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Original Article

Effect of exercise intensity after a single session of isocaloric aerobic exercise on the heart rate recovery

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Abstract:
The aim of this study was to compare heart rate recovery (HRR) and its association with energy systems of contributionafter isocaloric exercise sessions conducted at low (LI) and moderate (MI) intensity. Nine subjects randomly underwent two isocaloric exercise sessions, one conducted at LI (10% < velocity from anaerobic threshold) and other at MI (10% < velocity from respiratory compensation point). Blood lactate was measured pre- and post-exercise and rate perceived effort was assessed immediately after the exercise; during the exercise was estimated the contribution of oxidative, glycolytic and ATP-PCr systems. HRR30s, HRR60s (percentage of HR reduction after 30s and 60s of recovery), T30 (short-term time constant of HRR) and rMSSD30s (square root of the mean of the sum of the squares of differences between adjacent R-R intervals of subsequent 30s segments) were measured as parasympathetic reactivation markers. HRR300s (percentage of HR reduction after 300s of recovery) and HRRt (long-term time constant of the first order exponential fitting of HRR) were measured as sympathetic withdrawal markers. Paired t-test and two-way ANOVA were employed to compare LI and MI sessions and Pearson or Spearman correlations were used to analyze the association between energy contribution and HRR indexes, P<0.05. LI and MI did not present differences in HRR indexes. Regarding energy contribution, HRR30s was positively associated with oxidative contribution and negatively associated to ATP-PCr contribution, while HRR60s was negatively associated with glycolitc contribution, and T30 was negatively associated with oxidative contribution and positively associated with ATP-PCr contribution. No associations were observed after MI.Therefore, in recreationally active men, the caloric expenditure seems to play a role on HRR indexes after low and moderate intensity of a short matched-caloric expenditure aerobic exercise session,and parasympathetic reactivation is associated with greater oxidative, but lower glycolitic and ATP-PCr energy systems of contribution.
KeyWords*:* Physical activity; Parasympathetic reactivation; Sympathetic withdrawal; Energy systems.

Introduction

Chronic cardiovascular adaptations are dependent on a training of sufficient intensity and volume (Wilson et al., 2016; Iellamo et al., 2018). However, a high intensity exercise session promotes an overload on the cardiovascular system, which increases the risk of sudden cardiac death for up to 30 minutes after exercise, even in free from overt cardiovascular disease subjects (Albert et al., 2000).

The recovery of an exercise bout is a period at which occurs the integrated restoration of many systems to homeostasis and towards their resting state.Moreover, this approach helps to quantify the homeostatic stress generated by exercise assessing physiological markers during a dynamic recovery.The autonomic nervous system helps to regulate this process at different levels, such as regulating the secretory activity of glands, the vascular smooth muscle tone and cardiac work through blood pressure and heart rate (HR). Althoughno single measure represents the autonomic control of all organ and gland restoration of their functions during the recovery period from exercise, methods utilizing the HR have been shown as promising. Indeed, HR immediately after exercise has already been associated with different markers of stress, which reinforce its potential as a marker of restoring homeostasis post-exercise (Murrell et al.,2007). Among the known methods, HR recovery (HRR) after exercise has been suggested as noninvasive, applicable and a very common method to evaluate cardiac autonomic restoration post-exercise, making it possible to assess a combination of parasympathetic reactivation and sympathetic withdrawal (Peçanha et al., 2017; Kannankeril et al., 2004).

Previous studies have demonstrated that the higher intensities of exercise session affects negatively HRR during the recovery period (Mann et al., 2014;Matsuo et al., 2014;Peçanha et al., 2014;Al Haddad et al.,

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2011; Bartels et al.,2017; Hagberg et al. 1980).Regarding possible determinants for this difference, the higher level of metabolites produced (Peçanha et al., 2016), andthe augmented cardiac sympathetic activity observed during higher intensities of exercise areassociated with slower HRR (Ushijima et al., 2009).However, it is expected that higher intensities of exercise require greater caloric expenditure (Mann et al., 2014)increasing the contribution of each energy system in accordance with the demand(Gastin, 2001), and there is still no study conducted comparing different intensities after isocaloric exercise sessions. The absence of difference on HRR after an iscolaroic exercise session mayexplain the slower HRR after higher intensity.In addition, to whole caloric expenditure, the contribution of different energy systems may also contribute to understand the role of intensity in HRR.The restoration process after depleting phosphocreatine reserves includes a greater increase in body temperature a sympathetic activity represented by circulating catecholamine (Gaesser & Brooks, 1984; Sedlock et al., 2010). Besides the greater sympathetic activity to delay the HRR, body temperature can also influence HRR (Peçanha et al. 2017b; Peçanha et al., 2020), suggesting that the energy system of contribution may be a potential factor of influence on HRR post-exercise.In light of this, a previous study observed that glycolytic contribution, but not oxidative or ATP-PCr contribution during exercise was associated withHRR (Lopes-Silva et al. 2015). This is contradictory, since glycolytic contribution represents a larger relative contribution in higher intensities of exercise, but usually studies point to a slower HRR being associated with the intensity of exercise(Al Haddad et al., 2011; Bartels, 2017; Hagberg et al. 1980; Mann et al., 2014; Matsuo et al., 2014).Thus, to test the possible influence of expenditure uptake and to analyze the energy system of contribution during exercise on HRR might help to understand better the role of intensity of exercise on HRR. To the best of our knowledge, no study has been conducted to investigate whether the differences observed between high and moderate intensities remain between moderate and low intensity.

Thus, the aim of the study was to investigate whether different intensities conducted in an isocaloric aerobic exercise session affect HRR and its association with energy systems of contribution. The hypothesis was that moderate intensity exercise would promote a slower HRR than low intensity exercise.

Material & methods

Participants

Nine recreationally active and young adult men participated in this study. The criteria to take part of the study included, do not present any cardiorespiratory disease, smoking habits, neither taking over-the-counter nor prescription medications at the time of the study. The study was approved by Ethical Committee (Protocol 2.020.766). All the subjects signed the information consent form prior took part of the study, which was conducted in accordance with the latest review of Declaration of Helsinki.

Procedure

The experimental protocol was composed by four visits in different days. The first visit was used for screening the subjects, with preliminary exams to confirm the study criteria, which included the Physical Activity Readiness Questionnaire (PAR-Q) (Shephard et al., 1981), besides measurement of height, weight and a maximal cardiopulmonary exercise test until volitional fatigue in a treadmill (Super ATL, Inbraesporte, Brasil). The protocol was consisted of three minutes warm up at 9 $km.h^{-1}$, and followed by an increment of 1 km.h-1 each three minutes until voluntary exhaustion. This test was used to determine the VVO2max (e.g. the lower velocity that the VO2max remains longer than one minute (Davis, 1985), the anaerobic threshold (Lan), and the respiratory compensation point (RCP) were determined based on Skinner and McLellan (1980) criteria by two different evaluators, and a third one was consulted to solve discrepancies. Oxygen uptake was continuously measured by a metabolic cart (Metalyzer 3B, Cortex, Germany) and VO2max was determined as an increase in VO_2 lower than 2.1 $ml.kg^{-1}*min^{-1}$ during the last two stages, or alternatively following these criteria when a VO_2 plateau was not present: i) respiratory exchange >1.10; ii) HR ≥90% of the predicted by the age; and iii) until the subject could not keep the required velocity. The second visit was used to determine the caloric expenditure to be used during the experimental sessions. For this, a constant workload test was conducted at VVO_2max until the volitional fatigue. The oxygen consumption during this test was used to determine the duration of exercise for experimental sessions ensuring the same caloric expenditure. Then, the VO_2 was calculated for experimental sessions (mean of oxygen consumption*time of exercise).

The two experimental sessions were conducted in a crossover design, which the subjects underwent two different exercise sessions: one low intensity exercise session (LI) conducted at velocity 10% lower than velocity at Lan and; one moderate intensity exercise session (MI) conducted at velocity 10% lower than velocity at RCP. The sessions were performed in a randomized and balanced order (McArdle et al., 2001). In addition, oxygen consumption was measured during all exercise in the experimental sessions and the caloric expenditure was calculated assuming that each O_2 liter is equal than 5kcal (Gastin, 2001).

All exercise tests and experimental sessions were separated for at least 72 hours and maximal 7 days each, always during the same time of day, and under controlled temperature (~21ºC). All subjects were instructed to remain fasten from stimulating substances, such as alcoholic and caffeinated beverages 48 hours, as well exercise 24 hours before each test or experimental session.

Data collection and analysis

During the experiments, the RR intervals were continuously recorded during exercise and recovery using a HR monitor (Polar, RS800CX, Finland), and transmitted to the Polar Pro Trainer Software® (v. 5.0, Polar Inc., Kempele, Finland) where the signal was automatically inspected and corrected if needed by a moving average filter. Data was exported to Matlab (The Math Works, Natick, MA) for post-exercise HRR and HR variability (HRV). The HRR was assessed by calculating the following indexes: i) HRR30s; ii) HRR60s and HRR300s – percentage of HR reduction after 30s, 60s and 300s of recovery in relation to the peak of HR; T30 index - short-term time constant of HRR, obtained from the negative reciprocal of the linear regression line between HR and the time at the first 30 s of recovery; and HRRt index - long-term time-constant HRR obtained after exponential fitting of first order during the entire 300s of recovery (Peçanha et al., 2017). The HRV was assessed calculating the square root of the mean squared difference of 30-s non-overlapped segments of successive RR intervals (rMSSD30s). To avoid any transient outliers in the rMSSD30s plots, a median filter operation was employed on RR interval time series (Goldberger et al., 2006). Capillary blood samples of 25µl were collected from the earlobe to determine blood lactate concentrations pre-exercise, and during post-exercise recovery immediately after the exercise, at third and fifth minute (ACCUNTRED PLUS ® Roche, Basel, Switzerland). Regarding the highest value of blood lactate was considered during post-exercise recovery for analysis. The subjects reported their rate of perceived exertion (RPE) on a 6-20 Borg scale immediately after the completion of the exercise with the single question ("how hard were you working at the end of the exercise").

The ventilatory variables (i.e. total and mean oxygen consumption) were measured breath by breath using a metabolic cart (Metalyzer 3B, Cortex, Germany). The VO2 during exercise was determined by the average value and the total oxygen consumption during exercise, which was expressed as related to the body mass ($ml.kg^{-1}.min^{-1}$).

The energy systems of contribution, oxidative, glycolytic and ATP-PCr, were estimated by considering the VO_2 during exercise, the highest value of blood lactatepost-exercise and the fast phase of excess post-exercise oxygen consumption ($EPOC_{FAST}$). Oxidative system derived from the difference between the oxygen uptake assessed at resting($VO_{2\ baseline}$)and the VO_2 assessed during the exercise. The $VO_{2\ baseline}$ was measured after the subjects remain seated for 5 min quiet at rest, and then was considered the average of breath-by-breath values between 5^{th} and6^{th} min (Bertuzzi, et al., 2007). Glycolytic systemcontribution was calculatedbased on blood lactate concentration post-exercise assuming that each 1 $mmol*L^{-1}$ is equivalent to 3 ml $O_2kg^{-1}$of body mass (di Prampero& Ferretti,1999). The ATP-PCr system was estimated considering the $EPOC_{FAST}$(Ozyener2001).

Statistical analysis

Based on a previous study in the literature, a power analysis indicated the required sample size for a power of 80%, and alpha error of 5%, as a minimum of 7 subjects to show a difference of 8 ± 5 bpm18 in HRR60s (GPower V.3.1.5, Kiel, Germany) (Buchheit et al., 2007). Outlier data were checked by the box plot graphics. Normal or non-normal distribution of data was tested by Shapiro-Wilk (SPSS for windows, IBM, Chicago, IL). Paired t-test or Wilcoxon test was applied to compare HRR indexes (HRR30s; HRR60s; HRR300s; T30; HRRt) between LI and MI sessions. A two-way ANOVA for repeated measures was used to compare rMSSD30s during recovery time, regarding sessions (LI and MI) and stage (windows of 30s) as the main factors, blood lactate regarding sessions (LI and MI) and stage (pre vs. post-exercise); and energy system of contribution, main factor sessions (LI and MI) and energy systems (oxidative, glycolytic and ATP-PCr). Post hoc comparisons were made using the Newman-Keuls test (Statsoft, Statistic for windows, Tulsa, OK).

Pearson or Spearman correlations were used to analyze the association between energy system contribution and HRR indexes. All data are presented as mean±standard deviation, and $P<0.05$ was set as significant.

Results

The subjects' physical and function characteristics is shown in the table 1.

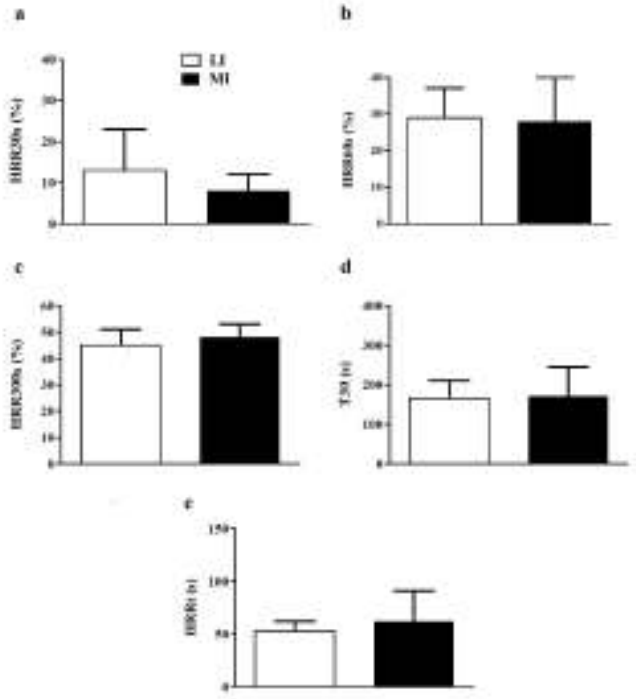
Table 1. Anthropometric and functional characteristics of the subjects.

N	9
Age (years)	27.2 ± 6.6
Weight (kg)	74.7 ± 5.8
Height (cm)	176 ± 4
Body mass index (Kg/m^2)	24.1 ± 5.4
Velocity 10%< Lan ($km.h^{-1}$)	11.6 ± 1.1
VO_2 10%< Lan ($ml.kg.min^{-1}$)	42.7 ± 3.8
Velocity 10%< RCP ($km.h^{-1}$)	14.8 ± 1.1
VO_2 10%< RCP ($ml.kg.min^{-1}$)	51.4 ± 3.4
VVO_2max ($km.h^{-1}$)	16.8 ± 1.2
VO_2max ($ml.kg.min^{-1}$)	61.3 ± 5.4

Note: VO2 - oxygen uptake; VVO2 – velocity related to VO2max – Lan - anaerobic threshold; RCP – respiratory compensation point. Values in mean±standard deviation.

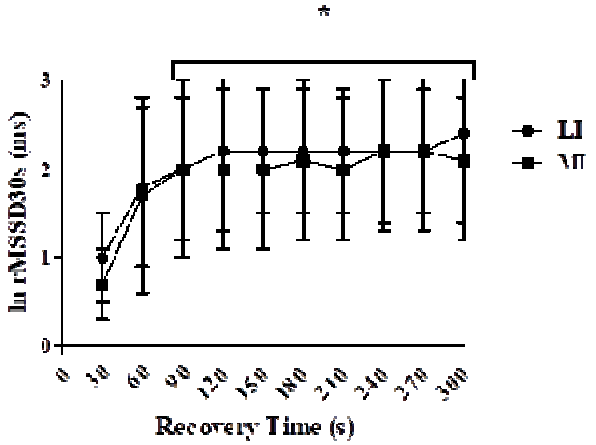
Caloric expenditure (69 ± 14 vs. 71 ± 13 kcal, P=0.07) were not different,while the duration of exercise was longer(4:08 ± 1:02 vs. 3:24 ± 0:45 m:s, P=0.00)in the LI and MI, respectively. Regarding, mean VO2 (39.7 ± 3.2 vs. 45.4 ± 2.9 ml.kg.min^{-1}, P=0.04), peak HR (154 ± 16 vs. 171 ± 11 bpm, P=0.00) and perceived exertion (8.9 ± 2.3 vs. 11.7 ± 1.8 a.u, P=0.04) were lower in the LI than MI, respectively. HRR analysis demonstrated that the indexes were not different between LI and MI intensities (HRR30s 13 ± 10 vs. 8 ± 4 %, P=0.19; HRR60s 29 ± 8 vs. 28 ± 12 %, P=0.98; HRR300s 45 ± 6 vs. 48 ± 5 %, P=0.11; T30 166 ± 44 vs. 171 ± 73 s, P=0.79; HRRt 53 ± 9 vs. 62 ± 29 s, P=0.84), respectively (Figure 1, panels A, B, C, D, and E).

Figure 1. Heart rate recovery indexes assessed post-exercise conducted at low intensity and moderate intensity.



Note: Heart rate recovery indexes assessed post-exercise conducted at low intensity (LI - white bar) and moderate intensity (MI - black bar). a - Heart rate percentage decrease after 30s of recovery (HRR30s); b - Heart rate percentage decrease after 60s of recovery (HRR60s); c - Heart rate percentage decrease after 300s of recovery (HRR300s); d – short-term time-constant of heart rate decay (T30); e – Long-term time-constant of heart rate decay (HRRt). HRV analysis presented no interaction between experimental session and time, but time was observed as a significant main factor. The rMSSD30s was significantly greater throughout the 60 s window (main factor session, P=0.00) (Figure 2).

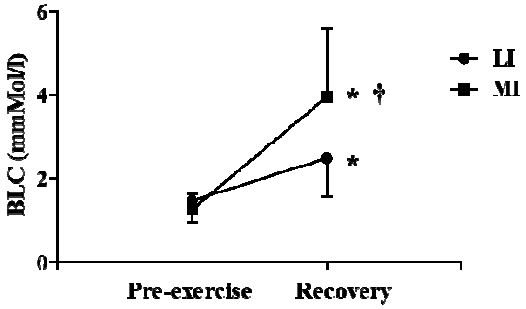
Figure 2. Post-exercise heart rate variability index evaluated across 300s of post-exercise recovery conducted at low intensity and moderate intensity.



Note: Post-exercise heart rate variability index evaluated across 300s of post-exercise recovery conducted at low intensity (LI ● -) and moderate intensity (MI -▲-). Square root of the mean of the sum of the squares of differences between adjacent RR intervals in segments of 30s (RMSSD). † Significant different from 30s and 60s windows, $p < 0.05$. Data showed as mean ± SD.

The blood lactate was increased during post-exercise period, and it was significantly greater in MI than LI (3.94 ± 1.64 vs. 2.48 ± 0.91 mmMol/l, P=0.01) (Figure 3).

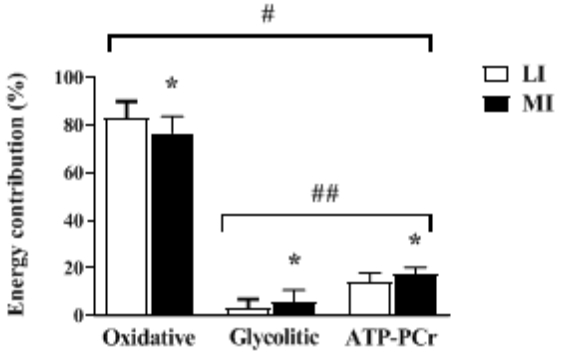
Figure 3. Blood lactate (BLC) measured pre-exercise and post-exercise recovery conducted at low intensity and moderate intensity.



Note: Blood lactate (BLC) measured pre-exercise and post-exercise recovery conducted at low intensity (LI ●-) and moderate intensity (MI -▲-). * Significantly different from pre-exercise in the same exercise session, † significantly different from recovery in LI, $P < 0.05$. Data showed as mean ± SD.

The energy contribution of systems demonstrated a greater oxidative contribution during LI, while glycolytic and ATP-PCr were greater during MI exercise session. Considering each exercise session separately, the oxidative contribution was greater followed by ATP-PCr energy system(Figure 4).

Figure 4. Estimative of each energy system of contribution during exercise.



Note: Estimated relative contribution of each energy system during exercise conducted at low intensity (LI - white bar) and moderate intensity (MI - black bar).* Significantly different from LI session, # significantly different from glycolytic and ATP-PCr contribution system, ## significantly different from ATP-PCr contribution system, $P < 0.05$. Data showed as mean ± SD.

Correlations between the relative contribution of each energy system and HRR indexes are shown in the table 2. After LI, HRR30s was positively associated with oxidative contribution and negatively associated to ATP-PCr contribution, while HRR60s was negatively associated with glycolitc contribution, and T30 was negatively associated with oxidative contribution and positively associated with ATP-PCr contribution. No associations were observed after MI.

Table 2. Correlations between estimated contribution of each energy systems and HRR indexes.

	LI						MI					
	Oxidative		Glycolytic		ATP-PCr		Oxidative		Glycolytic		ATP-PCr	
	R	p	R	**P**	**R**	p	**R**	P	R	**P**	**R**	P
HRR30s(%)	+0.80	0.02	-0.69	0.06	-0.77	0.02	+0.02	0.96	-0.21	0.59	+0.33	0.39
HRR60s(%)	+0.62	0.10	-0.71	0.04	-0.42	0.30	+0.27	0.48	-0.35	0.35	-0.05	0.90
HRR300s(%)	+0.49	0.91	-0.15	0.73	+0.05	0.91	+0.12	0.76	-0.28	0.47	+0.20	0.61
T30(s)	-0.71	0.47	+0.53	0.18	+0.77	0.02	-0.10	0.81	+0.20	0.60	-0.12	0.75
HRRt(s)	-0.62	0.14	+0.62	0.14	+0.52	0.23	+0.17	0.69	-0.09	0.84	-0.41	0.32

Note:HRR30s - Heart rate percentage decrease after 30s of recovery; HRR60s - Heart rate percentage decrease after 60s of recovery; HRR300s - Heart rate percentage decrease after 300s of recovery; T30 – short-term time-constant of heart rate decay; HRRt – Long-term time-constant of heart rate decay.

Discussion

The main findings in this study were thatcaloric expenditure seems to play a role in HRR, since no differences were observed between LI and MI sessions for HRR indexes and that greater oxidative contribution and lower ATP-PCr contribution are associated with cardiac parasympathetic reactivation.

Most studies investigating the effect of exercise intensity on HRR post-exercise responses compared high (i.e. above RCP or maximal exercise) with moderate exercises (Buchheit, 2014). However, it is unclear if intensity-mediated differences are still present when comparing low- and moderate-intensity exercise. In fact, the low-intensity session performed in the present study was below Lan, which is expected to not elicit a significant metabolic stress and, therefore, not stimulating the muscular metaboreflex, which may influence the HRR (Buchheit et al., 2007; Peçanha et al., 2017), or at least in lower level, which was observed in the current study. The difference between intensities was effective since the peak HR and the mean VO2 were lower in LI than MI and keeping the same caloric expenditure.

Immediately after a physical stress, such as an acute exercise bout, the cardiac autonomic control restoration depends on changes of some factors altered during exercise. The first minute is mainly determined by parasympathetic reactivation, known as fast phase of HRR, followed by sympathetic withdrawal to restore the cardiac autonomic control, known as the slow phase (Peçanha et al., 2017). Previous studies have suggested that high intensity exercise promotes a lower parasympathetic reactivation than moderate exercise (Mann et al., 2014; Peçanha et al., 2013), which may be related to the metabolites accumulated and the increase in temperature (Buchheit et al. 2007; Stanley et al., 2013). Exercises conducted at high intensities also promote higher sympathetic activity, which is known to blunt cardiac autonomic restoration (Ushijima et al., 2009). This pattern is usually observed in studies comparing different exercise intensities that may be observed through the higher values of HR, as we observed in the current study, comparing HR values during MI and LI. Nonetheless, the isocaloric exercise sessions were not able to be different in the current study that is probably because the short duration of exercise based on the constant workload test in VVO2max.The slight difference of intensity between exercise sessions may also explain the absence of differences in HRR, while previous studies compared moderate to high or even to supra maximal intensities (i.e. above VO_2max), the current study compared low with moderate intensity. The mean oxygen uptake during exercise is associated with HRR indexes (Buchheit et al., 2007); despite significantly different, the mean $VO_2$was only ~5 ml.kg.min^{-1}lower in LI compared with MI inthe current study, which may have masked differences between LI and MI.

It is worthy to note that none of those studies were controlled by the caloric expenditure (Bartels et al., 2018; Mann et al., 2014; Peçanha et al., 2013). Another important aspect is the duration of exercise, which might implicate in a higher reduction of plasma volume that influences cardiac autonomic control (Buchheit et al., 2009). The current study reported 44 seconds longer in LI, which despite statistically different was not enough toinfluence the results inHRR indexes. Moreover, although the hydration was not allowed during the study, a significant difference of liquid loss between sessions was not expected. Then, it suggests that the model of exercise used was enable to investigate the role of caloric expenditure on HRR avoiding liquid loss as a confounder. Regarding the energy system of contribution, the greater contribution of oxidative system than glycolytic and ATP-PCrwas observed in both exercise sessions. In addition, when each energy system was compared between sessions, LI presented a greater contribution of oxidative systemthan MI (83±7 vs. 77±7, %), while MI presented a greater contribution of glycolytic (3±4 vs. 6±5, %) and ATP-PCr (14±4 vs. 17±3, %) systems, as expected (McCrudden et al., 2017).The association among parasympathetic reactivation indexes (HRR30s, HRR60s and T30) and energy systems of contribution was a novelty of this study, which curiously was only observed in LI, suggesting that energy systems are associated with parasympathetic reactivation but not with sympathetic withdrawalafter an aerobic exercise of low intensity. Impaired parasympathetic reactivation was already associated with greater anaerobic contribution (i.e. Glycolytic and ATP-PCr) during high intensity exercise (i.e. supra PCR) and repeated sprints (all out) (Buchheit et al., 2007). However, oxidative contribution was still not explored. Thus, this result may have a practical application considering training sessions for active recovery inter-set effect (i.e. repeated sprints) during a training session or considering a training session for recovery. In addition, the absence of this association in MI might be explained by the greater levels of blood lactate and sympathetic activity expressed on the higher HR, both known for influencing HRR (Peçanha et al., 2016; Ushijima et al., 2009), and then may mask any association about HRR indexes and energy systems of contribution.

This study presents some limitations. The current study focused only on low and moderate intensities, and the results cannot be extrapolated to supra RCP exercise intensities. The exercise-model used was short to avoid any effect of liquid loss as a confounder, and we are not able to ensure that no differences would not be found after a longer exercise session. The subjects were recreationally active men, and adult women usually present better HRR indexes than men (de Mendonca et al., 2017; Mendonca et al., 2010). Since it was the first study investigating the effect of caloric expenditure in HRR and its association with energy systems of contribution, we opted to investigate only men, then our results should not extrapolated to women. Additionally, as an elite athlete group was not included (i.e. Olympic level), the results should not be attributed to all level of athletes. Future studies should investigate different group of athletes in a number of exercise conditions.

Conclusions

In recreationally active men, caloric expenditure seems to play a role incardiac autonomic restoration that reflects onHRR indexes after low and moderate intensity of a short matched-caloric expenditure aerobic exercise session.In addition,parasympathetic reactivationis associated with greater oxidative contribution, but lower glycolytic and ATP-PCr energy systems of contributionfavor a faster cardiac restoration only after exercise of low intensity. Thus, the intensity of exercise influences the association between energy systems of contribution during exercise and cardiac parasympathetic reactivation.

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References:

Albert, C. M., Mittleman, M. A., Chae, C. U., Lee, I. M., Hennekens, C. H., & Manson, J. E. (2000). Triggering of sudden death from cardiac causes by vigorous exertion. *The New England journal of medicine*, *343*(19), 1355–1361.

Al Haddad, H., Laursen, P. B., Chollet, D., Ahmaidi, S., &Buchheit, M. (2011). Reliability of resting and postexercise heart rate measures. *International journal of sports medicine*, *32*(8), 598–605.

Bartels, R., Prodel, E., Laterza, M. C., de Lima, J., & Peçanha, T. (2018). Heart rate recovery fast-to-slow phase transition: Influence of physical fitness and exercise intensity. *Annals of noninvasive electrocardiology: the official journal of the International Society for Holter and Noninvasive Electrocardiology, Inc*, *23*(3), e12521.

Bertuzzi, R. C., Franchini, E., Kokubun, E., & Kiss, M. A. (2007). Energy system contributions in indoor rock climbing. *European journal of applied physiology*, *101*(3), 293–300.

Buchheit, M., Laursen, P. B., &Ahmaidi, S. (2007). Parasympathetic reactivation after repeated sprint exercise. *American journal of physiology. Heart and circulatory physiology*, *293*(1), H133–H141.

Buchheit, M., Papelier, Y., Laursen, P. B., &Ahmaidi, S. (2007). Noninvasive assessment of cardiac parasympathetic function: postexercise heart rate recovery or heart rate variability? *American journal of physiology. Heart and circulatory physiology*, *293*(1), H8–H10.

Buchheit, M., Laursen, P. B., Al Haddad, H., &Ahmaidi, S. (2009). Exercise-induced plasma volume expansion and post-exercise parasympathetic reactivation. *European journal of applied physiology*, *105*(3), 471–481.

Buchheit M. (2014). Monitoring training status with HR measures: do all roads lead to Rome?.*Frontiers in physiology*, *5*, 73.

Daanen, H. A., Lamberts, R. P., Kallen, V. L., Jin, A., & Van Meeteren, N. L. (2012). A systematic review on heart-rate recovery to monitor changes in training status in athletes. *International journal of sports physiology and performance*, *7*(3), 251–260.

Davis J. A. (1985). Anaerobic threshold: review of the concept and directions for future research. *Medicine and science in sports and exercise*, *17*(1), 6–21.Gastin P. B. (2001). Energy system interaction and relative contribution during maximal exercise. *Sports medicine (Auckland, N.Z.)*, *31*(10), 725–741.

Goldberger, J. J., Le, F. K., Lahiri, M., Kannankeril, P. J., Ng, J., &Kadish, A. H. (2006). Assessment of parasympathetic reactivation after exercise. *American journal of physiology. Heart and circulatory physiology*, *290*(6), H2446–H2452.

Hagberg, J. M., Hickson, R. C., Ehsani, A. A., &Holloszy, J. O. (1980). Faster adjustment to and recovery from submaximal exercise in the trained state. *Journal of applied physiology: respiratory, environmental and exercise physiology*, *48*(2), 218–224.

Iellamo, F., Volterrani, M., Di Gianfrancesco, A., Fossati, C., &Casasco, M. (2018). The Effect of Exercise Training on Autonomic Cardiovascular Regulation: From Cardiac Patients to Athletes. *Currentsports medicine reports*, *17*(12), 473–479.

Lopes-Silva, J. P., Silva Santos, J. F., Branco, B. H., Abad, C. C., Oliveira, L. F., Loturco, I., & Franchini, E. (2015). Caffeine Ingestion Increases Estimated Glycolytic Metabolism during Taekwondo Combat Simulation but Does Not Improve Performance or Parasympathetic Reactivation. *PloS one*, *10*(11), e0142078.

Mann, T. N., Webster, C., Lamberts, R. P., & Lambert, M. I. (2014). Effect of exercise intensity on post-exercise oxygen consumption and heart rate recovery. *European journal of applied physiology*, *114*(9), 1809–1820.

Matsuo, T., Saotome, K., Seino, S., Eto, M., Shimojo, N., Matsushita, A., Iemitsu, M., Ohshima, H., Tanaka, K., & Mukai, C. (2014). Low-volume, high-intensity, aerobic interval exercise for sedentary adults: VO_2max, cardiac mass, and heart rate recovery. *European journal of applied physiology*, *114*(9), 1963–1972.

McArdle, W.D.; Katch, F.I.; Katch, V.L. (2001), Treinamento para potênciaanaeróbica e aeróbica. pag 470-512. In: McArdle, W. D.; Katch, F. I.; Katch, V. L. Fisiologia do Exercício: energia, nutrição e desempenho humano. Rio de Janeiro: Guanabara Koogan SA.

McCrudden, M. C., Keir, D. A., & Belfry, G. R. (2017). The effects of short work vs. longer work periods within intermittent exercise on $\dot{V}o_{2p}$ kinetics, muscle deoxygenation, and energy system contribution. *Journal of applied physiology (Bethesda, Md. : 1985)*, *122*(6), 1435–1444.

Mendonca, G. V., Heffernan, K. S., Rossow, L., Guerra, M., Pereira, F. D., &Fernhall, B. (2010). Sex differences in linear and nonlinear heart rate variability during early recovery from supramaximal exercise. *Applied physiology, nutrition, and metabolism = Physiologieappliquee, nutrition et metabolisme*, *35*(4), 439–446.

Mendonca, G. V., Teodósio, C., & Bruno, P. M. (2017). Sexual dimorphism in heart rate recovery from peak exercise. *European journal of applied physiology*, *117*(7), 1373–1381.

Murrell, C., Wilson, L., Cotter, J. D., Lucas, S., Ogoh, S., George, K., & Ainslie, P. N. (2007). Alterations in autonomic function and cerebral hemodynamics to orthostatic challenge following a mountain marathon. *Journal of applied physiology (Bethesda, Md.: 1985)*, *103*(1), 88–96.

Ozyener, F., Rossiter, H. B., Ward, S. A., &Whipp, B. J. (2001). Influence of exercise intensity on the on- and off-transient kinetics of pulmonary oxygen uptake in humans. *The Journal of physiology*, *533*(Pt 3), 891–902.

Pearson, M. J., & Smart, N. A. (2018). Exercise therapy and autonomic function in heart failure patients: a systematic review and meta-analysis. Heart failure reviews, 23(1), 91–108.

Peçanha, T., Bartels, R., Brito, L. C., Paula-Ribeiro, M., Oliveira, R. S., &Goldberger, J. J. (2017). Methods of assessment of the post-exercise cardiac autonomic recovery: A methodological review. *Internationaljournalofcardiology*, *227*, 795–802.

Peçanha, T., de Brito, L. C., Fecchio, R. Y., de Sousa, P. N., da Silva Junior, N. D., de Abreu, A. P., da Silva, G. V., Mion-Junior, D., & Forjaz, C. L. (2016). Metaboreflex activation delays heart rate recovery after aerobic exercise in never-treated hypertensive men. *The Journal of physiology*, *594*(21), 6211–6223.

Peçanha, T., Prodel, E., Bartels, R., Nasario-Junior, O., Paula, R. B., Silva, L. P., Laterza, M. C., & Lima, J. R. (2014). 24-h cardiac autonomic profile after exercise in sedentary subjects. *International journal of sports medicine*, *35*(3), 245–252.

diPrampero, P. E., & Ferretti, G. (1999). The energetics of anaerobic muscle metabolism: a reappraisal of older and recent concepts. *Respiration physiology*, *118*(2-3), 103–115.

Shephard, R. J., Cox, M. H., & Simper, K. (1981). An analysis of "Par-Q" responses in an office population. *Canadian journal of public health = Revue canadienne de sante publique*, *72*(1), 37–40.

Skinner, J. S., & McLellan, T. M. (1980). The transition from aerobic to anaerobic metabolism. *Research quarterly for exercise and sport*, *51*(1), 234–248.

Stanley, J., Peake, J. M., &Buchheit, M. (2013). Cardiac parasympathetic reactivation following exercise: implications for training prescription. *Sports medicine (Auckland, N.Z.)*, *43*(12), 1259–1277.

Ushijima, A., Fukuma, N., Kato, Y., Aisu, N., & Mizuno, K. (2009). Sympathetic excitation during exercise as a cause of attenuated heart rate recovery in patients with myocardial infarction. *Journal of Nippon Medical School = Nippon IkaDaigakuzasshi*, *76*(2), 76–83.

Wilson, M. G., Ellison, G. M., & Cable, N. T. (2015). Basic science behind the cardiovascular benefits of exercise. *Heart (British Cardiac Society)*, *101*(10), 758–765.