HIGH-INTENSITY INTERVAL TRAINING POSES NO RISK TO HYPERTENSIVE WOMEN

TREINAMENTO INTERVALADO DE ALTA INTENSIDADE NÃO APRESENTA RISCO PARA MULHERES HIPERTENSAS

EL ENTRENAMIENTO DE INTERVALOS DE ALTA INTENSIDAD NO PRESENTA RIESGO PARA MUJERES HIPERTENSAS

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ABSTRACT

Introduction: The aim of this study was to evaluate whether a single session of high-intensity interval training (HIIT) would promote a hypotensive effect and cardiovascular risk in hypertensive women, in addition to increasing the bioavailability of nitric oxide. Methods: The sample consisted of 10 hypertensive women (63.7 ± 10.34 years; 66 ± 7.67 kg and 153.7 ± 9.08 cm) and the training load was established at 60% of the maximum aerobic speed. Results: We observed a very high hypotensive effect between the interaction moments during the intervention (Int. Pre: 122.40 ± 18.58; Int. Post: 143.00 ± 24.90; Int. Post 60min: 121.40 ± 13.87; p<0.001, η2P = 0.569). No cardiovascular risk was observed during the intervention (DP = Int. Pre: 9138.20 ± 1805.34; Int. Post: 14849.70 ± 3387.94; Int. Post 60min: 9615.90 ± 1124.41, p< 0.001, η2P = 0.739) and there was no increase in the bioavailability of nitric oxide. Conclusion: In conclusion, this work reveals that an HIIT session is capable of generating a hypotensive effect while not posing cardiovascular risk in hypertensive women. ***Level of evidence I; High-quality randomized clinical trial with or without statistically significant difference, but with narrow confidence intervals.***

Keywords: High-intensity interval training; Hypertension; Post-exercise hypotension.

RESUMO

Introdução: O objetivo deste estudo foi avaliar se uma única sessão de treinamento intervalado de alta intensidade (HIIT) promoveria efeito hipotensor e risco cardiovascular em mulheres hipertensas, bem como aumentar a biodisponibilidade de óxido nítrico. Métodos: A amostra foi composta por 10 mulheres hipertensas (63,7 ± 10,34 anos; 66, ± 7,67 kg e 153,7 ± 9,08 cm) e a carga de treinamento foi estabelecida em 60% da velocidade aeróbica máxima. Resultados: Observamos um efeito hipotensor muito alto entre os momentos de interação durante a intervenção (Int. Pré: 122,40 ± 18,58; Int. Pós: 143,00 ± 24,90; Int. Pós 60 min.: 121,40 ± 13,87; p < 0,001, η2P = 0,569). Nenhum risco cardiovascular foi observado durante a intervenção (DP = Int. Pré: 9138,20 ± 1805,34; Int. Pós: 14849,70 ± 3387,94; Int. Pós: 60 min.: 9615,90 ± 1124,41, p < 0,001, η2P = 0,739) e não houve aumento da biodisponibilidade de óxido nítrico. Conclusões: Em conclusão, este trabalho revela que uma sessão de HIIT é capaz de gerar efeito hipotensor sem apresentar risco cardiovascular em mulheres hipertensas. ***Nível de evidência I; Estudo clínico randomizado de alta qualidade com ou sem diferença estatisticamente significativa, mas com intervalos de confiança estreitos.***

Descritores: *Treinamento intervalado de alta intensidade; Hipertensão; Hipotensão pós-exercício.*

RESUMEN

Introducción: El objetivo de este estudio fue evaluar si una única sesión de entrenamiento de intervalos de alta intensidad (HIIT) podría promover un efecto hipotensor y riesgo cardiovascular en mujeres hipertensas, así como aumentar la biodisponibilidad del óxido nítrico. Métodos: La muestra fue compuesta por 10 mujeres hipertensas (63,7 ± 10,34 años; 66 ± 7,67 kg y 153,7 ± 9,08 cm) y la carga de entrenamiento se estableció en el 60% de la velocidad aeróbica máxima. Resultados: Se observó un efecto hipotensor muy elevado entre los momentos de interacción durante la intervención (Int. Pre: 122,40 ± 18,58; Int. Post: 143,00 ± 24,90; Int. Post 60 min: 121,40 ± 13,87; p <0,001, η2P = 0,569). No se observó ningún riesgo cardiovascular durante la intervención (DP = Int. Pre: 9138,20 ± 1805,34; Int. Post: 14849,70 ± 3387,94; Int. Post 60 min: 9615,90 ± 1124,41, p <0,001, η2P = 0,739) y no hubo aumento de la biodisponibilidad de óxido nítrico. Conclusiones: En conclusión, este trabajo revela que una sesión de HIIT es capaz de generar efecto hipotensor sin presentar riesgo cardiovascular en mujeres hipertensas. ***Nivel de evidencia I; Estudio clínico aleatorizado de alta calidad con o sin diferencia estadísticamente significativa, pero con intervalos de confianza estrechos.***

Descriptores: *Entrenamiento de intervalos de alta intensidad; Hipertensión; Hipotensión posejercicio.*

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INTRODUCTION

Arterial Hypertension (AH) is the main risk factor for cardiovascular disease (CVD) and brain complications.[1] Its origin is multifactorial, and mostly ~95% are due to unknown factors related to other diseases.[2]

The daily practice of physical exercise induces beneficial responses in reducing the risk of cardiovascular diseases, as well as in controlling blood pressure (BP).[3] Consequently, it reduces mortality from these diseases and promotes greater cardiovascular health.[4]

One of the main factors responsible for this BP control during exercise is nitric oxide (NO). Generated by endothelial cells, it is a powerful vasodilator and consequently promotes lower peripheral resistance.[4] Its bioavailability is greater in physically active individuals than in sedentary ones. This is because the practice of physical exercise increases the volume of blood flow promoting greater endothelial shear stress, which is sensitive to NO production.[5,6]

Intensity as the exercise modality has direct effects on the bioavailability of NO and BP. In the work presented by Boutcher and Boutcher,[7] the authors report that an aerobic exercise session at 70% of the $Vo2_{max}$ does not produce a greater hypotensive effect than an aerobic exercise of moderate intensity. However, in intermittent exercises above 70% of the $Vo2_{max}$, it has been significantly associated with a greater hypotensive effect in hypertensive populations. This increase is associated with improved aerobic fitness, promoted in less time than in continuous training of moderate intensity. This modality also directly interferes with changes related to arterial stiffness, endothelial function, insulin resistance and mitochondrial biogenesis. All these factors contribute to an increased NO bioavailability, which also helps to reduce the levels of reactive oxygen species, increasing the antioxidant status.[8,9]

Given the above, this study aimed to evaluate whether a single session of high-intensity interval training (HIIT) would be able to promote a hypotensive effect and cardiovascular risk in hypertensive women, as well as increase the bioavailability of NO, justified by the increase in stress of endothelial shear in response to exercise, where it would result in a greater bioavailability of NO and, consequently, a hypotensive effect.

METHODS

Experimental design

This is a quasi-experimental study, with a cross-sectional character and a quantitative approach. The research met the requirements of the resolution 466/2012 of the National Health Council, regarding the guidelines and regulatory standards for research involving human beings, as well as the ethical principles expressed in the Declaration of Helsinki (1964, reformulated in 1975, 1983, 1989, 1996, 2000, 2008 and 2013) of the World Medical Association. It was approved by the Ethics Committee for Research with Human Beings at the Federal University of Sergipe under the number 1646486.

The sample consisted of 10 hypertensive women (63.7 ± 10.34 years; 66, ± 7.67 kg and 153.7 ± 9.08 cm) proven by medical reports and all were using antihypertensive drugs. The following inclusion criteria were adopted: 1) participating for at least 90 days in the extension project "Active Heart", linked to UFS; 2) being out of childbearing age and/or hysterectomized, facts confirmed through interviews. Pain or momentary incapacity that makes it impossible for the participant to carry out the intervention protocol and the subject's withdrawal were considered as exclusion criteria.

All participants signed an informed consent form, as recommended by Resolution no. 196/96 of the National Health Council.

Instruments

For physical evaluation, a mechanical anthropometric scale (Model 31, Filizola, Brazil) with 100 g precision was used to measure body mass and a ruler with a 0.5cm precision to measure height, with measurements taken in triplicate. For BP monitoring, an automatic oscillometric device (BP 3BTO-A, Microlife, China), validated (Cuckson et al., 2002) and calibrated, was used, following the recommendations for use present in the instruction manual.

Procedures

The sample was instructed to avoid foods and beverages that could directly interfere with the HR and BP responses, and not to smoke for at least two hours before the experiment, not to practice strenuous physical activities and not to suspend or change the use of medications to control BP during the study period.

Determination of the training load

Two familiarization sessions were carried out, with an interval of 48 hours between them, prior to the performance of the test protocols and, afterwards, an incremental test on a treadmill (Centurion 300, Micromed, Brazil) was carried out in order to establish the maximum aerobic velocity (MAV), used as a parameter to determine the intensity of the exercise protocol.

The protocol consisted of a 5 min warm-up at a speed of 3 km/h and inclination at 1%. After the warm-up, the inclination was maintained and the velocity was increased by 1 km/h every 1 min until exhaustion, with exhaustion criteria being HR values above 95% of the theoretical maximum, that is, 220-age[10]; perceived exertion above 09 on the adapted Borg scale[11] and/or if the volunteer requested to interrupt the test for any reason.

Training session

After the incremental tests, the sample was submitted to two protocols on non-consecutive days, carried out in the same period of the day and with a minimum interval of 48 h between them, as described below:

• Interval exercise protocol: a 5-minute warm-up was performed at a speed of 3 km/h and inclination of 1% on the treadmill, soon after the sample was instructed to walk and/or run for 16 min in a sequence alternating 30 s with an intensity of 90% of the MAV and 90 s at 60% of the MAV.

• Control protocol: the sample remained at rest (without exercise) in a sitting position for 30 min, with only verbalizations being allowed.

For the analysis of mean arterial pressure, double product and myocardial oxygen volume, the following equations were used: (mean arterial pressure = diastolic + (systolic - diastolic)/3);[12] double product = heart rate × systolic blood pressure;[13] To estimate the myocardial oxygen volume (MVO_2), a mathematical function was used, expressing the result in ml O 2 /100 g ventilations per minute (VE/min), as follows: MVO 2 = (double product × 0 .0014)-6.37.[14]

Statistical analysis

Descriptive statistics were performed using measures of central tendency, mean (X) ± standard deviation (SD) and a 95% confidence interval (CI95%). To verify the normality of the variables, the Shapiro-Wilk test was used, considering the sample size. Data for all analyzed variables were homogeneous and normally distributed. A repeated-measures analysis of variance was used to assess hemodynamic data and Nitric Oxide between groups, followed by the ANOVA (two-way) test, and Bonferroni's post hoc test. All statistical analyzes were performed using the computerized Statistical Package for Social Science (SPSS) version 22.0 (IBM Corp, Armonk, NY, USA). The level of significance adopted was $p < 0.05$. To verify the effect size, partial squared Eta ($\eta 2p$) was used, adopting values of low effect (≤0.05), medium effect (0.05 to 0.25), high effect (0.25 to 0 .50) and very high effect (> 0.50) for ANOVA.[15]

RESULTS

Table 1 shows the data on hemodynamic variables expressed as mean (X) and standard deviation (±). When analyzing the SBP, it is observed that there was a very high hypotensive effect when compared to the interactions of moments during the intervention (Int. Pre: 122.40±18.58; Int. Post: 143.00±24.90; Int. After 60min: 121.40±13.87; p<0.001, η2P = 0.569). In relation to DBP, there was a significant increase within the control groups (Pre: 69.70±8.62; Post: 69.90± 10.35; Post 60min: 70.00±6.02, p≥0.05, η2P = 0.597) and Intervention (Pre: 75.40±15.17; Post: 82.50±9.42; Post 60min: 76.30±13.18, p≥0.05, η2P = 0.597). For mean arterial pressure, it is observed that there was no significant increase when comparing the interactions between groups and moments (Contr. Pre: 85.67±8.50; Contr. Post.: 83.87±7.49; Contr. Post 60min; 84.93±6.21; Int. Pre: 91.07±14.83; Int. Post: 102.67±12.67; Int. Post 60min: 91.33±12.46b, p>0 .05). Heart rate was significantly increased immediately in the Post Intervention condition, followed by a significant decrease after 60min. For the control condition, there was a significant decrease between the Pre, Post and Post 60min moments (Contr. Pre: 76.80±6.00; Post Contr.: 70.60±4.72; Contr. Post 60min: 67.90± 5.61, p<0.001, η2P = 0.896), (Int. Pre: 74.40±6.52; Int Post: 103.50±11.77; Int. Post60min: 79.60±8.62, p <0.001, η2P = 0.844).

The double product and myocardial oxygen volume were only significantly increased immediately after the intervention moment, followed by a significant decrease after 60min. (DP= Int. Pre: 9138.20±1805.34; Int. Post: 14849.70±3387.94; Int. Post 60min: 9615.90±1124.41, p<0.001, η2P = 0.739) (MVO2= Int. Pre: 6.42±2.53; Int. Post: 14.42±4.74; Int. Post 60min: 7.09±1.57, p<0.001, η2P = 0.739).

Figure 1 presents data on Nitric Oxide before and after concerning the intervention and control groups. There were no differences between the Intervention Group and the Control Group at different times (p=0.254; η2p=0.141, mean effect).

DISCUSSION

This study aimed to analyze whether a single high-intensity aerobic interval exercise session would be able to promote a hypotensive effect as well as cardiovascular risk and greater NO bioavailability in hypertensive women. The main findings of this research focus on the hypotensive effect promoted by training after 60 minutes and that it did not offer cardiovascular risk in the investigated population.

Several studies corroborate our findings[16,17] hypotensive effect is largely induced by a decrease in vascular resistance, which is mediated by central and peripheral factors and the interactions between them - such as afferent and efferent signals that act peripherally and centrally to regulate the sympathetic control of peripheral resistance and may also act by impairing the transmission of norepinephrine from the sympathetic

Table 1. Hemodynamic variables (SBP; DBP, MBP, HR, DP and MVO2) (mean ± standard deviation, CI 95%).

Moments	SBP (X ± DP) (IC 95%)	DBP (X ± DP) (IC 95%)	MBP (X ± DP) (IC 95%)	HR (X ± DP) (IC 95%)	DP (X ± DP) (IC 95%)	MVO2 (X ± DP) (IC 95%)
Contr. Pré	117,60±12,90 (108,37-126,83)	69,70±8,62 (63,54-75,86)	85,67±8,50 (79,59-91,75)	76,80±6,00a,b,c (72,51-81,09)	9006,40±966,23 a,b,c (8315,20-9697,60)	6,24±1,35 a,b,c (5,27-7,21)
Int. Pré	122,40±18,58c (109,11-135,69)	75,40±15,17 (64,55-86,25)	91,07±14,83 (80,46-101,67)	74,40±6,52 a,b,c (69,74-79,06)	9138,20±1805,34 a,b,c (7846,74-10429,66)	6,42±2,53 a,b,c (4,62-8,23)
Contr. Pós	111,80±9,05 (105,32-118,28)	69,90±10,35b (62,49-77,31)	83,87±7,49b,c (78,51-89,22)	70,60±4,72 a,b,c (67,22-73,98)	7885,90±778,31 a,b,c (7329,13-8442,67)	4,67±1,09 a,b,c (3,89-5,45)
Int. Pós	143,00±24,90c (125,19-160,81)	82,50±9,42b (75,76-89,24)	102,67±12,67b,c (93,60-111,73)	103,50±11,77a,b,c (95,08-111,92)	14849,70±3387,94 a,b,c (12426,11-17273,29)	14,42±4,74 a,b,c (11,03-17,81)
Contr. 60min	114,80±10,41 (107,35-122,25)	70,00±6,02b (65,69-74,31)	84,93±6,21b,c (80,49-89,37)	67,90±5,61 a,b,c (63,89-71,91)	7775,60±795,59 a,b,c (7206,47-8344,73)	4,52±1,11 a,b,c (3,72-5,31)
Int. 60min	121,40±13,87c (111,48-131,32)	76,30±13,18b (66,87-85,73)	91,33±12,46b,c (82,42-100,25)	79,60±8,62 a,b,c (73,44-85,76)	9615,90±1124,41 a,b,c (8811,55-10420,25)	7,09±1,57 a,b,c (5,97-8,22)
p						
Moment	0,037a	0,247a	0,066a	<0,001a	<0,001a	<0,001a
Group	0,009b	0,005b	0,004b	<0,001b	<0,001b	<0,001b
Interaction	0,001c	0,215c	0,009c	<0,001c	<0,001c	<0,001c
η2p						
Moment	0,307##	0,144#	0,261##	0,896###	0,739###	0,739###
Group	0,549###	0,597###	0,617###	0,844###	0,779###	0,779###
Interaction	0,569###	0,157#	0,408	0,871###	0,818###	0,818###

Note: Subtitle: SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MBP: Mean Blood Pressure; HR: Heart Rate; DP: Double Product; MVO2: myocardial oxygen consumption. * p < 0.05 (ANOVA two-way, e Post Hoc de Bonferroni). # Medium Effect, ## High Effect, ### Very High Effect.

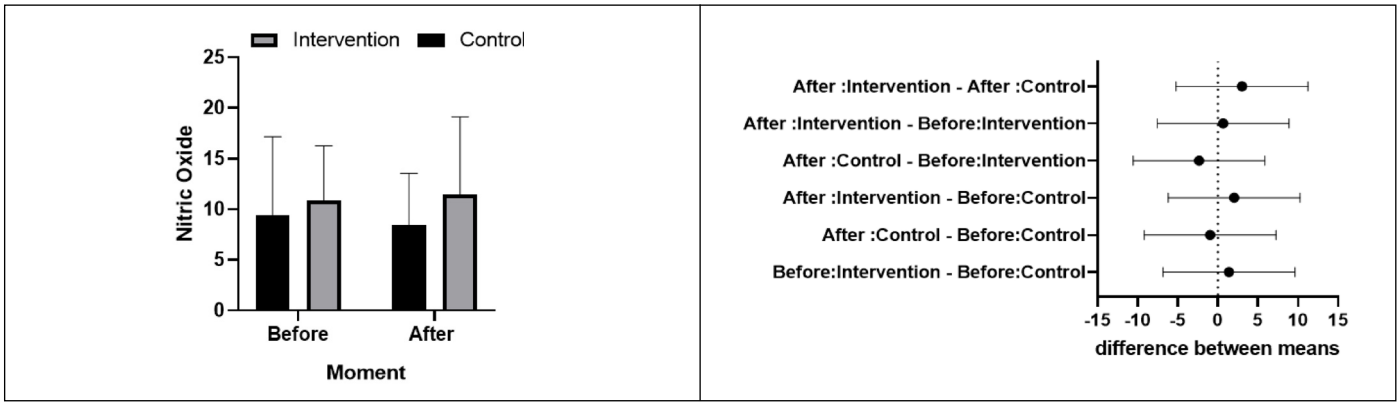

Figure 1. Nitric Oxide data.

nerves. Another important factor is the amount of muscle stimulated and the level of effort (intensity) caused by exercise.[16,18]

It is noteworthy that continuous low- or moderate-intensity training is also beneficial for this population.[19] However, when compared to the high-intensity intermittent model, the magnitude of the hypotensive effect is greater for high-intensity interval models up to 60 minutes post-exercise,[20] justifying the results found in our work.

When we analyze the information available in the literature on cardiovascular safety for HIIT, Pineda-García et al.,[21] by looking at people with heart disease at very high cardiovascular risk, it was identified that high--intensity interval aerobic training is safe and tends to improve exercise tolerance compared to continuous moderate-intensity aerobic training. Hannan et al.,[22] conducted a systematic review with a meta-analysis of high-intensity interval training compared to continuous moderate--intensity training in cardiac rehabilitation. The authors identified that high-intensity interval training promotes better results than continuous moderate-intensity training in cardiorespiratory function in ~6 weeks and that programs lasting longer than ~12 weeks would cause greater cardiorespiratory adaptations.

There are several reasons why this method is rated as safe even for specific populations. A wide range of factors, including skeletal muscle,[23] vasculature,[24] autonomic function,[25] cardiac function,[26] quality of life,[27] physiological markers such as peak VO2,[28] and endothelial function,[29] are presented with a greater magnitude when compared to continuous aerobic exercise.

Regarding NO bioavailability, no difference was found between the groups and the moments investigated. However, when looking at the literature on the subject, it appears that a significant supply of NO is found in longer studies ~6 to 12 weeks, a fact verified in the work of Hasegawa et al.,[8] The authors observed that the HIIT, after 6 weeks, promoted a greater NO bioavailability. In another study, promoted by Ghardashi Afousi et al.,[30] also when analyzing the HIIT after 12 weeks, a significant increase in the bioavailability of NO was observed. Perhaps, for this reason, it is justified why our work has not found any difference in its bioavailability between groups.

CONCLUSION

In conclusion, this work reveals that a HIIT session is capable of generating a hypotensive effect and does not offer cardiovascular risk in hypertensive women.

All authors declare no potential conflict of interest related to this article

AUTHORS' CONTRIBUTIONS: Each author made significant individual contributions to this manuscript. LMVS was the lead researcher, responsible for the concept/design, data collection, data analysis/interpretation, and drafting the article. MGM, ACM, GCR, LMVS, and JLS participated in the concept/design, data analysis/interpretation, and drafting of the article. FJA, CMALJ, and JLS performed statistical analysis and participated in the data interpretation, drafting, and critical review of the article. LMVS and ACM participated in the data analysis/interpretation, drafting, and critical review of the article. FJAS, JBS, DERA, JBS, MGM, and FJA contributed to the concept/design and participated in the data analysis/interpretation and critical review of the article. All authors read and approved the final manuscript.

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