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RESEARCH

Assessment of heart rate variability and infrared thermography in response to exercise-induced muscle damage

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Abstract

Objective The aim of this study is to investigate the behavior of autonomic nervous activation and infrared thermography (IR) after an eccentric exercise protocol.

Methods The sample consisted of ten physically active men (22.5 ± 3.3 years) who had not practiced plyometric training in the 6 months prior to the study. After a period of familiarization and assessment of body composition, the participants underwent a plyometric jumping protocol, including jumps over a 50 cm obstacle and jumps from a 50 cm box, after a 5-min warm-up on the treadmill. Subsequently, the following assessments were performed: assessments included measures of rating perceived fatigue (RPF), delayed-onset muscle soreness (DOMS), IRT and heart rate variability (HRV) performed at 24, 48 and 72 h after exercise.

Results The RPF results showed a significant increase from baseline at 24 h, 48 h, and 72 h after the muscle damage protocol. For DOMS, significant increases were observed at 24 h, 48 h, and 72 h compared to baseline, with values notably higher at 24 h and 48 h than at 72 h. Thermographic assessments of the posterior thigh region showed a higher concentration of pixels in the warm zone at 48 h and 72 h compared to baseline, as well as at 48 h and 72 h compared to 24 h. Regarding HRV, the LF/HF ratio and %LF were elevated at 24 h and 48 h compared to baseline and 72 h, while %HF decreased at 24 h and 48 h relative to baseline.

Conclusion The exercise-induced stress increased warm-zone pixels and elevated sympathetic activity (LF/HF ratio and %LF) up to 48 h post-exercise. This was followed by a shift to increased parasympathetic modulation (higher %HF), indicating recovery. These findings underscore the utility of IRT and HRV in monitoring recovery and optimizing training.

Keywords Autonomic modulation · Exercise recovery · Sympathetic activity · Parasympathetic modulation · Delayed-onset muscle soreness

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Introduction

Physical exercise is considered a stressor that disrupts homeostasis. Depending on the level of stress caused by physical exercise, the immune system generates an inflammatory response, signaling to the autonomic nervous system via pro-inflammatory cytokines [1]. This process is critical as it triggers a complex feedback loop between the immune system and the autonomic nervous system, which plays a pivotal role in maintaining and restoring physiological balance [2].

Among the systems involved in this feedback are the autonomic nervous system, which comprises the sympathetic nervous system, parasympathetic nervous system, and enteric nervous systems [3, 4]. The autonomic nervous system is primarily responsible for important physiological functions such as blood flow and pressure, metabolic demands of tissues, thermoregulation, motility, and gastrointestinal tract secretion, among others. These functions are essential for preserving homeostasis during both internal and external disturbances, particularly through the integrative relationship between the immune and neuroendocrine systems [1, 5].

The main communication between the systems is potentially organized by cytokines released by cells of the innate immune system, which provide signals to inhibit the immunomodulatory response through the sympathetic and vagal pathways that initiate pro-inflammatory, anti-inflammatory, and immunosuppressive actions [6, 7]. Notably, the sympathetic nervous system has a crucial role in immune function by innervating lymphoid organs, thereby influencing immune responses through norepinephrine released in peripheral lymphoid organs and adrenaline secreted by the adrenal medulla [6, 8].

In muscle damage induced by unusually strenuous physical exercise with greater eccentric demand, an inflammatory response occurs, initiating a cascade of physiological processes aimed at healing or repairing tissue damage. This response is an essential part of the body's defense mechanism and is marked by elevated cytokines and other inflammatory markers in the blood, which serve to regulate and coordinate the repair of affected muscle fibers [9–13].

The sympathetic and parasympathetic functions are altered due to the inflammatory process, caused by exercise-induced muscle damage. The sympathetic system has its predominant activity in conditions that control "fight-or-flight" responses, that is, emergency conditions or preparation for strenuous activity and during physical exercise. The parasympathetic system predominates in resting conditions, regulating basic bodily functions, conserving and storing energy [4, 14]. Mechanical hyperalgesia from peripheral nerve afferents sensitization following delayed-onset muscle soreness (DOMS) is linked with autonomic nervous system and may cause an increase in sympathetic activity and increased LF/HF ration [15]. Heart rate variability (HRV) is a valuable tool to assess the modulation between these two systems, providing insights into the balance between sympathetic, and parasympathetic activity by analyzing the ratio between low frequency (LF) and high frequency (HF) (LF/HF) [16].

Of the cardinal signs related to the inflammatory process, the increase in skin temperature (Tsk) observed following physical exercise is a complex phenomenon. While it may be partially influenced by exercise-induced muscle damage, it is also closely related to the increased work of active muscles and their greater production of energy, which is dissipated as heat. This heat dissipation triggers several thermoregulatory reflexes in the skin, mediated by the autonomic nervous system through sympathetic activation [17]. The interplay between these factors suggests that the increase in Tsk cannot be attributed solely to inflammation, but rather to a combination of muscle activity and thermoregulatory processes, making the phenomenon more intricate and not fully understood.

This supports the physiological explanation for the use of infrared thermography (IRT), as the inflammatory response induces an increase in blood flow and metabolic activity in the exercised region. This entire process has a direct influence on Tsk, which can be captured by IRT [18]. Given that the skin receives dual autonomic innervation, predominantly under sympathetic control, Tsk changes reflect the underlying autonomic activity, where sympathetic nervous system activation typically causes vasoconstriction and cooling, and peripheral nervous system activity can lead to vasodilation and warming [19, 20].

Such changes in Tsk resulting from an inflammatory process caused by physical exercise have been increasingly investigated in recent years, with a growing number of publications in sports and health using infrared thermography (IRT) [21, 22]. Despite this, the specific relationship between autonomic nervous system activation and these temperature fluctuations remains inadequately understood, and the effectiveness of IRT in tracking inflammatory responses warrants further investigation. Therefore, the aim of this study is to investigate the behavior of autonomic nervous activation and infrared thermography after an eccentric exercise protocol. To achieve this, we monitored changes in DOMS, HRV, and IRT.

Materials and methods

Participants

Ten male participants were recruited for this study (age 22.5 ± 3.3 years, weight 71.7 ± 11.0 kg, height

171.1 ± 5.3 cm, and fat mass 15.5 ± 4.7%). A sample size of participants was estimated using the G* Power 3.1 software (University of Düsseldorf, Düsseldorf, Germany) with a statistical power of 80%, α error of 0.05 and an effect size of 0.5 for data changes in skin temperature [23]. All participants were classified as physically active based on the International Physical Activity Questionnaire (IPAQ) short version [24], engaging in an average of 5 ± 1 training sessions per week, totaling 305 ± 102 min per week.

To control for potential external influences on performance, the participants were instructed to avoid strenuous physical activity for one week prior to and during the study. All participants were fully informed about the nature of the study, including potential risks and benefits, and provided written informed consent. The study was conducted in accordance with the Declaration of Helsinki (1975) and received approval from the Institutional Review Board of the Federal University of Maranhão (protocol number 3.429.057).

Inclusion and exclusion criteria

Regarding the inclusion criteria, the participants were included in the study only if they were classified as physically active and had no prior experience in plyometric training for at least 6 months before the study, to minimize any residual effects from previous training and ensure that baseline performance or recovery metrics were unaffected by prior plyometric exercises. Additionally, they needed to demonstrate overall physical health, defined as the absence of musculoskeletal injuries, cardiovascular conditions, or any other chronic health issues that could interfere with physical performance. As for the exclusion criteria, individuals with any history of musculoskeletal or cardiovascular conditions, or those who had participated in a plyometric training program within the 6 months before the study, were excluded from participation.

Study design

The experimental stage occurred at least 5 days after the sample characterization (measuring body composition and collecting data on physical activity levels) and familiarization phase. During the familiarization phase, the participants performed a total of five vertical jumps to become accustomed to the jumping protocol and reduce learning effects. Additionally, the participants completed the IPAQ to assess their physical activity levels. On the day of the experimental collections, the participants underwent a series of analyses including subjective ratings of perceived fatigue and muscle soreness, IRT, and HRV measurements. The recovery assessments were conducted at 24, 48, and 72 h following the prescribed exercise to evaluate the effects on the participants.

Body composition assessment

Body composition was assessed through measurements of body mass, height, and skinfold thickness. A Wellmy®W300 scale with a stadiometer was used to measure body mass and height, with an accuracy of 0.5 cm for height and 0.05 kg for weight. Skinfold thickness was measured at eight specific sites: subscapular, triceps, biceps, pectoral, subaxillary, suprailiac, abdominal, thigh, and calf, using a Sanny® caliper, calibrated in millimeters, following the protocol established by Jackson and Pollock [25]. The values from each skinfold were summed (Σ skinfolds) to estimate body fat percentage, calculated using the Inforsob® 1.0 software.

Plyometric exercise

The muscle damage protocol employed in this study was based on the protocol described by Tofas et al. [26]. Briefly, a warm-up was performed on a treadmill (5 min at 8 km/h). Following the warm-up, the volunteers began the jumping protocol. This protocol consisted of two types of jumps: 96 jumps over a 50 cm obstacle (eight sets of 12 repetitions) and 96 jumps starting from a 50-cm box and jumping until touching the ground (eight sets of 12 repetitions). The interval between sets for the obstacle jumps was 90 s, while the interval for the box jumps was 180 s.

Rating perceived fatigue

The rating perceived fatigue (RPF) scale was evaluated at 24, 48, and 72 h after the muscle damage protocol. This scale subjectively predicts the recovery status of athletes and/or individuals engaged in physical activity. It is graded on a scale from 0 to 10 and possesses both verbal and numerical properties [27]. Recent studies have increasingly utilized this scale in research on physical recovery and fatigue assessment [28, 29].

Delayed-onset muscle soreness

Each participant completed a muscle soreness scale for the anterior (quadriceps) and posterior (hamstrings) regions of the thigh before each training session. They rated their soreness on a scale from 0 (“no soreness”) to 10 (“very intense soreness”). Muscle soreness was assessed before and at 24, 48, and 72 h after the plyometric exercise, following standard procedures [30–33].

Infrared thermography evaluations

For the collection of thermographic images, the subjects were instructed not to perform vigorous activities in the 48 h prior to the procedures, not to consume alcohol or caffeine, and not to use any type of skin cream in the 6 h prior to the evaluation [34]. Further instructions included to avoid sunbathing and physiotherapy treatments (e.g., massages or cryotherapy)—all recommendations were to minimize any influence on Tsk measurements [21]. To obtain the thermograms, the volunteers remained at rest for 10 min inside the air-conditioned room for thermal equilibrium [18]. These images were captured by a FLIR T650sc camera (accuracy: ± 1.0 °C, sensitivity: < 0.02 °C; resolution: 640 × 480; temperature range: − 40 °C to 2000 °C; spectral range: 7.5–14 µm); FLIR System Inc. Model, Sweden). Before the thermographic images were collected, the IRT camera was calibrated by entering ambient temperature (20–22.0 °C), relative humidity (RH, 55–60%) and emissivity (ε = 0.98) [21, 35]. To analyze thermograms, the thermopixelgraphy method (TPG) proposed by Fernandes [34] was used, with APOLLO software (APOLLO®, v. 1.0, OMNI, Brazil) for thermal analysis and analyzed using the thermopixelgraphy method [34, 36], considering pixels compatible with temperatures ≥ 33 °C (warm zone) [34, 37].

Environmental variables

Temperature and relative humidity monitored by Heat Stress WBGT meter (FLIR Commercial Systems Inc, model HT30, Nashua, NH).

Heart rate variability

HRV was recorded with a 12-lead electrocardiogram from WinCardio 6.1.1 and with a 600 Hz electrocardiogram signal (Micromed Biotecnologia Ltda) in the supine position, for 10 min, at rest, with a spontaneous and normal respiratory rate (between 9 and 22 respiratory cycles per minute). Recorded at all times of analysis (24, 48 and 72 h). The indices were evaluated using the Kubios HRV Analysis software, version 2.0 (Kubios, Finland) [38, 39]. HRV was analyzed in the frequency domain using Fast Fourier Transform (FFT), where low frequency (LF 0.04–0.15 Hz), high frequency (HF 0.15–0.4 Hz) representing sympathetic and vagal modulations, respectively, and autonomic balance (LF/HF ratio) of the RR interval time series were measured. The beat-to-beat datasets were converted to equidistant time series before applying FFT to calculate and analyze the spectra. The LF component reflects sympathetic and vagal modulation, whereas the HF component appears to be the result of vagal modulation. Furthermore, the LF/HF component has been proposed as a measure of cardiac sympathovagal balance.

Data and statistical analyses

The data are presented as mean ± standard deviation (SD). The Shapiro–Wilk test was used to assess the normality of the variables. Given that the variables did not follow a normal distribution, comparisons among baseline, 24 h, 48 h, and 72 h post-exercise were conducted using the generalized estimating equations (GEE) model [33]. Based on the near information criterion adherence index (QIC) values, the model was adjusted to the gamma distribution for the statistical tests in GEE. Time was treated as an independent factor with a fixed effect, and Bonferroni post hoc corrections were applied when necessary for multiple comparisons. To assess the size of the effect, Cohen's d test was employed. The interpretation of effect sizes followed Cohen's thresholds (1998), as modified by Hopkins [34]: trivial = 0.0–0.2; small = 0.2–0.6; moderate = 0.6–1.2; large = 1.2–2.0; very large = 2.0–4.0; and extremely large > 4.0, with a 95% confidence interval. Data analysis was performed using Statistical Package for the Social Sciences (SPSS, Inc, Chicago, IL, USA) version 24.0, with significance set at $p < 0.05$. Graphs were created using GraphPad Prism version 8 (GraphPad, San Diego, CA, USA) (Fig. 1).

Results

The rating of RPF measurement used to assess the recovery status of the subjects after physical exercise showed a significant increase compared to the baseline at 24 h, 48 h, and 72 h after the muscle damage protocol. Additionally, the RPF values at 24 h and 48 h were significantly higher than those at 72 h, indicating a peak in fatigue at 48 h followed by partial recovery at 72 h ($p < 0.003$) (Fig. 2).

For DOMS (Fig. 3), there was a significant increase in the 24 h, 48 h, and 72 h moments ($p = 0.000$; $p = 0.000$; $p = 0.002$, respectively) compared to the baseline and in the 24 h and 48 h moments compared to the moment 72 h ($p = 0.000$ and $p = 0.000$) after muscle damage protocol.

When analyzing the temperature radiated from the skin, using infrared thermography, the results are presented in the distribution of pixels in each region of interest (ROI). Figure 4 represents Tsk changes in the anterior and posterior thighs, collected in each moment of this study (baseline, 24, 48, and 72 h). In the analysis of the anterior region of the thigh (Fig. 4A), there was no statistically significant difference in the warm zone, but a very large effect size was presented for the baseline moment vs 48 h post (TE = 2.9) (Table 1). For the IRT results in the posterior

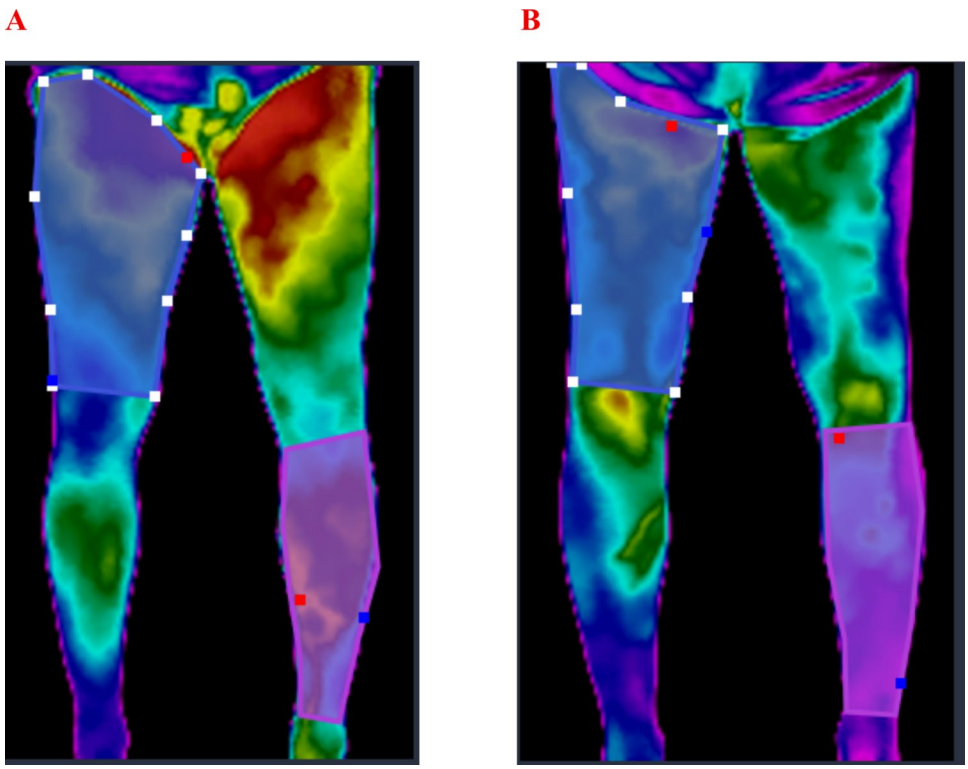

Fig. 1 Anterior (**A**) and posterior (**B**) thermopixelgraphy (TPG) of the low limbs (thigh and leg) with marked regions of interest (ROI's) taken for automatic analysis into three temperature zones (cold, neutral and warm zones)

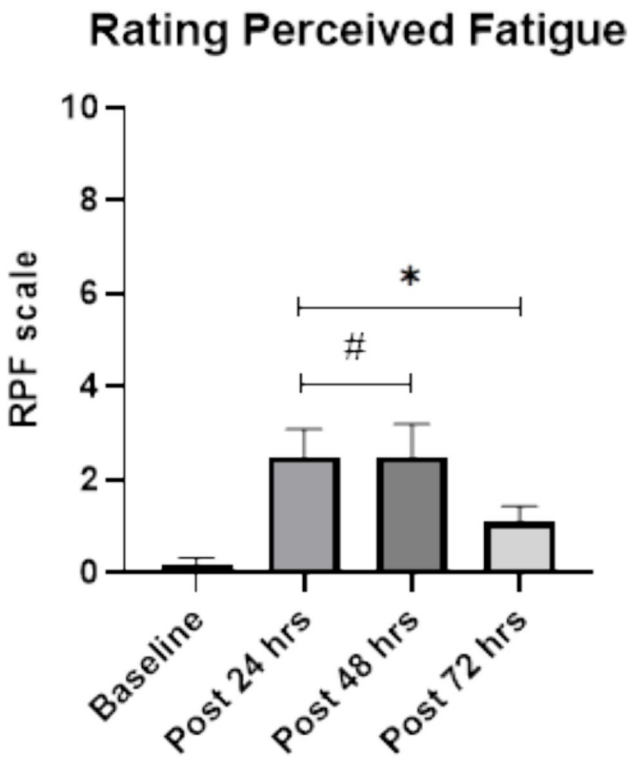

Fig. 2 Rating perceived fatigue for all moments. (* significant differences from baseline—$p < 0.05$. (# significant differences from post 72 h, $p < 0{,}05$)

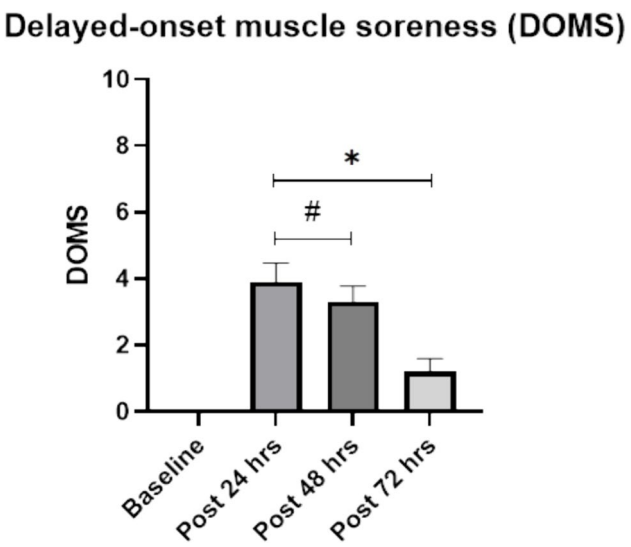

Fig. 3 DOMS for all moments. (* significant differences from baseline—p < 0.05. (# significant differences from post 72 h, p < 0.05)

region of the thigh (Fig. 4B), there was a higher concentration of the number of pixels in the warm zone there when comparing the baseline moment and the moments 48 h and 72 h ($p = 0.006$ and $p = 0.003$) and when comparing the moment 24 h post with the moments 48 h and 72 h ($p = 0.045$ and $p = 0.003$). Furthermore, a very large effect

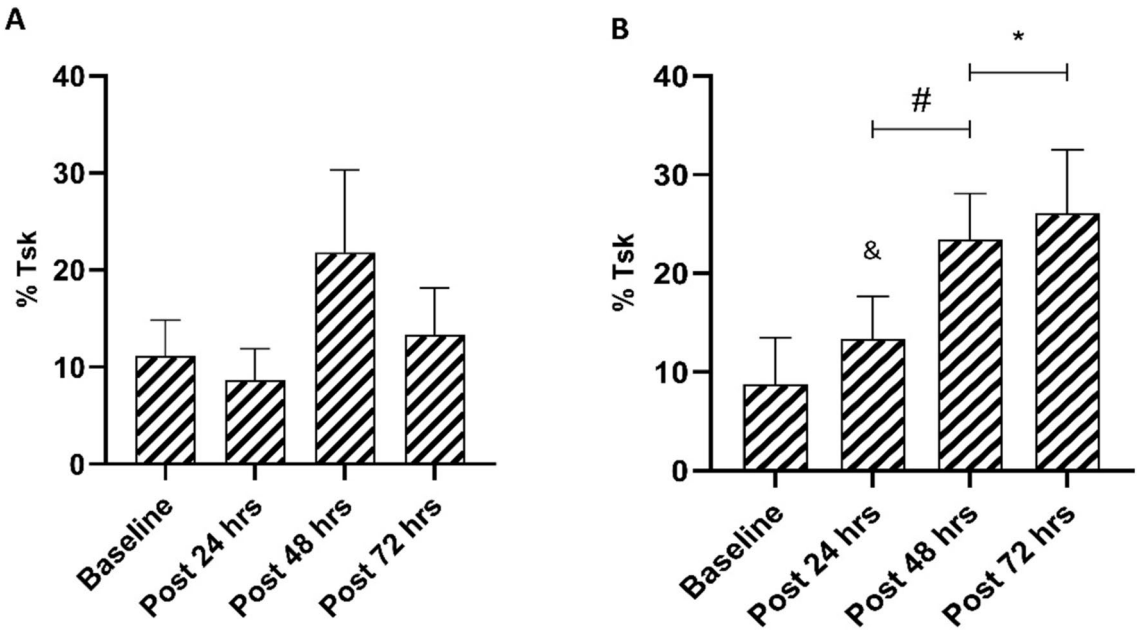

Fig. 4 Tsk changes in the anterior and posterior thighs at baseline and 24–72 h following plyometric exercise. Means (SD) for anterior thigh (**A**), posterior thigh (B). (* significant differences from baseline – $p < 0.05$; # significant differences from post 72 h, $p < 0.05$; and & significant differences from post 48 h)

Table 1 Effect size result for thigh pixel distribution zones

Zonas	Baseline *vs* 24 h	Baseline *vs* 48 h	Baseline *vs* 72 h
Warm zone anterior	−0.7	2.9	0.6
Warm zone posterior	1.0	3.1	3.6

Cohen's thresholds (1998) modified by Hopkins (2020), as trivial = 0.0–0.2; small = 0.2–0.6; moderate = 0.6–1.2; large = 1.2–2.0; very large = 2.0–4.0; and extremely large > 4.0

size was found for baseline vs 48 h post (TE = 3.1) and baseline vs 72 h post (TE = 3.6) (Table 1).

Figure 5 represents the Tsk in the anterior and posterior region of the legs, collected in each moment of this study (baseline, 24, 48, and 72 h). The distribution of pixels in the anterior region of the leg (Fig. 5A) showed that did not show differences in the warm zone, but an extremely large effect size was presented for the baseline vs 24 h post (TE = 9.5), baseline vs 48 h post (TE = 12.1) and baseline vs 72 h post (TE = 4.6) (Table 2). For the posterior region of the leg (Fig. 5B), the distribution of pixels showed that in the warm zone did not show any differences.

HRV measurements in the frequency domain included low frequency indices (ms and percentages), high frequency (ms and percentages), total values and LF/HF ratio, which correspond to the absolute and relative changes in the activities of the parasympathetic and sympathetic tone of the autonomic nervous system (HRV).

HRV absolute measurements in the frequency domain are presented in the Fig. 6. The low frequency index (LF) (Fig. 6A), which reflects the joint activity of the sympathetic and parasympathetic components, with sympathetic predominance, presented significant values at baseline and 24 h ($p = 0.000$), baseline and 48 h ($p = 0.045$) and at 24 h post and 72 h post ($p = 0.001$). Furthermore, a very large effect size was presented for the baseline vs 24 h post (TE = 2.9) and an extremely large effect size for the baseline vs 48 h post (TE = 4.2) (Table 3).

For the high frequency index (HF) (Fig. 6B), which corresponds to respiratory modulation and the tone of vagal activity over the heart, there were no statistical differences in absolute values between the moments analyzed.

Figure 6C shows the LF/HF ratio that represents the sympatho-vagal balance over the heart. There were differences between the baseline and 24 h moments ($p = 0.000$) and baseline and 48 h ($p = 0.011$), and also between the moments 24 h and 72 h ($p = 0.000$) and 48 h and 72 h ($p = 0.013$). A very large effect size was presented for the basal moment's vs 24 h post (TE = 3.0) and baseline vs 48 h post (TE = 4.1) (Table 3).

Figure 7 represents the HRV relative measurements in the frequency domain at baseline and 24–72 h following plyometric exercise. For the percentage values of the LF index (Fig. 7A), there was a statistical difference between the analyses at moments 24 and 72 h when compared to baseline ($p \leq 0.01$). As well as difference between 24 and 72 h post protocol ($p = 0.002$). And also, significant values

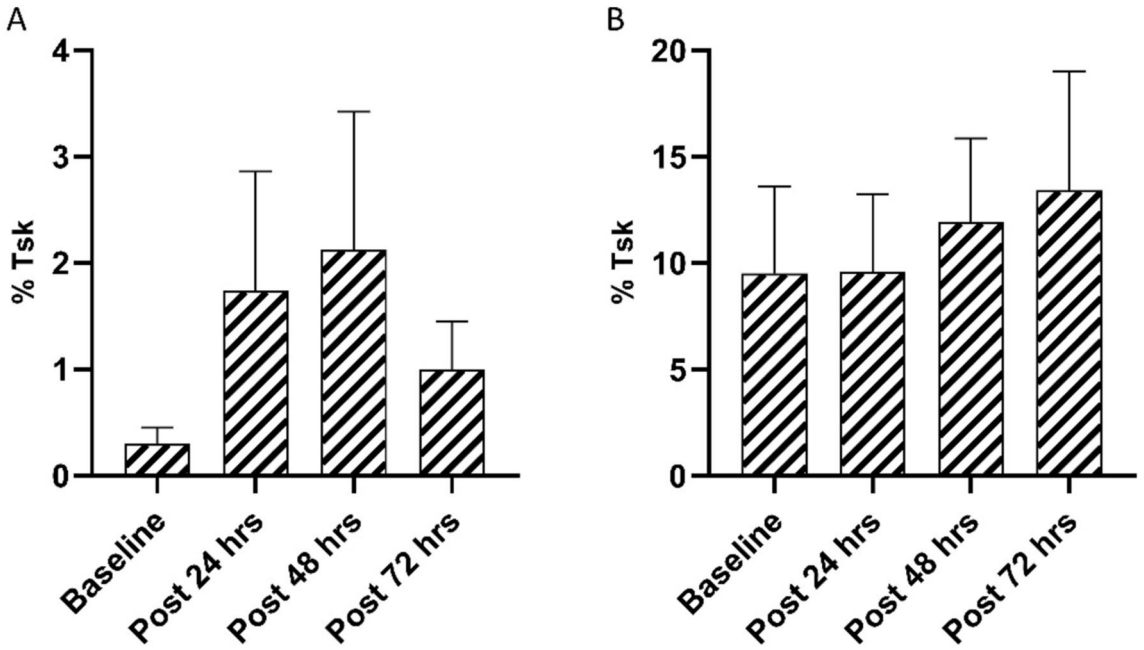

Fig. 5 Tsk changes in the anterior and posterior legs at baseline and 24–72 h following plyometric exercise. Means (SD) for anterior leg (**A**) and posterior leg (**B**) (no significant differences)

Table 2 Effect size result for leg pixel distribution zones

Zonas	Baseline *vs* 24 h	Baseline *vs* 48 h	Baseline *vs* 72 h
Warm zone anterior	9.5	12.1	4.6
Warm zone posterior	0.0	0.6	1.0

Cohen's thresholds (1998) modified by Hopkins (2020), as trivial = 0.0–0.2; small = 0.2–0.6; moderate = 0.6–1.2; large = 1.2–2.0; very large = 2.0–4.0; and extremely large > 4.0

when comparing the 48 h and 72 h moments ($p = 0.004$). An extremely large effect size was also presented for the basal moment's vs 24 h post (TE = 4.1) and very large for the baseline vs 48 h post (TE = 3.5) (Table 3).

Already for the percentage values of the HF index was identified a significant decrease in %HF at the 24 h moment ($p = 0.016$) and the 48 h moment ($p = 0.015$) compared to the 72 h moment after the muscle damage protocol (Fig. 7B). Moreover, a very large effect size was observed for the

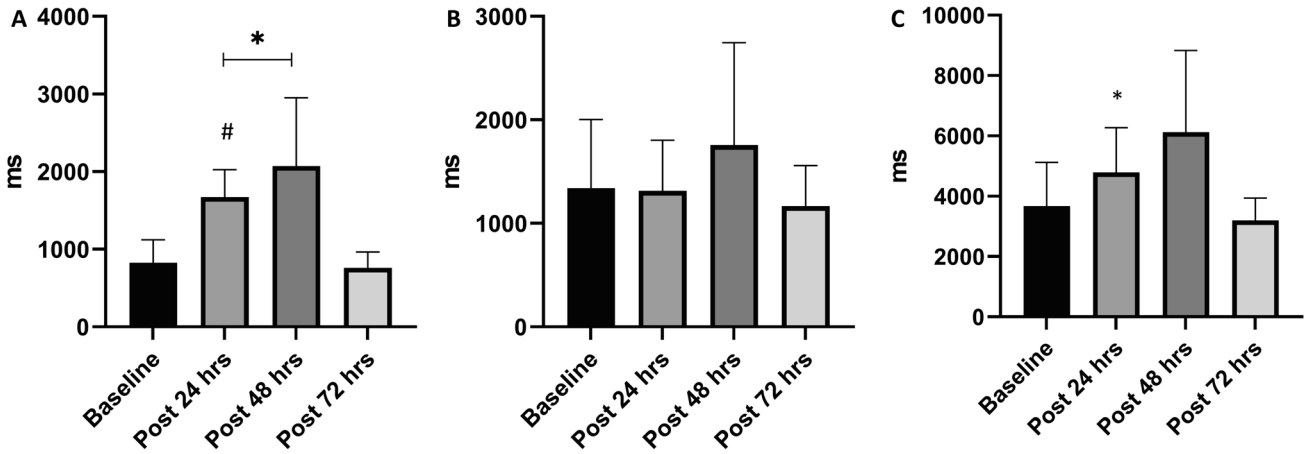

Fig. 6 HRV absolute measurements in the frequency domain at baseline and 24–72 h following plyometric exercise. Means (SD) for low frequency (**A**), high frequency (**B**), low frequency/high frequency ratio (**C**). (* significant differences from baseline—$p < 0.05$; # significant differences from post 72 h, $p < 0.05$)

Table 3 Effect size result for HRV measurements in the frequency domain

Indices	Basal *vs* 24 h	Basal *vs* 48 h	Basal *vs* 72 h
LF-HF ratio	3.0	4.1	−1.0
HF (ms)	−0.0	0.6	−0.3
% HF	−1.5	−1.1	2.2
LF (ms)	2.9	4.2	−0.2
% LF	4.1	3.5	−0.7
Total	0.8	1.7	−0.3

Cohen's thresholds (1998) modified by Hopkins (2020), as trivial = 0.0–0.2; small = 0.2–0.6; moderate = 0.6–1.2; large = 1.2–2.0; very large = 2.0–4.0; and extremely large > 4.0

comparison between baseline and 72-h post (TE = 2.2) (Table 3). Figure 7C shows the LF/HF ratio that represents the sympatho-vagal balance over the heart. There were differences between the baseline and 24 h moments ($p = 0.000$) and baseline and 48 h (p = 0.011), and also between the moments 24 h and 72 h ($p = 0.000$) and 48 h and 72 h (p = 0.013), shown in Fig. 7C. A very large effect size was observed for the comparison between baseline moments and 24-h post (TE = 3.0) and between baseline and 48-h post (TE = 4.1) (Table 3).

Discussion

The objective of this study was to investigate the behavior of autonomic nervous activation and infrared thermography after an eccentric exercise protocol. Our findings revealed that the stress induced by exercise with predominantly eccentric characteristics led to an increase in warm zone pixels, primarily observed after the initial 24 h in posterior region of the thigh, the area most affected by the eccentric characteristics of the exercise. This was accompanied by an elevation in LF% and the LF/HF ratio, which persisted until 48 h. Regarding cardiac modulation, significant differences in the values for %HF were found 72 h after the protocol (compared to 24 and 48 h).

The increase in DOMS at 24 and 48 h after the eccentric protocol is in line with what was found in previous studies that evaluated indirect markers of muscle damage and metabolic demand after eccentric exercise [33, 40–42]. For example, Peñailillo et al. [40] found that DOMS values were increased at 48 h and 72 h in young people after eccentric cycling. In the studies conducted by Arazi et al. [33], Kamandulis et al. [41], and Markovic et al. [42], which utilized a plyometric jumping protocol, VAS values exhibited an increase within 24 h, persisted at elevated levels for up to 48 h, and returned close to baseline values within 72 h—demonstrating a pattern similar to that observed in the present study. In contrast, the study by Korman et al. [43], which examined a continuous 10-day training protocol, identified a progressive decrease in resting skin temperature and an accumulated increase in creatine kinase levels, suggesting a potential long-term thermoregulatory adaptation to daily training. These results highlight that continuous exercise protocols may induce a different thermoregulatory response compared to single-session eccentric protocols, which, as shown in the present study, generate an acute, localized inflammatory response. It is also important to note that changes in skin temperature do not always correlate directly with biomarkers of muscle damage such as creatine kinase [44]. This suggests that temperature changes, while useful, may capture only part of the recovery and muscle

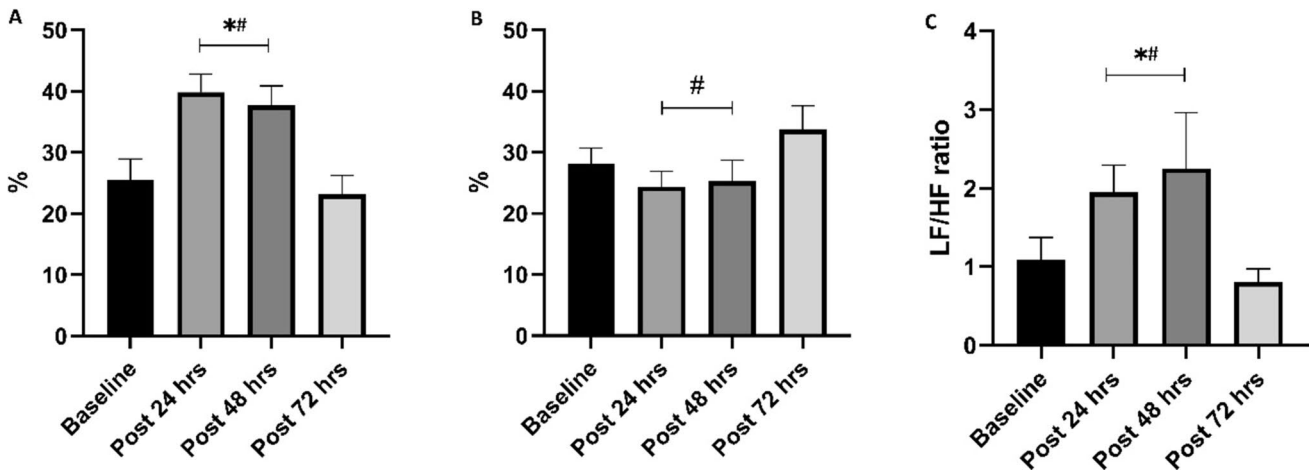

Fig. 7 HRV relative measurements in the frequency domain at baseline and 24–72 h following plyometric exercise. Means (SD) for % low frequency (**A**), % high frequency (**B**) and low frequency/high frequency ratio (**C**). (* significant differences from baseline—$p < 0.05$; # significant differences from post 72 h, $p < 0.05$)

damage process, reflecting factors like blood flow and metabolic activity in the tissue rather than direct markers of muscle fiber damage. Therefore, temperature assessments should ideally be combined with biochemical markers for a more comprehensive understanding of muscle recovery and damage [45].

The literature reports that eccentric muscular actions such as those performed during plyometric jumps, cause muscle damage [33, 40–42], accompanied by an inflammatory repair process, which generates increased blood flow and metabolic activity in the exercised region [18, 46]. This physiological stress can be measured by evaluating cardinal signs of inflammation, such as pain and heat.

The muscle damage generated resulted in a change in the distribution of pixels in the warm temperature zone. The increase in temperature, verified by the increase in the number of pixels in the warm zone occurred mainly in the posterior thigh region. The stress caused by the proposed protocol may also have caused a depression in parasympathetic/vagal tone, an increase in sympathetic activity and a reduction in HRV due to the corresponding increase in hypothalamic pituitary adrenal axis activity [47]. The analysis of Tsk, verified using infrared thermography, is associated with sympathetic nervous system modulation. The increase in the number of pixels in the warm zone after the protocol may be attributed to the rise in catecholamines, leading to heightened sympathetic activity. Over time, there is an augmentation in skin vascularization and heat exchange facilitated through the regulation of skin perfusion [48].

Additionally, the physiological mechanism linking thermal changes and HRV may involve peripheral sensitization, which plays a pivotal role in pain (soreness) perception, muscle blood flow, and muscle afferent gene expression. This peripheral sensitization can influence the autonomic nervous system response, particularly impacting HRV. As demonstrated by Zambolin et al. [15], the interaction between muscle damage and the resulting inflammatory response could lead to altered HRV via changes in muscle afferent activity and sympathetic modulation. This highlights the complex interplay between thermal responses, pain, and HRV suggesting that thermal imaging might reflect not only superficial circulation, but also deeper autonomic processes related to muscle recovery and inflammation.

In a recent study, Sillero-Quintana et al. [46] report the relationship between the activation of the sympathetic and parasympathetic systems with the Tsk of the lower limbs after a strength resistance exercise protocol, with PRE, POST, and POST-20 min measurements. An increase in sympathetic activation and an increase in Tsk were observed in the areas exercised in the POST and POST-20 min moment compared to the PRE moment. Although the collection times are different, it can be observed that both HRV and temperature are indicative of the individual's psychophysiological condition, and both are influenced by the activity of the autonomic nervous system [49, 50]. The temperature radiated from the skin can provide information about superficial circulation, which is modulated by the autonomic nervous system, responsible for thermoregulation processes, mainly due to venous return and cardiac output, which are also modulated by heat transfer from the skin to the blood and, subsequently, to the center of the body [51, 52].

The thermal changes observed in the present study may be linked to HRV through peripheral sensitization, which plays a critical role in pain perception, muscle blood flow, and the expression of muscle afferent genes. These factors can influence autonomic modulation, affecting both the increase in sympathetic activity and the depression of parasympathetic tone [15].

The findings of this study highlight the potential benefits of monitoring autonomic modulation and thermal responses in athletes to optimize recovery protocols. Health professionals, including physiologists, and athletic trainers should consider incorporating non-invasive methods such as IRT and HRV assessments in their practice. By using these tools, professionals can tailor recovery strategies to individual athletes, promoting more effective interventions that address physiological stress and enhance recovery.

The extrapolations of the present study's results should consider several limitations, including the lack of control over exercise intensity and the small number of participants. Although we used methods such as DOMS, HRV, and IRT to evaluate post-exercise effects, incorporating more precise tools like creatine kinase measurement could offer a clearer understanding of muscle damage. Additionally, it is important to assess muscle pain using more precise and consistently available methods, such as algesimeters, to improve the accuracy of pain measurement. Future research should utilize these advanced tools to better explore the relationship between exercise and physiological responses. By correlating these precise measurements with existing methods, researchers could address controversial findings and enhance the overall interpretation of results.

Conclusion

The present study demonstrated that exercise-induced stress resulted in a significant increase in warm-zone pixels, particularly in the posterior thigh region, alongside heightened sympathetic nervous system activity that lasted for up to 48 h post-exercise. This was followed by a notable shift towards increased parasympathetic nervous system modulation, indicating a recovery of vagal activity within the autonomic nervous system. This modulation may initially be linked to sympathetic nervous system-mediated vasoconstriction,

followed by vasodilation as Tsk rises, ultimately leading to a restoration of parasympathetic nervous system dominance. These findings highlighted the importance of monitoring sympathetic/parasympathetic activity and infrared thermography as a means of understanding the recovery process after exercise. The use of non-invasive methods such as IRT and HRV can be valuable, especially in the daily work of sports physiologists and coaches, as a way to assess physiological stress, load control, and recovery strategies in athletes, but they must be applied taking into account biological individuality and exercise conditions.

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Data availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest The authors declare no conflict interests.

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