

EFFECT OF RUNNING HIGH-INTENSITY INTERVAL TRAINING TYPE OF RECOVERY ON MUSCLE INJURY AND OXIDATIVE STRESS MARKERS

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ABSTRACT

Introduction: High Intensity Interval Training (HIIT) is understood as vigorous activity, with different intensities, interspersed with periods passive or active recovery, which induces an acute physiological response. Two running HIITs were analyzed, and biochemical markers of muscle damage (MD) and oxidative stress (OS) were measured. **Materials and Methods:** 15 soldiers were submitted to anthropometric and VO₂ max assessments. Subsequently, they performed treadmill running HIITs: (a) moderate to strong intensity and active recovery (HIIT AR) and, (b) moderate to strong intensity with passive recovery (HIIT PR). Venous blood samples were collected pre and post-tests for analysis of MD (lactate, creatine kinase - CK and lactate dehydrogenase - LDH), and OS (lipid peroxidation- LP, carbonyl protein - PC, total antioxidant activity -TAA and total sulfhydryl groups - TSG). **Results:** Comparing MD at baseline x HIIT PR, it was observed an increase in CK, LDH and lactate. At baseline x HIIT AR, a significant increase in CK, LDH and lactate. Comparing HIIT RP x HIIT AR, only lactate was significantly affected by HIITs. Comparing OS at baseline x HIIT PR, there was a significant increase in LP and PC. Baseline x HIIT AR, there was a reduction only at TSG, and comparing HIIT PR x HIIT AR, there was a reduction in LP/VO₂, TSG and PC. **Conclusion:** Both protocols increased blood levels of MD, but comparing HIIT PR and HIIT AR, lactate and OS were higher at HIIT PR. It is noteworthy that HIIT AR is more effective in removing lactate and modulating redox metabolism.

Key words: High intensity interval training. Running. Biochemistry markers. Oxidative stress.

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RESUMO

Efeito da execução de treinamento intervalado de alta intensidade tipo de recuperação sobre lesão muscular e marcadores de estresse oxidativo

Introdução: Treinamento Intervalado de Alta Intensidade (HIIT) entende-se como uma atividade vigorosa, com diferentes intensidades, intercalada com períodos de recuperação passiva ou ativa, que induz resposta fisiológica aguda. Foram analisados dois HIITs de corrida e mensurados marcadores bioquímicos de dano muscular (MD) e estresse oxidativo (OS). **Materiais e Métodos:** 15 militares foram submetidos a avaliação antropométrica e VO₂ max. Posteriormente realizaram HIITs de corrida em esteira: (a) intensidade moderada a forte com recuperação ativa (HIIT AR) e, (b) intensidade moderada a forte com recuperação passiva (HIIT PR). Amostras de sangue venoso foram coletadas pré e pós-testes para análise de MD (lactato, creatina quinase - CK e lactato desidrogenase - LDH), e de OS (peroxidação lipídica - LP, proteína carbonil - PC, atividade antioxidante total - TAA e grupos sulfidríla total - TSG). **Resultados:** Comparando MD em repouso x HIIT PR, observou-se aumento de CK, LDH e lactato. Em repouso x HIIT AR, aumento significativo em CK, LDH e lactato. Comparando HIIT RP x HIIT AR, apenas o lactato foi significativamente afetado pelos HIITs. Comparando OS em repouso x HIIT PR, verificou-se aumento significativo de LP e PC. Em repouso x HIIT AR, constatou-se redução apenas no TSG, e comparando HIIT PR x HIIT AR, observou-se redução no LP/VO₂, TSG e PC. **Conclusão:** Os protocolos aumentaram os níveis sanguíneos de MD, mas comparando o HIIT PR e HIIT AR, o lactato e OS foram maiores no HIIT PR. Destaca-se maior eficácia do HIIT AR na remoção de lactato e modulação do metabolismo redox.

Palavras-chave: Treinamento intervalado de alta intensidade. Corrida. Marcadores bioquímicos. Estresse oxidativo.

INTRODUCTION

High intensity interval training (HIIT), involves short or long intensive effort periods (5 sec – 5 min) interspersed with active or passive recovery, that is often used in athlete training programs for individual and/or team sports (Gibala and Jones, 2013).

HIIT induces metabolic and performance changes that stand out in traditional aerobic training, allowing athletes to maintain intense effort during competition for long durations and also a faster recovery (Cicioni-Kolsky and collaborators, 2013; Iaia, Ermanno, Bangsbo, 2009).

The intensity of physical activities with high energy demand favors the increase of oxidizing agents, such as reactive oxygen species (ROS). When there is an imbalance between the pro-oxidants and the antioxidant systems, and the pro-oxidants are predominant, it was considered an oxidative stress (OS) condition (Sies, 1986).

OS has been associated with decreased physical performance, fatigue, muscle damage and overtraining. Moreover, there is close relationship between exercise intensity and changes in total antioxidant activity (TAA) (Parker, McGunkin, Leicht, 2014), as a reduction in EO can improve exercise tolerance and physical performance (Cebula and collaborators, 2017; Powers, Ji and Leeuwenburgh, 1999; Radák and collaborators, 1999; U.S. National Library of Medicine, 2014).

Intracellular changes in redox status are commonly associated with cell membrane damage and possible muscle tissue injuries (Steinbacher and Eckl, 2015). This process is accompanied by an inflammatory incidence signaled by the augmented release of muscle enzymes into the blood plasma, evident histological changes, and muscle pain (Dekkers, van Doornen, Kemper, 1996; Nosaka and Clarkson, 1995).

Amongst muscle enzymes, creatine kinase (CK) and lactate dehydrogenase (LDH) plasma levels illustrate the acute effect of exercise (Martins, 2010; Puggina and collaborators, 2016).

HIIT with active recovery performed after exercise has been shown to be more effective when compared to passive recovery (Bonen and Belcastro, 1976; Hermansen and Stensvold, 1972).

Exercise intensity is the main factor that determines the degree of muscle damage and OS (Braun and collaborators, 2007) as the type of recovery can influence the blood lactate concentration.

Thus, understanding the dynamics of expression of biochemical markers of muscle damage, OS and fatigue may help to understand the body's adjustments and adaptations to HIIT at different intensities and with different recovery strategies (Córdova, Navas, Lazzoli, 2000).

The aim of this study was to compare biochemical markers of muscle damage and OS in two HIIT running approaches.

MATERIALS AND METHODS

Study Design

Experimental research with sample size estimated by G*Power 3.1 software (Faul and collaborators, 2007). The following information was inserted: ANOVA for repeated measures with intragroup interaction with three measures, Cohen's f effect size = 0.25, error α = 0.05, test power = 0.80, correlation coefficient between measures repeated = 0.7 and correction for non-sphericity = 1 (Beck, 2013). The sample size calculated was 17 participants.

The study admitted inclusion factors: a) physically active military of Physical Education College of Brazilian Army, Rio de Janeiro; b) under similar conditions during the study period (same scales of activities, practical and theoretical classes, physical tests), and c) similar patterns of eating and resting. Two militaries were excluded from this study due to muscle injuries.

Fifteen (15) male Brazilian Army soldiers participated in the study, with a "Very Good" index in the Physical Aptitude Test (TAF), corresponding to 2950 – 3050m of the Cooper test.

This research attends Helsinki Declaration for Research with Human Beings (WMA, 2014), and it was approved by the Ethics Committee of Galeão Air Force Hospital (nº 35458714.6.0000.5250).

Data collection

Data collection was divided in three moments with 48h to 72h interval as presented in figure 1.

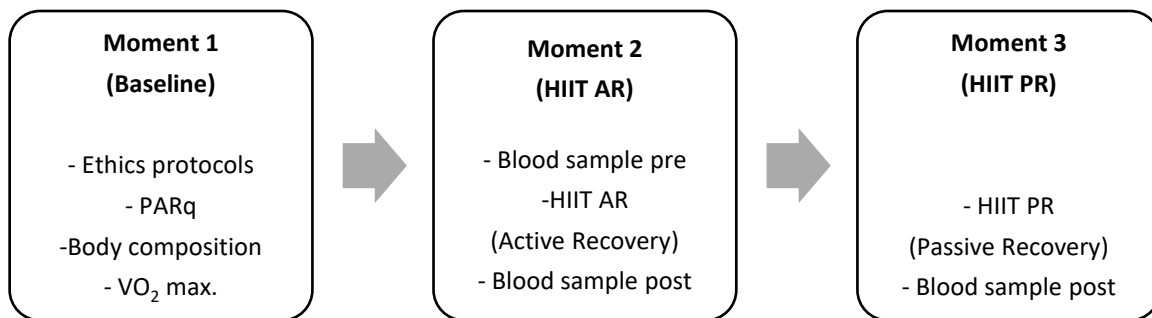


Figure 1 - Study design

Moment 1

Ethics protocols consisted by signing the Research Consent Term and answering the Physical Activity Readiness Questionnaire (PAR-Q).

The body composition was measured by ISAK (International Society the for the Advance of Kinanthropometry) (Stewart and collaborators, 2011) and Pollock 7-site skinfold protocol (Jackson e Pollock, 1985). Filizola® PL 2007 body weight scale, Sanny® stadiometer, and Cescorf® compass was used to measurement.

Cardiorespiratory fitness was assessed by maximum oxygen uptake (VO_{2max}) using ramp protocols in an ergoespirometry test performed on a treadmill. Participants performed a 3-minute warm-up at 9.0 km per hour, and a specific part with progressive accelerations of 0.5 km per hour by each 30 second until maximum fatigue.

The recovery phase was a 3-minute recovery performed at 40% of total speed. TechnoGym® Exite Run 900 and VO2000 (Medgraphics, USA) with Aerograph 4.3 (AeroSport Inc., USA) software was used. The effort intensity was controlled by heart rate (Forerunner 920xt Garmin Ltda., USA) and by perceived subjective effort (PSE Borg) with scores between 0 and 10 (Borg, 1998). This evaluation was used to calculate the individual HIIT speed.

Moments 2 and 3

Two running HIIT approaches were randomly applied on a treadmill: (a) HIIT AR - moderate to strong intensity (85% VO_2 max.) and 50% VO_2 max. with active recovery; and (b) HIIT PR - moderate to strong intensity (85% VO_2 max.) with passive recovery in the standing

position on the treadmill. The interval between protocols was 48 to 72 hours.

The HIIT approaches was performed in thermoneutral conditions (air temperature $20,6\pm 1,2^\circ\text{C}$ and air humidity 62% measured by MT-240 Minipa®) and consisted in 5-min. warm-up at 50% of VO_2 max., and a specific part with 6 sets of 1 minute and 30 seconds at 85% of VO_2 max. and 1-minute of recovery (active or passive). The recovery phase was a 3-minute recovery performed at 40% of total speed (Gillen and Gibala, 2014).

Biochemical evaluation was performed after blood sample collection 30 min before (pre) and immediately after the race (post) on a treadmill. Blood samples (14 ml) were collected with subjects remaining in a sitting position through the antecubital vein.

It was analyzed the biochemical markers of muscle damage through blood lactate, creatine kinase (CK) and lactate dehydrogenase (LDH), and oxidative stress markers (EO): lipid peroxidation (LP), carbonylated protein (PC), total antioxidant activity (TAA) by scavenging free radicals 2,2-diphenyl-1-picrylhydrazyl 2,2-diphenyl-1-picrylhydrazyl (DPPH*) and total sulfhydryl groups (TSG).

Plasma was harvested by centrifugation and stored at -80°C for further analysis of OS markers. An automated biochemical analyzer BT 3000 by Wiener Lab® Company was used to determine clinical serological markers. LP was evaluated through the formation of thiobarbituric acid reactive species (TBARS) during a heating reaction. PC was analyzed by the method that involves the derivatization of the carbonyl group with 2,4-dinitrophenylhydrazine (DNPH) and its relative amount considering control homogenates was expressed as 100 arbitrary units using carbonylated protein divided by maximum

oxygen uptake expressed by $\text{mlO}_2/\text{kg}/\text{min}$ (PC/VO_2). The exams were performed in duplicate and showed a coefficient of variation (CV) of less than 3%.

Data analysis

Samples normality and homogeneity were analyzed using the Shapiro-Wilk and Levene tests, respectively. Descriptive statistics and One Way ANOVA were used, followed by the adjusted Bonferroni post hoc. The equation $\Delta\% [(post\ intervention - baseline) * 100 / baseline]$

was used to determine the percentage difference. Data was analyzed by IBM SPSS Statistics 27 for Windows program. The value of $p < 0.05$ was adopted.

RESULTS

Body composition and cardiorespiratory fitness are presented at Table 1 to characterize the sample, and the muscle injury and OS markers are presented at Figure 2 and 3.

Table 1 - Body composition and cardiorespiratory fitness, (n=13).

Variables	Mean	SD	Minimum	Maximum
Age (yrs)	27.08	2.15	23.00	30.00
Height (m)	1.78	0.06	1.69	1.88
Lean Body Mass (kg)	80.22	9.20	61.00	92.00
Body Fatness (%)	10.74	3.03	7.49	17.01
VO_2 max. ($\text{mlO}_2/\text{kg}/\text{min}$)	56.56	6.99	45.67	67.33
Maximum speed (km/h)	19.50	1.40	18.00	22.00
Anaerobic threshold speed (km/h)	13.90	1.70	11.50	13.50
HIIT speed (km/h)	1.40	0.80	16.50	19.00

Legend: SD: standard deviation; %: percentual; VO_2 max: maximum oxygen uptake; m: meters; km/h: kilometers per hour.

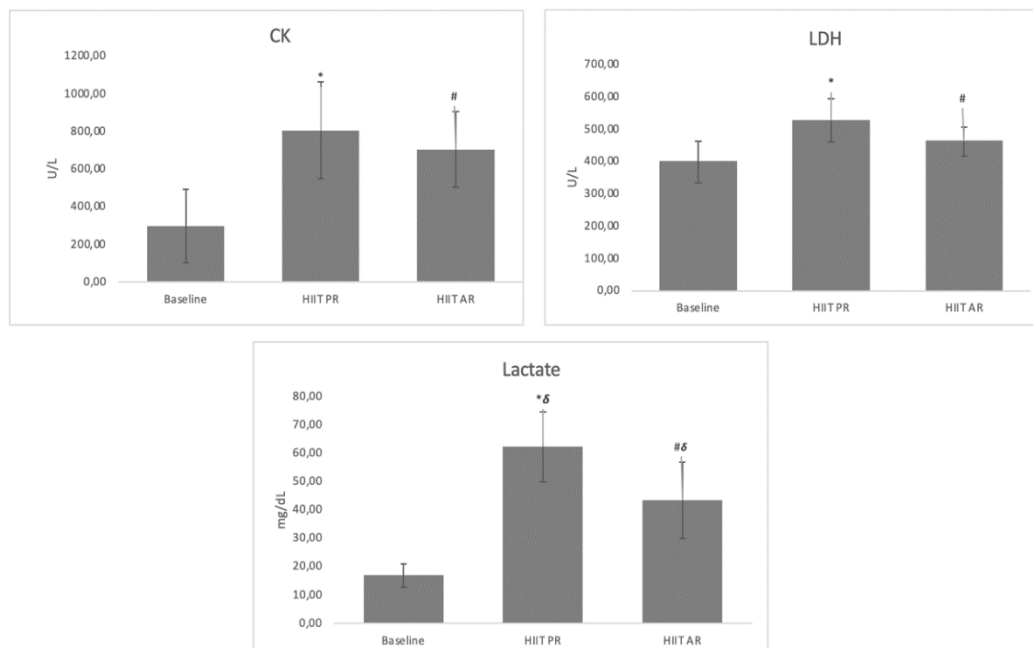


Figure 2 - Comparative analysis of muscle injury markers (CK, LDH and Lactate) between baseline and different interventions.

Legend: HIIT PR: HIIT Passive Recovery; HIIT AR: HIIT Active Recovery; CK: creatine kinase; LDH: lactate dehydrogenase; * Baseline x HIIT RP; # Baseline x HIIT AR; δ HIIT PR x HIIT AR. Significant level $p < 0.05$.

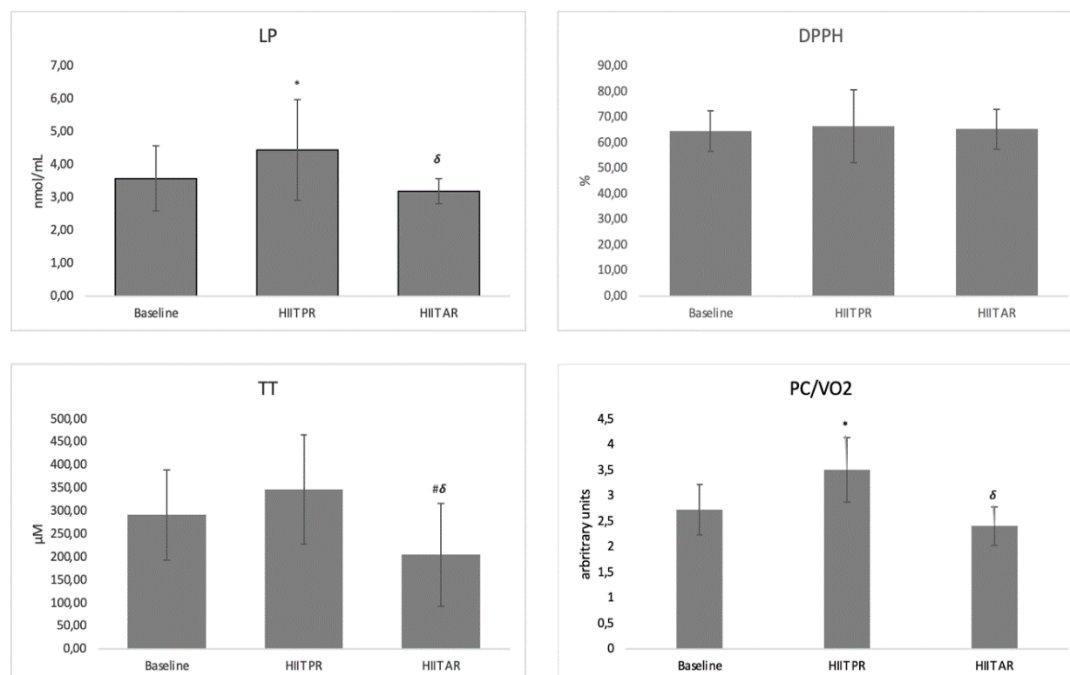


Figure 3 - Comparative analysis of oxidative stress markers between baseline and different interventions.

Legend: HIIT PR: HIIT Passive Recovery; HIIT AR: HIIT Active Recovery; LP: lipidic peroxidation; DPPH: difenil-1-picril-hidrazila; TT: total thiols; PC: protein carbonylated; VO₂: maximum oxygen uptake expressed by mlO₂ /kg/min; PC/VO₂: the relative amount of carbonylated protein considering control homogenates as 100 arbitrary units; * Baseline x HIIT RP; # Baseline x HIIT AR; ^δ HIIT PR x HIIT AR. Significant level p<0.05.

Comparing muscle injury markers at baseline x HIIT PR, it was observed a significant increase in CK ($\Delta\%$ = 170,66; p<0,001), LDH ($\Delta\%$ = 32,28; p<0,001) and lactate ($\Delta\%$ = 258,51; p<0,001). At baseline x HIIT AR, a significant increase in CK ($\Delta\%$ = 136,44; p<0,001), LDH ($\Delta\%$ = 15,99; p=0,028) and lactate ($\Delta\%$ = 156,87; p<0,001). Comparing HIIT RP versus HIIT AR, the only significant difference was the reduction in Lactate ($\Delta\%$ = -30,29; p = 0,008) at active recovery.

Concerning to OS markers, at baseline x HIIT PR, it was observed a significant increase in LP ($\Delta\%$ = 24,09; p=0,035) and PC/VO₂ ($\Delta\%$ = 28,57 p=0,030). At baseline x HIIT AR, a reduction only at TSG ($\Delta\%$ = -29,75; p=0,038) and at HIIT PR x HIIT AR, it was observed a reduction in LP ($\Delta\%$ = -28,34; p=0,001), TSG ($\Delta\%$ = -40,89; p<0,001) and PC ($\Delta\%$ = -31,54; p=0,001). No difference was found at DPPH.

DISCUSSION

The aim of this study was to compare biochemical markers of muscle damage and OS

in two HIIT running approaches. And knowing that the high intensity physical exercise has been associated with fatigue and muscle damage, it could be observed that the blood lactate levels immediately increase after HIIT approaches tested herein. Our results corroborate to scientific evidence reported by pointing out that it has its peak concentration in high-intensity exercises lasting between 5 and 10 min (Bird, Linden, Hawley, 2014).

It reflects the short-term metabolic stress, with the lactate threshold being the transition point between the predominance of aerobic to anaerobic energy (Delsmann and collaborators, 2021).

The difference in blood lactate between the HIIT protocols in our study is similar to the results reported by Germano e collaborators, (2022) when comparing HIIT in active and passive recovery, it was presented that the blood lactate was lower after performing with active recovery, but it is noteworthy that the recovery time used in this study was considerably longer (8 min) compared to our study (1 min).

Muscle injury biomarkers, CK and LDH levels presents significant increase in moderate-intensity exercises, such as 5 or 10 km sprints (Munjal and collaborators, 1983).

It is noteworthy that continuous training can result in an increase in CK levels and, therefore, athletes tend to have higher CK levels at rest than untrained individuals (Branccacio and collaborators, 2008), that also can be explained by the constant accelerations and decelerations during the sprints performed (Gastin and collaborators, 2019).

Wiewelhove and collaborators, (2015) who demonstrated that HIIT can elevate the levels of this biomarker as observed in both protocols in our study.

The magnitude of the increase in CK levels must be highlighted because it was significant compared to baseline, but it was subtle when compared to severe exercise, as observed in an ultramarathon (Bird, Linden, Hawley, 2014).

And it has been reported that CK and LDH levels are influenced by factors such as race, color, age, gender, muscle mass, physical fitness level and weather conditions interfere in its plasma concentration, with higher values in young men (Branccacio, Lippi, Manfulli, 2010; Brewster and collaborators, 2012).

Furthermore, eccentric exercises promote a higher level than the concentric ones (Fernandez-Lázaro and collaborators, 2020). And LDH plasma increase is slower than CK after physical exertion or muscle injuries (Branccacio, Lippi, Manfulli, 2010).

Significant increase in LDH plasma immediately after HIIT protocols with different duration of stimulus and passive recovery was observed (Brandão and collaborators, 2020; Cipryan, 2017). And in a high-intensity exercise protocol it was reported that the increase in injury biomarkers is more related to exercise duration than to training intensity (Shin and collaborators, 2016).

About OS biomarkers, the significant increase observed in LP and PC levels in HIIT RP without any change in TSG and TAA demonstrates that the type of rest used in HIIT exercise was not enough to activate the antioxidant defense system of ROS attack. In HIIT RA, PL and PC significantly reduced together with the total GS, which could protect the biomolecules from the attack of ROS generated by the exercise. In this context, different physiological pathways were stimulated by each HIIT protocol that causes

different individual responses, through distinct effects on redox metabolism (Braun and collaborators, 2016).

In fact, HIIT is beneficial by reducing OS and increasing antioxidant system. Active individuals undergoing to nine sessions of 30 seconds on a cycle ergometer with 4 minutes of recovery, presented reduction in PL and PC associated with increased antioxidant glutathione peroxidase (GPx) and catalase enzymes (Bogdanis and collaborators, 2013).

A study with thirty individuals submitted to two sessions of 30 minutes treadmill HIIT with intervals of six and two minutes, observed an increase in AAT and IL-6 in physically active individuals (Cipryan, 2018).

And other, the response of three different levels of HIIT protocols with different times (15, 30 and 60 seconds), with an immediate increase in IL-6, TAC, CK, myoglobin and LDH (Cipryan, 2017).

It should be noted that, in similar protocols, the recovery time is very important, especially in exercises with supramaximal intensity, that require a longer recovery time to achieve a higher performance (Germano and collaborators, 2022).

A short recovery period can impair performance in supramaximal sprints, so we suggest that an interval less than 30 seconds could be harmful to energy production in this type of exercise (Schoenmakers and collaborators, 2019).

The present study has some limitations as: i) sample group size, which may have contributed on biomarkers biological individuality responses; ii) non-evaluation of food ingestion; iii) non-analysis of some important biomarkers in the understanding of redox metabolism, such as the antioxidant enzymes catalase, superoxide dismutase (SOD) and glutathione peroxidase (GPx).

CONCLUSION

It could be concluded that both HIIT protocols increased the blood levels of muscle injury markers, but when comparing the passive and active recovery the lactate and the oxidative stress markers LP, TT and PC was higher at HIIT PR. So, the HI IT AR was more efficient on lactate remotion and redox metabolism for this sample. And it must be considered for training prescription.

The monitoring of tissue injury and oxidative stress biomarkers proved to be great

for monitoring training loads, being useful as a tool in injury prevention. Further studies are needed to verify if other HIIT protocols with other type of volume and intervals produce different effects.

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Recebido para publicação em 15/03/2022
Aceito em 04/06/2022

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