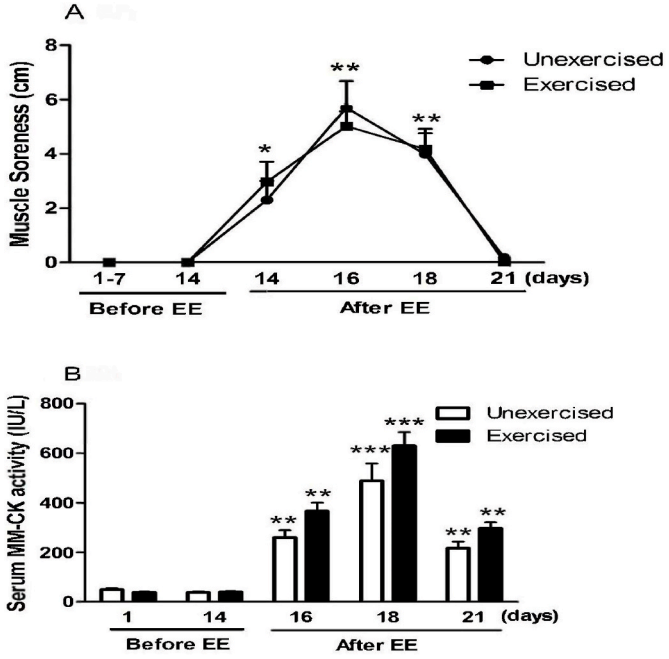

Fig. 3. (A–B): Muscle Soreness (MS, Fig. 3A) and creatine kinase activity (MM-CK, Fig. 3B) activity of young university student's before-EC, after-EC, 16°, 18°, and 21° days after the EC for subjects provided exercise ($n = 10$) or unexercised ($n = 9$). The values are presented as mean ± SEM and the results expressed in cm (MS) and IU/L (MM-CK). *P < 0.05 or **P < 0.01 or ***P < 0.001 were considered to establish a statistically significant difference between groups from ANOVA, Bonferroni's post hoc test. The x^2 test for nonparametric analyses was also used.

cm) ($P < 0.01$) compared to baseline conditions. However, no significant differences ($P > 0.05$) in any parameters were detected after aerobic exercise sessions (Fig. 3A). MM-CK activity was significantly enhanced in both groups on days 16 (267 ± 54 u·L^{-1}; 419 ± 63u·L^{-1}) ($P < 0.01$), 18 (518 ± 68u·L^{-1} 601 ± 61u·L^{-1} ($P < 0.001$), and 21 (232.8 ± 29u·L^{-1}; 293.2 ± 30u·L^{-1} ($P < 0.01$) in relation to baseline parameters (47±8u·L^{-1}; 36±6u·L^{-1}). Notably, MM-CK activity was not altered after aerobic exercise sessions (Fig. 3B).

3.3. Oxidative damage

As shown in Fig. 4a, xylenol levels in both groups were increased on days 16 (1.6 ± 0.8nnmol/mg protein; 1.5 ± 0.5 nnmol/mg protein), 18 (3.2 ± 0.4nnmol/mg protein; 3.1 ± 0.9nnmol/mg protein) and 21 (2.1 ± 0.8nnmol/mg protein; 2 ± 0.6nnmol/mg protein) ($P < 0.01$) compared to baseline conditions (0.3 ± 0.1nnmol/mg protein; 0.3 ± 0.2nnmol/mg protein). Carbonyl levels (Fig. 4b) were significantly augmented in both groups on days 16 (3.1 ± 0.3 nnmol/mg protein; 3.2 ± 0.2nnmol/mg protein), 18 (5 ± 0.7nnmol/mg protein; 5.5 ± 0.5nnmol/mg protein) ($P < 0.05$) and 21 (6.3 ± 0.9nnmol/mg protein; 6.7±1nnmol/mg protein) ($P < 0.01$) in relation to the pre-exercise period (1.8 ± 0.2nnmol/mg protein; 1.3 ± 0.1nnmol/mg protein), however no differences between the two groups were detected ($P > 0.05$). According to Fig. 4c, the total thiol content significantly decreased in both groups on days 16(16.3±1nnmol/mg protein; 15 ±1nnmol/mg protein), 18(9.9±2nnmol/mg protein; 12.2 ± 0.4nnmol/mg protein) and 21 (7.3 ± 0.9nnmol/mg protein; 8.6 ± 0.8nnmol/mg protein) ($P < 0.01$) in comparison to the pre-exercise period (28.6 ±2nnmol/mg protein; 30.2±3nnmol/mg protein). No differences in any variables were observed after aerobic exercise sessions ($P > 0.05$).

3.4. Antioxidant enzyme activity

Fig. 5A shows increased SOD activity in both groups on days 16 (2.42 ± 0.2Units of SOD/mg protein; 2.47 ± 0.4 Units of SOD/mg protein), 18 (2.33 ± 0.1 Units of SOD/mg protein; 2.41 ± 0.9 Units of SOD/mg protein) and 21 (2.99 ± 0.3Units of SOD/mg protein; 3.22 ± 0.3Units of SOD/mg protein) ($P < 0.05$). CAT activity (Fig. 5B) was also significantly higher in both groups but only on day 16 (6.1±1Units of CAT/mg protein; 7.2 ± 1 Units of CAT/mg protein) ($P < 0.001$) as than in the pre-exercise period (2.47 ± 0.2Units of CAT/mg protein; 2.5 ± 0.1Units of CAT/mg protein). GPx activity was up regulated in both groups on days 18 (1.17 ± 0.2Units of GPx/mg protein; 1.38 ± 0.2Units of GPx/mg protein) and 21 (1.43 ± 0.2 Units of GPX/mg protein; 1.56± Units of GPX/mg protein) ($P < 0.01$) (Fig. 5C). No differences in any parameters were observed after aerobic exercise sessions.

3.5. Cytokines

Both groups had significantly increased TNF-α (Fig. 6a) and IL-1β levels (Fig. 6b) on days 16 (1.14 ± 0.1 pg mL^{-1};1.17 ± 0.2 pg mL^{-1}; 0.04 ± 0.002 pg mL^{-1}; 0.04 ± 0.01 pg mL^{-1}) and 18 (1.4 ± 0.1 pg mL^{-1}; 1.3 ± 0.1 pg mL^{-1}; 0.05 ± 0.003 pg mL^{-1}; 0.04 ± 0.005 pg mL^{-1}), while IL-10 concentrations (Fig. 6c) were augmented on days 16 (0.55 ± 0.05 pg mL^{-1}; 0.44 ± 0.04 pg mL^{-1}), 18 (0.6 ± 0.004 pg mL^{-1}; 0.05 ± 0.03 pg mL^{-1}) and 21 (0.55 ± 0.01 pg mL^{-1}; 0.43 ± 0.03 pg mL^{-1}) after EE, as compared to the pre-exercise period (0.25 ± 0.02 pg mL^{-1}; 0.21 ± 0.04 pg mL^{-1}) ($P < 0.05$). As with other parameters, no significant differences in any variables were observed after aerobic exercise sessions.

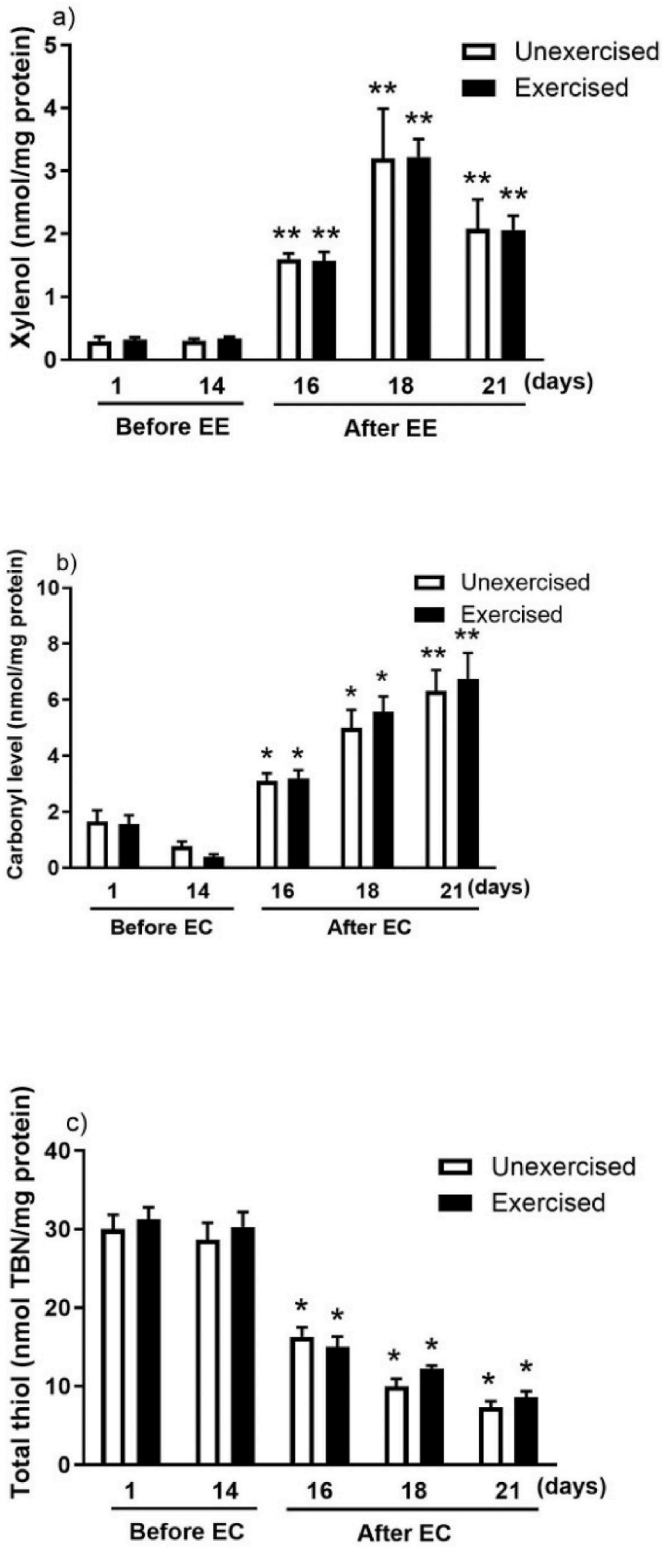

Fig. 4. (a–c): Damage oxidative in lipids (xylenol content, 4a), protein carbonylation (carbonyl content, 4b), and protein modification (total thiol, 4c) of young university students before before-EE, after-EC, 16th, 18th, and 21st days after the EC for subjects provided exercise ($n = 10$) or unexercised ($n = 9$). The values are presented as mean ± SEM, and the results are expressed in nmol/mg/protein. *P < 0.05 or **P < 0.01 were considered to establish a statistically significant difference between groups from ANOVA and Bonferroni's post hoc test. The x^2 test for nonparametric analyses was also used.

4. Discussion

In this study, the effect of aerobic physical exercise on parameters of muscle performance, oxidative stress, and inflammation during recovery after EC was examined. It is known that the anti-inflammatory and oxidative defense response is systemic. This is produced in various organs and tissues and migrates to the injured muscle tissue to repair it. We hypothesize that low-intensity aerobic exercise can activate the anti-inflammatory and antioxidant system, helping to speed up the recovery process. The results obtained indicated that aerobic physical exercise during the recovery period does not improve muscle performance as measured by isometric strength and fails to affect muscle fatigue. Muscle damage, gauged by muscle soreness scores and CK activity, oxidative stress parameters (lipid peroxidation, protein oxidation and antioxidant enzyme activity), and inflammatory cytokines (IL-1β, TNF-α, IL-10) were not altered by aerobic physical exercise.

It has been demonstrated that a period of active recovery, as opposed to passive recovery, improved muscle recovery (Ahmaidi et al., 1996; Sairyo et al., 2003). We observed a reduction in isometric strength and enhanced muscle fatigue (Fig. 2A and B) similarly in both groups during the recovery period after EC. These results corroborate a previous study (Nosaka et al., 2002), which showed a reduction of isometric strength within a similar period after EC. These results may suggest that seven days of rest was not enough time to allow complete recovery of muscle strength and fatigue following EC, resulting in muscle damage. The addition of aerobic exercise during the rest period did not accelerate physiological recovery.

It is a fact that during eccentric exercises muscle microlesions occur (Chen et al., 2005). Biomechanically, in these exercises, there is a phase called eccentric contraction. During this phase, muscle sarcomeres stretch and contract at the same time. This generates greater tension on the muscle fiber, causing myofibral ruptures that generate pain, edema, and reduced force (Chen et al., 2007; Martin et al., 2004). Muscle injury induced by EC causes muscle soreness and increased CK activity in the plasma (Silva et al., 2010, 2014; Theodorou et al., 2011). Our results show no effect of aerobic exercise on muscle soreness or CK activity (Fig. 3A and B) during the recovery process. Enhanced muscle soreness and CK activity on days 2 and 4 after EC have been reported (Lee et al., 2002). Another study (Stupka et al., 2000) showed an increase in CK activity levels for as long as 6 days after EC. Mixed findings have been reported in the literature, depending on the recovery strategy investigated and its localization (Duffield et al., 2010; Gill et al., 2006).

An imbalance between oxidative stress and antioxidant capacity in the blood induced by eccentric exercise has been postulated by many studies. (Silva et al., 2010, 2014). An increase in oxidative damage (Fig. 4A–C) and antioxidant enzyme activity (Fig. 5A–C) in both experimental groups during the recovery period after EC was found in this study. This is caused by oxidative stress, which results from the protocol of EC used in the present study, as reported in previous studies. (Childs et al., 2001; Silva et al., 2008). Repeated EC during the recovery period have been shown to be reduced by oxidative stress according to previous studies (Nikolaidis et al., 2007). This phenomenon was referred to as the repeated effect. However, aerobic exercise during the recovery period had no effect on these markers. Our results could not bear this notion. In our opinion, the differences between our findings and results from the last study can be explained by the different models of exercises (localized vs. not located).

Eccentric contraction induces an increase in the number of cytokines, such as TNF-α, interleukin IL-1β, and IL-10, for several days (Silva et al., 2010; Petersen and Pedersen, 2005). We report an increase in levels of TNF-α, IL-1β, and IL-10 during the recovery period in both groups after EC (Fig. 6a-c), and aerobic exercise did not change these results. The recovery metabolic properties are an important part of the muscular regeneration process. During cellular prolifiration in differentiation requires stimulation of mitochondrial biogenesis (Herzberg et al; Rochard et al). Mitochondrial biogenesis is one of the most striking responses

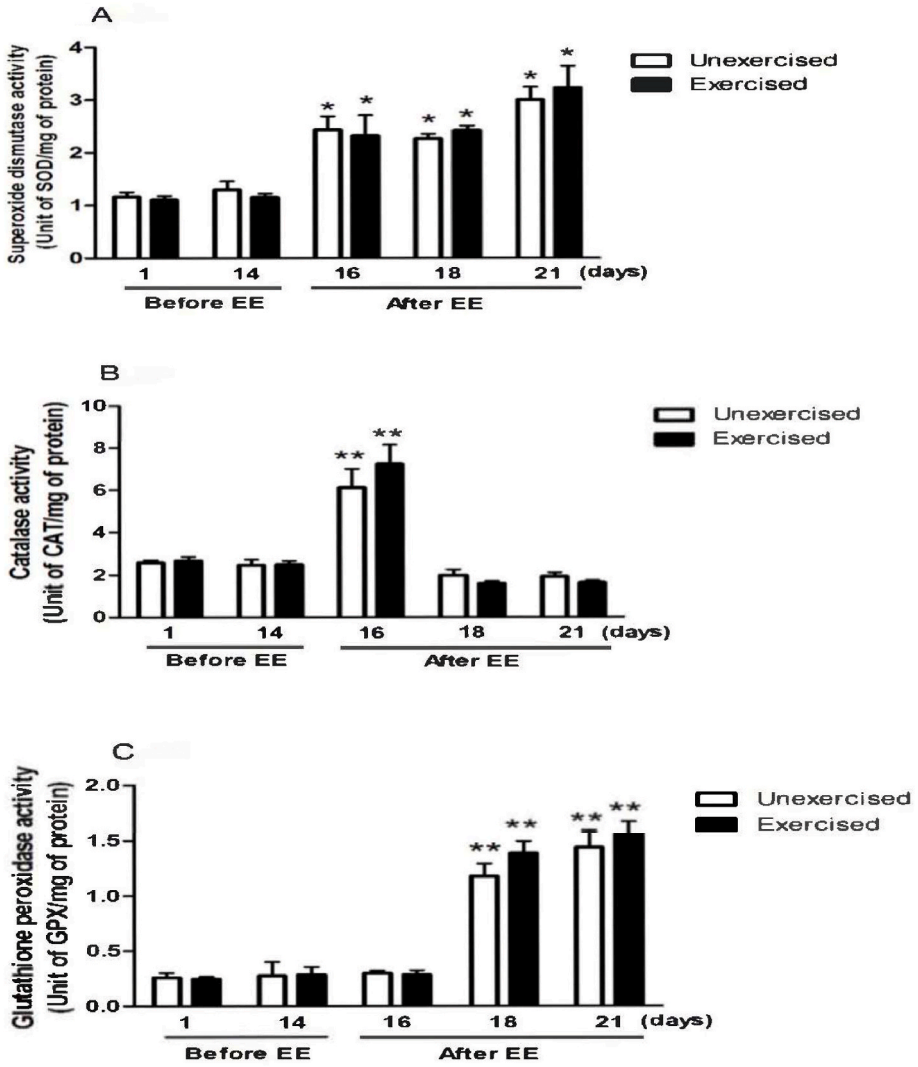

Fig. 5. (A–C): Superoxide dismutase (SOD, 5A), catalase (CAT, 5B) and glutathione peroxidase (GPX, 5C) activities of young university students before-EC, after-EC, 16°, 18°, and 21° days after the EC for subjects provided exercise ($n = 10$) or unexercised ($n = 9$). The values are presented as mean ± SEM, and the results are expressed in Units/mg/protein. *P < 0.05 or **P < 0.01 were considered to establish a statistically significant difference between groups from ANOVA and Bonferroni's post hoc test. The x^2 test for nonparametric analyses was also used.

observed in skeletal muscle after endurance training (Freyssenet et al). As aerobic exercise stimulates mitochondrial biogenesis, we hypothesized that increased contractile activity during muscle regeneration could also improve recovery and decrease inflammation. However, this did not occur. These results support the thesis that the adaptive response of the exercises performed during the recovery period is specific according to the model used in the injury protocol.

5. Conclusion

We suggest that using aerobic exercise (low-intensity running) during the recovery period has no effect on parameters of muscle performance, oxidative stress, and inflammation caused by localized EC. Contrary to our hypothesis, it is concluded that aerobic exercise does not improve performance or reduce oxidative stress and inflammation during the recovery period after muscle injury induced by EC exercise.

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CRediT authorship contribution statement

Luciano A. da Silva: Conceptualization, Writing – original draft. **Daniel Boeira:** Methodology, Data curation, Software, Validation. **Ramiro Doeynart:** Methodology, Data curation, Software, Validation.

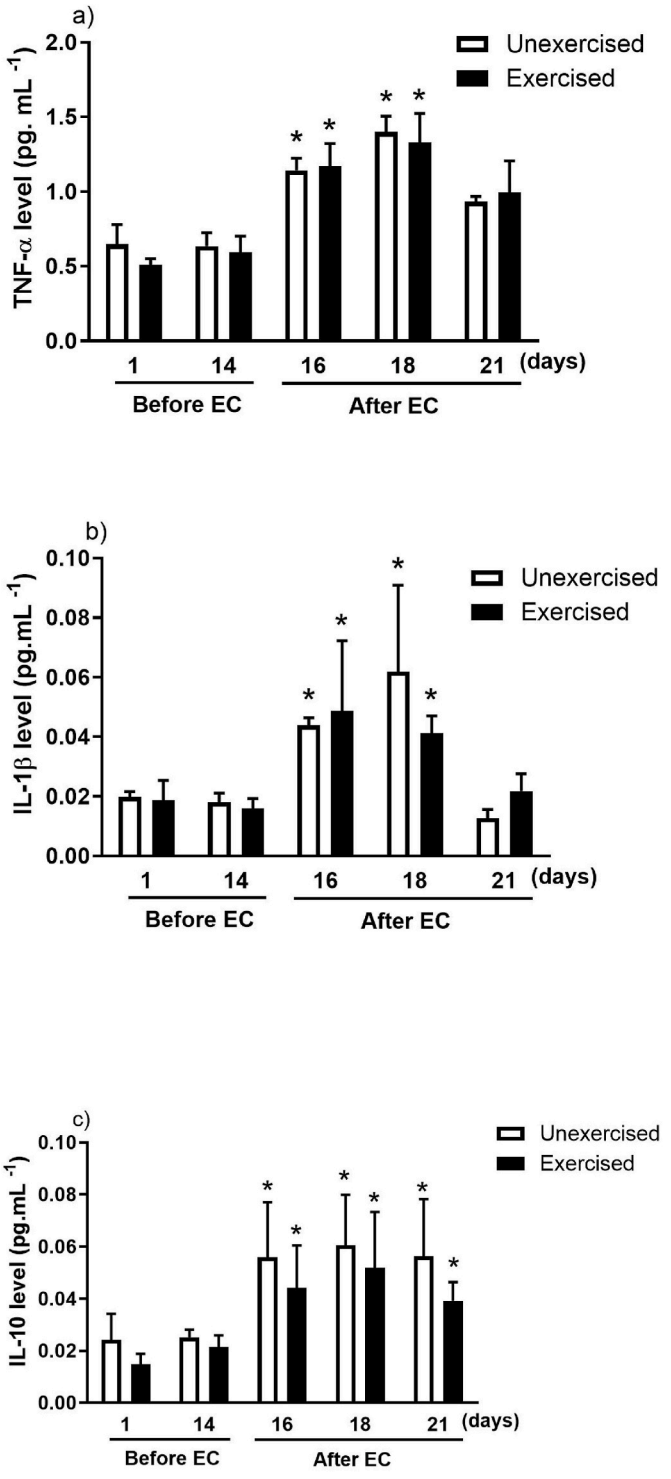

Fig. 6. (a–c): Tumor necrosis factor alpha (TNFα, 6a), interleukin 1β (IL-1β, 6b) and interleukin 10 (IL10, 6c) of young university students before-EC, after-EC 16°, 18°, and 21° days after the EC for subjects provided exercise ($n = 10$) or unexercised ($n = 9$). The values are presented as mean ± SEM, and the results are expressed in pg/ml. *P < 0.05 or **P < 0.01 were considered to establish a statistically significant difference between groups from ANOVA and Bonferroni's post hoc test. The x^2 test for nonparametric analyses was also used.

Willian C. Longen: Methodology, Data curation, Software, Validation. **Luiz Felpe Marqueze:** Methodology, Data curation, Software, Validation. **Paulo C.L. Silveira:** Writing – review & editing. **Anand Thirupathi:** Writing – review & editing. **Yaodong Gu:** Writing – review & editing. **Ricardo A. Pinho:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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