ORIGINAL RESEARCH

Impact of Moderate Exercise Training on Heart Rate Variability in Obese Adults

Breanna L. Davidson, MS[1], Kristen A. Byrne, MS[2], Brittany L. Rood, MS[1], Elizabeth S. Edwards, PhD[1], Jeremy D. Akers, PhD, RD[2], David L. Wenos, PED[1], Trent A. Hargens, PhD, FACSM, ACSM-CEP, CEPA-RCEP[1]

ABSTRACT

Background: Exercise has been shown to improve heart rate variability (HRV) at rest in healthy subjects. HRV response during and immediately after acute exercise in obese individuals following aerobic exercise training has not been evaluated. The purpose of this study was to examine the effect of 12 weeks of moderate intensity aerobic exercise training on HRV during acute exercise and active postexercise recovery in obese individuals.

Methods: Eleven obese individuals (5 men, 6 women; body mass index = 39.2 ± 6.3 kg·m^{-2}] underwent 12 weeks of exercise training at 60% of predicted VO_{2max}, determined via a submaximal treadmill test. Body composition was assessed with dual-energy x-ray absorptiometry. HRV was measured during the final minute in each exercise stage and in recovery and analyzed with Kubios HRV software.

Results: Predicted VO_{2max} (baseline: 28.2 ± 3.5 mL·kg^{-1}·min^{-1} and posttraining: 27.4 mL·kg^{-1}·min^{-1}, $P > 0.05$) was unchanged and body fat % decreased (46.2% ± 2.2 vs 45.5% ± 7.2, respectively). Initial stage heart rate and postexercise recovery heart rate was lower after training. The high frequency component was greater during the initial exercise stage after training. The low frequency component and the standard deviation of instantaneous beat-to-beat variability were greater during the final exercise stage after training. During cooldown, the root-mean-square of differences between adjacent RR intervals and high frequency normalized units were greater after training.

Conclusion: HRV markers were improved during acute exercise stage and active recovery in obese individuals following 12 weeks of moderate intensity exercise training. These results suggest improvements in autonomic function can be seen with reductions in adiposity, independent of cardiorespiratory fitness changes in obese adults. *Journal of Clinical Exercise Physiology*. 2021;10(1):12–19.

Keywords: HRV, obesity, autonomic function, exercise intervention

INTRODUCTION

As of 2020, an estimated 38% of American adults are classified as obese (1). Obesity is a well-known independent risk factor for the development of metabolic disease, obstructive sleep apnea, dyslipidemia, cardiovascular disease (CVD), among many other metabolic disorders (1). The physiological mechanisms linking obesity to chronic disease are complex and multifaceted. One such mechanism is obesity-induced hypertension (2). It is estimated that 65% to 75% of essential hypertension is due to obesity (2,3).

One potential pathway in which obesity leads to hypertension, and ultimately to the development of CVD, is through autonomic nervous system (ANS) dysregulation (3,4). Evidence suggests that excess adiposity increases

[1]James Madison University, Human Performance Laboratory, Department of Kinesiology, Harrisonburg, VA 22807 USA
[2]James Madison University, Department of Health Professions, Harrisonburg, VA 22807 USA

Address for correspondence: Trent A. Hargens, Ph.D., FACSM, Department of Kinesiology, 261 Bluestone Drive MSC 2302, James Madison University, Harrisonburg, VA 22807; (540) 568 5844 (voice), (540) 810 1310 (mobile), (540) 568 3338 (fax); e-mail: hargenta@jmu.edu.

Conflicts of Interest and Source of Funding: Sentara Rockingham Memorial Hospital Foundation Community Grant Program and James Madison University, College of Health and Behavioral Studies Collaborative Grant. No conflicts of interest.

sympathetic nervous system (SNS) activation to several areas including the kidneys, muscles, and vascular endothelium (4–6). Heart rate variability (HRV) is the beat-to-beat variation in time between consecutive heartbeats and is a noninvasive method to assess ANS function (7). A decrease in HRV indicates that there is either a decrease in parasympathetic nervous system activation or an increase in SNS activation and is associated with an increased risk of CVD, acute coronary events such as myocardial infarction and sudden cardiac death, and all-cause mortality (8,9). Obese individuals have decreased HRV at rest because of an increase in SNS activity (10–12).

Exercise improves HRV during rest in healthy subjects by increasing parasympathetic nervous system activity (13–17). Additionally, improvements in HRV are measured at rest in obese individuals following moderate-to-vigorous intensity exercise training (18,19). What has not been examined is the HRV response during and immediately after acute exercise in obese individuals following aerobic exercise training. The use of graded exercise testing and the acute exercise response is an established tool for the diagnosis and prognosis of CVD (3,13,17–24). The prognostic value of heart rate drop at 1 and 2 minutes after acute exercise is established (22,24), but the HRV response during and immediately after exercise is not. Studies examining postexercise HRV after aerobic exercise training in nonobese individuals have equivocal results (13,25–27). Therefore, the purpose of this study was to examine the effect of 12 weeks of moderate intensity aerobic exercise training on autonomic function during acute exercise and immediately postexercise, as measured by HRV in obese individuals. We hypothesized that HRV measures would be increased following exercise training during acute exercise and active recovery, particularly those related to parasympathetic function, as regular physical activity increases vagal influence on the heart rate (28).

METHODS

Experimental Procedures

Obese individuals were recruited to undergo 12 weeks of aerobic exercise training, 3 days per week, at a moderate intensity. Prior to participation, all subjects provided their written informed consent. All study procedures were approved by the Institutional Review Board of James Madison University. Prior to the exercise intervention, subjects underwent body composition analysis, a fasting blood draw, and a cardiorespiratory fitness test. Upon completion of the exercise intervention, subjects repeated the same assessments.

Subjects

Sixteen obese individuals volunteered to participate in the study. Inclusion criteria was a body mass index (BMI) ≥ 30 $kg \cdot m^{-2}$. Exclusion criteria included any orthopedic limitation precluding participation in regular exercise, women who were pregnant, or any health condition that did not clear them for exercise according to the American College of Sports Medicine (ACSM) (20).

Body Composition

Body weight and height were measured on a Med Vue Detecto Medical Weight Analyzer (Detecto, Webb City, Missouri) with minimal clothing and no shoes and were recorded to the nearest 0.1 kg and 0.1 cm. Waist circumference was measured with a cloth tape measure with a spring-loaded handle, following ACSM guidelines. Percent body fat, fat-free mass, and fat mass were determined via total body dual-energy x-ray absorptiometry (Norland at Swiss-ray Illuminatus DXA, Fort Atkinson, Wisconsin).

Blood Analysis

A blood sample was obtained following an 8 to 10 hour fast via venipuncture and analyzed for total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, and glucose via the Chemwell 2910 (Awareness Technology, Inc., Palm City, Florida) using manufacturer's standards of analysis.

Cardiorespiratory Fitness Assessment

Subjects underwent a submaximal graded exercise test on a treadmill (Tuff Tread HS Elite, Tuff Tread, Conroe, Texas) using a modified Balke treadmill test protocol (20). All subjects completed the same initial exercise stage during the submaximal graded exercise treadmill test (2.0 mph and 0% grade). Each subsequent stage was personalized according to individual ability. Gas exchange measurements were obtained continuously via the Parvo Trueone 2400 Metabolic Measurement System (ProvoMedics, Sandy, Utah), which uses a high efficiency mixing chamber. Resting heart rate (HR) was obtained prior to exercise, and exercise HR was continuously monitored via Polar RS800CX watch and Polar H10 HR strap (Polar Electro Inc., Bethpage, New York). Blood pressure and ratings of perceived exertion were obtained every 2 minutes. The test was terminated when the subject achieved 85% of age-predicted HR max or experienced any adverse signs or symptoms, following ACSM guidelines (20). All subjects completed an active cooldown at 2.0 mph and 0% grade.

To calculate the predicted VO_{2max} for each test, the slope of the HR and VO_2 relationship was determined by using the VO_2 values obtained during the final 30 seconds of each exercise stage, and the HR values obtained at the end each stage. Two stages in which the HR values were between 110 and 150 $b \cdot m^{-1}$ were used. The slope was calculated with the following equation: slope = $([VO_2 2 - VO_2 1] / [HR2 - HR1])$, where $VO_2 1$ is the submaximal VO_2 from stage 1, $VO_2 2$ is the submaximal VO_2 from stage 2, HR1 is the HR from stage 1, and HR2 is the HR from stage 2. Predicted VO_{2max} was then calculated from the following equation: VO_{2max} $(mL \cdot kg^{-1} \cdot min^{-1})$ = slope (Predicted HR_{max} − HR2) + $VO_2 2$. This procedure was conducted following ACSM recommendations (29).

HRV Assessment

For HRV assessment, RR interval (RRi) data was obtained with the Polar RS800CX (Polar Electro Oy, Kempele,

Finland), which has previously been validated relative to electrocardiography-obtained RRi data (21,30–32). The final 1 minute of RRi data during the initial 2-minute exercise stage, when all subjects were at the same speed and grade on the treadmill, the final 1 minute of the final exercise stage before recovery, when all subjects were at 85% of their age-predicted maximal HR, and the final 1 minute of active recovery, when all subjects were again at the same speed and grade on the treadmill, were used for HRV analysis (30,33). The RRi data was uploaded using Polar Pro Trainer 5® software. HRV analysis was performed using Kubios Standard software (version 3.1.0.1, Biosignal Analysis and Medical Imaging Group, Kuopio, Finland) (34).

Time and frequency domains were used for HRV analysis. Time domain measures were HR (beats·min^{-1}), the standard deviation of the normal-to-normal intervals (between adjacent heartbeats) time series (SDNN [ms]), and the square root of the mean squared differences of successive normal-to-normal intervals (RMSSD [ms]). The frequency domain measures, using the fast Fourier Transformation, were low frequency (LF [ms^2], 0.04 – 0.15 Hz), high frequency (HF [ms^2], 0.15 – 0.40 Hz) and low frequency-to-high frequency ratio (LF/HF). Normalized units of LF and HF were also calculated, as well as log transforming LF and HF measures for analysis because of nonnormal distribution.

The time domain measures of SDNN and RMSSD assess overall autonomic function, as they cannot distinguish between changes in HRV due to sympathetic or vagal tone (35). For the frequency domain variables, traditionally LF is considered a marker of sympathetic modulation, while HF represents parasympathetic control (7). In more recent years, however, it is suggested that LF is due to both sympathetic and parasympathetic control (33,35). And the LF/HF ratio is considered a measure of sympathovagal balance (7,35).

Exercise Intervention

All subjects performed walking on a treadmill for the duration of the 12-week intervention. All subjects completed a 5-minute warm-up, a 20-minute exercise session, and a 5-minute cooldown, 3 times per week. For the initial 2 weeks of training, the exercise intensity was set at 50% of HR reserve. Weeks 3 to 6, the training intensity was increased to 60% of HR reserve, and for weeks 7 to 12, the training intensity was 65% of HR reserve. Predicted maximal HR and measured HR were used to calculate training intensities. During each exercise session, HR was monitored continuously via Polar watch and chest strap to ensure that subjects were adhering to the prescribed training intensity. All exercise sessions were monitored by study staff. The goal for exercise adherence was to attend at least 85% of all exercise sessions.

Statistical Analysis

All analyses were performed using SPSS (IBM SPSS version 25.0, IBM Corporation, Armonk, New York). The distribution normality of all variables was assessed using a Shapiro-Wilk test. Those variables that deviated from normality were log transformed for statistical analysis. Data are presented as mean ± SD. Paired sample *t* tests were used to compare cardiorespiratory fitness, body composition, blood lipids, and HRV variables between pretraining and posttraining. HR, VO_2, and HRV data from the initial exercise stage of the submaximal treadmill test was used as the early exercise stage, as all subjects performed the identical workload. HR, VO_2, and HRV data from the last stage of exercise prior to test termination were used as the late exercise stage, as all subjects were at/near 85% of age-predicted maximal HR. Pearson correlation coefficients were calculated for body composition and HRV variables. Statistical significance for all analyses was set at an alpha of 0.05.

RESULTS

Of the 16 subjects who volunteered for the study and began training, 5 subjects dropped out. One subject dropped out because of an injury that occurred separate from the exercise intervention, 3 dropped out because of scheduling conflicts, and 1 dropped out because of contracting mumps. There was no significant difference between the subjects who dropped out compared to those who did not for age, weight, BMI, or waist circumference. Only 1 subject who dropped out underwent baseline predicted VO_{2max} assessment, so a comparison could not be made on this variable. Mean adherence for the 11 subjects who completed exercise training was 92%, with a range of 86% to 100%. Figure 1 shows the mean exercise intensity achieved each week of exercise training as a function of HR reserve.

Body composition and blood lipid data and cardiorespiratory fitness data from before and after aerobic training are presented in Table 1. There was a small but significant decrease in percent body fat after aerobic training. Bodyweight, BMI, lean body mass, fat mass, blood lipids, and glucose did not change following training. Additionally, predicted VO_{2max} did not change with aerobic training when assessed relative to total body weight or to lean body mass.

From before exercise training to after training, initial exercise stage VO_2 values (10.6 ± 2.5 vs 10.1 ± 1.1 mL·kg^{-1}·min^{-1}, $P = 0.56$) and final exercise stage VO_2 values (20.1 ± 2.9 vs 20.2 ± 3.3 mL·kg^{-1}·min^{-1}, $P = 0.96$) did not differ. Resting, exercise, and cooldown HR data is presented in Figure 2. Following exercise training, there was a trend ($P = 0.07$) for resting HR to be lower (Figure 2A). Initial exercise stage HR was lower following exercise training ($P = 0.002$, Figure 2B), as was the percent of HR max with the initial exercise stage (65.0% ± 7.9 vs 59.6% ± 7.3, $P = 0.002$). The final exercise stage HR did not differ ($P = 0.26$, Figure 2C), nor did percent of HR max (84.6% ± 2.5 vs 82.6 ± 6.1, $P = 0.32$). The 2-minute, active cooldown HR was lower following exercise training ($P = 0.02$) (Figure 2D), as was the percent of HR max during cooldown (69.8% ± 5.1 vs 64.9% ± 6.7, $P < 0.01$). The difference between peak HR achieved and the 2-minute recovery HR was not significantly different after training. (24.7 ± 11.6 vs 27.7 ± 7.4; $P = 0.2$).

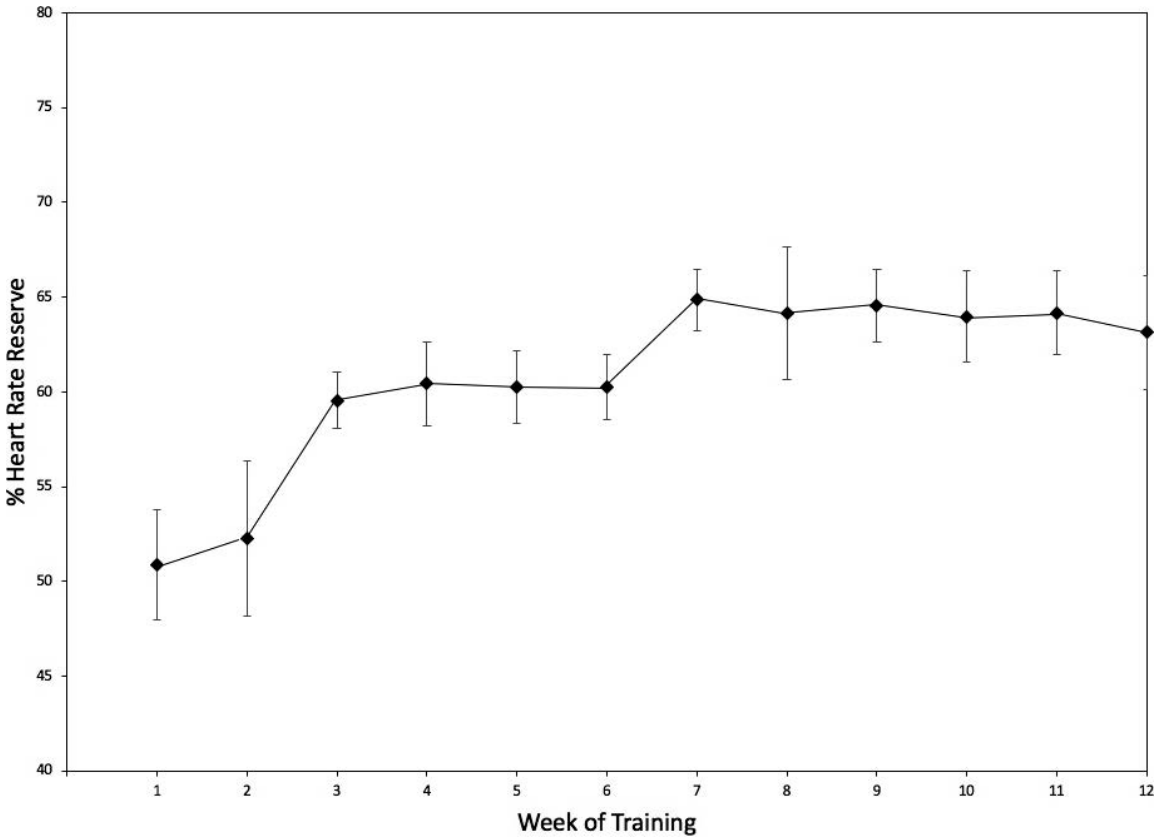

FIGURE 1. Mean exercise intensity achieved during each week of exercise training, as a function of heart rate reserve.

TABLE 1. Subject characteristics ($N = 11$) before and after aerobic training.

	Before	After
Age (yr)	41.1 ± 9.8	—
Height (cm)	172.3 ± 11.3	—
Weight (kg)	113.8 ± 26.1	113.6 ± 25.1
Body mass index ($kg \cdot m^2$)	39.3 ± 6.3	39.2 ± 5.9
% fat	46.2 ± 2.2	45.5 ± 7.2*
Fat mass (kg)	55.3 ± 17.0	54.3 ± 16.5
Lean body mass (kg)	63.4 ± 15.1	64.1 ± 14.2
Total cholesterol ($mg \cdot dL^{-1}$)	212.5 ± 32.5	207.5 ± 30.2
LDL cholesterol ($mg \cdot dL^{-1}$)	134.4 ± 35.6	131.6 ± 34.6
HDL cholesterol ($mg \cdot dL^{-1}$)	47.5 ± 12.0	46.0 ± 9.8
Triglycerides ($mg \cdot dL^{-1}$)	153.4 ± 92.6	149.7 ± 97.7
Glucose ($mmol \cdot L^{-1}$)	5.6 ± 1.0	5.8 ± 1.4
Predicted VO_{2max} body mass ($mL \cdot kg^{-1} \cdot min^{-1}$)	28.2 ± 3.5	27.4 ± 4.5
Predicted VO_{2max} lean body mass ($mL \cdot kg^{-1} \cdot min^{-1}$)	49.8 ± 7.5	49.3 ± 7.4

HDL = high-density lipoprotein; LDL = low-density lipoprotein. Data are given as mean ± standard deviation
*$P = 0.02$ compared to before

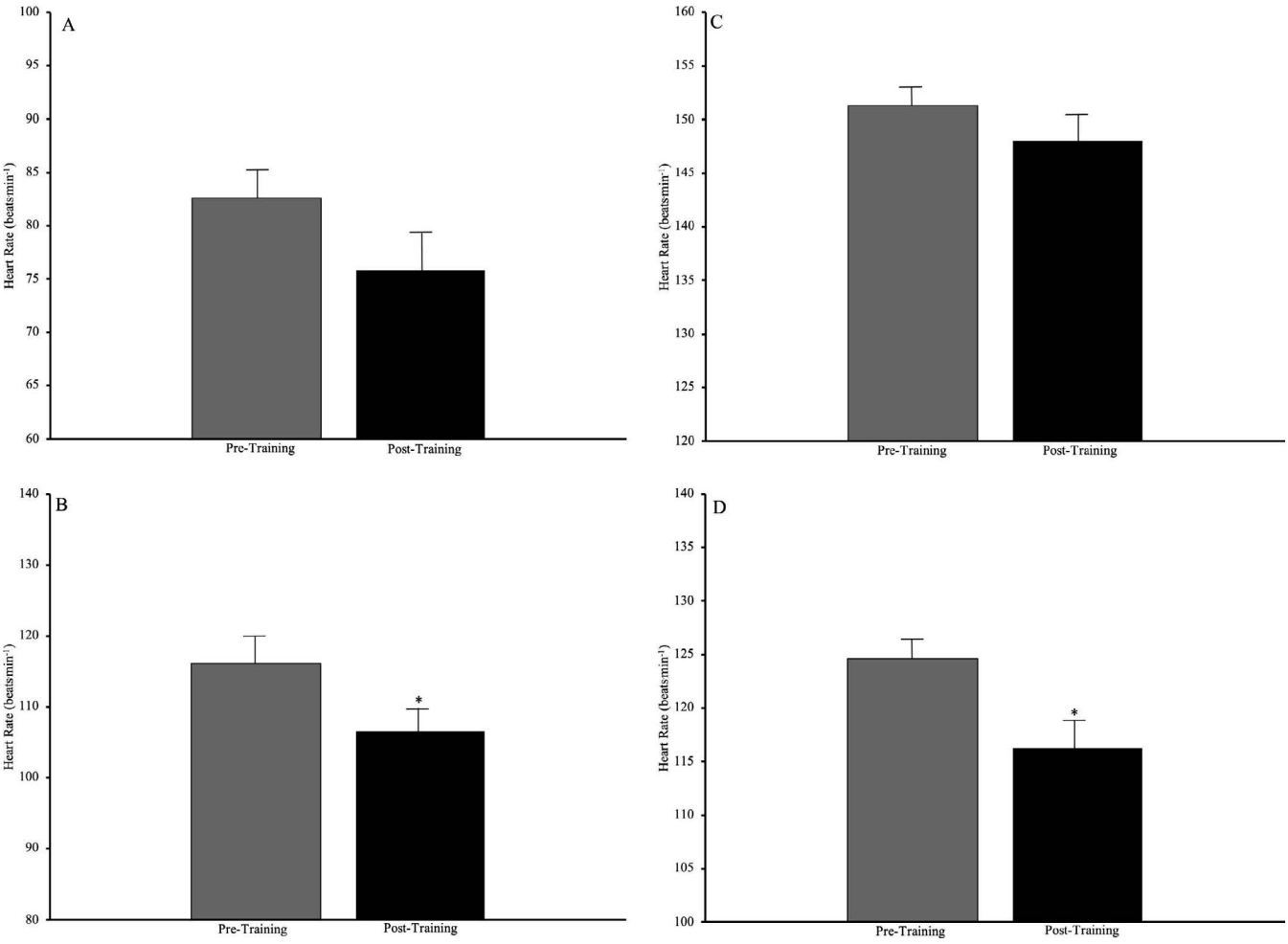

FIGURE 2. Pretraining and posttraining heart rates in obese individuals A) at rest, B) during initial stage exercise, C) during final stage of exercise, and D) during the active recovery period. * indicates $P < 0.05$ from pretraining to posttraining.

HRV data during exercise and cooldown from the submaximal treadmill tests from before and after exercise training are presented in Table 2. After exercise training, HF was significantly increased during the initial exercise stage. There was a trend for SDNN and RMSSD to increase, but they did not reach statistical significance ($P = 0.07$ for each). No other HRV variable was different following exercise training during this initial stage. For the final exercise stage of the submaximal treadmill tests, LF increased after training. During active cooldown, RMSSD and normalized units of HF were significantly increased, while SDNN showed a trend toward an increase ($P = 0.06$ for each). No other HRV variable during cooldown differed following exercise training.

There was no association between body composition and any HRV variable at baseline. However, following exercise training, percent body fat was inversely associated with RMSSD ($r = -0.67$, $P = 0.02$), normalized units of HF ($r = -0.67$, $P = 0.02$), SD1 ($r = -0.67$, $P = 0.02$), and positively associated with normalized units of LF ($r = 0.67$, $P = 0.02$) during the cooldown phase. There was no association between body composition and HRV during the initial or final exercise stage.

DISCUSSION

The current study found that measures of HRV were improved following 12 weeks of moderate aerobic exercise training in obese individuals during the acute exercise and active recovery, independent of changes in cardiorespiratory fitness. Further, we observed a small but significant improvement in percent fat, suggesting a potential link between adiposity and autonomic function. The benefits of exercise training, independent of body fat, could play a role in improvements in autonomic function (25,36). The mechanisms behind such have yet to be elucidated, and further investigation is needed. To our knowledge, this is the first study to examine the effect of training on exercise and immediate postexercise HRV.

We observed improved HRV during the initial stage of exercise primarily, with some changes in HRV during the final stage of the exercise bout. Little research has examined the effect of aerobic training on HRV during exercise. Perini et al. conducted an 8-week progressive interval-based aerobic training program and found no changes in exercising HRV following training in older adults (27). Perini et al. started participants cycling at 40% of their pretraining max workrate in 2 to 4 minute stages totaling 30 minutes of

TABLE 2. Exercise HRV parameters before and after aerobic training.

	Before	After
Initial exercise stage		
SDNN (ms)	7.8 ± 3.8	10.4 ± 6.4
RMSSD (ms)	6.6 ± 3.7	8.7 ± 5.2
LF (ms^2)[a]	30.7 ± 25.3	51.3 ± 47.4
LF (nu)	60.0 ± 22.9	55.0 ± 23.3
HF (ms^2)[a]	28.4 ± 30.0	83.1 ± 162.7*
HF (nu)	39.6 ± 22.9	44.9 ± 23.3
LF/HF ratio	4.1 ± 6.7	1.8 ± 1.5
Final exercise stage		
SDNN (ms)[a]	2.5 ± 0.5	2.6 ± 1.3
RMSSD (ms)[a]	3.3 ± 0.9	3.3 ± 1.1
LF (ms^2)[a]	1.3 ± 1.1	1.7 ± 3.5*
LF (nu)	55.3 ± 26.0	49.3 ± 27.3
HF (ms^2)[a]	0.9 ± 0.8	2.6 ± 6.8
HF (nu)	25.3 ± 7.6	27.1 ± 8.2
LF/HF ratio[a]	4.0 ± 1.2	2.4 ± 0.7
Cooldown stage		
SDNN (ms)	5.4 ± 2.3	8.3 ± 5.3
RMSSD (ms)[a]	5.2 ± 2.2	8.3 ± 6.1*
LF (ms^2)[a]	34.6 ± 72.8	28.3 ± 47.8
LF (nu)	60.9 ± 15.8	59.2 ± 19.5
HF (ms^2)[a]	20.6 ± 33.2	20.3 ± 21.6
HF (nu)[a]	38.7 ± 15.7	41.5 ± 19.7*
LF/HF ratio[a]	2.1 ± 1.6	1.9 ± 1.1

HF = high frequency; LF = low frequency; nu = normalized units; RMSSD = square root of the mean squared differences of successive normal-to-normal intervals; SDNN = standard deviation of the normal-to-normal intervals. Data are given as mean ± standard deviation

[a]Log transformed for analysis, raw data presented

*$P < 0.05$ compared with Before

exercise. Over 8 weeks the resistance was gradually increased from 40% to 60% up to 80% to 100% of pretraining maximal work rate with participants cycling for 36 minutes each session. Differences in these findings compared to ours could be because of differences in participant age and body composition, or because of the length of training as our study employed 4 additional weeks of training. The Perini et al. study examined an older population (70–80 years old), and their highest BMI was less than 30 $kg \cdot m^{-2}$ whereas in the present study participants were aged 41.1 ± 9.8 years, all with a BMI ≥ 30 $kg \cdot m^{-2}$. Autonomic dysfunction increases with age (37), however, the mechanisms between the age-related and obesity-related decline in ANS modulation are not the same. Therefore, exercise training may only impact the mechanisms related to excess adiposity. The present study observed improvements in HRV primarily during the first stage of exercise, with less impact on the final stage. This difference is likely because of increases in metabolic demand and upregulation of sympathetic control and parasympathetic withdrawal, as is expected (38,39). We did, however, see an improvement in LF variable, which is influenced by both the sympathetic and parasympathetic systems (7).

The present study observed improvements in autonomic function during active recovery in obese individuals following aerobic training and is the first study to examine this. Previous exercise interventions in obese individuals have found improvements in HRV at rest following training (18,19). Amano et al. measured improved ANS activity at rest in obese individuals following 12 weeks of aerobic exercise training on a cycle ergometer (3 sessions per week, 20 minutes at ventilatory threshold) (19). Whereas a study by Figueroa et al. found improvements in autonomic balance during the 20-minute postexercise passive recovery period without changes in resting HRV following a 16-week exercise training in obese individuals with and without type 2 diabetes (36).

Studies on postexercise HRV following aerobic training with nonobese individuals have shown inconsistent results (13,25–27). Two studies examining middle-aged, healthy, sedentary men showed no impact following aerobic training (10 weeks and 1 year of training, respectively) at light-to-moderate intensities on HRV variables (13,26). Perini et al. examined both men and women and found no impact of 8 weeks of training on HRV variables (27). In contrast, 1 study found that 14 weeks of low-intensity and high-intensity training resulted in greater HRV in the frequency domain variables of HRV during during acute exercise (25). They did not report, however, body composition variables, so it is not known whether a change in body composition occurred. Studies in nonobese individuals that reported no change in postexercise HRV following training also reported no significant changes in body weight or mass (13,26,27). Conversely, the present study did not result in any changes in body mass or weight but did note improvements in body fat percentage and HRV during exercise and active recovery. This suggests that changes in body adiposity play a role in autonomic function.

Observations of ANS dysfunction in obese individuals has been reported (4). A potential pathway in which obesity influences ANS dysregulation is through elevated plasma leptin concentrations (40–42). Leptin is an important regulator in lipid metabolism and is released in proportion to body fat concentration (37). Additionally, leptin upregulates catecholamine activity in animal models and may directly increase SNS activity in humans (23,41,43). Higher leptin concentrations occur with elevated adiposity and, therefore, would modulate autonomic function (40,42). It is important to note that the present study did not measure leptin concentration and therefore cannot conclude that this is the acting mechanism of improved HRV. However, reductions in body fat percentage were observed from pretraining to

posttraining. This could have potentially led to a decreased leptin and leptin-driven effects on SNS activity, thus improving ANS balance and HRV.

A potential limitation of the present study was the use of individualized submaximal tests to predict VO_{2max}. This limited the ability to truly observe changes that may have occurred in cardiorespiratory fitness from pretraining to posttraining. This also limited the ability to draw comparisons in HRV at each workload during exercise, aside from the initial and final stages of exercise because of the same workload and relative intensity, respectively. However, using an individualized submaximal protocol as opposed to a maximal graded exercise test was safer in the population being observed and allowed every participant to achieve their submaximal goal. As previously discussed, the literature surrounding cardiac autonomic regulation during exercise has found that from moderate to high intensities there is decreased autonomic activity due to shifts from predominantly parasympathetic to sympathetic cardiac control to meet metabolic demands (38,39). Because of this it is unlikely that changes would have occurred in the subsequent stages following the initial stage of the graded submaximal exercise test because of increasing intensity. Additionally, we did not measure HRV at rest. This limited our ability to compare our findings to other studies that have examined HRV changes with training at rest. Our focus, however, was to explore changes during acute exercise and active recovery with aerobic training.

CONCLUSIONS

In summary, the present study found that HRV during exercise and an active recovery were improved following 12 weeks of moderate aerobic exercise training in obese individuals. Future research should further examine the impact of exercise training and acute exercise on HRV variables, as well as to examine the effect of exercise on leptin concentration during physical activity and exercise training. Specifically, the role leptin concentrations may have on ANS function following exercise training in obese individuals.

For the practicing clinical exercise physiologist, this study provides evidence to suggest that obese individuals can improve autonomic dysregulation, a powerful pathway to the development of CVD risk factors, thereby potentially improving long-term risk. For individuals in which vigorous intensity exercise is not possible because of low fitness, orthopedic limitations, etc., and where significant weight loss may be challenging as a result, this study suggests that improvements can still be achieved.

Acknowledgments: The authors would also like to thank the individuals who volunteered to participate in this study. This work was supported by grants from the Sentara Rockingham Hospital Foundation and the James Madison University College of Health and Behavioral Studies Collaborative Research Grant Program.

REFERENCES

1. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, Chamberlain AM, Chang AR, Cheng S, Delling FN, Djousse L, Elkind MSV, Ferguson JF, Fornage M, Khan SS, Kissela BM, Knutson KL, Kwan TW, Lackland DT, Lewis TT, Lichtman JH, Longenecker CT, Loop MS, Lutsey PL, Martin SS, Matsushita K, Moran AE, Mussolino ME, Perak AM, Rosamond WD, Roth GA, Sampson UKA, Satou GM, Schroeder EB, Shah SH, Shay CM, Spartano NL, Stokes A, Tirschwell DL, VanWagner LB, Tsao CW, American Heart Association Council on E, Prevention Statistics C, Stroke Statistics S. Heart disease and stroke statistics-2020 update: a report from the American Heart Association. Circulation. 2020;141(9):e139–e596.
2. Garrison RJ, Kannel WB, Stokes J 3rd, Castelli WP. Incidence and precursors of hypertension in young adults: the Framingham Offspring Study. Prev Med. 1987;16(2):235–51.
3. Bell BB, Rahmouni K. Leptin as a mediator of obesity-induced hypertension. Curr Obes Rep. 2016;5(4):397–404.
4. Fidan-Yaylali G, Yaylali YT, Erdogan C, Can B, Senol H, Gedik-Topcu B, Topsakal S. The Association between central adiposity and autonomic dysfunction in obesity. Med Princ Pract. 2016;25(5):442–8.
5. Hall JE, do Carmo JM, da Silva AA, Wang Z, Hall ME. Obesity-induced hypertension: interaction of neurohumoral and renal mechanisms. Circ Res. 2015;116(6):991–1006.
6. Grassi G, Mark A, Esler M. The sympathetic nervous system alterations in human hypertension. Circ Res. 2015;116(6): 976–90.
7. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Eur Heart J. 1996;17(3): 354–81.
8. Huikuri HV, Stein PK. Heart rate variability in risk stratification of cardiac patients. Prog Cardiovasc Dis. 2013;56(2):153–9.
9. Thayer JF, Yamamoto SS, Brosschot JF. The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. Int J Cardiol. 2010;141(2):122–31.
10. Yadav RL, Yadav PK, Yadav LK, Agrawal K, Sah SK, Islam MN. Association between obesity and heart rate variability indices: an intuition toward cardiac autonomic alteration - a risk of CVD. Diabetes Metab Syndr Obes. 2017;10:57–64.
11. Laederach-Hofmann K, Mussgay L, Ruddel H. Autonomic cardiovascular regulation in obesity. J Endocrinol. 2000; 164(1):59–66.
12. Karason K, Mølgaard H, Wikstrand J, Sjöström L. Heart rate variability in obesity and the effect of weight loss. Am J Cardiol. 1999;83(8):1242–7.
13. Cornelissen VA, Verheyden B, Aubert AE, Fagard RH. Effects of aerobic training intensity on resting, exercise and post-exercise blood pressure, heart rate and heart-rate variability. J Hum Hypertens. 2010;24(3):175–82.
14. Pichot V, Roche F, Denis C, Garet M, Duverney D, Costes F, Barthelemy JC. Interval training in elderly men increases both heart rate variability and baroreflex activity. Clin Auton Res. 2005;15(2):107–15.
15. Pichot V, Roche F, Gaspoz J-M, Enjolras F, Antoniadis A, Minini P, Costes F, Busso T, Lacour J-R, Barthèlèmy JC. Relation between heart rate variability and training load in

middle-distance runners. Med Sci Sports Exerc. 2000; 32(10):1729–36.
16. Levy WC, Cerqueira MD, Harp GD, Johannessen K-A, Abrass IB, Schwartz RS, Stratton JR. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. Am J Cardiol. 1998;82(10):1236–41.
17. De Meersman RE. Heart rate variability and aerobic fitness. Am Heart J. 1993;125(3):726–31.
18. Castello V, Simões RP, Bassi D, Catai AM, Arena R, Borghi-Silva A. Impact of aerobic exercise training on heart rate variability and functional capacity in obese women after gastric bypass surgery. Obes Surg. 2011;21(11):1739–49.
19. Amano M, Kanda T, Ue H, Moritani T. Exercise training and autonomic nervous system activity in obese individuals. Med Sci Sports Exerc. 2001;33(8):1287–91.
20. Riebe D, Ehrman JK, Liguori G, Magal M. ACSM's guidelines for exercise testing and prescription. T10th ed. Philadelphia: Wolters Kluwer Health; 2018.
21. Barbosa MP, da Silva NT, de Azevedo FM, Pastre CM, Vanderlei LC. Comparison of Polar(R) RS800G3 heart rate monitor with Polar(R) S810i and electrocardiogram to obtain the series of RR intervals and analysis of heart rate variability at rest. Clin Physiol Funct Imaging. 2016;36(2):112–7.
22. Fletcher GF, Ades PA, Kligfield P, Arena R, Balady GJ, Bittner VA, Coke LA, Fleg JL, Forman DE, Gerber TC, Gulati M, Madan K, Rhodes J, Thompson PD, Williams MA, American Heart Association Exercise CR, Prevention Committee of the Council on Clinical Cardiology CoNPA, Metabolism CoC, Stroke N, Council on E, Prevention. Exercise standards for testing and training: a scientific statement from the American Heart Association. Circulation. 2013;128(8):873–934.
23. Beltowski J. Role of leptin in blood pressure regulation and arterial hypertension. J Hypertens. 2006;24(5):789–801.
24. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. N Engl J Med. 1999;341(18):1351–7.
25. Martinmaki K, Hakkinen K, Mikkola J, Rusko H. Effect of low-dose endurance training on heart rate variability at rest and during an incremental maximal exercise test. Eur J Appl Physiol. 2008;104(3):541–8.
26. Verheyden B, Eijnde BO, Beckers F, Vanhees L, Aubert AE. Low-dose exercise training does not influence cardiac autonomic control in healthy sedentary men aged 55-75 years. J Sports Sci. 2006;24(11):1137–47.
27. Perini R, Fisher N, Veicsteinas A, Pendergast DR. Aerobic training and cardiovascular responses at rest and during exercise in older men and women. Med Sci Sports Exerc. 2002;34(4):700–8.
28. Bhati P, Shenoy S, Hussain ME. Exercise training and cardiac autonomic function in type 2 diabetes mellitus: A systematic review. Diabetes Metab Syndr. 2018;12(1):69–78.
29. Liguori G. ACSM's health-related physical fitness assessment manual. 5th ed. Philadelphia: Wolters Kluwer; 2018.
30. Hernando D, Garatachea N, Almeida R, Casajus JA, Bailon R. Validation of heart rate monitor Polar RS800 for heart rate variability analysis during exercise. J Strength Cond Res. 2018;32(3):716–25.
31. Giles D, Draper N, Neil W. Validity of the Polar V800 heart rate monitor to measure RR intervals at rest. Eur J Appl Physiol. 2016;116(3):563–71.
32. Gamelin FX, Baquet G, Berthoin S, Bosquet L. Validity of the Polar S810 to measure R-R intervals in children. Int J Sports Med. 2008;29(2):134–8.
33. Esco MR, Williford HN, Flatt AA, Freeborn TJ, Nakamura FY. Ultra-shortened time-domain HRV parameters at rest and following exercise in athletes: an alternative to frequency computation of sympathovagal balance. Eur J Appl Physiol. 2018;118(1):175–84.
34. Niskanen JP, Tarvainen MP, Ranta-Aho PO, Karjalainen PA. Software for advanced HRV analysis. Comput Methods Programs Biomed. 2004;76(1):73–81.
35. Draghici AE, Taylor JA. The physiological basis and measurement of heart rate variability in humans. J Physiol Anthropol. 2016;35(1):22. doi:10.1186/s40101-016-0113-7
36. Figueroa A, Baynard T, Fernhall B, Carhart R, Kanaley JA. Endurance training improves post-exercise cardiac autonomic modulation in obese women with and without type 2 diabetes. Eur J Appl Physiol. 2007;100(4):437–44.
37. Parashar R, Amir M, Pakhare A, Rathi P, Chaudhary L. Age related changes in autonomic functions. J Clin Diagn Res. 2016;10(3):CC11–5.
38. Michael S, Graham KS, Davis GMO. Cardiac autonomic responses during exercise and post-exercise recovery using heart rate variability and systolic time intervals-a review. Front Physiol. 2017;8:301. doi:10.3389/fphys.2017.00301
39. White DW, Raven PB. Autonomic neural control of heart rate during dynamic exercise: revisited. J Physiol. 2014;592(12): 2491–500.
40. Ekmen N, Helvaci A, Gunaldi M, Sasani H, Yildirmak ST. Leptin as an important link between obesity and cardiovascular risk factors in men with acute myocardial infarction. Indian Heart J. 2016;68(2):132–7.
41. Rahmouni K. Leptin-induced sympathetic nerve activation: signaling mechanisms and cardiovascular consequences in obesity. Curr Hypertens Rev. 2010;6(2):104–209.
42. Ostlund RE Jr, Yang JW, Klein S, Gingerich R. Relation between plasma leptin concentration and body fat, gender, diet, age, and metabolic covariates. J Clin Endocrinol Metab. 1996;81(11):3909–13.
43. Satoh N, Ogawa Y, Katsuura G, Numata Y, Tsuji T, Hayase M, Ebihara K, Masuzaki H, Hosoda K, Yoshimasa Y, Nakao K. Sympathetic activation of leptin via the ventromedial hypothalamus: leptin-induced increase in catecholamine secretion. Diabetes. 1999;48(9):1787–93.