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1 Body composition influences blood pressure during submaximal
2 graded test in women

3
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11

12

13 Abstract

14 This study aimed to determine whether body composition affected cardiovascular responses
15 of normal, overweight and obese women by comparing heart rate (HR) and blood pressure
16 (BP) during a graded submaximal treadmill exercise. Thirty five healthy non-smoking,
17 premenopausal women (age 30.8 ± 9.5 yr; height 1.66 ± 0.05 m; weight 73.8 ± 18.9 kg; body fat
18 $36.2 \pm 9.5\%$; maximum oxygen uptake 33.5 ± 8.1 mL·min·kg⁻¹) volunteered for this study, and
19 were classified into three groups based on BMI: normal weight (NOR; ≤ 24.9 kg·m²),
20 overweight (OVW; 25-29.9 kg·m²) and obese (OBE; ≥ 30 kg·m²). Participants performed a
21 submaximal graded test at 30%, 40%, 50% and 60% of maximal oxygen uptake, during
22 which HR, systolic BP (SBP), diastolic BP (DBP) and mean arterial pressure (MAP) were
23 measured. At rest, participants of OBE group had higher BP than NOR and OVW. During the
24 submaximal graded test, HR, SBP and MAP increased as a function of intensity. There were
25 no group differences in HR, but greater SBP, DBP and MAP in OBE compared to NOR and
26 OBW at all intensities except 60%. Overall, our study further contributes to underscore the
27 importance of stress testing specifically to evaluate the increased risks apparently healthy
28 obese women are placed at to developing diseases from the chronic exposure to raised BP,
29 despite normal resting BP and HR responses during exercise.

30

31 **Introduction**

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

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

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

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

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

71

72 **Methods**

73 **Ethics statement and participants**

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

77 written informed consent prior to inclusion in the study. Participants consisted of healthy,
78 non-menopausal female individuals aged 18 to 50 years of age (n=35; age 30.8±9.5 yr; height
79 1.66±0.05 m; weight 73.8±18.9 kg; maximum oxygen uptake 33.5±8.1 mL·min⁻¹·kg⁻¹) who
80 were **not smoking and** not taking any medications known to affect cardiovascular measures in
81 addition to being free from both mental and physical impairments.

82 Participants were invited for a preliminary visit for screening against underlying health
83 conditions potentially affecting the study procedures and findings (Medical Health
84 Questionnaires, lung function assessed using spirometry) and familiarisation with the
85 procedures and equipment. Resting anthropometric characteristics of participants was
86 recorded (height, weight, body mass index [BMI]=weight/height², hip and waist
87 circumferences, waist-to-hip ratio [W:H]) and their body composition using dual-energy x-
88 ray absorptiometry (DEXA; Lunar Prodigy Advance, GE Healthcare, Buckinghamshire, UK)
89 (total and regional body fat, muscle mass, percentage body fat, bone mineral density [BMD]
90 and bone mineral content [BMC], and blood pressure, blood glucose and cholesterol were
91 also measured during the initial visit.

92 Methodology

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

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

107 The second session consisted in a submaximal test including five exercise stages, each lasting
108 3 min. After an initial warm-up stage at $4.5 \text{ km}\cdot\text{h}^{-1}$ (baseline), a graded test with four intensity
109 stages were performed at relative intensities corresponding to 30%, 40%, 50% and 60% of
110 $\dot{V}O_{2\text{MAX}}$. Heart rate (HR) was measured continuously at 0.2 Hz using a chest strap (Polar
111 Electro, Kempele, Finland). Blood pressure responses were measured twice at the end of each
112 stage of exercise during the graded test using a manual blood pressure cuff, aneroid and
113 stethoscope (Welch Allyn, New York USA).

114 Statistical analyses

115 Participants were classified into three groups based on BMI: normal weight (NOR; ≤ 24.9
116 $\text{kg}\cdot\text{m}^2$), overweight (OVW; $25-29.9 \text{ kg}\cdot\text{m}^2$) and obese (OBE; $\geq 30 \text{ kg}\cdot\text{m}^2$). For comparison
117 purposes, participants were also grouped into tertiles of percentage of body fat. Contingency
118 tables showed strong association ($p < 0.001$) between the two approaches and BMI groups
119 were used for analyses.

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

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

136

137 **Results**

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

146

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

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

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

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

155 The two measures of SBP, DBP and MAP performed at each exercise intensity were in high
156 agreement and concordance (Table 3). No participant achieved SBP characteristic of HRE
157 (>190 mmHg), and all participants with resting pre-hypertension and hypertension had
158 normal exercise BP (maximum 172/80 mmHg). The dynamics of BP response were markedly
159 different between OBE and the two other groups (Figure 1): minimum BP responses were
160 observed at 30% in NOR (SBP: 116 ± 15 mmHg; DBP: 81 ± 5 mmHg; MAP: 93 ± 8 mmHg) and
161 OVW groups (SBP: 112 ± 11 mmHg; DBP: 79 ± 6 mmHg; MAP: 90 ± 6 mmHg) compared to
162 60% in OBE (SBP: 134 ± 13 mmHg; DBP: 82 ± 6 mmHg; MAP: 100 ± 7 mmHg), and maximum
163 BP responses were observed at 60% in NOR (SBP: 141 ± 19 mmHg; DBP: 84 ± 10 mmHg;
164 MAP: 103 ± 12 mmHg) and OVW (SBP: 141 ± 15 mmHg; DBP: 86 ± 9 mmHg; MAP: 104 ± 11
165 mmHg) compared to 50% in OBE (SBP: 148 ± 17 mmHg; DBP: 95 ± 10 mmHg; MAP: 112 ± 11
166 mmHg).

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

168 For SBP, there were significant and large main effects of exercise intensity ($p<0.001$;
169 $\eta^2_p=0.389$) and group ($p=0.001$; $\eta^2_p=0.370$), with no main effect of interaction ($p=0.570$;
170 $\eta^2_p=0.052$). There was no significant effect of age ($p=0.894$; $\eta^2_p=0.001$). Post-hoc testing

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

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

178 For MAP, there were significant and large main effects of exercise intensity ($p<0.001$;
179 $\eta^2_p=0.232$) and group ($p<0.001$; $\eta^2_p=0.430$), with no main effect of interaction ($p=0.160$;
180 $\eta^2_p=0.099$). There was no significant effect of age ($p=0.812$; $\eta^2_p=0.002$). Post-hoc testing
181 revealed DBP in OBE group was significantly different from NOR ($p=0.004$) and OVW
182 ($p<0.001$), with no significant differences between NOR and OVW ($p=0.977$).

183 Percentage body fat was significantly inversely correlated with HR% only at 60% ($r=-0.464$,
184 $p=0.006$), with SBP at all intensity levels (30%: $r=0.500$, $p=0.004$; 40%: $r=0.448$, $p=0.012$;
185 50%: $r=0.471$, $p=0.008$; 60%: $r=0.522$, $p=0.003$), with DBP at all intensity levels (30%:
186 $r=0.634$; 40%: $r=0.644$; 50%: $r=0.695$, 60%: $r=0.682$; all correlations $p<0.001$), and with
187 MAP at all intensity levels (30%: $p=0.594$, $p<0.001$; 40%: $r=0.585$, $p<0.001$; 50%: $r=0.630$
188 ; 60%: $r=0.649$; all correlations $p<0.001$).

189 Discussion

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

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

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

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

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

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

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

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

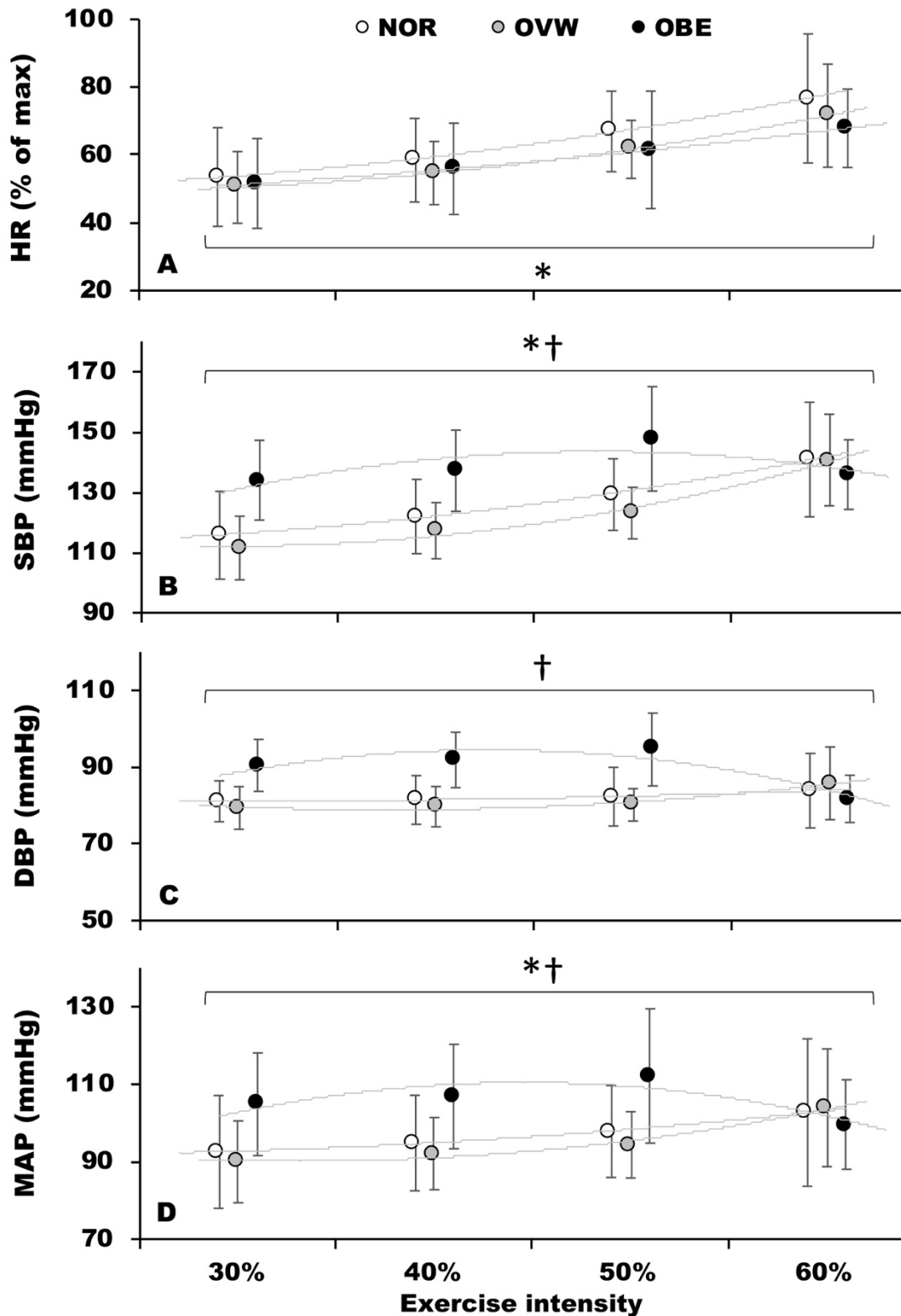
252

253 **Perspectives**

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

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268

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

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

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276 **Tables**

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

	Normal	Overweight	Obese	ANOVA	
	<24.9 kg·m ²	25-29.9 kg·m ²	>30 kg·m ²	p	η 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

279 Data are mean ± SD

280 ^A p < 0.05 from BMI <24.9 kg·m²

281 ^B p < 0.001 from BMI <24.9 kg·m²

282 ^C p < 0.001 from BMI >30 kg·m²

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

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

		Normal <24.9 kg·m ² (n = 15)	Overweight 25-29.9 kg·m ² (n = 13)	Obese >30 kg·m ² (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

288 Data are mean ± SD

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

292

		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

293

^A p < 0.001

294

Abbreviations used: SBP (systolic blood pressure); DBP (diastolic blood pressure);

295

MAP (mean arterial pressure).

296

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