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Parasympathetic modulation during sleep time is reduced after maximal exercise, correlated with aerobic fitness in young women

A modulação parassimpática durante o sono é reduzida após o exercício máximo e está correlacionada com a aptidão aeróbia em mulheres jovens

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Abstract – It is known that cardiovascular risk is increased during exercise and recovery. Thus, it is necessary to assess all the risk associated with exercise to minimize the possibility of cardiovascular events. The aim of this study was to verify whether a maximal exercise alters ambulatory cardiac autonomic modulation in untrained women and whether aerobic fitness is correlated to cardiac autonomic modulation. Twelve women (25.35 ± 5.44 years) were outfitted with the Holter monitor on an experimental (after maximum exercise) and a control day to heart rate variability (HRV) evaluation. Maximal exercise increased 24 h heart rate (82 ± 14 vs 77 ± 11 bpm; $p = 0.04$) and during sleep time (72 ± 14 vs. 65 ± 9 bpm; p = 0.01), reduced parasympathetic modulation (HF – n.u. 49.96 ± 11.56 vs 42.10 ± 14.98; $p = 0.04$), and increased low-frequency/high-frequency ratio (2.88 ± 3.24 *vs* 1.31 ± 0.60; $p = 0.03$) during sleep time compared to the control day. Aerobic fitness was correlated positively with LF, HF, and HF (n.u.) indices ($r = 0.61$ to 0.73, $p < 0.05$) and correlated negatively with LF (n.u.) and LF/HF ratio (*Rho* = - 0.57 to - 0.69; $p < 0.05$). Maximal exercise alters parasympathetic modulation during sleep time in untrained women. Ambulatory cardiac autonomic modulation after exercise is related to aerobic fitness.

Keywords: Heart rate; Heart rate variability; Intense exercise; Maximum oxygen uptake.

Resumo – *Sabe-se que o risco cardiovascular aumenta durante o exercício e sua recuperação. Assim, é necessário avaliar todo o risco associado ao exercício para minimizar a chance de eventos cardiovasculares. Objetivou-se verificar se um exercício máximo altera a modulação autonômica cardíaca ambulatorial em mulheres não treinadas e se a aptidão aeróbia está correlacionada à modulação autonômica cardíaca. Doze mulheres (25,35 ± 5,44 anos) foram equipadas com monitor Holter em um dia experimental (após exercício máximo) e dia controle para avaliação da variabilidade da frequência cardíaca (VFC). O exercício máximo aumentou a frequência cardíaca de 24 h (82 ± 14 vs 77 ± 11 bpm; p = 0,04) e durante o sono (72 ± 14 vs 65 ± 9 bpm; p = 0,01), bem como reduziu a modulação parassimpática (HF - nu 49,96 ± 11,56 vs 42,10 ± 14,98; p = 0,04) e aumentou a razão de baixa frequência / alta frequência - LF/HF (2,88 ± 3,24 vs 1,31 ± 0,60; p = 0,03) durante o período do sono em comparação com o dia controle. A aptidão aeróbia foi correlacionada positivamente com os índices LF, HF e HF (nu) (r = 0,61 a 0,73, p <0,05) e negativamente correlacionada com LF (nu) e razão LF / HF (Rho = - 0,57 a - 0,69; p <0,05). O exercício máximo altera a modulação parassimpática durante o sono em mulheres não treinadas. A modulação autonômica cardíaca ambulatorial após o exercício foi correlacionada com a aptidão aeróbia.*

Palavras-chave: *Frequência cardíaca; Variabilidade da frequência cardíaca; Exercício intenso; Consumo máximo de oxigênio*

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INTRODUCTION

Heart rate variability (HRV) and heart rate (HR) after exercise are non-invasive monitoring tools used to assess cardiac autonomic functioning, and important in the cardiac risks stratification[1] even in asymptomatic[2] or healthy[3] individuals. A sleep variables analysis can be useful in evaluating responses to exercise and for the prescription of an appropriate training program[4]. A recent study has verified that autonomic nervous system alterations during sleep may be present a long time before the onset of a cardiovascular event[5].

Aerobic fitness has been recognized as responsible for adaptations in the cardiovascular and cardiac autonomic system, enabling the individual to adapt and respond more effectively to post-exercise[6,7]. On the other hand, individuals with little aerobic fitness have a larger cardiovascular window of exposure after physical exercise[8], as well as a need for greater recovery time between exercise sessions. The maximal incremental test is commonly used in clinical practice[9] and for the determination of the maximum oxygen uptake to characterize health status or training[1,10].

Although women present an autonomic balance in favor of parasympathetic modulation compared to men[11], minor differences in aerobic fitness may be relevant to improve the autonomic cardiac modulation response to physiological stress recovery in untrained women. One previous study[10] has reported an attenuated parasympathetic nervous modulation in sleep time after high-intensity exercise. On the other hand, a recent study[12] verified attenuated parasympathetic nervous modulation only in the first-hour post maximal exercise. A third study[13] observed that the nighttime period was more responsive to the positive effects of moderate-intensity exercise on cardiac autonomic modulation.

Considering that sex is a significant predictor of differences in HRV[14], that the responsiveness to exercise might be dependent on the resting status, and that several studies analyzed only men[1,10,12,13,15], we suggest here that studies to understand all the risks associated with recovery from maximum exercise be designed to specifically consider sex in order to increase their ecological validity. This study aimed to verify whether maximal exercise alters the ambulatory cardiac autonomic modulation in untrained women and whether aerobic fitness is correlated to cardiac autonomic modulation. We hypothesized that maximal exercise reduces ambulatory parasympathetic modulation and that aerobic fitness is correlated with these changes.

METHODS

Participants

We recruited the participants through advertisements on the university campus, and 49 individuals showed interest. Of these, 17 women 18 to 35 years old met the inclusion criteria, and 12 performed all assessments. The exclusion criteria were: smoking, use of medications continuously (except for oral contraceptives), muscle and joint problems, chronic-degenerative diseases, shift workers, engagement in regular physical exercise programs

in the previous four months, being pregnant, breastfeeding, or during the first week of the follicular phase of the menstrual cycle. The study procedures followed the ethical guidelines outlined in the Declaration of Helsinki and were approved by the Committee for Ethics in Research in Humans (nº. 65485217.5.0000.5541). All participants signed an Informed Consent Form.

Procedures

Two visits, with a minimum interval of 72 h and between 4 and 8 pm, were made to the laboratory. The test area was kept at a temperature of 23.17 ± 1.64 ˚C, and relative air humidity of 44.17 ± 5.51%, for both the control and experimental days, which occurred randomly (Figure 1). The participants were instructed not to drink alcohol or stimulants (*i. e.* coffee, tea, and soft drinks) and not to practice vigorous physical activities in the 24 h before both visits to the laboratory. On the first visit, a health history questionnaire about smoking status, medication use, and previous diseases was carried out. Anthropometric measures, and blood pressure were measured as well.

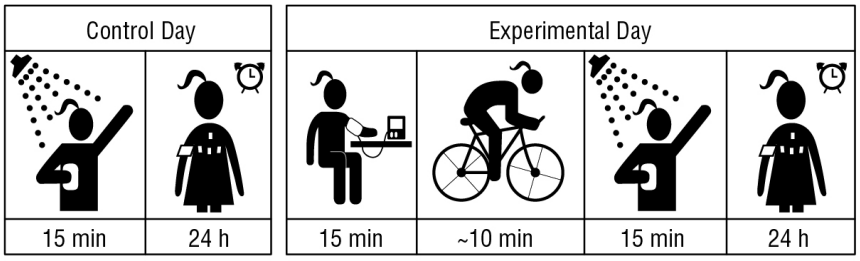

Figure 1. Experimental design.

Anthropometric measures and body composition

Body mass (CAMRY® scale, 100 g, Brazil) and height (SANNY stadiometer, 0.1 cm, Brazil) were measured to determine body mass index; abdominal circumference was obtained (CARDIOMED® tape measure, 0.1 cm, Brazil). The percentage (%) of body fat was determined through bioimpedance, with the participant in dorsal decubitus on a stretcher with no interferences for the electrical conduction (MALTRON, BF 907®, Australia).

Blood pressure

Systolic and diastolic blood pressure were determined in the left arm after 15 min at resting, using an automatic sphygmomanometer (Microlife, BP 3BT0-A®, Brazil) as has been previously described[16].

Control day

The Holter monitor was placed with no previous physical exercise. After skin asepsis, using gauze soaked in 70% alcohol, the participants were outfitted with a 3-channel Holter monitor (Cardios, CardioMapa, Brazil) by fixing

electrodes, using four precordial derivations in specific anatomical points on the trunk, following the manufacturer's recommendations. The Holter monitor was placed on all participants by the same researcher.

The participants were instructed to obtain 6 to 10 h of sleep at night time, to maintain their usual sleep habits and daily activities (such as hours for meals and sleep periods). The participants were instructed no take off the monitor at the end of the 24 h recording; when wearing the monitor: not to take a shower; not to practice physical exercise; not to sleep during the day, to go to bed before midnight, to record hours of sleep, and when sleeping not to adopt a prone position, and to put the monitor on the bed, next to the head or chest.

Experimental day

After an initial 15 min rest, the incremental exercise test was performed on a cycle ergometer (INBRAMED, CG-04, Brazil), with an initial workload of 30 W and an addition of 15 W every minute, keeping a cadence of 60 rpm, until voluntary exhaustion[17]. The HR maximal was identified at the end of the maximal incremental test (POLAR, RS800CX, Finland). Direct acquisition of exhaled gases was performed every 10 s, with analysis of the volumes relating to oxygen uptake (VO_2) and carbon dioxide production (VCO_2), obtained through spirometry (VO2000 MEDICAL GRAPHICS, USA). Aerobic fitness was determined through peak oxygen uptake (VO_2 peak) from the highest value reached in the final moments of the test. There was no difference between test and retest to the VO_2 peak determined through this protocol[17]. The recovery after the maximal incremental test was performed for five minutes with 15 W at 60 rpm.

All participants reached the criteria adopted to consider the incremental exercise test as maximum[9]: inability to maintain 60 rpm for more than 10 s despite verbal encouragement; 90% predicted maximum HR (220 - age); values in the Borg scale > 17 points; respiratory exchange ratio > 1.1. Then, after 15 min intended for personal hygiene, the participants were equipped with the Holter monitor (Cardios, CardioMapa, Brazil), as described in the control day section.

Data analysis

Ambulatory heart rate variability (HRV)

The HRV analysis was performed using software CardioSmart 540CS (Cardios, Brazil – 800 pulses per second, with a resolution of 12bits). On both days, participants went to bed on average at ~11 p.m., woke up at ~ 6 a.m. For the data analysis, the calculation of the simple arithmetic average was used for continuous segments of 24 h, awake (after maximal exercise up to ~ 11 p. m. and between 6 a.m. and 4 to 8 p. m.), and sleep (~ 11 p. m. until 6 a. m.). The HRV indices were analyzed in the frequency domain, by the Fast Fourier Transform method: LF ms^2 - low-frequency component, with a variation of 0.04 to 0.15Hz, which represents the baroreflex activity[18]; HF

(high-frequency component, with a variation of 0.15 to 0.4Hz) in ms^2 and normalized units (n. u.), representing the parasympathetic modulation[19]; and LF/HF ratio component of low and high-frequency. The power of each spectral component was normalized by dividing the power of each spectrum component by the total variance minus the value of the very-low-frequency component and multiplying the result by 100. The data were analyzed by the software CardioSmart 540CS (Cardios, Brazil). The simple arithmetic averages of the 24 h, awake, and sleep periods were measured. The differences (Δ) between sleep and awake periods on control and experimental days, for each variable, were calculated as being: Δ: sleep – awake.

Statistical analysis

The data normality and homogeneity were tested through the Shapiro-Wilk and Levene's test, respectively. Variables between the experimental and control days were compared using paired the Student's t-test and Wilcoxon for parametric and non-parametric data, respectively. Effect size (ES) was calculated by the Cohen's measure, adopting the following values for its interpretation: small (< 0.5), moderate (> 0.5 and < 0.8), and large (> 0.8). The statistical power (Pr = 1—β) posterior to the main analysis was shown. Pearson's linear correlation was used for parametric data and Spearman's Rank for non-parametric data. The significance level adopted was 5% ($p \leq 0.05$).

RESULTS

Table 1 displays the characteristics of the subjects and the variables performed at maximal exercise.

Table 1. Subjects characteristics and the variables at maximal exercise.

	Mean ± standard deviation
Age (years)	25.35 ± 5.44
Body weight (kg)	56.76 ± 7.56
Body weight index ($kg \cdot m^{-2}$)	21.91 ± 2.33
Abdominal circumference (cm)	77.63 ± 6.71
Body fat (%)	29.05 ± 6.96
Systolic blood pressure (mmHg)	106 ± 11
Diastolic blood pressure (mmHg)	68 ± 11
Maximal incremental test	
Time (min)	9.95 ± 2.08
Maximal workload (w)	150.75 ± 30.56
Maximal heart rate (%)	97.67 ± 5.02
Respiratory quotient	1.17 ± 0.06
Effort perceived	19.83 ± 0.58
VO_{2peak} ($mL \cdot kg^{-1} \cdot min^{-1}$)	31.70 ± 7.32

Note. VO2 peak: peak oxygen uptake.

24 h HR (Figure 2A - 82 ± 14 vs 77 ± 11 bpm; p= 0.04; Pr= 0.34) and during sleep time was higher (72 ± 14 vs. 65 ± 9 bpm; p= 0.01, Pr= 0.67) after maximal exercise compared to the control day, as well as, there was a parasympathetic modulation reduction - Figure 2C and 2D (HF 1132.93 ± 1089.59 *vs* 1802.61 ± 1690.70 - p= 0.04; Pr= 0.45; and HF n. u. 49.96 ±

11.56 vs 42.10 ± 14.98 - *p*= 0.04; Pr= 0.61); and increased the LF n. u. - Figure 2F (57.90 ± 14.98 *vs* 50.03 ± 11.55; p= 0.04; Pr= 0.61) and LF/HF ratio - Figure 2B (2.88 ± 3.24 *vs* 1.31 ± 0.60; *p*= 0.03; Pr= 0.70) during sleep time after maximal exercise.

There were no differences in the delta (Δ: sleep - awake) between the control and experimental day for HR and HRV indices: ΔHR: -18 ± 8 and -14 ± 11 bpm, *p*: 0.01; ES: 0.4; ΔHF: 20.59 ± 10.48; 14.52 ± 10.12 n. u., *p*: 0.11, ES: 1.0, and ΔLF/HF: -2.19 ± 1.14 and -1.49 ± 1.41, *p*: 0.22; ES: 0.6. The experimental day (8.08 ± 1.44 h) presented no difference (p= 0.67) from the control (7.92 ± 1.73 h) in sleep time.

LF (ms^2), HF (ms^2), and HF (n. u.) indices were correlated positively with aerobic fitness (*r*= 0.61 to 0.73; *p*< 0.05), while LF (n. u.) and LF/HF ratio correlated negatively (*Rho*= -0.57 to -0.69; *p*< 0.05) (Figure 3B-D). There was no significant correlation between 24 h HR in experimental day (*r*= -0.53; *p*= 0.07) with aerobic fitness (Figure 3A).

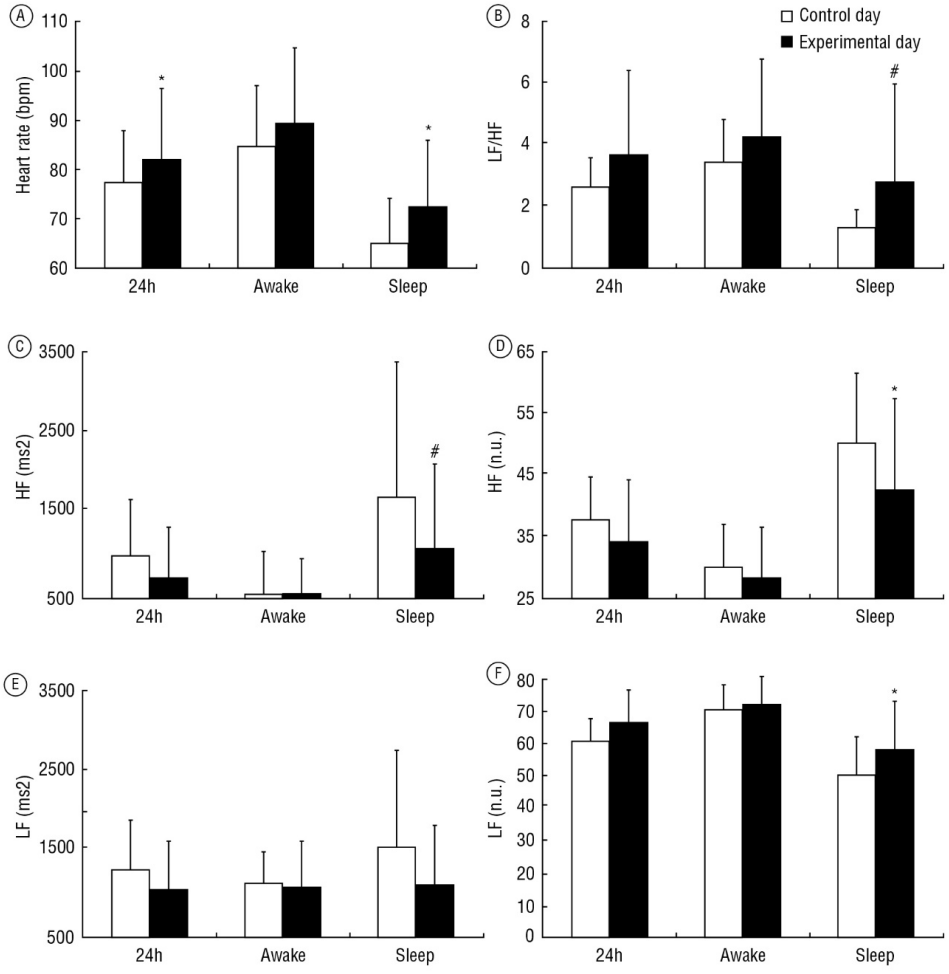

Figure 2. Heart rate and heart rate variability indices at control and experimental day. LF = low-frequency; HF = high-frequency; LF/HF = low and high-frequency ratio; n.u. = normalized units. *Significant difference between days by paired Student's t-test (p ≤ 0.05). #Significant difference between days by Wilcoxon test (p ≤ 0.05). (A) 24 h: p = 0.04; ES: 0.38; Awake: p = 0.16; ES: 0.28; Sleep: p = 0.01; ES: 0.64; (B) 24 h: p = 0.21; ES: 0.52; Awake: p = 0.18; ES: 0.41; Sleep: p = 0.03; ES: 0.67; (C) 24 h: p = 0.14; ES: 0.35; Awake: p = 0.95; ES: 0.02; Sleep: p = 0.04; ES: 0.47; (D) 24 h: p = 0.17; ES: 0.71; Awake: p = 0.55; ES: 0.22; Sleep: p = 0.04; ES: 0.59; (E) 24 h: p = 0.25; ES: 0.36; Awake: p = 0.74; ES: 0.09; Sleep: p = 0.14; ES: 0.47; (F) 24 h: p = 0.13; ES: 0.64; Awake: p = 0.55; ES: 0.22; Sleep: p = 0.04; ES: 0.59.

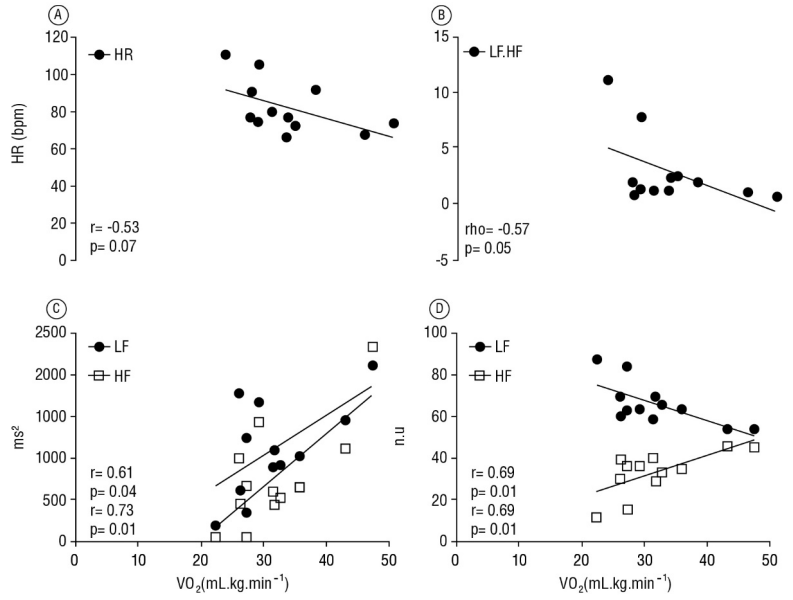

Figure 3. Correlation coefficients (r/Rho) of aerobic fitness with heart rate (A) and HRV indices (B-D) at 24 h at experimental day. LF = low-frequency; HF = high-frequency; LF/HF = low and high-frequency ratio; n.u. = normalized units.

DISCUSSION

The main finding of this study was that, in untrained young women, maximal exercise causes an increase in ambulatory cardiac stress and reduced parasympathetic modulation during sleep time. On the other hand, aerobic fitness was correlated with cardiac autonomic modulation.

The importance of this study is highlighted by the evaluation of HRV considering awake and sleep time, which enables a better understanding of healthy variation across the day, since short periods and clinic values are only indicative of the HRV status of a brief fraction of the entire day. The parasympathetic and sympathetic influence on HR is mediated via cholinergic vagal nerve fibers and by catecholamines, respectively. Although differences in cardiac autonomic modulation between the sexes are well established[11,14], most studies analyze cardiac autonomic modulation exclusively in men[1,10,12,13,15] or trained women[7,20]. To our knowledge, there is no information about ambulatory cardiac autonomic modulation responses to maximal exercise in women, especially those with little aerobic fitness, who represent the majority of the population[21]. The differences between the sexes in HRV indices are due to the female hormones (i. e β-estradiol)[22], involved in the facilitation of cardiac vagal activation[23], and also lower levels of circulating catecholamines, especially under a stressful condition[24].

In the present study, a maximal exercise performed in the afternoon period caused a greater cardiovascular overload in the sleep period. Previously, a parasympathetic modulation reduction during the sleep period after intense exercise in physically active men has been reported[1,10,15]. On the other hand, a recent study[12] verified attenuated parasympathetic nervous modulation only in the first hour post maximal exercise, while and another study[13] observed

that the nighttime period was more responsive to the positive effects of moderate-intensity exercise on cardiac autonomic modulation. However, both studies[12,13] were performed in the morning and with men. The differences in subject characteristics such as physical fitness and sex, the varied duration and intensity of exercise protocols, may also contribute to the differing results reported. Michael et al. observed that longer exercise duration (32 vs 8 min)[25] and higher intensity (high vs moderate or low intensity)[26] attenuated the post-exercise cardiac parasympathetic reactivation.

Aerobic fitness seems to be responsible for adaptations in the cardiovascular system[6,27] enabling the individual to adapt and respond more effectively to situations of physiological stress. In a study[12], the 24 h, awake and asleep HR significantly correlated with the HRV threshold workload[18]. In our study, even in untrained individuals, aerobic fitness correlated with parasympathetic modulation after the maximal exercise. Previously, nocturnal HRV changes were related to the changes in VO_2max due to aerobic training. The authors suggest that sleep HRV analysis can provide a useful parameter in evaluating individual responses to physical training and prescription for an optimal training program for untrained participants[4].

The mechanisms by which aerobic fitness promotes alterations in autonomic control remain unclear. However, it was suggested that aerobic fitness improved cardiac parasympathetic activity since atropine treatment eliminated the differences in the HR response to exercise, and the indices of cardiac parasympathetic activity observed between the trained and sedentary animals[28]. The cardioprotective effects of aerobic fitness imply reduction of β2-adrenoceptor expression or responsiveness[29]. Structurally, aerobic training results in an increase in myocardial cell size, which can enhance stroke volume and decreases HR due to higher myocardial contractility[30].

Our results suggest that the cardiac autonomic modulation changes due to afternoon physical stress may impair sleep quality and an adequate recovery in women with little aerobic fitness. In this way, organizing a training program (*i. e.,* time of day, and intensity of exercise) to improve sleep routines can optimize recovery in untrained individuals, who represent the majority of people starting training programs for health promotion. Santos[27] observed a significant correlation between VO_{2max} and HRV indices in policemen. Moreover, the number of sleep hours was negatively correlated with LF and LF/HF ratio and positively correlated with HF.

The present study presents some limitations, such as the lack of control of the menstrual cycle regularity, hormone levels, chronotype, food intake before bedtime, sleep quality and a schedule of activities to indicate stress situations. Although ambulatory measures have less control than laboratory measures, they reflect ecologically valid configurations (e.g. diary life settings). Additionally, the time of day and the modality of the exercise performed make it impossible to generalize the results to a morning or evening performed exercise, or to a traditional exercise session, given that these factors can have an important influence on the cardiac autonomic responses. On the other hand, due to little aerobic fitness, these individuals may need greater recovery time between sessions of intense exercise[10].

CONCLUSION

In summary, maximal exercise caused a reduced parasympathetic modulation during sleep in untrained young women. The cardiac autonomic modulation was correlated with aerobic fitness.

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COMPLIANCE WITH ETHICAL STANDARDS

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Ethical approval

Ethical approval was obtained from the local Human Research Ethics Committee (UFMT) and protocol (nº. 65485217.5.0000.5541) was written in accordance with standards set by the Declaration of Helsinki.

Conflict of interest statement

The authors have no conflict of interests to declare

Author Contributions

Author LTC and GA have given substantial contributions to the conception or the design of the manuscript; author GKT, JAA and FIN to acquisition, analysis and interpretation of the data. All authors have participated to drafting the manuscript; author GMP revised it critically. All authors read and approved the final version of the manuscript.

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