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# Obesity and parasympathetic reactivation of the heart following exercise testing in young male adults: a pilot study

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**BACKGROUND:** In elderly people, obesity may induce changes in the autonomic nervous system via alteration of the balance between the sympathetic and parasympathetic nervous systems. Little is known about obesity and parasympathetic reactivation following exercise testing, particularly in young people in Saudi Arabia, and its relationship with body composition parameters.

**OBJECTIVES:** Compare parasympathetic reactivation using heart rate recovery (HRR) following the exercise test between young obese and nonobese people and explore the association between body composition parameters with HRR.

**DESIGNS:** Cross-sectional study.

**SETTING:** University research lab.

**PATIENTS AND METHODS:** Twenty-seven young male college students underwent anthropometric measurements and treadmill exercise testing, during which the heart rate was monitored via 12-lead electrocardiography. Participants were divided into a group (n=15) with high body fat percentage (>30%), and a group (n=12) with a normal body fat percentage (<30%) to compare multiple parameters including HRR, which was defined as the absolute change from heart rate (HR) peak during exercise to 1-minute post-HR peak.

**MAIN OUTCOME MEASURES:** HRR, body mass index, waist-to-hip ratio, fat percentage, and trunk fat.

**SAMPLE SIZE AND CHARACTERISTICS:** n=27, mean (SD) age=22.4 (0.98) years, range 21–25 years.

**RESULTS:** There was no significant difference in HRR between the groups (32.20 [13.42] bpm for high body fat percentage vs 35.42 [13.35] bpm for normal body fat percentage) (*P*=.54). We found a non-significant inverse correlations of HRR with BMI (r=-0.18, *P*=.37), WHR (r=-0.04, *P*=.86), fat percentage (r=-0.18, *P*=.38) and trunk fat (r=-0.23, *P*=.25).

**CONCLUSION:** HRR was preserved in our young obese people and was not different from nonobese people. Furthermore, it seems that obese people with higher body composition parameters may have slower HRR, or slower recovery indicating poorer parasympathetic reactivation.

**LIMITATIONS:** Need a larger sample to confirm the findings of this pilot study.

**CONFLICT OF INTEREST:** None.

Obesity is a major epidemic challenge for global public health. The prevalence of obesity has been consistently increasing in recent decades.[1] Thirty-four percent of US adults aged 20 and over are obese and almost 75% are overweight.[2] These numbers are relatively similar in Saudi Arabia, with an overall prevalence of 35.5% for obesity and 70-75% for overweight.[3,4] This is alarming considering the complications and comorbidities associated with overall excessive fatty weight gain.

There is ample evidence that obesity increases the risk for cardiovascular diseases (CVD)[5,6] via multiple mechanisms that eventually lead to CVD including dyslipidemia, hypertension, diabetes, atherosclerosis.[7] These mechanisms are known to have adverse effects on the cardiovascular system. Development of obesity in early life can lead to premature death.[2]

Obesity also may induce an alteration in the balance between the activity of the two branches of the autonomic nervous system (ANS): the sympathetic and parasympathetic, which may result in alteration in the hemostasis of blood and the metabolic rate leading to increased risk of CVD.[8] Obesity alters the functioning of the ANS hemodynamically and metabolically, which increases the risk of CVD by increasing the risk of developing metabolic diseases such as hypertension, insulin resistance, and dyslipidemia. Indeed, autonomic imbalance has been found among obese and overweight people in both the elderly[9] and in adolescents[10] when compared to non-obese people. Furthermore, using multiple noninvasive tests for ANS function in middle-aged people, it was found that obese people have impaired parasympathetic function when compared to nonobese people.[11] In a recent study using heart rate variability (HRV) as a measure for ANS function in young adults, obese people had higher HR values at rest (indicating sympathetic dominance) and reduced parasympathetic activity when compared to nonobese.

Heart rate recovery (HRR) following exercise testing is one of many methods to assess autonomic imbalance. A slower HRR is an indication of reduced activity of the parasympathetic nervous system.[12] The use of HRR has been widely used due to its simplicity in application and a reduced need for sophisticated software and interpretation. Furthermore, several studies have found that obesity, as measured by body mass index (BMI), has adverse effects on HRR.[13-15] However, there has been minimal research in this area in Saudi Arabia.

To the best of knowledge, only one study was conducted in Saudi Arabia that compared ANS function between obese and nonobese people. The study used HRV as well as HRR postexercise to compare ANS functioning among 55 young female students; the obese group had autonomic imbalance in comparison to nonobese.[16] Studies comparing ANS functioning between obese and nonobese people among young adults are limited and little is known about the relationship between excessive fat and ANS functioning especially with parasympathetic reactivation following exercise. Therefore, the study aimed to compare parasympathetic reactivation using HRR following exercise testing, between obese people with excessive fat and nonobese young adults with less fat, and investigate the relationship between multiple body composition parameters with HRR. It was hypothesized that obese people would have poorer parasympathetic reactivation than nonobese people.

## PATIENTS AND METHODS

### *Study population*

Twenty-seven college male students from Prince Sattam bin Abdulaziz University, Alkharj, Saudi Arabia, volunteered to participate in the study. They were invited by word of mouth and invitation posters. Participants were divided into two groups based on the classification of body fat percentage.[17] One group of participants (n=15) with high body fat percentage >30%, and another group participants (n=12) with body fat percentage <30% were included in the study. Inclusion criteria were apparently healthy young adults aged from 18 to 30 years old. Exclusion criteria were comorbidities incompatible with exercise as per American College of Sports Medicine (ACSM) guidelines.[18] In addition, participants with a history of medical (acute medical illness that limits physical exertion), surgical (recent surgeries in the upper or lower extremities or surgery where physical exertion is contraindicated), mental problems (illness that may affect their understanding of commands during exercise) affecting their exercise performance were excluded. The study was approved by the ethical committee at Prince Sattam bin Abdulaziz University and all participants were given informed consent prior to participation.

### *Study protocol*

Participants were invited to the exercise research lab at the College of Applied Medical Sciences for one visit. During the visit, participants completed the information sheet prior to testing. Height was measured using a stadiometer to the nearest 0.5 cm and weight was measured using a weight analyzer (DETECTO, USA). Body composition was analyzed using bioelectrical impedance analysis (Biacorpus RX 4000; Medical HealthCare Gmbh, Karlsruhe, Germany) while participants were ly-

ing in a supine position. Then, waist and hip ratio were measured using a non-stretchable tape at the midpoint between the last palpable rib and top of the iliac crest (approximately at the umbilical line below the 12th rib) for the waist circumference, and at the widest portion of the buttocks for the hip circumference.[19] Brachial blood pressure was measured while seated and resting using an electronic sphygmomanometer (Wollex Blood Pressure Monitor (ARM)/WXT-5902, Cigli Izmir, Turkey). Subsequently, 12-lead electrocardiogram (ECG) electrodes were placed on the bare chest to monitor heart rate activity before, during and after the exercise test.

### *Exercise tolerance test protocol*

All participants performed an exercise tolerance test (ETT) on the treadmill (HP Cosmos Mercury, Nussdoerf-Traunstien, Germany). An individualized ramp test was used and modified according to the physical abilities of each participant.[20] The starting speed was set to serve as a warming and familiarization stage (approximately 3.5–4 km/h) without inclination. Then, the speed was gradually increased to reach a maximum brisk walking. The first two minutes of the ETT served as familiarization and warming up phase for participants. Then, on the third minute after maximum brisk walking reached based on participant's ability, the test started with 1% of inclination. Thereafter, the speed was constant and inclination was progressively increased by 1% every minute. During the test, heart rate was recorded using ECG up to recovery period after termination of the ETT. The test was terminated if the participant reached volitional exhaustion and could no longer complete the test, or if any of the relative or absolute contraindications of the ACSM criteria were met.[18] Upon termination of the test, heart rate and blood pressure were monitored continuously for six minutes or until vital signs regained normal ranges while participants were seated comfortably on a chair next to the treadmill.

### *Outcome variables*

Heart rate recovery (HRR) was measured as the absolute difference between peak heart rate achieved during ETT, in which the test was terminated, and the heart rate after 60 seconds following peak heart rate. HRR was defined as (HRR=Peak heart rate–heart rate 60 seconds post-peak heart rate). Body mass index (BMI) was measured using the weight in kg divided by the square of the height in meters.

Waist-to-hip ratio (WHR) was measured by dividing the measured waist circumference in centimeters using the standard method by the measured hip circumference in centimeters. The measurement was done while the participants were standing. Fat mass and body fat percentage were measured using a single-frequency bioelectrical impedance analysis device (Biacorpus RX 4000; Medical HealthCare Gmbh, Karlsruhe, Germany) while in the supine position with pronated and slightly abducted legs. After application of alcohol swabs on the dorsum of the hands and feet, eight electrodes were attached to the participant, two on each extremity over the third metacarpal bone of the hands and feet. A minimum distance of 5 cm was kept between the signaling and measuring electrode on each extremity. Before measurement, the participant was free from any metallic or electrical conducting item. Once measurement was obtained, the results were transferred to interpretation software provided by the manufacturer (BodyComp V 8.3; Medical HealthCare GmbH, Karlsruhe, Germany).

### *Statistical analysis*

Statistical analysis was performed using IBM SPSS (Armonk, NY: IBM Corp) version 20. Normality of the variables was tested using the Kolmogorov-Smirnov test. All variables were normally distributed and presented as mean and standard deviation. To compare parasympathetic activation between participants with excessive fat percentage (≥30%) versus those with less fat percentage (<30%), the unpaired t test was used for parametric variables (all the variables), and the Mann-Whitney U test was used for non-parametric variables (smoking distribution between groups). To investigate the relationship between HRR and other body fat measures, the bivariate correlation was used with Pearson product-moment correlation analysis. The level of significance for all analysis was set at .05.

## RESULTS

The mean (SD) age of the entire sample (n=27) was 22.4 (1.1) (range 21–25 years) (**Table 1**). About twice the percentage of the normal fat percentage group were smokers compared to the excessive fat percentage group. Smoking was not an exclusion criterion, as it was difficult to find a sample free from smoking for either group. However, both groups were comparable in the distribution of smokers (*P*=.347). There were no statistically significant differences between the groups in measures of blood pressure (BP) including presystolic BP (*P*=.65) and prediastolic BP (*P*=.235) either before or after exercise testing (**Table 2**). Likewise, there were no detectable between-group differences in the HR variables, including resting HR (*P*=.261), maximum HR (*P*=.229) and HRR (*P*=.541). For the combined data from the two fat groups, there were negative correlations of HRR with BMI (*P*=.371), with waist-to-hip ratio (*P*=.856), with to-

**Table 1.** Demographic characteristics and anthropometric measurements of the participants.

| Variable | Excessive fat (n=15) | Normal fat (n=12) | *P* value |
|---|---|---|---|
| Age, years | 22.5 (1.3) | 22.4 (0.67) | .81 |
| Weight, kg | 109.8 (20.5) | 70.8 (10.6) | **<.001** |
| Height, m | 1.8 (0.18) | 1.7 (0.17) | **.15** |
| Waist circumference, cm | 80.86 (8.07) | 109.18 (15.12) | **<.001** |
| Hip circumference, cm | 95.59 (5.38) | 116.86 (11.76) | **<.001** |
| Body mass index, kg/m$^2$ | 35.5 (7.1) | 24.2 (2.9) | **<.001** |
| Waist-to-hip ratio | 0.93 (0.6) | 0.85 (0.6) | **.003** |
| Overall fat mass, kg | 42.6 (13.7) | 16.5 (5.3) | **<.001** |
| Fat percentage, % | 38.2 (5.9) | 22.9 (4.9) | **<.001** |
| Fat free mass, kg | 67.3 (10.2) | 54.1 (6.1) | **.001** |
| Body cell mass, kg | 37.5 (7.6) | 31.9 (7.3) | .06 |
| Total body water, % | 48.5 (7.7) | 36.9 (7.3) | **.001** |
| Fat mass, trunk area, kg | 27.7 (7.1) | 11.0 (3.3) | **<.001** |
| Smokers, n (%) | 3 (20.0) | 5 (41.7) | .347 |

Data are number (%) or mean (SD).

**Table 2.** Cardiac parameters before and after exercise testing.

| Variable | Excessive fat (n=15) | Normal fat (n=12) | *P* value |
|---|---|---|---|
| Presystolic BP, mm Hg | 129.9 (8.4) | 127.9 (13.4) | .650 |
| Prediastolic BP, mm Hg | 88.3 (7.5) | 84.5 (8.9) | .235 |
| Resting HR, beat/min | 75.7 (10.4) | 71.0 (10.6) | .261 |
| Maximum HR, beat/min | 172.3 (11.1) | 166.6 (12.4) | .229 |
| HR recovery | 32.2 (13.4) | 35.4 (13.4) | .541 |
| Postsystolic BP, mm Hg | 172.5 (22.7) | 167.4 (24.8) | .581 |
| Postdiastolic BP, mm Hg | 96.4 (24.6) | 92.2 (6.9) | .569 |

Data are expressed as mean ± standard deviation (SD), BP: blood pressure, HR: heart rate.

tal fat percentage (*P*=.384), and with trunk fat (*P*=.252) (**Figure 1**). However, these correlations were not statistically significant.

## DISCUSSION

Our study investigated the impact of several parameters for excessive fatty weight gain on parasympathetic reactivation of the heart between people with excessive fat and people with normal fat. The study showed no difference in parasympathetic reactivation using HRR following ETT. In addition, the study showed no significant association between excessive fat gain parameters including BMI, WHR, fat percentage, and trunk fat with HRR.

HRR reflects reactivation of the parasympathetic nervous system and withdrawal of the sympathetic nervous system after dominance of the latter during physical exertion.[21] HRR is also known to be a predictor of mortality[22] as well as a marker of the risk of CVD.[23] An HRR of 12 bpm or slower has been established as a cut-off point for the increased risk of CVD and mortality.[22] Due to its simplicity as a noninvasive tool for vagal tone, HRR has been widely used as a measure of parasympathetic reactivation following exercise. In the current study, none of our participants reached this cut-off point of slow HRR, possibly due to the age group of our participants, as the cut-off point of 12 bpm has been es-

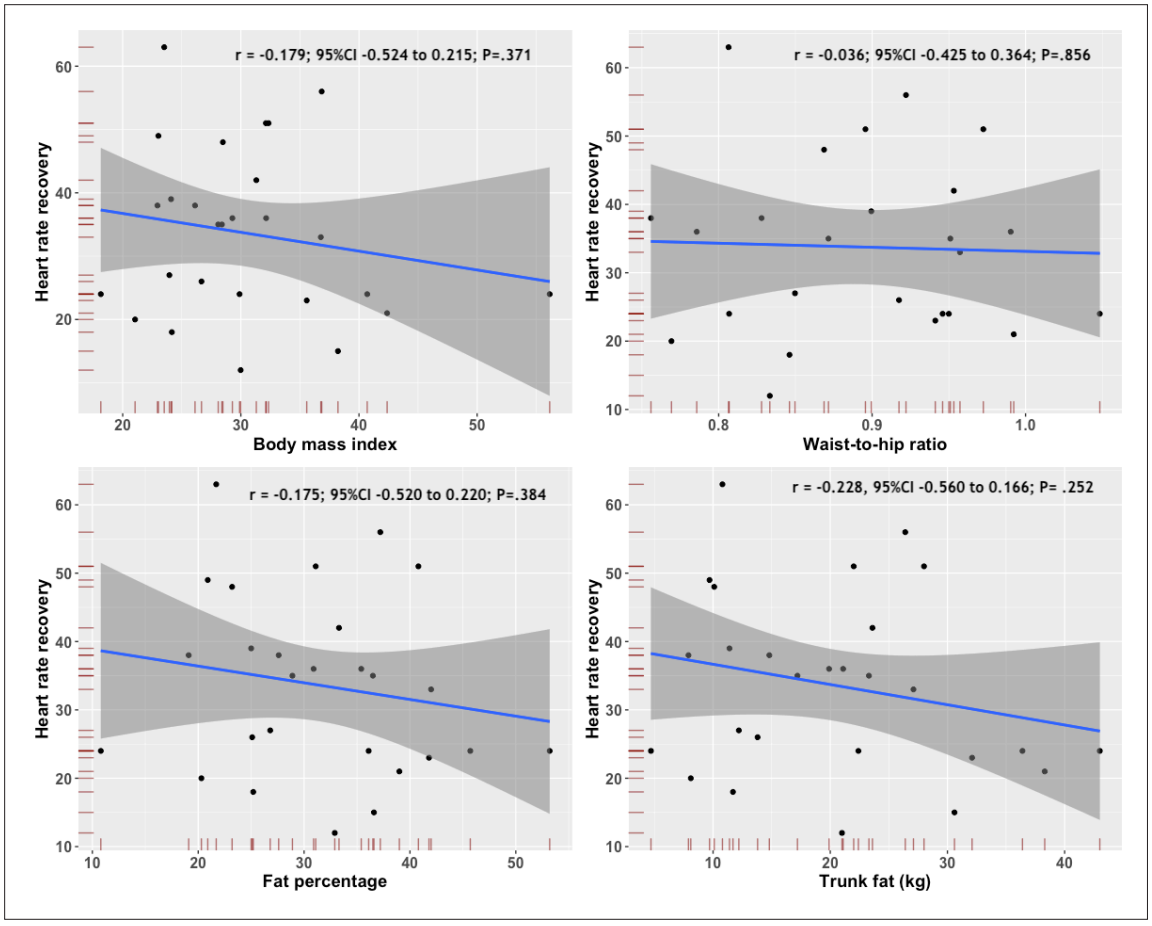


**Figure 1.** Correlation of heart rate recovery with body mass index, waist-to-hip ratio, total body fat percentage and trunk fat.

tablished for apparently healthy but older individuals.[22]

In a recent study, HRR was reduced among elderly people with a high body fat percentage (n=45) when compared with low body fat percentage (n=45).[9] The same findings were reported in a study comparing HRV and HRR among young obese (n=28) and nonobese people (n=27).[16] Interestingly, although both studies looked into different age categories and used different obesity parameters (the earlier used fat percentage, the later used BMI), both study participants were female. In the current study, we recruited males only due to cultural reasons. Therefore, we cannot relate or compare the current study's findings to the aforementioned ones. No significant difference in HRR between the groups was found in the current study. However, it is worth noting that HRR in the normal fat group had a slightly but not significantly better HRR than the excessive fat group. Although our hypothesis was not confirmed statistically by the findings of this study so far, the difference between the two groups may indicate a trend toward a bigger difference that would become apparent in a larger sample size. As mentioned earlier, the participants were young healthy adults; therefore, we may speculate that the reason for not finding significant differences between the two groups might be that the sample size was too small to detect a statistically significant difference, as in other studies, or the age group of participants was too young to show clear manifestations of the adverse effect of excessive fat on the reactivation of the parasympathetic nervous system.

The grouping in this study was based on fat percentage. There is no clear consensus about the cut-off points used for fat percentage. However, a range between 25% to 30% was for males was suggested by the World Health Organization.[24] These ranges showed some variation based on geographic region and ethnicity.[25] We grouped our sample based on the maximum threshold, which is above 30% to separate the extreme from those who might have a bit extra fat (overweight), but do not fall in the category of high excessive fat. It is

worth noting that the use of body fat ≥40% as high excessive fat have been used before using the dual energy X-ray absorptiometry scan.[9]

Several studies reported a negative association between obesity/body fat parameters and HRR. In a study of Malaysian male and female adolescents (n=1071), HRR was inversely associated with multiple body composition parameters such as BMI, WHR, and body fat.[26] Slower HRR was reported to be associated with higher BMI in males and females of different age categories (n=2443) in another study.[14] In the current study, despite that statistical significance was not reached, the results show similar trends toward an inverse association between HRR and body fat parameters including BMI, WHR, body fat percentage, and trunk fat, meaning that the higher body fat the poorer or slower HRR. The mechanism behind this inverse association between HRR and body fat parameters can be explained by an alteration in the function of the ANS caused by obesity, which includes an increase the activity of the renin-angiotensin complex among obese people.[27] It has also been suggested that obesity may have a negative effect on baroreflex sensitivity (which reduces the responsiveness to shift from sympathetic dominance to parasympathetic takeover) via insulin-mediated routes[28] and disturbed adipocyte production from visceral fat,[29] which may cause sympathetic dominance over parasympathetic. These mechanisms may also explain why obese people may exhibit a higher basal heart rate at rest when compared to nonobese people. Indeed, our sample showed that excessive fat participants had a higher resting heart rate (**Table 2**).

It is noteworthy that smoking is widely increased among young male students locally. Smoking is a major public concern that is related to an increased risk of CVD.[30] Both obesity and smoking have been identified as a common risk factor for CVD worldwide. They are also positively associated with one another.[31] Strategies to quit smoking such as aerobic exercise are effective not only in withdrawal from smoking but also in reducing other CVD risk factors.[32,33]

To our knowledge, this is one of few studies that has used bioelectrical impedance as a measure for body fat percentage as well as trunk fat in Saudi Arabia, as the majority of studies rely on BMI. BMI is the most commonly used anthropometric measure for obesity.[14,26] BMI is well known for either overestimation/underestimation of body fat in different people,[34] mainly because it does not differentiate between fatty mass and nonfatty mass. Bioelectrical impedance overcomes this problem by distinguishing the fat mass from the fat-free mass.

Strategies should be implemented to reduce obesity and its associated complications. One of the reported associated problems with obesity is depression.[35] Outdoor physical activity with Vitamin D supplementation are effective in the improvement of depression symptoms among obese people.[35] Another study investigated the effect of aquatic exercise on low back pain, where significant improvement was also found in body composition parameters.[36] These studies are suggestive of different activities that can help obese people to reduce obesity and its associated problems. Thus, exercise programs and different types of physical activities should be encouraged to reduce the burden of obesity.

There are a number of limitations learned from this pilot study. The data showed a possible trend toward confirming our hypothesis despite the limited sample size. This was further investigated via power calculation analysis (GPower version 3.1). Using a post hoc test based on the mean and standard deviation of HRR in the current sample, the effect size was d=.24 and achieved power (1-$\alpha$ err probability)=0.15. For a Pearson correlation between HRR with BMI, total fat percentage and trunk fat, the effect size of current sample=0.45 and achieved power (1-$\alpha$ err prob)=0.67. This small sample size may have increased the possibility of a type 2 statistical error, which led to not finding significant difference in HRR between the two groups. This may explain the contradictory results where no significant difference was noted between the participants with excessive fat and those with normal fat, but there was an inverse association between body fat parameters and HRR in the correlation analysis. Using a power calculation to achieve power (1-$\alpha$ err probability)=0.81 the required sample size for each group is 51.

Another limitation is that the study was gender-biased due to cultural reasons that make it difficult for a male to recruit a female. However, this can be modified by including a female in the research team to be responsible for recruiting female participants. We could not include smoking as an exclusion criterion, although smoking can have a negative impact on HRR.37 However, in this pilot study, there was no difference in smoking rate between the two groups.

In conclusion, obese people with excessive fatty weight gain had a slower but not statistically significantly parasympathetic reactivation than nonobese people. There was a trend towards an inverse association between HRR and body composition parameters including BMI, WHR, fat percentage, and trunk fat but no statistically significant association. These findings need to be confirmed in a larger and more representative sample size, and further research needs to include female young people as well.

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