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# Body composition influences blood pressure during submaximal graded test in women

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# Abstract

This study aimed to determine whether body composition affected cardiovascular responses of normal, overweight and obese women by comparing heart rate (HR) and blood pressure (BP) during a graded submaximal treadmill exercise. Thirty five healthy non-smoking, premenopausal women (age 30.8±9.5 yr; height 1.66±0.05 m; weight 73.8±18.9 kg; body fat 36.2±9.5%; maximum oxygen uptake 33.5±8.1 mL·min·kg$^{-1}$) volunteered for this study, and were classified into three groups based on BMI: normal weight (NOR; ≤24.9 kg·m$^2$), overweight (OVW; 25-29.9 kg·m$^2$) and obese (OBE; ≥30 kg·m$^2$). Participants performed a submaximal graded test at 30%, 40%, 50% and 60% of maximal oxygen uptake, during which HR, systolic BP (SBP), diastolic BP (DBP) and mean arterial pressure (MAP) were measured. At rest, participants of OBE group had higher BP than NOR and OVW. During the submaximal graded test, HR, SBP and MAP increased as a function of intensity. There were no group differences in HR, but greater SBP, DBP and MAP in OBE compared to NOR and OBW at all intensities except 60%. Overall, our study further contributes to underscore the importance of stress testing specifically to evaluate the increased risks apparently healthy obese women are placed at to developing diseases from the chronic exposure to raised BP, despite normal resting BP and HR responses during exercise.


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# Introduction

Hypertension is one of the most prevalent non-communicable diseases, affecting more than 1 in 5 people worldwide [1]. It is a significant predictor of cardiovascular risk characterised by abnormally elevated resting blood pressure (BP), typically diagnosed with systolic BP (SBP) above 140 mmHg and diastolic BP (DBP) above 90 mmHg. Chronic exposure to pressure overload can lead to pathologic cerebrovascular, retinovascular and cardiovascular trauma, as well as secondary functional impairments such as altered thermoregulation from impaired peripheral vasodilation.

Measuring BP during exercise is also used as a significant predictor of cardiovascular risk: despite being normotensive at rest, individuals exhibiting a hypertensive response to exercise (HRE; SBP>210 mmHg in men and 190 mmHg in women) are at an increased risk of developing hypertension later in life [2-5]. Furthermore, blood pressure during exercise has been found to be more strongly related to mortality and morbidity from myocardial infarction than post exercise BP [6]. Therefore, having a thorough understanding of the blood pressure response to exercise in normotensive and hypertensive individuals is of clinical significance for practitioners and those working in the health and fitness industry.

Although overweight and obese people are at an increased risk of hypertension [7-9], few studies have investigated exercise BP in this population. An increased myocardial wall thickness was found to correlate significantly with raised exercise SBP in overweight, mildly hypertensive individuals [10]. Elevated SBP during submaximal cycling has also been reported regardless of insulin sensitivity or cardiorespiratory fitness in individuals with a higher waist circumference [11]. However, while the prevalence of obesity and its associated risk factors such as hypertension has been found to be higher in men [12], little is currently

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known on the specific pathophysiology of hypertensive overweight or obese women during exercise despite the known effects of pregnancy, menopause and contraception on hypertension [13].

The main physiological mechanisms influencing the hypertensive response during exercise in overweight and obesity could originate in autonomic system dysregulation. At the commencement of and during exercise, an exercise pressor reflex dictates that sympathetic nervous system (SNS) activity overrides parasympathetic vagal tone, and results in increased heart rate, stroke volume and cardiac output in conjunction with a simultaneous redirection of blood flow to high priority visceral organs and skeletal muscles [14] to meet the increased metabolic needs of the active musculature, and drive SBP and mean arterial pressure (MAP) upward [15]. In overweight and obese people, an impaired response of SNS activity at onset of exercise may alter blood redistribution, and therefore pressure, through decreased endothelial responsiveness, increased arterial stiffness and increased hormonal response (mainly of the renin-angiotensin-aldosterone system) [16, 17]. Therefore, the purpose of this study was to compare the heart rate (HR) and BP responses of normal, overweight and obese women during a graded submaximal treadmill exercise, to determine whether body composition selectively affects cardiovascular responses to exercise.

# Methods

## Ethics statement and participants

The procedures described in this study were granted ethical clearance by the local ethics committee on human research. Participants for this study were recruited in the local community through the use of advertisements and e-mails, and were required to provide

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written informed consent prior to inclusion in the study. Participants consisted of healthy, non-menopausal female individuals aged 18 to 50 years of age (n=35; age 30.8±9.5 yr; height 1.66±0.05 m; weight 73.8±18.9 kg; maximum oxygen uptake 33.5±8.1 $mL \cdot min^{-1} \cdot kg^{-1}$) who were not smoking and not taking any medications known to affect cardiovascular measures in addition to being free from both mental and physical impairments.

Participants were invited for a preliminary visit for screening against underlying health conditions potentially affecting the study procedures and findings (Medical Health Questionnaires, lung function assessed using spirometry) and familiarisation with the procedures and equipment. Resting anthropometric characteristics of participants was recorded (height, weight, body mass index [BMI]=weight/height$^2$, hip and waist circumferences, waist-to-hip ratio [W:H]) and their body composition using dual-energy x-ray absorptiometry (DEXA; Lunar Prodigy Advance, GE Healthcare, Buckinghamshire, UK) (total and regional body fat, muscle mass, percentage body fat, bone mineral density [BMD] and bone mineral content [BMC], and blood pressure, blood glucose and cholesterol were also measured during the initial visit.

## Methodology

Each participant completed two experimental sessions separated by more than three days to prevent from potential fatigue effects between tests. Participants reported to the laboratory after a 10-12 hour overnight fast and no later than 10 AM. Hyperglycaemia was controlled using resting blood glucose, measured from a drop of blood collected at the fingertip using a single use lancet and portable analyser (Accu-Chek Advantage, Roche Diagnostics, Indianapolis, IN, USA). The first session consisted in a maximal incremental exercise test to volitional exhaustion on a treadmill (TMX425C, Trackmaster, Full Vision Inc., Newton, KS). The rate of oxygen utilization ($\dot{V}O_2$), was measured continuously using a two-way, non-

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rebreathing valve (series 2700, Hans-Rudolph, Kansas City, USA) and an automated open-circuit spirometry metabolic analysis system (True One 2400, Parvo Medics, Sandy UT). After a two minute warm-up walking at 4 km·h$^{-1}$ or 6 km·h$^{-1}$ (depending on the participant's exercise history and physical characteristics), speed was increased every minute until the participant signalled they had reached a comfortable speed, whereupon the grade of the treadmill was increased by 1-2% every minute until volitional exhaustion.

The second session consisted in a submaximal test including five exercise stages, each lasting 3 min. After an initial warm-up stage at 4.5 km·h$^{-1}$ (baseline), a graded test with four intensity stages were performed at relative intensities corresponding to 30%, 40%, 50% and 60% of $\dot{V}O_{2MAX}$. Heart rate (HR) was measured continuously at 0.2 Hz using a chest strap (Polar Electro, Kempele, Finland). Blood pressure responses were measured twice at the end of each stage of exercise during the graded test using a manual blood pressure cuff, aneroid and stethoscope (Welch Allyn, New York USA).

## Statistical analyses

Participants were classified into three groups based on BMI: normal weight (NOR; ≤24.9 kg·m$^2$), overweight (OVW; 25-29.9 kg·m$^2$) and obese (OBE; ≥30 kg·m$^2$). For comparison purposes, participants were also grouped into tertiles of percentage of body fat. Contingency tables showed strong association ($p<0.001$) between the two approaches and BMI groups were used for analyses.

Data are expressed as mean ± standard deviation (SD). All statistical analyses were performed using jamovi [18], and the level of significance was set at $p<0.05$. Differences in resting characteristics, body composition and fitness level between groups were determined using one-way ANOVAs with Bonferroni post-hoc tests. Agreement and concordance between the two measures of blood pressure taken at each level of the graded exercise were

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evaluated using Pearson's coefficient of correlation (r) and Kendall's rank correlation coefficient (tau-b) for SBP, DBP and MAP. The effect of exercise intensity (30%, 40%, 50% and 60%), BMI group (NOR, OVW, OBE) and their interaction (intensity×group) on HR%, SBP, DBP and MAP were assessed using 2-way, repeated measures ANCOVAs using age as a covariate of cardiovascular measures, with Bonferroni post-hoc tests. Pearson's r was used to calculate simple correlations between percentage body fat and HR%, SBP, DBP and MAP. Effect sizes for pairwise comparisons were assessed using Cohen's d, calculated in the standard manner and interpreted according to Cohen's scale (small effect: $0.2<d<0.5$, moderate effect: $0.5<d<0.8$, and large effect: $d>0.8$). For ANOVAs and ANCOVAs, we reported effect size using partial eta-squared ($\eta^2_p$) interpreted according to Cohen's scale (small effect: $0.01<\eta^2_p<0.06$, moderate effect: $0.06<\eta^2_p<0.14$, and large effect: $\eta^2_p>0.14$).

## Results

All participants completed the study procedures. The participant groups were homogenous in age, height, W:H ratio, DBP and resting blood glucose (Table 1). Participants in OVW and OBE group had higher weight, trunk fat and body fat (in g and %) compared to NOR (Table 1). Participants in OBE group also had higher weight, trunk fat and body fat compared to both OVW and NOR (Table 1). Participants in OBE group had greater LBM, and resting SBP and MAP compared to NOR only (Table 1). At rest, all but one participant in OBE group were pre-hypertensive, and one participant of OVW group was hypertensive (147/100 mmHg).

**** Insert Table 1 about here ****

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Absolute speed and gradient achieved at each stage of the submaximal graded test are presented in Table 2. Results for HR% are presented in Figure 1. There was a significant and large main effect of exercise intensity on HR% ($p<0.001$; $\eta^2_p=0.473$), with no significant differences across groups ($p=0.078$; $\eta^2_p=0.152$) and no significant interaction between group and exercise intensity ($p=0.232$; $\eta^2_p=0.082$). There was no significant effect of age as a covariate of HR% ($p=0.326$; $\eta^2_p=0.031$).

**** Insert Table 2 about here ****

**** Insert Figure 1 about here ****

The two measures of SBP, DBP and MAP performed at each exercise intensity were in high agreement and concordance (Table 3). No participant achieved SBP characteristic of HRE (>190 mmHg), and all participants with resting pre-hypertension and hypertension had normal exercise BP (maximum 172/80 mmHg). The dynamics of BP response were markedly different between OBE and the two other groups (Figure 1): minimum BP responses were observed at 30% in NOR (SBP: 116±15 mmHg; DBP: 81±5 mmHg; MAP: 93±8 mmHg) and OVW groups (SBP: 112±11 mmHg; DBP: 79±6 mmHg; MAP: 90±6 mmHg) compared to 60% in OBE (SBP: 134±13 mmHg; DBP: 82±6 mmHg; MAP: 100±7 mmHg), and maximum BP responses were observed at 60% in NOR (SBP: 141±19 mmHg; DBP: 84±10 mmHg; MAP: 103±12 mmHg) and OVW (SBP: 141±15 mmHg; DBP: 86±9 mmHg; MAP: 104±11 mmHg) compared to 50% in OBE (SBP: 148±17 mmHg; DBP: 95±10 mmHg; MAP: 112±11 mmHg).

**** Insert Table 3 about here ****

For SBP, there were significant and large main effects of exercise intensity ($p<0.001$; $\eta^2_p=0.389$) and group ($p=0.001$; $\eta^2_p=0.370$), with no main effect of interaction ($p=0.570$; $\eta^2_p=0.052$). There was no significant effect of age ($p=0.894$; $\eta^2_p=0.001$). Post-hoc testing

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revealed SBP in OBE group was significantly different from NOR (p=0.015) and OVW (p=0.002), with no significant differences between NOR and OVW (p=0.958).

For DBP, there was no significant effect of intensity (p=0.912; $\eta^2_p$=0.006) but a significant and large main effect of group (p<0.001; $\eta^2_p$=0.416), with no significant effect of interaction (p=0.219; $\eta^2_p$=0.089). There was no significant effect of age (p=0.792; $\eta^2_p$=0.002). Post-hoc testing revealed DBP in OBE group was significantly different from NOR (p=0.004) and OVW (p<0.001), with no significant differences between NOR and OVW (p=1.000).

For MAP, there were significant and large main effects of exercise intensity (p<0.001; $\eta^2_p$=0.232) and group (p<0.001; $\eta^2_p$=0.430), with no main effect of interaction (p=0.160; $\eta^2_p$=0.099). There was no significant effect of age (p=0.812; $\eta^2_p$=0.002). Post-hoc testing revealed DBP in OBE group was significantly different from NOR (p=0.004) and OVW (p<0.001), with no significant differences between NOR and OVW (p=0.977).

Percentage body fat was significantly inversely correlated with HR% only at 60% (r=-0.464, p=0.006), with SBP at all intensity levels (30%: r=0.500, p=0.004 ; 40%: r=0.448, p=0.012 ; 50%: r=0.471, p=0.008 ; 60%: r=0.522, p=0.003), with DBP at all intensity levels (30%: r=0.634 ; 40%: r=0.644 ; 50%: r=0.695, 60%: r=0.682 ; all correlations p<0.001), and with MAP at all intensity levels (30%: p=0.594, p<0.001 ; 40%: r=0.585, p<0.001 ; 50%: r=0.630 ; 60%: r=0.649 ; all correlations p<0.001).

## Discussion

The aim of this study was to determine and compare the cardiovascular responses of normal, overweight and obese women to a graded submaximal treadmill exercise. The main findings of this study were that besides anthropometric and body composition differences, i) there were no differences across groups for HR, ii) there were no significant differences in BP

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responses during exercise between NOR and OVW, but iii) BP responses in OBE women were significantly different to NOR at rest and to both NOR and OVW during exercise.

In the current study, HR responses can be considered normal as they increased as a function of exercise intensity, and reached maximum values within 10% of age-predicted maximum during the incremental test in all participant groups. Maximum HR values were consistent with previous literature [19]. Participants also did not report adverse perceptual responses during or after exercise. We did not observe group differences in HR at the same relative exercise intensities between groups during the submaximal graded test, and age was not a significant factor of HR response. Still, despite the lack of significant differences across groups (p=0.078), visual inspection of Figure 1 indicates that HR responses to exercise appear of a lesser magnitude in the obese compared to the two other groups. Further, HR was significantly and inversely correlated with body fat only at the highest intensity (60%). Since HR is a dependent variable with a large dispersion (coefficient of variation 7-12%), the possibility of a type II error remains and warrants future research specifically aiming to measure exercise HR at higher than moderate exercise intensities.

Contrasting with normal and expected HR responses, BP characteristics differed markedly between OBE and the two other groups. Participants in OBE group were pre-hypertensive at rest [20] and had overall higher exercise BP than NOR and OVW, although no participant reached levels characteristic of HRE [16]. As could be expected, percentage body fat was a strong predictor of blood pressure during exercise. While overall elevated BP responses in individuals with higher fat mass are consistent with previous literature [11], we also observed altered BP dynamics in OBE group, with higher SBP, DBP and MAP between 30% and 50% compared to both NOR and OVW, followed by significantly lower BP responses at the highest exercise intensity (60% of $\dot{V}O_{2MAX}$), the highest exercise intensity currently

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recommended in overweight and obese individuals with underlying health conditions (diabetes, hypertension, dyslipidemia, CVD) [20].

Observations of unchanged HR with simultaneous exaggerated BP responses and altered BP dynamics during exercise, such as those observed specifically in the OBE group in our study, are consistent with obesity-specific SNS activity dysregulation. The main mechanism for specific SNS alterations may originate in adipose tissue inflammation in obese individuals, which has been shown to increase systemic inflammatory response and markers of oxidative stress, such as angiotensin II adipokine [21]. Angiotensin is known to play a major role in the regulation of BP, as it has been shown to drive the hypertensive responses to exercise [22]. Additionally, blocking angiotensin II receptors reduces SBP during exercise [23]. Previous studies have also reported altered SNS activity specifically in mildly hypertensive individuals, as characterized by a reduced propensity to postexercise hypotension (the decrease in BP following dynamic exercise) [24, 25]. Therefore, together with the inconclusive observations we previously mentioned regarding HR responses to exercise, these observations provide a basis warranting future exercise studies in obese people specifically targeting responses at exercise above moderate intensities to explore SNS responses. The effect of even higher intensity exercise should also be further investigated, since high intensity interval exercise has been shown to be a time-efficient training model promoting body composition changes [26] and hypotensive exercise response [27] comparable to the traditionally recommended moderate-intensity exercise [20], but the potential for SNS dysregulation occurring specifically in obese individuals has been insufficiently described.

It is important to acknowledge our findings are limited to the obese population under investigation, limited to seven, white female participants, in which we could not account for the effect of menstrual phase. However, we observed no effect of age on BP responses, no

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violations of the assumption of equality of variances, and the two measures of BP performed at each stage were in high agreement and concordance. In addition, although we determined participant groups based on BMI, which is known to be inaccurately representing body composition, results were unchanged when using tertiles of percentage body fat. For increased practical applicability, we used BMI classification for all analyses. Finally, for practical reasons we did not measure SNS activity directly or indirectly, and we did not measure postexercise BP. These limitations should be taken in consideration before extrapolating our results to a wider population or derive conclusive evidence on the role of adipokines and SNS activity from our discussion.

## Perspectives

Our study further contributes to underscore the increased risks apparently healthy obese women are placed at to developing diseases from the chronic exposure to raised BP despite apparently normal HR responses. As such, it is important that exercise testing in overweight and obese individuals should be accompanied by measures of blood pressure, as these might discriminate more efficiently at-risk individuals. Furthermore, our findings indicate that more research is required to investigate the benefits of training above moderate exercise intensities, such as high intensity exercise training, for pre-hypertensive obese individuals with no underlying health diseases, as we observed an altered response at the highest intensity of the submaximal graded test (60%). Such endeavors could have large implications for exercise recommendations to prevent the development of further health complications in pre-hypertensive populations.

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# Figure

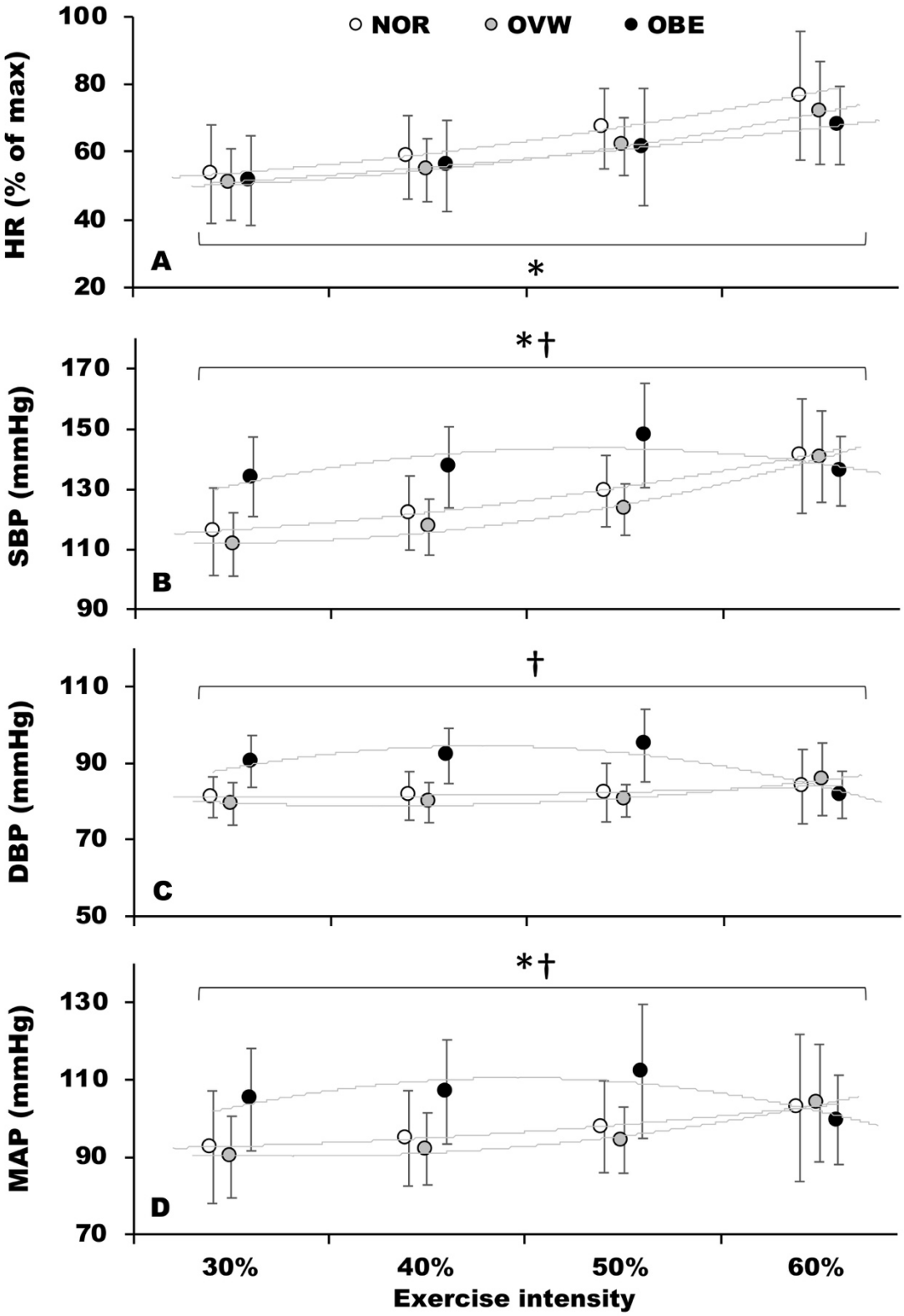


**Figure 1.** Measures of heart rate (HR, **A**), systolic blood pressure (SBP, **B**), diastolic blood pressure (DBP, **C**) and mean arterial pressure (MAP, **D**) during the graded submaximal exercise, in the three study groups (normal weight, overweight and obese participants).

Symbols * and † denote significant main effects ($p<0.01$) of exercise intensity and group, respectively.

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# Tables

**Table 1.** Participants' resting anthropometry and blood pressure profiles classified according to BMI.

| | Normal | Overweight | Obese | ANOVA | |
|---|---|---|---|---|---|
| | <24.9 kg·m² | 25-29.9 kg·m² | >30 kg·m² | p | $\eta^2_p$ |
| | (n = 15) | (n = 13) | (n = 7) | | |
| Weight (kg) | 60.9 ± 7.8 | **72.3 ± 3.5** [A C] | **104.4 ± 18.4** [B] | **<0.001** | **0.751** |
| Height (m) | 1.65 ± 0.05 | 1.65 ± 0.05 | 1.68 ± 0.06 | 0.484 | 0.044 |
| Age (yr) | 28.3 ± 10.1 | 33.4 ± 8.9 | 31.6 ± 8.8 | 0.362 | 0.062 |
| W:H ratio | 0.74 ± 0.04 | 0.77 ± 0.05 | 0.78 ± 0.04 | 0.065 | 0.166 |
| Trunk fat (kg) | 5.0 ± 2.5 | **8.1 ± 2.2** [A C] | **18.7 ± 5.2** [B] | **<0.001** | **0.760** |
| Body fat (kg) | 17.6 ± 5.7 | **25.6 ± 4.0** [A C] | **51.6 ± 10.2** [B] | **<0.001** | **0.798** |
| Body fat (%) | 30.0 ± 7.0 | **36.8 ± 5.3** [A C] | **51.2 ± 2.5** [B] | **<0.001** | **0.660** |
| LBM (kg) | 40.2 ± 4.3 | 43.9 ± 4.2 | **49.1 ± 8.5** [A] | **0.020** | **0.230** |
| HR max (%) | 188 ± 9 | 184 ± 7 | 186 ± 7 | 0.451 | 0.049 |
| SBP (mmHg) | 110 ± 11 | 117 ± 12 | **132 ± 18** [B] | **0.003** | **0.327** |
| DBP (mmHg) | 70 ± 9 | 74 ± 10 | 79 ± 10 | 0.164 | 0.114 |
| MAP (mmHg) | 84 ± 9 | 88 ± 10 | **96 ± 11** [A] | **0.027** | **0.214** |
| Glucose (mmol·L$^{-1}$) | 5.2 ± 0.5 | 5.3 ± 0.4 | 5.5 ± 0.4 | 0.348 | 0.064 |

Data are mean ± SD

[A] $p < 0.05$ from BMI <24.9 kg·m²

[B] $p < 0.001$ from BMI <24.9 kg·m²

[C] $p < 0.001$ from BMI >30 kg·m²

Abbreviations used: W:H (waist-to-hip); LBM (lean body mass); SBP (systolic blood pressure); DBP (diastolic blood pressure).

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**Table 2.** Speed, gradient and heart rate (% of maximum) achieved at each stage of the graded test and classified according to BMI.

| | | **Normal** <24.9 kg·m² (n = 15) | **Overweight** 25-29.9 kg·m² (n = 13) | **Obese** >30 kg·m² (n = 7) |
|---|---|---|---|---|
| **Speed (km·h⁻¹)** | | | | |
| | 30% | 4.6 ± 1.0 | 4.0 ± 0.8 | 2.0 ± 0.5 |
| | 40% | 5.6 ± 0.9 | 5.1 ± 0.8 | 3.7 ± 1.0 |
| | 50% | 6.6 ± 1.0 | 6.1 ± 0.9 | 4.9 ± 0.7 |
| | 60% | 7.5 ± 1.2 | 6.0 ± 0.7 | 5.7 ± 0.6 |
| **Gradient (%)** | | | | |
| | 30% | 0.5 ± 0 | 0.5 ± 0 | 0.5 ± 0 |
| | 40% | 0.5 ± 0 | 0.5 ± 0 | 0.5 ± 0 |
| | 50% | 0.7 ± 0.5 | 0.5 ± 0 | 0.5 ± 0 |
| | 60% | 1.1 ± 1.4 | 1.0 ± 0.9 | 0.5 ± 0 |

Data are mean ± SD

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**Table 3.** Indicators of agreement and concordance between measures of blood pressure during the graded test.

| | SBP | DBP | MAP |
|---|---|---|---|
| **Baseline** | | | |
| Pearson' r | 0.936 [A] | 0.889 [A] | 0.935 [A] |
| Kendall's tau-b | 0.762 [A] | 0.739 [A] | 0.766 [A] |
| **30% $VO_{2MAX}$** | | | |
| Pearson' r | 0.821 [A] | 0.838 [A] | 0.868 [A] |
| Kendall's tau-b | 0.627 [A] | 0.778 [A] | 0.672 [A] |
| **40% $VO_{2MAX}$** | | | |
| Pearson' r | 0.886 [A] | 0.833 [A] | 0.905 [A] |
| Kendall's tau-b | 0.729 [A] | 0.707 [A] | 0.697 [A] |
| **50% $VO_{2MAX}$** | | | |
| Pearson' r | 0.933 [A] | 0.892 [A] | 0.931 [A] |
| Kendall's tau-b | 0.766 [A] | 0.715 [A] | 0.707 [A] |
| **60% $VO_{2MAX}$** | | | |
| Pearson' r | 0.931 [A] | 0.891 [A] | 0.945 [A] |
| Kendall's tau-b | 0.782 [A] | 0.732 [A] | 0.809 [A] |

[A] $p < 0.001$

Abbreviations used: SBP (systolic blood pressure); DBP (diastolic blood pressure); MAP (mean arterial pressure).

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# References


[1] World Health Organization. Global Heal Observatory (GHO) data. Blood pressure. 2015.

[2] Manolio TA, Burke GL, Savage PJ, Sidney S, Gardin JM, Oberman A. Exercise Blood Pressure Response and 5-Year Risk of Elevated Blood Pressure in a Cohort of Young Adults: The CARDIA Study. American Journal of Hypertension. 1994;7(3):234-41. doi: 10.1093/ajh/7.3.234.

[3] Miyai N, Arita M, Morioka I, Miyashita K, Nishio I, Takeda S. Exercise BP response in subjects with high-normal BP: Exaggerated blood pressure response to exercise and risk of future hypertension in subjects with high-normal blood pressure. Journal of the American College of Cardiology. 2000;36(5):1626-31. doi: https://doi.org/10.1016/S0735-1097(00)00903-7.

[4] Singh JP, Larson MG, Manolio TA, O'Donnell CJ, Lauer M, Evans JC, et al. Blood Pressure Response During Treadmill Testing as a Risk Factor for New-Onset Hypertension. Circulation. 1999;99(14):1831-6. doi: 10.1161/01.CIR.99.14.1831.

[5] Wilson NV, Meyer BM. Early prediction of hypertension using exercise blood pressure. Preventive Medicine. 1981;10(1):62-8. doi: https://doi.org/10.1016/0091-7435(81)90006-2.

[6] Mundal R, Kjeldsen Sverre E, Sandvik L, Erikssen G, Thaulow E, Erikssen J. Exercise Blood Pressure Predicts Mortality From Myocardial Infarction. Hypertension. 1996;27(3):324-9. doi: 10.1161/01.HYP.27.3.324.

[7] World Health Organization. Global Health Observatory (GHO) data. Overweight and obesity. 2015.

[8] Hall JE. Pathophysiology of obesity hypertension. Current Hypertension Reports. 2000;2(2):139-47. doi: 10.1007/s11906-000-0073-4.

[9] Hall JE, Crook ED, Jones DW, Wofford MR, Dubbert PM. Mechanisms of obesity-associated cardiovascular and renal disease. The American Journal of the Medical Sciences. 2002;324(3):127-37. doi: 10.1097/00000441-200209000-00003.

[10] Pierson LM, Bacon SL, Sherwood A, Hinderliter AL, Babyak M, Gullette EC, et al. Relationship between exercise systolic blood pressure and left ventricular geometry in overweight, mildly hypertensive patients. Journal of Hypertension. 2004;22(2):399-405. PubMed PMID: 00004872-200402000-00026.

[11] Huot M, Arsenault Benoit J, Gaudreault V, Poirier P, Pérusse L, Tremblay A, et al. Insulin Resistance, low cardiorespiratory fitness, and increased exercise blood pressure. Hypertension. 2011;58(6):1036-42. doi: 10.1161/HYPERTENSIONAHA.111.180349.

[12] Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. Circulation. 1983;67(5):968-77. doi: 10.1161/01.CIR.67.5.968.

[13] Wenger NK, Arnold A, Bairey Merz CN, Cooper-DeHoff RM, Ferdinand KC, Fleg JL, et al. Hypertension Across a Woman's Life Cycle. Journal of the American College of Cardiology. 2018;71(16):1797-813. doi: 10.1016/j.jacc.2018.02.033.

[14] Le V-V, Mitiku T, Sungar G, Myers J, Froelicher V. The Blood Pressure Response to Dynamic Exercise Testing: A Systematic Review. Progress in Cardiovascular Diseases. 2008;51(2):135-60. doi: https://doi.org/10.1016/j.pcad.2008.07.001.

[15] Myers JN. The physiology behind exercise testing. Primary Care: Clinics in Office Practice. 2001;28(1):5-28. doi: https://doi.org/10.1016/S0095-4543(05)70005-1.

[16] Kim D, Ha J-W. Hypertensive response to exercise: mechanisms and clinical implication. Clin Hypertens. 2016;22:17-. doi: 10.1186/s40885-016-0052-y. PubMed PMID: 27468357.


ACCEPTED FOR PUBLICATION

[17] MacKnight JM. Exercise considerations in hypertension, obesity, and dyslipidemia. Clinics in Sports Medicine. 2003;22(1):101-21. doi: 10.1016/S0278-5919(02)00069-8.
[18] jamovi project. jamovi (Version 0.9). Retrieved from https://www.jamovi.org2018.
[19] Steffan HG, Elliott W, Miller WC, Fernhall B. Substrate utilization during submaximal exercise in obese and normal-weight women. European Journal of Applied Physiology and Occupational Physiology. 1999;80(3):233-9.
[20] Pescatello LS, Arena R, Riebe D, Thompson PD. ACSM's guidelines for exercise testing and prescription. 9th edition ed. Medicine ACoS, editor. Baltimore, MD: Lippincott Williams & Wilkins; 2014.
[21] Ramalingam L, Menikdiwela K, LeMieux M, Dufour JM, Kaur G, Kalupahana N, et al. The renin angiotensin system, oxidative stress and mitochondrial function in obesity and insulin resistance. Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease. 2017;1863(5):1106-14. doi: https://doi.org/10.1016/j.bbadis.2016.07.019.
[22] Shim CY, Ha J-W, Park S, Choi E-Y, Choi D, Rim S-J, et al. Exaggerated Blood Pressure Response to Exercise Is Associated With Augmented Rise of Angiotensin II During Exercise. Journal of the American College of Cardiology. 2008;52(4):287-92. doi: https://doi.org/10.1016/j.jacc.2008.03.052.
[23] Warner JG, Metzger DC, Kitzman DW, Wesley DJ, Little WC. Losartan improves exercise tolerance in patients with diastolic dysfunction and a hypertensive response to exercise. Journal of the American College of Cardiology. 1999;33(6):1567-72. doi: https://doi.org/10.1016/S0735-1097(99)00048-0.
[24] Kenney MJ, Seals DR. Postexercise hypotension. Key features, mechanisms, and clinical significance. Hypertension. 1993;22(5):653-64. doi: 10.1161/01.HYP.22.5.653.
[25] Hagberg JM, Park J-J, Brown MD. The Role of Exercise Training in the Treatment of Hypertension. Sports Med. 2000;30(3):193-206. doi: 10.2165/00007256-200030030-00004.
[26] Wewege M, van den Berg R, Ward RE, Keech A. The effects of high-intensity interval training vs. moderate-intensity continuous training on body composition in overweight and obese adults: a systematic review and meta-analysis. Obesity Reviews. 2017;18(6):635-46. doi: 10.1111/obr.12532.
[27] Bonsu B, Terblanche E. The training and detraining effect of high-intensity interval training on post-exercise hypotension in young overweight/obese women. European Journal of Applied Physiology. 2016;116(1):77-84. doi: 10.1007/s00421-015-3224-7.

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