

Active recovery does not accelerate cardiac autonomic recovery in young men with obesity, regardless of metabolic recovery

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Abstract

Active recovery may improve metabolic recovery, but its effects on cardiac autonomic recovery in young men with obesity are still unknown. **Purpose:** To assess whether active recovery accelerates cardiac autonomic recovery in young men with obesity. **Methods:** Sixteen young men with obesity (26.21 ± 3.05 years; BMI: 34.12 ± 2.46 kg·m⁻²; resting blood pressure: $124 \pm 8/77 \pm 5$ mmHg) completed two experimental sessions (passive and active recovery) involving a maximal incremental test on a cycle ergometer followed 90 min recovery. (I) Passive recovery: 10 min of sitting on a cycle ergometer (short-term recovery) + 80 min of sitting on a chair (long-term recovery); (II) active recovery: 10 min of pedaling on a cycle ergometer + 80 min of sitting on a chair. **Results:** Systolic blood pressure was higher during active recovery than in passive recovery ($p \leq 0.01$, main effect of recovery) in long-term recovery. During short-term recovery, active recovery resulted in a higher HR ($p \leq 0.01$, recovery vs. time), lower lnRMSSD ($p = 0.04$, main effect for recovery), and lower lnSDNN ($p = 0.01$, recovery vs. time), without affecting long-term cardiac autonomic recovery ($p > 0.05$). Regardless of recovery, HR and lnRMSSD returned to resting values at 60 min and lnSDNN at 45 min ($p \leq 0.01$). Blood lactate concentration ([Lac]) was lower in active recovery at three, 10, and 20 min ($p \leq 0.03$) than in passive recovery. Furthermore, the [Lac] returned to rest values at 30 min in active recovery and at 40 min in passive recovery. **Conclusion:** In young men with obesity, active recovery, regardless of metabolic recovery, did not accelerate the cardiac autonomic recovery.

Key Words: blood lactate, blood pressure, cardiac autonomic modulation, heart rate variability, obesity, parasympathetic reactivation.

Introduction

Currently, more than 1.9 billion adults have shown excess body mass. Of these, over 650 million are obese (World Health Organization, 2018). In 2015, the high body mass index was responsible for ~ four million deaths worldwide, more than two-thirds due to cardiovascular diseases (GBD 2015 Obesity Collaborators, 2017), which have been related to the impairment of cardiac autonomic modulation at rest (Yadav et al., 2017). Regular exercise has been shown to protect against cardiovascular diseases (Lavie, Ozemek, Carbone, Katzmarzyk, & Blair, 2019) associated with obesity. Although monitoring autonomic responses to a stressor may be of prognostic/diagnostic value (Araújo, Novelli, Arsa, & Cambri, 2023; Junior, Araújo, Okuno, & Schamne, 2025; Meireles et al., 2020), the effect of acute physical exercise on cardiac autonomic response in individuals with obesity remains poorly understood. Additionally, individuals with obesity are more likely to develop ventricular arrhythmias during physical exercise (Sabbag et al., 2016).

In recent years, heart rate variability (HRV) analysis (Task Force, 1996) has been suggested as a noninvasive method for assessing cardiac autonomic modulation. The root mean square of successive RR interval (RRi) differences (RMSSD) has been pharmacologically validated as a marker of parasympathetic reactivation after physical exercise (Goldberger et al., 2006), and has demonstrated clinical applicability (Lahiri et al., 2012; Wang et al., 2011).

The autonomic nervous system responses after exercise result from the integration of central (i.e., central command) and peripheral mechanisms involving skeletal (i.e., mechanoreflex and metaboreflex) and smooth muscles (arterial baroreceptors) (Carter, Watenpaugh, Wasmund, Wasmund, & Smith, 1999; Fisher et al., 2010; Hartwich, Dear, Waterfall, & Fisher, 2011; Peçanha, Forjaz, & Low, 2017). Additionally, some events (e.g., ventricular ectopy) that occur during post-exercise recovery are better predictors of cardiovascular risk than those occurring during exercise itself. Indeed, physical exercise induces rapid physiological changes that can lead to cardiovascular instability during recovery (Halliwill, Sieck, Romero, Buck, & Ely, 2014; Romero,

Minson, & Halliwill, 2017). In this context, some studies (Bastos et al., 2012; Soares et al., 2017) have evaluated strategies for optimizing the restoration of physiological systems to pre-exercise levels.

Active recovery due to the promotion of efficient venous return (American College of Sports Medicine, 2013; Carter et al., 1999; Halliwill et al., 2014; Romero et al., 2017) and thus maintaining cardiac output and cerebral blood flow (Carter et al., 1999; Halliwill et al., 2014), has been recommended, especially for individuals with symptoms of neurocardiogenic syncope. Besides, active recovery facilitates the removal of circulating metabolites (Bastos et al., 2012; Gmada et al., 2005; Mota et al., 2017; Spierer, Goldsmith, Baran, Hryniewicz, & Katz, 2004), which may have implications on cardiac autonomic modulation (Fisher et al., 2010; Hartwich et al., 2011; Peçanha et al., 2016). However, whether these responses are also observed in young obese men with low aerobic fitness remains unclear.

Although active recovery post-exercise (Bastos et al., 2012; Soares et al., 2017) did not promote a higher reactivation of cardiac autonomic modulation in physically active men, interventions that directly accelerate cardiac autonomic recovery or indirectly, through the restoration of other physiological systems, must be highlighted, especially in obese individuals who have an increased risk of cardiovascular events.

Therefore, the present study aimed to evaluate whether active recovery accelerates cardiac autonomic recovery after maximal exercise in young men with obesity. We hypothesized that active recovery would promote greater clearance of circulating metabolites and consequently accelerate cardiac autonomic recovery.

Material & methods

Participants

Sixteen men with obesity (26.21 ± 3.05 years; body mass index: $34.12 \pm 2.46 \text{ kg}\cdot\text{m}^{-2}$; body fat: $35.70 \pm 3.79\%$; blood pressure - BP: 124 ± 8 and 77 ± 5 mmHg), who had not regularly engaged in physical exercise in the four months prior to the study, took part in this investigation. The exclusion criteria were cardiovascular or metabolic diseases, smoking, use of medication, any substances that would interfere with the main outcomes, and locomotor problems that prevented physical exercise.

The study procedures were approved by the Institutional Human Research Ethics Committee (no. 76551917.7.0000.8124), and all participants were informed of the nature, purpose, potential risks, and benefits of the study. Those who chose to participate signed the informed consent form. This study was performed in accordance with the ethical guidelines outlined in the Declaration of Helsinki.

Study design

All subjects randomly performed two experimental sessions (passive and active recovery), which were conducted with a minimum interval of 72 hours between them. All the procedures occurred in the morning period (7:00 to 12:00 a.m.), in a temperature-controlled (22.92 ± 0.94 °C), and the relative humidity-monitored ($44.89 \pm 3.72\%$) laboratory. The sessions were performed at the same time of the day to avoid variability resulting from circadian rhythm (Brito et al., 2015). For all visits, participants were advised to avoid vigorous physical activity and not to ingest stimulating or alcoholic beverages in the previous 24 hours.

After a preliminary evaluation, the experimental session started with resting measurements (HRV and BP) in the seated position after 15 min, and then the participants were subjected to the maximal exercise test. After exercise, in passive recovery, participants immediately stopped pedaling and remained seated in the cycle ergometer for 10 min (short-term recovery), while active recovery was performed for 10 min. In both sessions, after short-term recovery, the participants adopted a sitting position on an upholstered chair for another 80 min (long-term recovery). All the participants were evaluated by the same evaluator.

Experimental protocol

After the resting measurements, a maximal incremental exercise test was performed using a cycle ergometer (Inbrasport, model CG-04, Brazil) with an initial workload of 15 watts (W) and increments of 15 W every min, with 60 revolutions per minute (rpm) until volitional exhaustion (Queiroz et al., 2018). Oxygen consumption and carbon dioxide production were evaluated using a portable gas analyzer (VO2000 Medical Graphics, USA), which was calibrated using ambient air, according to the manufacturer's recommendations. During the entire protocol (rest, exercise, and recovery), the HR was recorded beat-to-beat using an HR monitor (Polar RS800CX, Kempele, Finland, sampling frequency = 1000 Hz). The rate of perceived exertion was monitored during the last 10s of each stage using the 15-point Borg scale (Borg, 1982).

The test was considered maximal in both experimental sessions because all participants achieved at least three of the following criteria: inability to maintain 60 rpm for more than 10 s, at least 90% of maximal HR predicted by age (220-age), Borg scale values > 17 points, and respiratory quotient > 1.1 (American College of Sports Medicine, 2013).

In passive recovery, participants remained seated on cycle ergometer for 10 min, without pedaling (short-term recovery), and then adopted the sitting position in a padded chair for another 80 min (long-term recovery). Active recovery was performed at 15 W and 60 rpm for 10 min, after which participants adopted a seated position in a padded chair for an additional 80 min.

Data acquisition and analysis

Blood pressure

BP was measured twice using an automatic sphygmomanometer (MICROLIFE, BP 3BT0-A®, Brazil), with a two-minute interval between measurements. When a 5 mmHg difference was found between the measurements, a third measurement was performed, and the mean between the two closest values was calculated (Cardiology, Hypertension, & Nephrology, 2010). After maximal exercise, BP was measured every 10 min (10, 20, 30, ..., and 90 min).

Heart rate variability

During rest, exercise, and recovery, the RRi between consecutive heartbeats was recorded using an HR monitor (Polar, model RS800CX, Kempele, Finland, sampling rate of 1000 Hz) and following the recommendations of the Task Force (Task Force, 1996). To not disturb the gradual return of HR to pre-exercise levels, respiratory breathing rate was not controlled (Fang, Sun, Li, Poon, & Wu, 2008).

The RRi time series was analyzed using the Kubios HRV® program (KubiosOy, Version 2.1, Kuopio, Finland). The artifacts were filtered using a moderate filter in which artifacts and ectopic beats were automatically detected (i.e., any RRi 0.25 s larger or smaller than the signal average) and replaced by interpolated values using a cubic spline function (Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014). The percentage of observed errors was < 2% for all participants.

Pre-exercise HRV

At rest, HR and HRV were evaluated using consecutive RRi with the greatest signal stability within the last 10 min interval recorded in this condition. HRV was assessed by calculating the RMSSD, which reflects parasympathetic modulation, and the standard deviation of normal-to-normal RRi (SDNN), which reflects overall autonomic nervous system activity (Task Force, 1996).

Time-varying HRV indices

During the entire 10 min of recovery after post-exercise, because of the non-stationary behavior of RRi during this period, the RMSSD and SDNN indices were evaluated in successive non-overlapped 30 s segments (e.g., RMSSD30s; RMSSD60s ... RMSSD600s and SDNN30s, SDNN60s ... SDNN600s) (Goldberger et al., 2006). Moreover, for HR, RMSSD, and SDNN, the total area under the curve (AUC) was calculated using the equation $\sum (1 + 2 + \dots + 20)$, where each area was calculated as $[(\text{larger base} + \text{smaller base}) \times \text{height}] / 2$. A height value of 30 s was considered.

Post-exercise HRV indices

In the long-term recovery, HR, RMSSD, and SDNN were analyzed in the last 5 min of each 15 min interval (RMSSD15min; RMSSD30min ... RMSSD90min, and SDNN15min, SDNN30min ... SDNN90min).

Blood lactate concentration

Twenty-five microliters of blood were collected from the earlobe using capillary tubes and transferred to Eppendorf tubes containing 50 μL of sodium fluoride (1%). The samples were analyzed in triplicate by enzymatic assay using specific reagent kits (Labtest Ref.: 138, Minas Gerais, Brazil). The blood lactate concentration [Lac] was determined at rest, 2 min before exercise, and at the maximal post-exercise recovery at 3, 5, 7, 10, 20, and 90 min.

Statistical analysis

Normal distribution of the data was assessed using the Shapiro-Wilk test. The HRV indices were transformed into natural logarithms to enable parametric analysis. Parametric data are presented as mean $\pm$ SD and nonparametric data are presented as median and interquartile range (Q1-Q3). To compare variables between experimental sessions (passive and active recovery), the paired t-test was used for parametric data, and the Wilcoxon test for nonparametric data. A two-way repeated measures analyses of variance (ANOVA) (recovery vs. time) was used to evaluate the BP, [Lac], HR, and HRV indices at rest, at the end of physical exercise, and during recovery. When the main effect or interaction was significant, a Bonferroni post hoc test was used. To measure the magnitude of the difference between the analyzed variables for the recovery type, the effect size was evaluated using the partial eta squared (η^2_{partial}) calculated by two-way ANOVA with repeated measures. The effect size was considered small (≥ 0.01), medium (≥ 0.06), or large (≥ 0.14) (Cohen, 1988). The significance level was set at $p \leq 0.05$.

Results

There were no differences ($p > 0.05$) among the variables at rest (Figs. 1, 2, 3, and 4) or during physical exercise (Table 1) between the experimental sessions. These data indicate that participants performed post-exercise recovery under similar physiological conditions.

Table 1. Maximal incremental test variables

<i>n</i> = 16	Passive recovery	Active recovery	p value
Time (min)	14.18 ± 2.09	14.16 ± 2.56	0.949
Maximal workload (W)	206.63 ± 31.41	206.41 ± 38.42	0.946
VO ₂ peak (ml·kg ⁻¹ ·min ⁻¹)	24.97 ± 5.07	24.05 ± 3.56	0.336
Maximal heart rate (%)	93.93 ± 4.40	94.32 ± 4.41	0.448
Rate perceived exertion (points)*	20 (20 – 20)	20 (20 – 20)	0.564
Respiratory exchange ratio	1.17 ± 0.08	1.20 ± 0.19	0.604

* Values are median, interquartile range (Q1-Q3) and Wilcoxon test for nonparametric data were employed. The other variables are presented as mean ± standard deviation, and Student's t-test for parametric data was employed.

Effect of active recovery on post-exercise BP

SBP was higher during active recovery (the main effect of recovery). Regardless of the recovery, the SBP was reduced compared to the pre-exercise SBP at 40, 60, and 90 min (main effect for time) (Fig. 1A). DBP was reduced compared to pre-exercise only at 80 min (main effect for time) (Fig. 1B).

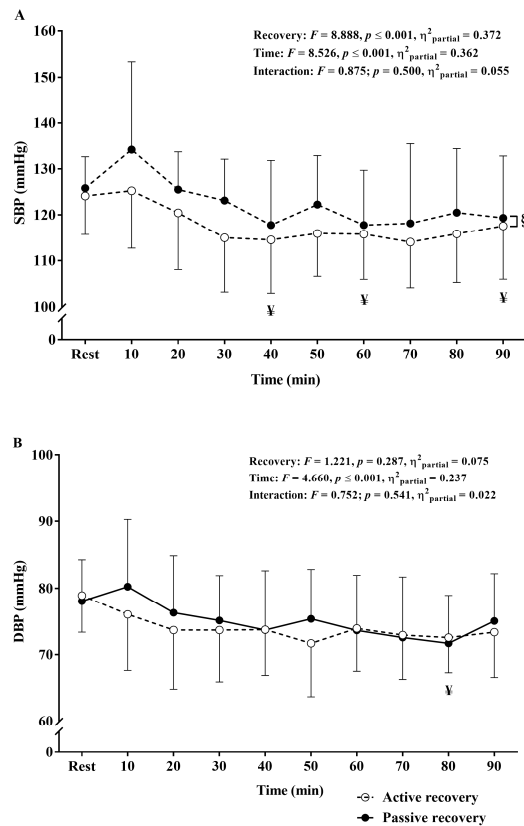


Figure 1: Blood pressure at rest and during 90 min post-maximal exercise. Panel A: systolic blood pressure. Panel B: diastolic blood pressure. Values are means ± standard deviation. Two-way ANOVA: § $p \leq 0.05$ main effect for recovery; ¥ $p \leq 0.05$ main effect for time.

Short-term cardiac autonomic recovery

During short-term recovery (10 min, i.e., 600 s), there were main effect for recovery and time, as well as a significant interaction between recovery and time for HR (Figure 2A) and lnSDNN index (Figure 2C). There was only a main effect of recovery and time on the lnRMSSD index (Figure 2B). As expected, active recovery resulted in a higher HR values from 180 to 600 s and lower lnSDNN values from 90 to 600 s as opposed to passive recovery. In addition, the AUC for HR was higher, and the AUCs for lnRMSSD and lnSDNN were lower for active recovery than for passive recovery.

Regardless of the type of recovery, during the initial 10 min, the HR (Figure 2A) decreased, and the lnSDNN index (Figure 2C) increased from peak exercise to 600s, without returning to the resting levels.

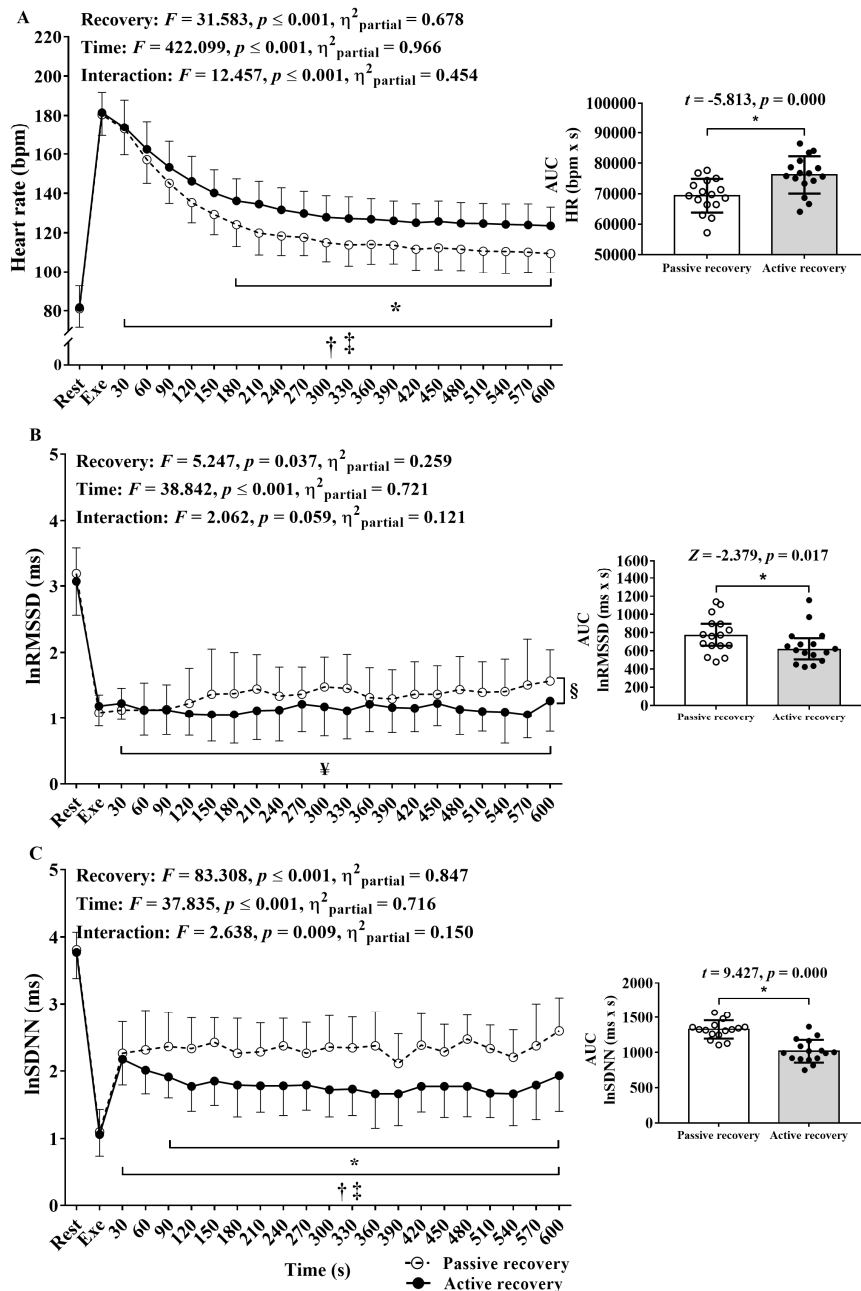


Figure 2: Short-term cardiac autonomic recovery. Panel A: Heart rate. Panel B: Time-varying lnRMSSD index. Panel C: Time-varying lnSDNN index. Values are means $\pm$ standard deviation, and AUC lnRMSSD in median with interquartile range.

Two-way ANOVA: § $p \leq 0.05$ main effect for recovery; ¥ $p \leq 0.05$ main effect for time in relation to rest; * $p \leq 0.05$ passive recovery session vs. active recovery session; † $p \leq 0.05$ in relation to rest in passive recovery; ‡ $p \leq 0.05$ in relation to rest in active recovery.

Long-term cardiac autonomic recovery

Active recovery did not influence the recovery of HR (Fig. 3A) or the lnRMSSD index (Figure 3B), without a main effect on recovery and significant interaction. However, the lnSDNN index (Figure 3C) was higher after 15 min of active recovery than after passive recovery.

There was a main effect of time on post-exercise responses. Regardless of the type of recovery, the HR (Figure 3A) and lnRMSSD index (Figure 3B) returned to rest values at 60 min of recovery. Regardless of the type of recovery, the lnSDNN index (Figure 3C) returned to rest after 45 min of recovery.

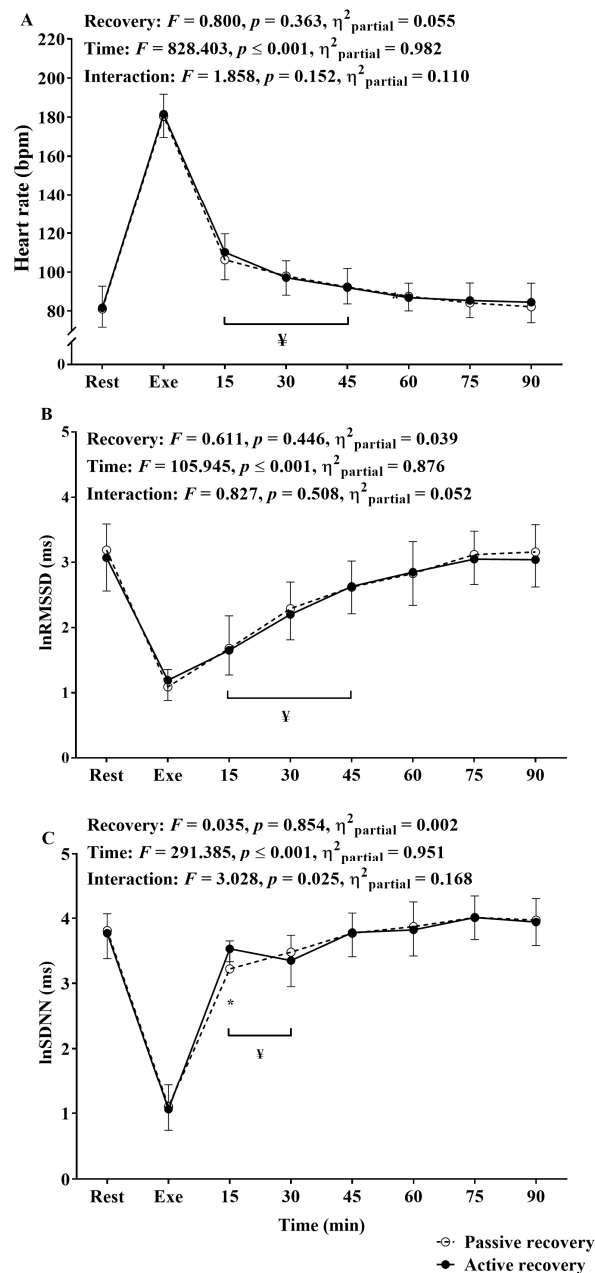


Figure 3: Long-term cardiac autonomic recovery. Panel A: Heart rate. Panel B: Time-varying lnRMSSD index. Panel C: Time-varying lnSDNN index. Values are means $\pm$ standard deviation. Two-way ANOVA: $\$$ $p < 0.05$ main effect for time. * $p \leq 0.05$ passive recovery session vs. active recovery session;

Effect of active recovery on post-exercise [Lac]

For [Lac] over 90 min following exercise, there were main effects of recovery and time, as well as a significant interaction between recovery and time. Active recovery resulted in lower [Lac] levels at 3 ($p = 0.03$), 10 ($p < 0.01$), and 20 min ($p = 0.01$) compared to passive recovery. In addition, active recovery accelerated [Lac] recovery, with values returning to resting levels at 30 min ($p = 0.02$). During passive recovery, [Lac] returned ($p < 0.01$) to resting values only at 40 min (Figure 4).

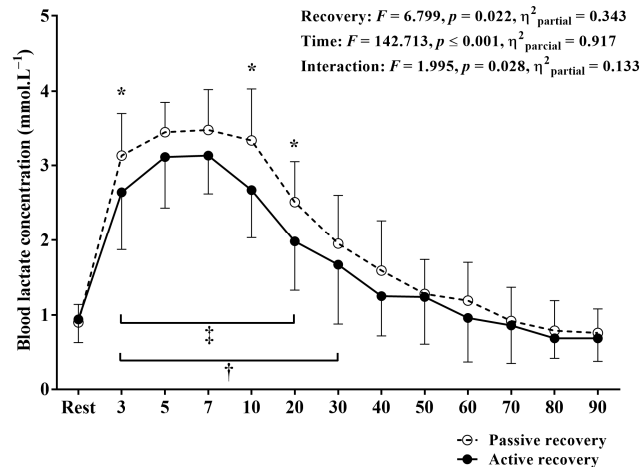


Figure 4: Blood lactate concentration at rest and during 90 min post-maximal exercise. Values are means $\pm$ standard deviation. Two-way ANOVA: * $p \leq 0.05$ passive recovery session vs. active recovery session; † $p \leq 0.05$ in relation to rest in passive recovery; ‡ $p \leq 0.05$ in relation to rest during active recovery.

Discussion

The main findings of the present study are that performing low-intensity exercise immediately following maximal exercise promoted higher blood lactate removal than passive recovery but did not accelerate autonomic cardiac recovery in young men with obesity.

In the present study, active recovery favored a smaller reduction in post-exercise SBP, which was not observed in physically active men, possibly due to the shorter recovery time (Soares et al., 2017). Contraction of the gastrocnemius muscle (i.e., the muscle pump) facilitates venous return during active recovery (Carter et al., 1999; Halliwill et al., 2014; Romero et al., 2017), which may have led to a lower reduction in post-exercise blood pressure in men with obesity.

To the best of our knowledge, only a few studies have investigated the influence of active recovery on cardiac autonomic recovery. In addition, the differences in the modality (running (Bastos et al., 2012); cycling (Soares et al., 2017)), exercise intensity (submaximal (Soares et al., 2017); maximal (Bastos et al., 2012)), active recovery time performed (5-6 min (Bastos et al., 2012; Soares et al., 2017)), and recovery time evaluated (30 (Soares et al., 2017) and 90 min (Bastos et al., 2012)) made comparisons with the present study difficult. In this context, studies (Michael, Jay, Graham, & Davis, 2017) demonstrated that both intensity and modality exercise influence HRV responses in recovery. We also believed that the reduced duration of active recovery (i. e. 5-6 min) used in previous studies (Bastos et al., 2012; Soares et al., 2017) was not enough to accelerate the metabolic recovery, thus explaining the lack of differences in autonomic recovery between the active and passive sessions. Therefore, we chose to use 10 min of active recovery.

The active recovery protocol was characterized by mechanical stress to maintain exercise at light-intensity exercise. As expected, during active recovery, the HR was higher, and the lnSDNN index was lower than during passive recovery, corroborating the findings of Bastos et al. (2012). This can be explained by the maintenance of neural responses because, in addition to central command activation, muscle contraction stimulates nerve endings that are sensitive to mechanical changes (i.e., mechanoreflex), which avoids a higher increase in parasympathetic modulation and reduction in HR (Carter et al., 1999; Gladwell & Coote, 2002; O'Leary, 1993).

To investigate the benefits of active recovery after exercise, participants remained seated for 80 minutes. After the active recovery protocol, that is, after the cessation of central and peripheral stimuli (i.e., mechanoreflex), HRV indices were similar to those observed during passive recovery. Similarly, 5-6 min of active recovery did not accelerate the reestablishment of cardiac autonomic modulation during either 30 (Soares et al., 2017) or 90 min (Bastos et al., 2012) of recovery period investigated. Additionally, unlike these studies (Bastos et al., 2012; Soares et al., 2017), we also investigated whether the type of recovery affected the time required for HRV indices to return to pre-exercise levels. Regardless of the recovery type, metabolic and overall HRV recovery (SDNN, 45 min) preceded vagal restoration (RMSSD, 60 min), corroborating a previous study that investigated passive recovery (Hoshi et al., 2017).

Active recovery is an important strategy for accelerating the removal of blood lactate (Bastos et al., 2012; Gmada et al., 2005; Mota et al., 2017; Peçanha et al., 2016; Spierer et al., 2004). The lower [Lac] and anticipated restoration in active recovery can be attributed to higher oxidation by active skeletal muscles, heart, and liver. Possibly, this greater clearance of accumulated blood lactate occurs due to the increase in blood flow, which promotes H^+ buffering and pH restoration (Bangsbo, Gollnick, Graham, & Saltin, 1991; Oyono-Enguelle et al.,

1990). This is mainly attributed to oxidative type I muscle fibers, as well as myocardial oxidation and gluconeogenesis via the Cori cycle, whereas lactate production predominantly occurs in glycolytic type II muscle fibers (Draper, Bird, Coleman, & Hodgson, 2006; Gladden, 2004). In another study, the enhanced lactate clearance observed during active recovery was primarily attributed to increased lactate uptake by the skeletal muscles. Furthermore, monocarboxylate transporter 1 (MCT1) has been suggested to play a key role in lactate transport, especially during active recovery when low-intensity exercise predominantly recruits type I muscle fibers, thereby facilitating lactate uptake and oxidation (Cupeiro et al., 2016). However, our study was limited to the measurement of [Lac]; therefore, it was not possible to determine the exact fate of lactate in our subjects.

Metaboreceptor activation in skeletal muscles elicits reflex autonomic responses that influence cardiovascular control (Fisher et al., 2010; Hartwich et al., 2011; Peçanha et al., 2016). A study of β 1-adrenergic and parasympathetic blockade conditions verified that muscle metaboreflex activation-mediated increases in HR were principally attributable to increased cardiac sympathetic activity, with a contribution from the partial withdrawal of cardiac parasympathetic tone (Fisher et al., 2013). Therefore, we expected that a lower [Lac] active recovery would accelerate cardiac autonomic recovery. A previous study (Guerra et al., 2019) demonstrated that metaboreflex control is reduced in obesity, as obese women exhibit lower muscle sympathetic nerve activity responses during circulatory arrest than lean women. Nevertheless, the mechanism underlying the relationship between active recovery, metabolite removal, and metaboreflex activation, particularly in individuals with obesity, remains unclear. Therefore, further studies are needed.

The present study has some limitations. First, the findings are limited to apparently healthy obese young men. Second, we did not evaluate frequency-domain indices because they have been suggested to show lower reproducibility than the time-domain indices during short-term recovery after exercise (Araújo et al., 2020). These indices are influenced by respiratory parameters, mechanical events, and baroreflex sensitivity, regardless of changes in cardiac autonomic nervous activity (Billman, 2013; Goldstein, Benth, Park, & Sharabi, 2011). Third, cardiovascular control mechanisms associated with the autonomic nervous system were not been evaluated. Finally, we did not use post-exercise circulatory occlusion, which is the most widely used method for evaluating metaboreflex responses.

Conclusion

In summary, in young men with obesity, active recovery, despite promoting greater blood lactate clearance, did not accelerate cardiac autonomic recovery after maximal exercise. In addition, active recovery resulted in a smaller reduction in post-exercise SBP. Although the maximal exercise session used in this study is not typically prescribed for aerobic training, short-term high-intensity exercise is widely practiced among young adults and can be help reduce total session time. Therefore, our findings do not support the adoption of active recovery after high-intensity efforts in asymptomatic young men with obesity, as it prolonged the overall session duration without providing additional benefits to cardiac autonomic recovery. Consequently, passive recovery may be a more time-efficient strategy in this context and could potentially favor adherence to exercise programs.

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Conflicts of Interest The authors declare that there is no conflict of interest.

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Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used ChatGPT in order to improve the clarity, grammar, and readability of the manuscript. After using this tool, the authors carefully reviewed and edited the content and assume full responsibility for the content of the publication.

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