Original Research Article

BLOOD PRESSURE AND HEART RATE RESPONSES IN YOUNG NIGERIAN WOMEN DURING PASSIVE AND ZERO-RESISTANCE ACTIVE EXERCISE TESTS COMPARED TO NON-OBESE CONTRO

ABSTRACT

Aim: To compare blood pressure (BP) and heart rate (HR) responses to passive and unloaded active leg movements and the neural control mechanisms between obese young female adults and their nonobese control.

Study Design: A case control study

Place and Duration of Study: Department of Human Physiology, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria, between July 2015 to January 2016.

Methodology: Thirty normotensive young women were randomly selected for this study and assigned into two groups- obese (n =15) and non-obese (n =15). Subjects performed a sub-maximal ergometer exercise test. Each group performed separate bouts of 5-minutes passive and unloaded active cycling exercises. BP and HR were recorded at baseline and during the two exercise conditions. All statistics were done using SPSS (version 25). Comparative analyses were done using independent sample t-test (for normally distributed data) or Mann Whitney-U (for abnormally distributed data) or a two-way analysis of variance (ANOVA).

Results: The obese women indicated significantly ($p < 0.05$) higher HR, systolic BP, diastolic BP, mean arterial pressure (MAP), and rate pressure product (RPP) compared with the non-obese control at baseline, during passive and unloaded active exercise conditions. The magnitude of increase (Δ) in BP from baseline to passive leg movement and ΔHR elicited from passive to unloaded active movement showed no significant differences between the two groups.

Conclusions: The higher mean BP and HR observed in the obese women, at baseline, during passive and unloaded active cycle movements were attributed to poor cardio-respiratory fitness and greater myocardial metabolic demand imposed by excessive adipose tissue. The lack of significant differences in $\Delta BP_{baseline\text{-}passive\ exercise}$ and $\Delta HR_{passive\text{-}active}$, between the two groups, suggests that both mechanoreceptors and central neural command played limited roles in increased BP and HR drive during low-intensity dynamic exercise tests.

Keywords: Blood Pressure, Heart Rate, Rate Pressure Product, Oxygen Uptake, Passive Exercise, Unloaded Active Exercise.

INTRODUCTION

The rising prevalence of obesity worldwide is fast becoming a severe global epidemic [1, 2]. Similarly, the impact of obesity on the risk of developing chronic diseases such as type 2 diabetes, stroke, and cardiovascular diseases, particularly heart failure, hypertension, and coronary heart disease is also a major health concern [3]. More worrisome is the steadily increasing trend of obesity among young adults, especially college and university students [4, 5]. Currently, young adults are being recognized as a 'vulnerable group' for unhealthy lifestyles (inactivity, eating large portions of processed food and sugary drinks, long hours of watching television and playing computer games), which lead to overweight and obesity and put them at high risk of heart disease earlier in life [6]. Due to the high prevalence of obesity among young adults, clinicians and investigators are faced with the challenge of unraveling the link between obesity and early chronic pathologies including diseases such as hypertension, diabetes, and atherosclerosis, that were once limited to older adult populations.

It is consistently reported that obesity is more prevalent amongst females compared to males [7, 8, 9]. Females are also reported to be more vulnerable to factors that lead to obesity such as sedentary lifestyles and cultural values favoring the wrong notion that a larger body size among women is a sign of fertility, healthfulness, or prosperity [10, 11]. Furthermore, obese females are more likely to show poor hemodynamic responses during exercise [12, 13], and to perform less daily physical activity compared with males [14, 15]. Hence the need to evaluate hemodynamic responses during exercise in young obese females and the mechanisms behind these responses.

Exercise stresses the body's physiological systems, which can lead to a variety of physiologic changes needed to supply enough oxygen and nutrients to meet the demands of muscular effort. A rise in HR and BP are two immediate physiological responses to exercise. It is also established that exercise intensity is one of the factors influencing these hemodynamic responses elicited by exercise. However, there is a paucity of comparative studies regarding HR and BP responses to involuntary (passive) and voluntary (zero resistance active) exercise tests between normotensive young obese females and non-obese control, and to the best of our knowledge, no such study has been done in the Nigerian population. Although previous studies have examined hemodynamic responses following exercise in obese individuals, most of these investigations were carried out using loaded active exercise tests or among older adults and often directed towards clinical application in diagnosing cardiovascular abnormalities. Moreover, some of these studies made use of complex, invasive, high-cost, and not easy-to-administer instruments. This study was therefore undertaken to assess the BP and HR responses to involuntary (passive) and voluntary (zero resistance active) ergometer exercise tests and to compare them between obese adult females and their nonobese control, using simple, non-invasive, low-cost, easily administered

instruments. We also sought to determine the possible mechanisms behind the BP and HR responses during such extremely low or no-intensity exercise tests and compare their inputs between the obese females and their non-obese controls. To identify and understand the reflex pathways mediating the hemodynamic responses in the present study, we employed a 'reductionist' approach which has been used previously to assess the HR and BP responses from baseline to passive exercise and from passive to unloaded active exercise.

We hypothesized that 1) obesity would cause an increase in BP and HR responses to both passive and unloaded active exercise tests (2) both the passive and unloaded active cycle movements will elicit augmented BP and HR responses in normotensive obese women versus nonobese control (3) the mechanoreflex and central neural command activation would be greater in obese females vs. nonobese control.

METHODS

Subjects

Thirty apparently healthy young female volunteers from the students' population and within the age brackets of 18 to 30 years, were assigned into two groups – obese (body mass index (BMI) ≥ 30 kg/m^2; n = 15) and normal weight (BMI, <25 kg/m^2; n = 15). A structured health and lifestyle questionnaire was administered to the participants to ascertain their health statuses and determine if they were qualified to participate in the study or not. Subjects were excluded from the study if their BMI were below 18.5 kg/m^2, or if they had a history of cardiovascular, respiratory, or musculoskeletal diseases, exercise limitations, medical problems, and confounding results. The participants were also excluded if they were smokers or alcoholics and if they were ill, experiencing menstrual flow, or using drugs that may affect cardiovascular functions.

Anthropometric Measurements

The anthropometric measurements of participants such as height and weight were recorded. The height of the participants without footwear was measured to the nearest 0.1 centimeters with a portable stadiometer (SECA, Hamburg, Germany). Body weight was measured to the nearest 0.1kg with the participants in an upright position, with light clothes and without footwear using a calibrated weighing scale. The body mass index (BMI) was calculated as body weight in kg/height in m^2.

Exercise Test Protocol

The study was conducted at the Human Physiology Laboratory, Nnamdi Azikiwe University, Nigeria. Experimental tests were conducted in a well-ventilated room at the same period of the day (between 7 to 11 am) for each participant. Ambient room temperature during the study ranged between 23-25℃. The participants were informed about their right to decide whether to participate in the study or not. They were also asked to refrain from exercise, alcoholic drinks, and stimulants like coffee and caffeinated drinks 24 hours before the exercise test. The

investigation conformed to the principles outlined in the Declaration of Helsinki except for registration in a database.

The two experimental groups were assigned to two kinds of exercise tests – passive and unloaded active cycle exercise. During the passive exercise, the participant's feet were well secured to the cycle ergometer pedals, which were rotated for them at the rate of 60 revolutions per minute. In order not to contribute to or resist the pedal rotation, the participants were instructed and constantly reminded to keep their legs fully relaxed. The passive exercise test was selected to examine the role of mechanoreceptors during leg movement without the accompanying participation of the central neural command or metaboreceptors [16].

In the unloaded active exercise test, the cycle ergometer was set at zero point so that the participant pedaled with no resistance or load at the rate of 60 revolutions per minute. The unloaded active test involves the central neural command and the mechanoreceptors; thus, the passive cycling test was performed to serve as a control for the central neural command effect. Both exercise tests lasted for 5 minutes each. The peak values of the central hemodynamic parameters including HR, systolic blood pressure (SBP), and diastolic blood pressure (DBP) were measured and recorded at the end of the 5 minutes, and then the exercise was terminated. The exercise tests were separated by at least 10 minutes for rest to allow the hemodynamic variables to return to baseline.

Measurement of Hemodynamic Parameters

The hemodynamic parameters – HR, SBP, and DBP were measured using an electronic BP and HR monitor (Omron Health Care Inc., Vernon Hills, Illinois). Other parameters such as mean arterial pressure (MAP) and rate pressure product (RPP) were estimated using standard equations.

(a) Blood Pressure and Heart Rate Measurements:

The resting BP and HR of participants were measured twice after 10 and 15 minutes of rest in a quiet room and with the participants in a sitting position, using the Omron electronic monitor. The average resting BP and HR measurements were used for the baseline data analysis. The peak exercise BP and HR measurements were done immediately before the termination of the passive and unloaded active exercise tests, and used as peak exercise data analysis.

(b) Mean Arterial Pressure

The mean arterial pressure was calculated using the formula; [systolic blood pressure + (2 x diastolic blood pressure)] / 3 [17].

(c) Rate Pressure Product

The rate pressure product, a valuable marker of the oxygen requirement in the heart in a given condition, was calculated as HR × SBP.

(d) Resting Oxygen Consumption (VO_2)

The resting VO_2 was determined by direct calorimetry using a modified Weir equation for resting energy expenditure (REE) to generate a simplified equation based on carbon dioxide elimination (VCO_2).

REE (kcal/day) = [3.941(VO_2) + 1.106(VCO_2)] × 1440 [18]

Given that Respiratory Exchange Ratio (RER) = VCO_2/VO_2 and RER = 0.82 (for mixed diet), then rearrange for VCO_2 = VO_2 x 0.82 and substitute in the equation.

REE (kcal/day) = [3.941(VO_2) + 1.106(VO_2 x 0.82)] x 1440

REE (kcal/day) = [3.941(VO_2) + 1.106(VO_2) x 0.907)] x 1440

REE (kcal/day) = [5.047 (VO2) x 0.907] x 1440

VO_2 (L/min) = REE (kcal/day) / [5.047 x 0.907] x 1440

VO_2 (L/min) = REE / 6590.9

Given that REE is calculated as; 655.1 + (9.56 x body weight in kg) + (1.86 x height in cm) – (4.68 x age in years) using Harris Benedict's equation for females [19], we determined the resting VO_2 (L/min) using the above equation. The absolute resting VO_2 (L/min) values were converted to relative values (ml/kg/min) using the formula; (Absolute VO_2 x 1000) / Body weight.

Data Analysis

Data were presented as mean and standard deviation or median (range). The test for normality was done using Shapiro-Wilk's normality test. Comparative analysis involving two groups was done using an independent sample t-test (for normally distributed data) or Mann Whitney-U (for abnormally distributed data). Comparison among multiple groups was done using a two-way analysis of covariance (ANCOVA) and analysis of variance (ANOVA) as appropriate. Pearson's bivariate test (for normally distributed data) and Spearman's rho test (for abnormally distributed data) were used in assessing the relationships between two groups of variables where appropriate. $P < 0.05$ was considered to be statistically significant. All statistics were done using SPSS (version 25).

RESULTS

The demographic characteristics of the study population are expressed in Table 1. Data indicated significantly greater body weight ($p < 0.001$), BMI ($p < 0.001$), WC ($p < 0.001$), HC ($p < 0.001$), and WHR ($p = 0.003$) in obese individuals compared with the non-obese controls. In contrast, there were no significant differences found in age ($p = 0.446$) and height ($p = 0.441$) between the two groups.

Table 2 shows the comparative analysis of the hemodynamic variables between obese and non-obese participants at baseline, peak of passive, and unloaded active cycle exercise tests. The obese women exhibited significantly higher ($p < 0.05$, $p < 0.01$, $p < 0.001$) mean HR, SBP, DBP, MAP, and RPP compared with the non-obese controls at rest and during passive and unloaded active exercise tests. The non-obese women indicated higher resting VO_2 ($p < 0.001$) compared with their obese counterparts.

Considering the experimental conditions, data indicated that during passive exercise, the non-obese individuals showed significantly higher mean HR ($p = 0.003$), SBP ($p = 0.020$), MAP ($p < 0.001$), and RPP ($p = 0.003$) compared with the baseline. Diastolic blood pressure did not differ between the two experimental conditions. The passive exercise also elicited higher MAP ($p = 0.05$) compared with the baseline in obese women. In contrast, no significant difference was seen in mean HR, SBP, DBP, and RPP between the passive exercise and baseline values in obese women. Following the unloaded active exercise, non-obese women showed significantly ($p < 0.001$) higher mean HR, SBP, DBP, MAP, and RPP compared with the baseline values. Similarly, the obese women exhibited significantly higher mean HR ($p < 0.001$), SBP ($p = 0.008$), DBP ($p = 0.014$), MAP ($p = 0.003$), and RPP ($p < 0.001$) compared with baseline.

Furthermore, during the unloaded active exercise test, the non-obese women indicated significantly greater mean peak HR ($p = 0.002$), SBP ($p = 0.001$), DBP ($p < 0.001$), MAP ($p < 0.001$) and RPP ($p = 0.001$) compared with the passive exercise condition. The obese women also presented higher ($p < 0.001$) peak HR and RPP, but similar SBP ($p = 0.065$), DBP ($p = 0.379$), and MAP ($p = 0.074$), during the unloaded active exercise compared with passive exercise.

Table 3 shows changes in HR and BP from baseline to passive and unloaded active exercise conditions and from passive to unloaded active exercise conditions. No significant differences ($p > 0.05$) were detected in the magnitude of ΔHR, ΔSBP, ΔMAP and ΔRPP between obese and non-obese women at baseline to peak passive exercise, baseline to peak unloaded active exercise, and passive to unloaded active exercise conditions respectively. Significant differences were observed in $\Delta DBP_{\text{baseline-unloaded active}}$ ($p = 0.033$) and $\Delta DBP_{\text{passive – unloaded active}}$ ($p = 0.021$), but not in $\Delta DBP_{\text{baseline – passive}}$ ($p = 0.460$).

Correlation analysis between resting VO_2 and BMI in the non-obese, obese and overall data of the study population is shown in Figure 1. Results indicated significant negative correlations ($p < 0.001$) between resting VO_2 and BMI in all the regimens. In addition, Table 4 indicated

significant negative correlations between resting VO_2 and HR, SBP, DBP, MAP, and RPP respectively, at rest ($p < 0.001$), during passive exercise ($p < 0.001$), and during unloaded active exercise ($p < 0.05$; $p < 0.01$; $p < 0.001$).

DISCUSSION

The main findings of this study are as follows; (i) the obese women indicated significantly higher HR, SBP, DBP, MAP and RPP compared with the non-obese control at baseline, and during passive and unloaded active exercise conditions; on the other hand, the non-obese women indicated higher resting VO_2 compared with their obese counterparts (ii) the passive cycle leg movement elicited an increase in the hemodynamic variables (HR, SBP, MAP, RPP) in non-obese control, but not in obese women, compared with baseline (iii) the unloaded active exercise movement resulted in augmented HR, SBP, DBP, MAP and RPP in both obese and non-obese women compared with baseline (iv) unloaded active exercise movement also elicited increase in HR and RPP in both obese and non-obese women compared with passive movement (v) the hemodynamic responses elicited during passive and unloaded active cycle movements were not different between obese and non-obese women (vi) negative correlations were observed in subjects' resting VO_2 vs. BMI, HR and BP parameters.

The present study, which indicated significantly higher mean resting SBP, DBP, and MAP in obese compared with nonobese women, is in agreement with the findings of Martin et al., 2003; Roopa et al., 2011 and Chrysohoou et al., 2010 [20, 21, 22]. Regarding the resting HR values, the result of the obese group was also higher compared to the non-obese group, which concurs with previous findings that indicated the existence of a linear association between the level of adiposity and resting HR in obese individuals [23]. We could not find any previous study comparing HR, SBP, DBP, MAP, and RPP between the obese and non-obese women following passive and unloaded active cycling tests. However, in agreement with a previous study involving individuals of normal weight passive cycling resulted in an increase in BP and HR [24]. Nobrega et al also reported that passive cycling at 60rpm by healthy non-obese individuals elicited an increase in BP, but with no change in HR; on the other hand, unloaded active cycling resulted in an HR increase with no change in BP [25]. The higher BP and HR observed in obese individuals at baseline and during passive and unloaded active exercise conditions may be related to altered autonomic functions and poor cardio-respiratory fitness. A reduction in parasympathetic activity and predominance of sympathetic activity has been reported in the obese group compared to normal-weight individuals [26]. A lower, but normal RPP level is considered to be an indicator of increased parasympathetic nervous outflow and increased parasympathetic tone and is also believed to be cardio-protective [27]. RPP, an index of myocardial oxygen demand, is also useful in determining the physical fitness of a healthy person at rest and in stressed conditions [28]. Interestingly, we observed higher RPP levels among the obese group compared with the non-obese. The higher RPP, especially at the resting level, is mainly due to higher HR levels observed among obese individuals, which is considered an indicator of poor aerobic fitness and cardiac function [28]. Another contributory factor to the greater hemodynamic responses in obese persons could be the higher myocardial metabolic demand imposed by their excessive adipose tissue. The higher myocardial metabolic demand is indicated by the greater RPP values observed in obese persons compared with the non-obese group. The RPP, therefore,

offers an integrative perspective concerning autonomic function, cardiovascular health, and hemodynamic responses to exercise [29].

Furthermore, the VO_2, a very important index of cardio-respiratory fitness in individuals at rest as well as in stressed conditions, was significantly greater in non-obese compared with the obese individuals at baseline. This was an indication that the non-obese individuals had greater cardio-respiratory fitness and functional capacity compared with the obese group. Previous studies have also shown strong negative correlations between cardio-respiratory fitness levels of individuals and their BMI [30] as well as their HR and BP levels [31]. These findings are in agreement with the present study which indicated significant negative correlations between the baseline VO_2 and BMI and the hemodynamic parameters at different conditions. The present findings, therefore, add to the knowledge of the link between obesity and alterations in BP and HR at rest and during voluntary and involuntary exercise tests and suggest the importance of improving the cardio-respiratory fitness statuses of obese individuals to improve their hemodynamic outcomes.

Exercise pressor reflexes (mechanoreflex and metaboreflex) and the baroreflex work together to tightly regulate the BP and HR responses to exercise [32]. Alterations in these reflexes have been described in obesity, but it is difficult to isolate the exact contribution of each of these components in humans [33]. Passive exercise, an involuntary physical activity, engages the mechanoreceptors without the concomitant participation of central neural command or metaboreceptors [16]. Stimulation of the group III afferent nerve activates the exercise mechanoreflex to contribute to increases in HR, SV, and sympathetic nerve activity. These three factors work together to raise CO, redistribute blood volume, and maintain MAP [34]. We could not assess the mechanoreflex mechanism directly during the protocols, however, using the reductionist approach, the principal difference in BP between baseline and passive pedaling was taken as the mechanoreceptor input during the passive exercise as previously demonstrated in previous studies [25, 35]. Compared with the baseline, our data indicated that the passive cycle leg movement elicited an increase in MAP in both non-obese control and obese women, thus suggesting the activation of the muscle mechanoreceptors by passive cycling movement and in agreement with previous studies [25, 35]. However, the blood pressure responses (increase (Δ) in SBP, DBP, and MAP) did not show significant differences between the obese women and their non-obese counterparts, suggestive of similar mechanoreflex sensitivity between the two groups. The similar ΔBP observed in obese and non-obese women may be attributed to the equal ΔHR indicated by the two groups. In a previous study [32] of a differing methodology, obese women exhibited a greater BP response compared with their non-obese control, during involuntary contractions involving whole-body vibration suggestive of an exaggerated mechanoreflex.

When subjected to an unloaded active (voluntary) exercise test, both the obese and non-obese women indicated significantly higher mean HR, SBP, DBP, MAP, and RPP compared with the baseline values as well as higher HR and RPP compared with passive cycling movement. The unloaded active pedaling engages both the mechanoreceptors and central neural command, thus the increase in BP and HR variables relative to baseline suggests that the two neural components were activated during the unloaded active cycling test. The passive pedaling test was therefore performed to serve as a control for the central neural command effect since passive movement is mediated by the muscle mechanoreflexes, while the unloaded active pedaling engages both the

mechanoreceptors and central neural command. We tried to demonstrate the role of the central command in the hemodynamic responses among the subjects, by comparing HR response between the passive (involuntary) and active zero-load (voluntary) exercise tests, as supported by pieces of literature evidence [33, 36, 37]. Evidence also suggests that HR is controlled mainly by central command, hence it is a linear measure of central command during exercises [38, 39]. The unloaded active cycle leg movement, which elicited an increase in HR in both obese and non-obese women, therefore suggests that the central neural control was activated by the active cycling leg movements. After comparing the net HR increase from passive to unloaded active exercise between the two study groups, our study indicated a similar HR response ($\Delta HR_{passive-active}$) between the obese and non-obese women. This suggests an equal influence of the central neural command inputs in control of HR responses during the voluntary dynamic exercise test. The lack of significant difference in HR response may also explain the similar BP response ($\Delta BP_{passive-active}$) noted between the two groups. The present finding seems to contradict previous speculations of increased central neural command control in obese young individuals compared with non-obese individuals in a previous study [40], which methodology is quite different from ours.

The above findings lead us to speculate that both mechanoreceptors and central neural command may play limited roles in increased BP and HR drive during passive and unloaded dynamic exercises in obese women. In other words, the mechanism behind the overactive hemodynamic responses often associated with obese individuals may be mediated by other obesity-related factors beyond the scope of this study, such as elevated sympathetic drive, hyperinsulinemia, hyperleptinemia, activation of Renin-Angiotensin-Aldosterone System (RAAS), mitochondrial dysfunction, increased adipokines, dyslipidemia or arterial baroreflex dysfunction [33, 40, 41, 42]. We recommend further studies to determine the role of some of these factors in contributing to increases in BP and HR responses during the same experimental conditions.

Conclusion: The higher mean BP and HR observed in the obese women, at baseline, during passive and unloaded active cycle movements were attributed to poor cardio-respiratory fitness and greater myocardial metabolic demand imposed by excessive adipose tissue. The lack of significant differences in $\Delta BP_{baseline-passive\ exercise}$ and $\Delta HR_{passive-active}$, between the two groups, suggests that both mechanoreceptors and central neural command played limited roles in increased BP and HR drive during low-intensity dynamic exercise tests.

Limitations of Study

We could not confirm the lack of leg muscle activation during passive exercise pedaling using electromyography (EMG). This confirmation would have given the confidence that no central command signals existed during passive pedaling. We could not also investigate the influence of other mechanisms such as metaboreflexes and respiratory pumps as this would have also ruled out their influences during the experimental conditions of subjects. This is particularly important for the metaboreflex mechanism, since, in all forms of movements, whether passive or active,

metaboreceptor reflexes may be activated to some extent. However, all participants stated that they did not add any voluntary contraction to the passive movements and rated the exertion as lower than that of the zero-resistance active exercise, but somewhat higher than the resting condition. This suggests that the central command and metaboreceptors played a very trivial role during the passive exercise. Furthermore, we could not measure the resting VO_2 directly, but used a predictive equation derived from important and validated resting energy equations [18, 19]. Indirect predictive VO_2 equations have been shown to present a very interesting alternative for the assessment of cardio-respiratory fitness since they exhibit significant benefits such as low operating costs, the convenience of application, access to testing sites, and the ability to evaluate several participants at once [43]. We could not however find in the literature, any predictive VO_2 equations for passive and zero-resistant exercise tests. This study is also limited to only females, who are mildly obese and of age between 18 – 30 years, and to exercise tests of low intensity, which are influenced by non-exercise factors which are difficult to control, thus the application, interpretation, and conclusions from the present study should not be generalized.

Ethical Approval and Consent:

Ethical approval of the experimental procedures and protocols was obtained from the Ethical Committee of the Faculty of Basic Medical Sciences, College of Health Sciences, Nnamdi Azikiwe University, Anambra State, Nigeria, through a Letter of Approval (REF: NAU/CHS/NC/FBMS/104) issued by the chairperson of the committee. The informed written consent of all the participants was obtained after they were provided with a detailed explanation of the experimental procedures and the nature of the exercise.

Conflict of Interest

The authors declare that there is no conflict of interest.

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Author Contributions

- CWC: Acquisition, analysis, and interpretation of data for the work; Drafting of the work or revising it critically for important intellectual content.
- UD: Conception or design of the work; Analysis and interpretation of data for the work; Revising of the work critically for important intellectual content.
- IFO: Revising the work critically for important intellectual content.

- AJU: Revising the work critically for important intellectual content.

All Authors approved the final version of the manuscript and also agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Table 1. Demographic and baseline characteristics of the study population

Variables	Non-Obese N = 15	Obese N = 15	t-Statistics	P-Value
Age (years)	21.3 ± 1.7	20.93 ± 1.0	0.77	0.446
Height (meters)	1.64 ± 0.05	1.65 ± 0.03	-0.78	0.441
Weight (kg)	57.2 ± 4.6	93.3 ± 14.7	-9.04	<0.001
BMI (kg/m^2)	21.2 ± 1.9	33.8 ± 3.1	-13.35	<0.001
WC	28.4 ± 2.6	38.7 ± 3.8	-8.64	<0.001
HC	35.7 ± 2.5	44.6 ± 4.4	-6.80	<0.001
WHR	0.79 ± 0.06	0.87 ± 0.05	-3.19	0.003

Abbreviations: BMI = Body Mass Index; WC = Waist Circumference; HC = Height Circumference; WHR = Waist Hip Ratio.

Table 2. Hemodynamic characteristics of study participants at baseline, during passive exercise and unloaded active exercise conditions

Variables	Baseline		P Value	Passive		P Value	Unloaded Active		P Value
	Non-Obese	Obese	<0.001	Non-Obese	Obese		Non-Obese	Obese	
	N = 15	N = 15		N= 15	N = 15		N = 15	N = 15	
HR (bpm)	63.0 (20) †	83 (31) †	<0.001	69.8 ± 8.9[a]	84.4 ± 9.2	<0.001	85.3 ± 12.3 [a, b]	94.9 ± 11.9 [a, b]	0.039
SBP (mmHg)	100.8 ±11.7	124.6 ± 13.2	<0.001	104.4 ± 11.9[a]	129.3 ± 17.7	<0.001	117.0 ± 8.1 [a, b]	138.7 ± 20.7[a]	0.001
DBP (mmHg)	68.1 ± 7.1	84.5 ± 6.7	<0.001	69.0 ± 7.5	86.7 ± 8.2	<0.001	78.4 ± 8.9 [a, b]	88.9 ± 7.5[a]	0.002
MAP (mmHg)	79.0 ± 7.5	97.9 ± 8.1	<0.001	80.8 ± 7.5[a]	100.9 ± 8.4[a]	<0.001	91.3 ± 7.6 [a, b]	105.5 ± 10.2[a]	<0.001
RPP (10^3)	6.6 ± 1.2	10.0 ± 1.7	<0.001	7.3 ± 1.5[a]	10.9 ± 2.1	<0.001	10.0 ± 1.6[a]	13.2 ± 2.8[a]	0.001
VO_2 (ml/kg/min)	3.7 ± 0.2	2.9 ± 0.2	<0.001	-	-	-	-	-	-

Abbreviations: HR = Heart Rate; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; MAP = Mean Arterial Pressure; RPP = Rate Pressure Product; VO_2 = Oxygen Consumption. † Data is Median (Range); [a] $P < 0.05$, significant difference vs. baseline; [b] $P < 0.05$, significant difference vs. passive.

Table 3. Changes in heart rate and blood pressure from baseline to passive and unloaded active exercise conditions and from passive to unloaded active exercise conditions.

Variables	Baseline to Passive Exercise			Baseline to Unloaded Active Exercise			Passive to Unloaded Active Exercise		
	Non-Obese N = 15	Obese N = 15	P Value	Non-Obese N= 15	Obese N = 15	P Value	Non-Obese N = 15	Obese N = 15	P Value
ΔHR (bpm)	4.1 ± 4.4	4.2 ± 9.7	0.962	19.6 ± 15.4	14.7 ± 8.7	0.296	15.5 ± 15.5	10.5 ± 6.9	0.264
ΔSBP (mmHg)	3.6 ± 5.3	4.7 ± 11.8	0.752	16.2 ± 11.3	14.1 ± 17.8	0.698	12.6 ± 11.9	9.4 ± 18.2	0.574
ΔDBP (mmHg)	0.86 ± 2.7	2.2 ± 6.3	0.460	10.3 ± 8.3	4.3 ± 5.9	0.033	9.4 ± 7.1	2.1 ± 9.1	0.021
ΔMAP (mmHg)	1.8 ± 1.5	3.0 ± 5.5	0.408	12.2 ± 7.1	7.6 ± 8.1	0.106	10.5 ± 7.2	4.5 ± 9.1	0.059
ΔRPP (10^3)	0.68 ± 0.7	0.9 ± 1.9	0.650	3.3 ± 2.2	3.2 ± 2.3	0.872	2.7 ± 2.4	2.3 ± 1.7	0.633

Abbreviations: HR = Heart Rate; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; MAP = Mean Arterial Pressure; RPP = Rate Pressure Product.

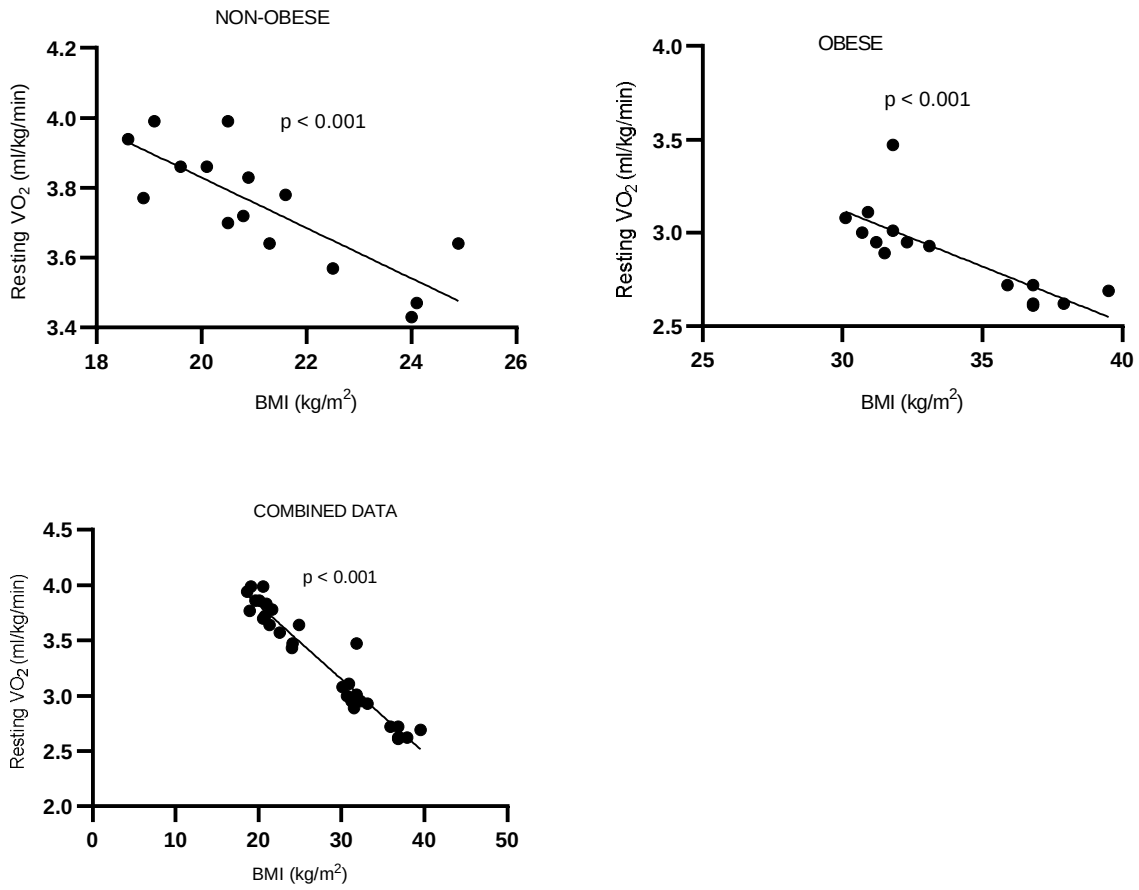

Figure 1. Scatterplot diagrams showing the correlations between resting VO_2 and BMI in non-obese, obese, and overall data.

Table 4. Correlation analyses between peak VO_2 and participants' heart rate and blood pressure parameters at rest, during passive and unloaded active exercise conditions.

Variables Peak VO_2 vs.	Resting Condition			Passive Exercise			Unloaded Active Exercise		
	R	P - Value	95% CI	R	P - Value	95% CI	R	P - Value	95% CI
Heart Rate	-0.619	<0.001	-0.800 to -0.331	-0.647	<0.001	-0.815 to -0.371	-0.377	0.040	-0.648 to -0.018
Systolic Blood Pressure	-0.766	<0.001	-0.882 to -0.559	-0.697	<0.001	-0.844 to -0.448	-0.673	<0.001	-0.831 to -0.412
Diastolic Blood Pressure	-0.769	<0.001	-0.884 to -0.564	-0.788	<0.001	-0.894 to -0.598	-0.540	0.002	-0.763 to -0.222
Mean Arterial Pressure	-0.807	<0.001	-0.904 to -0.628	-0.835	<0.001	-0.919 to -0.679	-0.678	<0.001	-0.834 to -0.420
Rate Pressure Product	-0.778	<0.001	-0.776 to -0.578	-0.748	<0.001	-0.872 to -0.527	-0.642	<0.001	-0.813 to -0.365

Abbreviations: CI, Confidence Interval; R, Correlation Coefficient