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Acute exercise modulates the mental stress-induced responses in healthy and obese young adults

O exercício agudo modula as respostas induzidas pelo estresse mental em adultos jovens saudáveis e obesos

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Abstract – The aim of this study was to verify whether obesity and acute physical exercise could influence the reactivity to mental stress. Twelve normal weight (NW) and ten obese (Ob) (31.82% women), normotensive individuals between 18 and 40 years old were evaluated. The Stroop color test was performed under two conditions: 1) Baseline and 2) 30 min after the end of aerobic physical exercise. Individuals performed 30 min of physical exercise (NW: 3.38 and Ob: 2.99 km·h^{-1}; p< 0.05) between 50-60% of heart rate reserve (NW: 139±7 and Ob: 143±7 bpm). The reactivity to mental stress for systolic blood pressure - SBP (Δ2 min) and diastolic blood pressure - DBP (Δ2 and Δ4 min) was lower (p< 0.05) in Ob compared to NW group. The SBP reactivity to mental stress was less (p< 0.05) post-exercise in all moments (Δ2 and Δ4 min), regardless of obesity, while the DBP reactivity to mental stress was less post-exercise, only in NW. BP reactivity to stress correlated negatively with obesity indicators (r/Rho = – 0.42 to – 0.64), under the baseline condition, but not post-physical exercise. Obesity blunted BP reactivity to mental stress in normotensive young adults. Moderately-intensity acute aerobic exercise reduced SBP reactivity to mental stress, regardless of obesity, while it reduced DBP reactivity to mental stress, only in NW. In summary, acute physical exercise may be a one means to prevent cardiovascular changes.

Key words: Aerobic exercise; Autonomic nervous system; Blood pressure; Obesity; Stroop test.

Resumo – *O objetivo deste estudo foi verificar se a obesidade e o exercício físico agudo podem influenciar a reatividade ao estresse mental. Foram avaliados 12 indivíduos eutróficos (E) e dez obesos (Ob) (31,82% mulheres), normotensos entre 18 e 40 anos. O teste de cor Stroop foi realizado em duas condições: 1) Linha de base e 2) 30 min após o término do exercício físico aeróbio. Os indivíduos realizaram 30 min de exercício físico (E: 3,38 e Ob: 2,99 km•h–1; p<0,05) entre 50–60% da frequência cardíaca de reserva (E 139±7 e Ob: 143±7 bpm). A reatividade ao estresse mental para pressão arterial sistólica – PAS (Δ2 min) e pressão arterial diastólica – PAD (Δ2 e Δ4 min) foi menor (p< 0,05) no grupo Ob comparado ao grupo E. A reatividade da PAS ao estresse mental foi menor (p< 0,05) pós-exercício em todos os momentos (Δ2 e Δ4 min), independente da obesidade, enquanto a reatividade da PAD ao estresse mental foi menor pós-exercício, apenas no E. A reatividade da PA ao estresse correlacionou-se negativamente com os indicadores de obesidade (r/Rho = – 0,42 a – 0,64), na condição basal, mas não pós-exercício físico. A obesidade embotou a reatividade da PA ao estresse mental em adultos jovens normotensos. O exercício aeróbico agudo de ientesidade moderada reduziu a reatividade da PAS ao estresse mental, independentemente da obesidade, enquanto reduziu a reatividade da PAD ao estresse mental, apenas no E. Em resumo, o exercício físico agudo pode ser um meio de prevenir alterações cardiovasculares.*

Palavras-chave: *Exercício aeróbico; Sistema nervoso autonômico; Pressão arterial; Obesidade; Teste de Stroop.*

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INTRODUCTION

Among traditional cardiovascular diseases (CVD) risk factors, obesity has become a major research target due to its epidemic prevalence[1]. Another independent risk factor for CVD is psychological stress[2]. Acute mental stress tasks have been used as a laboratory models to examine cardiovascular response to psychological stress[3-5]. Evidence has suggested that the association between systolic blood pressure (SBP) reactivity to mental stress and increased risk for future cardiovascular events is independent of baseline BP values[6].

Although there is evidence that exaggerated cardiovascular responses to mental stress are able to predict the development of both cardiovascular disorders[3] and hypertension[5, 7], the link between obesity and stress response remains unclear. It seems probable that obesity, acting as a chronic stressor, may augment cardiovascular response to a psychological stressor. However, some studies[8,9] have hypothesized that obesity, a chronic stressor, may decrease rather than increase cardio response to psychological stress. This suggests that there is a need for research on to what degree acute stress alters reactivity in obese people and what factors can mitigate its effects on cardiovascular response.

Acute physical exercise appears to have a significant impact on BP responses to psychosocial stress; this may have important implications for cardiovascular health. A lower post-aerobic exercise mental stress reactivity has been demonstrated in pre-hypertensive/hypertensive individuals[10,11], and trained cyclists[12], but no in individuals with a family history of hypertension[13]. Most studies exploring an acute exercise cardioprotective role in stress reactivity have involved healthy[12-17] populations, while studies in populations with cardiovascular risks (e. g., obesity) are scarce, since few have involved pre-hypertensive/hypertensive individuals[10,11,18]. To the best of our knowledge, no study has evaluated stress response in young obese adults without hypertension, nor the influence of physical exercise on these people. This information would enable better management to prevent development of hypertension in obese people. As well, this research has important implications for physical exercise as an effective therapy in combating the deleterious impact of mental stress and hypertension that accelerate CVD.

The purpose of this study was to verify whether obesity and physical exercise could influence reactivity to mental stress. In addition, the study evaluated the correlation between obesity indicators with the reactivity of hemodynamic variables to mental stress, both baseline and after acute physical exercise. Finally, we hypothesized obese people have lower reactivity to mental stress and that acute physical exercise promotes lower reactivity to mental stress.

METHODS

Participants

Subjects were recruited through advertisements in the university and in social media. A total of 28 volunteers were interviewed. Of these, 22 (7 women) young adults met the inclusion criteria, of whom 12 had normal-weight (NW) and 10 were obese (Ob).

The inclusion criteria were: adults of both sexes, aged 18 to 40 years, NW ($20 \leq 24.9\ kg \cdot m^{-2}$) and Ob ($30 \leq 39.9\ kg \cdot m^{-2}$), no diagnosed diseases, non-smokers

and no regular physical exercise. The exclusion criteria were: use of continuous medications and any substances that could interfere with the variables under study, or presenting cardiovascular and metabolic diseases, being pregnant, breastfeeding. The study was performed in accordance with the ethical standards of the institutional ethics committee in Human Research (CAAE 76816917.4.0000.8124).

Study design

An afternoon visit (4:30 to 7:00 p.m.) was carried out to evaluate physical activity level, obesity indicators, hemodynamic variables, as well as to undergo a mental stress test (Stroop color test), and a physical exercise session.

Experimental session

The participants were oriented not to perform physical exercise, not to drink stimulants or alcoholic beverages 24h before the visit. Women were evaluated between the 6th and the 12th day of their menstrual cycle. After the initial clinical screening, the participants answered a health history and medication use questionnaire to confirm the absence of diagnosed diseases and the fact that they were not using continuous medicines.

Subjects were placed in the sitting position in a quiet air-conditioned room (approximately 24°C) and asked to rest quietly for 10 min. Then they were exposed to a mental stress challenge test both before (baseline) and 30 min after a single bout of continuous physical exercise. All participants were evaluated by the same evaluator.

Level of physical activity

The level of physical activity was evaluated using the International Physical Activity Questionnaire (IPAQ) short-form, that estimates the weekly time spent in various activities of everyday life. The sum of weekly physical activity, such as walking (3.3 metabolic equivalents - METs), and moderate (4 METs), and vigorous activities (8 METs), was used to determine each participant's METs min•week^{-1} scores.

Obesity indicators

Body mass (OMRON Corporation, Kyoto, Japan) and height (Sanny stadiometer, 0.1 cm, Brazil) were measured to determine body mass index (BMI). Abdominal circumference was measured at the level of the umbilical scar (Cardiomed® tape; 0.1 cm, Brazil). The percentage (%) of body fat was estimated by bioimpedance (OMRON Corporation HBF-514C, Kyoto, Japan), following the manufacturer's instructions. The participants were asked to empty their bladders and not ingest any liquids 30 min before this procedure.

Baseline hemodynamic variables

Blood pressure

BP was determined in a seated position by an automatic sphygmomanometer (Microlife, BP 3BT0-A®, Brazil), after resting for 10 min, based on three

readings, with a one minute interval between them; the average of the two last readings was considered.

Cardiac autonomic modulation

The RRi recorded (Polar model V800, Kempele, Finland) at the last 5-min interval at baseline condition was used for the HR variability (HRV) indices calculation (Kubios Oy, Version 2.1, Kuopio, Finland). The artifacts were filtered using the software's moderate filter, in which the artifacts and ectopic beats were automatically detected and substituted by interpolated values using a cubic spline function[19]. The percentage of observed error was ~ 0.51 ± 0.63% in all records. The HRV indices were calculated using spectral analysis and an autoregressive algorithm model (order 16). The HRV indices analyzed were: 1) linear time-domain indices: root mean square of successive RR interval differences (RMSSD); and the standard deviation of normal-to-normal RRi (SDNN). 2) frequency-domain indices: low frequency (LF); high frequency (HF, expressed in n. u.) and LF/HF ratio.

Stroop color test

At the end of the baseline and after 30 min of aerobic exercise recovery the Stroop color test was administered. A panel with the names of 4 colors (blue, green, red, and yellow) had each name of a color written with letters of a different color. This was shown to the participants over a period of four minutes: the words were changed each second. The participant had to say, as quickly as possible, the color of the letters without reading the word. A continuous auditory conflict with different names of colors was used to increase the amount of mental stress. Each participant was asked to assess the difficulty of the task according to previously established levels of difficulty: 0 = not stressful; 1 = slightly stressful; 2 = stressful; 3 = very stressful; and 4 = extremely stressful. To avoid the learning effect, tests were performed with different color sequences (test 1, and test 2) at baseline and after physical exercise. The order of the tests was performed by a draw (random and balance). BP and HR were recorded at minutes two and four during test.

Physical exercise

Participants performed 30 min of walking/running at 50-60% of reserve HR. HR was monitored during all 30 min of physical exercise.

Post-exercise recovery

After physical exercise the participants remained in a seated position for 30 min. The BP values at 30 min were considered the pré Stroop test values in post-exercise condition.

Statistical analysis

The data normality was checked by the Shapiro–Wilk test. Data were expressed as mean and standard deviation, when parametric data, while nonparametric data

were presented in median, minimum, and maximum values. The variables between the NW and Ob groups were compared through the Unpaired Student's t-test or Wilcoxon test. To compare hemodynamic responses to mental stress at baseline and after physical exercise between groups a three-way repeated measures ANOVA was used, considering the following factors and levels: group (NW/Ob); condition (baseline/post-exercise); time during Stroop color test (0, 2 and 4 min). Bonferroni's post hoc was used when there was an interaction between factors. The reactivity to mental stress (Δ) at 2 and 4 min during mental stress on the baseline and post-exercise condition, for BP and HR, were calculated as being: Δ: 2 and 4 min – pre-test values. To compare reactivity to mental stress at baseline and post-exercise between groups a two-way repeated measures ANOVA was used, considering the following factors and levels: group (NW/Ob); condition (baseline/post-xercise). ES measures of the ANOVA factor analysis were calculated by the partial eta square (η^2): ($\eta^2 \geq 0.01$ were considered small; ≥ 0.06 medium, and ≥ 0.14 large ES)[20]. The statistical power (Pr= 1—β) to a posteriori of the main analysis was shown. Obesity indicators (body mass, BMI, abdominal circumference, and body fat percentage), and physical activity (MET's) were correlated to reactivity to mental stress (Δ2: BP and HR) at baseline and post-exercise. These variables were correlated through Pearson's linear correlation to parametric data, and Spearman's Rank to non-parametric data. Correlation coefficients were adjusted to SBP and DBP values at baseline. The significance level adopted was 5% ($p \leq 0.05$).

RESULTS

The Ob group showed higher obesity indicators (body mass, BMI, abdominal circumference, and body fat), as would be expected. Additionally, clinical SBP values were higher in the obese group. There were no significant differences ($p > 0.05$) related to age, DBP, METs score, or for cardiac autonomic modulation between groups (Table 1).

Table 1. Obesity indicators, physical activity, hemodynamic and physical exercise parameters

	Normal weight (n=12)	Obese (n=10)	p
Sex (F/M)	4/8	3/7	
Age (years)*	27.53 (20.77-36.12)	32.39 (20.95-36.61)	0.47
Body mass (kg)	65.57±8.79	102.50±10.96	**<0.01**
Body mass index (kg·m^{-2})	22.56±1.79	34.25±2.60	**<0.01**
Abdominal circumference (cm)	77.23±7.14	105.70±8.70	**<0.01**
Body fat (%)	22.79±5.27	38.92±8.32	**<0.01**
Total MET's (min·week^{-1})*	1039 (0-8199)	305 (0-6720)	0.47
Systolic BP (mmHg)	107±10	116±8	**0.04**
Diastolic BP (mmHg)*	65 (59-79)	66 (59-88)	0.26
RMSSD (ms)*	37.26 (16.88-58.42)	19.39 (3.71-78.39)	0.19
SDNN (ms)	41.28±9.36	29.31±16.93	0.06
HF (n. u.)	37.98±9.35	43.25±20.96	0.46
LF/HF*	1.37 (1.10-3.98)	1.84 (0.19-4.27)	0.83
Physical exercise parameters			
Heart rate (bpm)	139±7	143±7	0.26
Distance (km)*	3.38 (2.86-5.14)	2.99 (2.35-3.42)	**0.02**
Velocity (km·h^{-1})*	6.75 (5.71-10.27)	5.96 (4.69-6.84)	**0.01**

Note. BP: blood pressure; HF = high-frequency; LF/HF = low and high-frequency ratio; MET's: metabolic equivalents; ms: milliseconds; n.u. = normalized units; RMSSD: the root mean square of successive RR differences, SDNN: the standard deviation of normal-to-normal RRi. The data are presented as mean and standard deviation. * Mann Whitney U test for nonparametric data, with median, minimum, and maximum values.

Finally, there was no significant difference in HR during physical exercise performance; the Ob group, however, performed at a lower velocity and less distance during the 30 min (Table 1). A single acute exercise did not reduce SBP and DBP during the recovery period (30 min), regardless of obesity. There was no difference in the difficulty of the task perceived during the Stroop test between groups or conditions [baseline - NW: 2 (1-3) vs Ob: 2 (1-3); post-exercise: NW: 2 (0-4) vs Ob: 2 (1-3)].

The SBP was higher at 2 and 4 min compared to 0 during mental stress in both groups, only under baseline condition (time x condition interaction, $p< 0.01$, $Pr= 0.97$), Table 2. Furthermore, SBP was lower in NW compared to Ob group, only under post-exercise condition (group x condition interaction, $p= 0.04$, $Pr= 0.54$)

The DBP, in the NW group, was higher at 2 and 4 min compared to 0 during mental stress, under the both conditions. However, in the Ob group, the DBP was higher at 2 min compared to 0 during mental stress, only under the baseline condition (group x time x condition interaction; $p= 0.02$; $Pr= 0.65$). Furthermore, DBP was lower at 2 and 4 min post-exercise in NW group, and at 2 min in Ob at post-exercise compared to baseline condition, Table 2.

The HR was higher (time main effect; $p< 0.01$; $Pr= 1.00$) at 2 and 4 min compared to 0 during mental stress, in both groups and, regardless of condition. Furthermore, HR was higher (condition main effect; $p< 0.01$. $Pr= 1.00$) at post-exercise compared to baseline condition, at all moments, regardless of obesity (Table 2).

The reactivity to mental stress to SBP (Δ2 min) and DBP (Δ2 and Δ4 min) was lower ($p< 0.05$; $Pr= 0.50$ to 0.57) in Ob compared to NW group, regardless of condition (baseline and post-exercise). The SBP reactivity to mental stress was reduced ($p< 0.05$; $Pr= 0.85$ to 1.00) under the post-exercise condition when compared to baseline in both moments (Δ2 and Δ4 min), regardless of obesity. However, DBP reactivity to mental stress was reduced ($p< 0.05$; $Pr= 0.65$ to 0.78) in both moments (Δ2 and Δ4 min), only to NW group (Table 3).

BP reactivity to stress correlated negatively ($p< 0.05$) with obesity indicators ($r/Rho= -0.42$ to -0.64), under baseline conditions, but not after acute physical exercise (Figure 1A to 1D). HR reactivity to stress correlated with abdominal circumference ($Rho= -0.45$; $p < 0.05$), under baseline conditions, but not after acute physical exercise. There were no significantly correlations between BP and HR reactivity to stress with METs. After adjusting to SBP and DBP values at baseline, BP reactivity to stress remained correlated negatively ($p< 0.05$) with obesity indicators (-0.48 to -0.60).

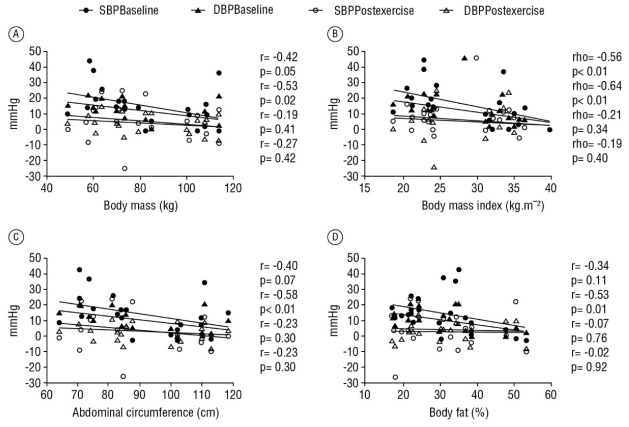

Figure 1. Correlation coefficients (*Rho/r*) of body mass (A), BMI (B), abdominal circumference (C) and body fat (D) with systolic and diastolic blood pressure reactivity to stress (Δ2 min) at baseline and post-exercise.

Table 2. Hemodynamic responses to mental stress at baseline and after an exercise bout in normal weight and obese group.

	Times	Normal weight (n=12)			Obese (n=10)				Three-Way ANOVA						
		0	2	4	0	2	4		Group	Time	Condition	Group x Time	Group x Condition	Time x Condition	Group x Time x Condition
Systolic BP (mmHg)	**Baseline**	107±10[a]	128±11[b]	122±9[b]	116±8[a]	125±17[b]	125±14[b]	*p*	0.17	< 0.01	< 0.01	0.13	**0.04**	**< 0.01**	0.64
								ES	0.09	0.51	0.61	0.11	0.20	0.45	0.01
	Post-Exercise	105±10	111±12	110±9	116±7*	119±8*	117±11*	*Pr*	0.27	0.99	1.00	0.32	0.57	0.97	0.07
Diastolic BP (mmHg)	**Baseline**	67±7[a]	82±5[b]	79±9[b]	74±14[a]	81±15[b]	78±16[ab]	*p*	0.51	< 0.01	< 0.01	0.04	0.15	< 0.01	**0.02**
								ES	0.02	0.58	0.30	0.20	0.10	0.32	0.23
	Post-Exercise	69±7[a]	74±5b[#]	73±7[b#]	74±11	78±15[#]	77±13	*Pr*	0.10	1.00	0.80	0.55	0.30	0.84	0.65
Heart rate (bpm)	**Baseline**	74±13[a]	84±17[b]	84±17[b]	84±12[a]	88±16[b]	90±15[b]	*p*	0.39	**< 0.01**	**< 0.01**	0.08	0.61	0.10	0.70
								ES	0.04	0.58	0.69	0.14	0.01	0.13	0.01
	Post-Exercise[#]	86±15[a]	92±14[b]	93±13[b]	93±16[a]	96±15[b]	96±15[b]	*Pr*	0.13	1.00	1.00	0.41	0.08	0.39	0.07

Note. ES: effect size; Pr: Statistical power to a posteriori. Note. BP: blood pressure; ES: effect size; Pr: Statistical power to a posteriori. Different letters indicate significant differences between times (pré, 2 and 4 min) during the Stroop color test. *Significant difference between obese and normal weight group at post-exercise condition. [#]Significant difference between post-exercise and baseline condition.

Table 3. Delta - Δ (exercise – baseline condition values) of hemodynamic responses to mental stress in normal weight and obese group.

		Baseline		Post-Exercise		Two-Way ANOVA			
		Normal weight (n=12)	Obese (n=10)	Normal weight (n=12)	Obese (n=10)		Group	Condition	Group x Condition
Systolic BP (mmHg)	Δ2	21±10	9±11	6±14	4±10	*p*	**0.05**	**0.01**	0.14
						ES	0.17	0.34	0.11
						Pr	0.50	0.85	0.31
	Δ4	15±6	9±10	5±7	1±11	*p*	0.13	**< 0.01**	0.61
						ES	0.11	0.47	0.01
						Pr	0.33	0.98	0.08
Diastolic BP (mmHg)	Δ2	15±4	7±6	5±6#	4±7	*p*	0.04	< 0.01	**0.01**
						ES	0.20	0.63	0.29
						Pr	0.57	1.00	0.78
	Δ4	13±6	4±8	4±6#	3±4	*p*	0.04	< 0.01	**0.02**
						ES	0.19	0.34	0.23
						Pr	0.55	0.85	0.65
Heart rate (bpm)	Δ2	10±8	4±6	7±7	3±3	*p*	0.09	0.07	0.61
						ES	0.14	0.16	0.01
						Pr	0.39	0.46	0.08
	Δ4	10±8	6±7	8±8	3±4	*p*	0.09	0.10	0.70
						ES	0.14	0.13	0.01
						Pr	0.41	0.37	0.07

Note. BP: blood pressure; ES: effect size; Pr: Statistical power to a posteriori. #Significant difference between post-exercise and baseline condition in normal weight group.

DISCUSSION

The main findings of this study were that a) obese young individuals showed lower BP reactivity to mental stress; b) moderate-intensity aerobic exercise reduced SBP reactivity to mental stress, regardless of obesity; and it reduced DBP reactivity to mental stress, only in NW group; c) BP reactivity to stress correlated negatively with obesity indicators at baseline condition, but not after acute physical exercise. The highlight of this study was to explore the acute exercise cardioprotective role in stress reactivity in populations with cardiovascular risks such as obesity.

Several studies have shown that BP reactivity to mental stress predicts hypertension at different periods of follow-up time (e.g., 8, and 12 years) and with different groups of people (young, middle-aged, and older people without a previous history of hypertension or CVD)[4,5,7]. SBP reactivity was a stronger predictor of hypertension in the youngest (24 years) cohort study than it was in the middle and older cohort studies[5]. The risk of hypertension was higher for those with a higher BMI, and those with elevated baseline SBP[7]. The increase in BP during the Stroop color test can be partly explained by increased systemic vasoconstriction caused by increased sympathetic-adrenal activity. Sympathetic activation is responsible for increasing circulating levels of adrenaline and noradrenaline. The adrenaline increases triggers neurogenic vasoconstriction that, associated with the action of presynaptic receptors, can facilitate the release of norepinephrine, increasing peripheric vascular resistance, consequently increasing HR and BP[13,21,22].

Some studies[8,23] have supported the hypothesis that low, not high cardiac and cortisol stress reactivity is related to adiposity. Those subjects with a greater BMI, and skinfold thicknesses, or who were categorized as obese, displayed lower

cardiac reactions to acute stress and cortisol reactivity. In prospective analyses, low cardiac reactivity has been associated with an increased likelihood of becoming or remaining obese in the subsequent four to seven years[9]. In obese individuals, although the baseline SBP was higher than in non-obese individuals regardless of hypertension, the stress-induced increase of SBP was lower compared to the non-obese individuals[8]. The key pathways responsible for stress-induced cardiovascular reactivity are a result of intermediate changes in the sympathetic and parasympathetic nervous system and hypothalamic-pituitary-adrenal axis outflow to the heart and vasculature, resulting in changes to cardiovascular parameters such as cardiac output, HR, vascular resistance, and BP[24]. In obese people, studies have suggested that the low cardiovascular reactivity may be attributable to a blunted sympathetic nervous system response to acute challenge[9,23]. Evidence shows that the sympathetic nervous system of obese individuals is less responsive to stimulation[23]. A previous study[18] found no difference in mental stress scores before and after exercise within or between groups.

In our study, BP and HR reactivity to stress correlated negatively with obesity indicators under the baseline condition in agreement with a previous study[8], but not after acute physical exercise. BMI was associated with generalized blunting of cardiovascular activity (HR; stroke volume, cardiac output, SBP; total peripheral resistance) and endocrine responses to stress[25]. The consequence of reduced responsiveness may be less ability to cope with challenging situations.

In agreement with previous studies[15,17], the present study showed that aerobic exercise did not cause hypotension, probably because individuals were normotensive[26] or because the recovery time was short (30 min). Post-exercise hypotension is a phenomenon most often seen in people with cardiometabolic diseases[26]. Regardless of the absence of post-exercise hypotension, both the mean absolute values and the response to mental stress (Δ) were attenuated. A single bout of maximal exercise attenuated the BP response to mental stress in healthy individuals, along with lower stroke volume and cardiac output, denoting an acute modulatory action of exercise on the central hemodynamic response to mental stress (60 min after exercise)[15]. On the other hand, physical exercise did not reduce BP reactivity, regardless of a family history of hypertension. However, the stress task was performed only 10 min after physical exercise[13]. The timing of stress exposure in relation to exercise completion (e. g. 10[13], 15[17], 30[12], 60 min[15]) was the most controversial factor among the studies consulted.

The mechanisms through which physical exercise reduces reactivity to mental stress are still unclear. Lower BP reactivity to mental stress following exercise can be due to peripheral mechanisms, such as peripheral vascular resistance reduction[13], catecholamines[27], or changes in baroreflex sensitivity and the consequent attenuation of sympathetic tone. However, some studies did not register changes in total peripheral resistance[15,17,18]. In this case, reduced BP responses to stress after exercise can be strongly linked to a decrease in the sympathetic drive due to a reduced plasma norepinephrine response and elongation of the pre-ejection period[28]. Medeiros et al.[18] showed that acute exercise reversed an impaired vasodilator response to mental stress in pre-hypertensive individuals, as indicated by a lower BP increase. The attenuating effect of acute exercise on the BP response to mental stress may accumulate over time to create a condition of lower risk for cardiovascular events. Georgiades et al.[29] showed that in obese individuals with elevated BP, exercise training alone or in combination with a behavioral weight loss program lowed BP, peripheral resistance, and HR, both at rest and during mental

stress. Thus, it is important to highlight that the investigation of hemodynamic responses after a single bout of exercise can contribute to an understanding of the process of decreasing the risk of CVD as a result of physical training.

As for HR responses to stress, although HR values were higher after the physical exercise session, there were no differences in HR responses to stress, maybe due to the timing of stress exposure in relation to exercise completion. HR values after physical exercise had not returned to baseline levels before participants were exposed to mental stress, thus interfering with the actual HR response during stress[16]. Other studies have also reported this[10,12,17].

Some limitations were observed in this study: 1) We did not measure psychological health; 2) We did not performed a control session. On the other hand, this study shows great ecological validity, since this physical exercise session, mental stress test, and hemodynamic measures perform are simple and inexpensive.

CONCLUSION

Obesity blunted BP reactivity to mental stress in normotensive young adults. Moderately intensity acute aerobic exercise reduced SBP reactivity to mental stress, regardless of obesity, and DBP reactivity to mental stress, only in NW group, denoting an acute modulatory action of exercise on the hemodynamic response to mental stress.

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. 76816917.4.0000.8124) 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

Authors' contributions

Author LTC has given substantial contributions to the conception or the design of the manuscript, author AC, JAA and LCN to acquisition, analysis and interpretation of the data. All authors have participated to drafting the manuscript, author JCF revised it critically. All authors read and approved the final version of the manuscript.

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