Published in final edited form as:

J Pediatr. 2015 August ; 167(2): 378–383.e1. doi:10.1016/j.jpeds.2015.04.058.

Sympathetic Activity Assessed During Exercise Recovery in Young Obese Females

R. Lee Franco, Ph.D.¹, Stacey H. Privett, M.S.¹, Mary K. Bowen, M.S.¹, Edmund O. Acevedo, Ph.D.¹, James A. Arrowood, M.D.², Edmond P. Wickham, M.D.³, and Ronald K. Evans, Ph.D.¹

¹Department of Kinesiology and Health Sciences, College of Humanities and Sciences, Virginia Commonwealth University, 1020 West Grace Street, Suite 111, Richmond, VA, USA 23284-3021

²Department of Internal Medicine, Division of Cardiology, School of Medicine, Virginia Commonwealth University, 1001 East Broad Street, Suite 405, Richmond, VA, USA 23298-0036

³Departments of Internal Medicine and Pediatrics, Division of Endocrinology and Metabolism, School of Medicine, Virginia Commonwealth University, 10001 East Broad Street, Suite 405, Richmond, VA, USA 23298-0036

Abstract

Objective—To evaluate differences in sympathetic activity, as assessed by an exercise recovery index (ERI; heart rate/VO₂ plateau), between black and white obese female adolescents. An additional aim was to determine the association of ERI with insulin resistance (HOMA-IR), cardiovascular fitness per fat-free mass (VO₂FFM), systolic blood pressure (SBP), and percent body fat (%FAT) in both black and white obese adolescents.

Study design—Sixty-one females volunteered to participate in this study. HOMA-IR, SBP and %FAT were assessed during resting conditions in black (n=49, 13.7±1.6 yrs, 38.1±6.1 kg/m²) and white (n=12, 13.3±2.2 yrs, 34.3±4.9 kg/m²) obese adolescents. An ERI was calculated during a 5-minute passive recovery period immediately following a graded treadmill exercise test to exhaustion.

Results—The ERI was significantly greater in black compared with white obese adolescent females (29.8 ± 6.4 vs. 24.1 ± 3.1 bpm·mLO₂⁻¹·min⁻¹, P = 0.004). Using multiple linear regression modeling, there was a significant independent association between ERI and VO₂FFM (r = -0.310, P = 0.027) and %FAT (r = 0.326, P = 0.020) in black obese adolescents after controlling for HOMA-IR and SBP.

Conclusions—These results suggest that black obese adolescent females have greater sympathetic activity, as assessed by an ERI, than white obese adolescent females. These findings

Corresponding Author: R. Lee Franco, 1020 West Grace Street, 500 Academic Centre, Room 111, Virginia Commonwealth University, Richmond, VA 23284-3021, Telephone: 804-827-9348, francorl@vcu.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

support the need for weight management efforts aimed at both reducing %FAT and improving fitness in obese adolescents, specifically black females.

Trial registration—Registered with Clinicaltrials.gov: NCT00562293

Keywords

Autonomic Nervous System; Exercise Recovery Index; Adolescents; Racial Differences; Adolescents

A common detriment of obesity is cardiovascular autonomic nervous system (ANS) dysfunction (1). In obese populations, disequilibrium of the ANS is reflected by reduced parasympathetic nervous system (PNS) function and/or sympathetic nervous system (SNS) overactivity (2, 3). Over-activation of the SNS specifically targeting the heart, blood vessels, and kidneys appears to exacerbate metabolic consequences associated with obesity by predisposing individuals to the development of hypertension and other cardiovascular disease risk factors (2). Although adolescent cardiovascular ANS activity has been investigated extensively, the relationship between SNS activity and obesity in children and adolescents has received little attention (4–9). Studies evaluating the impact of obesity on SNS function during adolescence has revealed equivocal findings of both over (4, 6–8) and under-activation of the SNS (5, 9).

Recently, a novel exercise recovery index (ERI) was introduced as a minimally invasive method that could potentially assess SNS control within individuals at risk of developing cardiovascular- or diabetes-related disorders (11). To date, no studies have directly examined the effect of ethnicity on SNS function in obese adolescents. Therefore, the primary aim of this study was to explore the relationship between ethnicity and SNS function in obese adolescent females, assessed with the simple HR/VO₂ plateau ERI. It is hypothesized that SNS activity, as measured by the ERI, is higher in black obese adolescent females than white obese adolescent females.

METHODS

Obese female adolescents between 11 and 18 years of age [Body Mass Index (BMI) 95th percentile for age and sex according to the 2000 CDC Growth Charts] were recruited to participate in this study. Additionally, all participants met the BMI international cut off points for obesity by sex between ages 2 and 18 years, defined to pass through BMI of 30 kg/m² at age 18 (12). Because autonomic function in adolescents has been shown to be mediated by leptin (13), this study chose to investigate only females. Leptin, which has been shown to be inhibited by testosterone and stimulated by estradiol and dexamethasone, can stimulate hypothalamus-induced thermogenesis through SNS mechanisms (14 – 16). Prior to enrollment in the study, volunteers were not participating in an exercise program. Study procedures were explained, and parents provided written, informed consent and adolescents provided written assent before participation. Following enrollment in the study, participants underwent a comprehensive anthropometric and metabolic assessment. A complete medical history, physical examination, and evaluation for participation in exercise testing was conducted by a physician. Individuals diagnosed with any known cardiovascular or

metabolic disorders [e.g., diabetes and obstructive sleep apnea] or taking medications that could potentially influence cardiovascular or endocrine function were excluded from the study. In addition, physical activity levels were assessed utilizing the interviewer-administered 7-day physical activity recall (17). All procedures were approved by Virginia Commonwealth University's Institutional Review Board.

Following an overnight fast, each adolescent underwent a comprehensive anthropometric and metabolic assessment at the Virginia Commonwealth University's Clinical Research Services Unit. Measurements included height (to the nearest 0.5 cm), weight (to the nearest 0.25 kg), and body composition via bioelectrical impedance analysis (Quantum II, RJL Systems, Clinton Township, MI) for determination of fat and lean mass. After being seated for 5 minutes, determination of systolic (SBP) and diastolic (DBP) blood pressure was obtained using an automated device (Dynamap Pro 100, General Electric). Fasting venous blood samples were collected for analysis of total cholesterol, triglycerides (TAG), LDL cholesterol, HDL cholesterol, glucose, and insulin. Total cholesterol, TAG, and HDL cholesterol were measured using a Roche automated clinical chemistry analyzer. LDL cholesterol was calculated by the Friedewald equation ($\text{LDL} = \text{total cholesterol} - \text{HDL cholesterol} - [\text{triglycerides}/5]$) (18). Plasma glucose and insulin levels were determined using a glucose oxidase methodology (YSI 2300 Stat Plus Glucose Analyzer Yellow Springs Instruments), and an enzyme-linked immune-assay (ALPCO Diagnostics), respectively. To compute the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), a simple mathematical approximation was employed using the product of fasting insulin and glucose values ($\text{HOMA-IR} = [(\text{fasting plasma glucose} \times \text{fasting plasma insulin})/22.5]$) (19).

Within one week of baseline testing, subjects were scheduled for a maximal graded exercise test. Subjects were asked to refrain from exercise 24 hours before the exercise test. Exercise testing was conducted at the Virginia Commonwealth University Exercise Physiology Research Laboratory, with participants at least 4 hours postprandial. Peak VO_2 was determined using a maximal graded exercise test to exhaustion on a treadmill (Trackmaster TMX425C, Full Vision, Inc., Newton KS) (20). During exercise, breath by breath gas exchange variables were measured using a VMAX Spectra Sensormedics gas analyzer (Sensormedics Corp., Yorba Linda, CA). The ventilatory expired gas analysis system was calibrated prior to each exercise session according to manufacturer specifications. Additionally, heart rate responses were recorded at each minute during the test via heart rate monitors (Model E600, Polar Electro, Lake Success, NY) and rating of perceived exertion (6–20 Borg Scale) was documented near the end of each stage.

Following a 3-min rest period of standing gas exchange, subjects began a step transition into a 4-min stage at 2.5 mph and 0% grade. The progressive protocol continued with a 2-min stage at 3.0 mph and 0% grade. Subsequent 2-min stages were held constant at 3.0 mph while grade was increased to 2.0, 5.0, 8.0, 11.0, 14.0, and 17%. Subjects were verbally encouraged to give maximal effort during the test until volitional exhaustion was achieved. Breath-by-breath data were averaged into 10 second intervals to reduce noise and enhance the underlying physiological response characteristics. Peak VO_2 was taken at the highest recorded 10s average during the maximal exercise test. The attainment of peak VO_2 was determined by participants satisfying at least two of the following criteria: i) a respiratory

exchange ratio = 1.00, ii.) a maximum HR = 90% of age predicted maximum HR, and iii) rating of perceived exertion = 18.

Immediately following attainment of peak VO_2 , subjects completed a 5-minute passive recovery while standing as still as possible with their hands by their sides. During this recovery period, HR was recorded every 30 seconds and VO_2 was recorded continuously. It has been suggested that HR recovery from exercise is initially mediated by vagal reactivation up to 120 seconds (21). By extending the recovery period from a maximal exercise test beyond the initial rapid PNS response (~ 2 minutes), the ratio taken during the time-point into recovery where the degree of heart rate (HR) recovery normalized for oxygen consumption (VO_2) initially plateaus (HR/ VO_2 plateau) defines the ERI (11). Specifically, the 2 minutes change in HR following cessation of the exercise test, measured as the difference between maximal HR attained and the HR at 2-minutes post-exercise, was used to evaluate PNS activity. To assess SNS activity, the ERI was calculated from dividing HR (bpm) by VO_2 ($\text{mLO}_2 \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) for every 30-second interval during exercise recovery and graphed against each respective time point using Microsoft Excel. Two technicians evaluated the graphs on separate occasions to determine the ERI value representing the time point where the ratio initially reached a plateau (11). A representative curve, with a point of interest used as a functional index of SNS activity, is provided in Figure 1 (available at www.jpeds.com) as a graphical example.

Statistical Analyses

Due to differences in group sample sizes, a one-way ANOVA (SPSS, Chicago, IL: V19.0) was used to demonstrate homogeneity of subject demographics between the two groups. In addition, a one-way ANOVA was used to compare variables of interest within the study. Simple associations between ERI and health characteristics were evaluated using Pearson product-moment correlations. Multiple linear regression was performed on the black female group to assess the independent variability of ERI with percent body fat (%FAT), HOMA-IR, fasting insulin, fasting TAG, VO_2 per fat free mass ($\text{VO}_{2\text{FFM}}$), and SBP. Log transformations of the insulin sensitivity indices were computed within SPSS and used to satisfy assumptions of the statistical methods (11). All data are expressed as mean $\pm$ SD unless otherwise noted. Statistical significance was set at $P = 0.05$ for all analyses.

RESULTS

Sixty-one adolescents participated in the study (Black: $N = 49$; White: $N = 12$) from December 9, 2009, to August 25, 2011. Subject characteristics for both black and white females are presented in Table I. Group sample sizes were unequal due to attrition of recruited participants not meeting inclusion criteria. In addition, the group differences in sample size reflects the region's demographic distribution of race. Equal variances were observed in both groups, therefore results of the one-way ANOVA analyses were provided to indicate observed differences between the obese adolescent females. There were no significant differences with regard to age (Black females, 13.7 ± 1.7 years vs. White females, 13.3 ± 2.1 years; $P = 0.506$, $d = 0.21$), BMI (Black females, $38.2 \pm 6.2 \text{ kg/m}^2$ vs. White females, $35.1 \pm 5.4 \text{ kg/m}^2$; $P = 0.119$, $d = 0.53$), or %FAT (Black females, 52.1 ± 4.6

% vs. White females, 49.9 ± 5.1 %; $P = 0.168$, $d = 0.45$) between the two groups. Furthermore, there were no statistical differences between the two groups when comparing intensity levels of physical activity reported (White vs. Black Females: Moderate 1.7 ± 2.1 vs. 1.2 ± 1.4 hrs/week, $P = 0.347$; Hard 0.0 ± 0.0 vs. 0.1 ± 0.4 hrs/week, $P = 0.449$).

Insulin concentrations could not be determined in two black females. Therefore, the black obese female group had 47 subjects for all fasting insulin and HOMA-IR data analyses. The black females displayed significantly higher SBP values (119.2 ± 9.7 vs. 111.0 ± 9.3 mmHg; $P = 0.012$, $d = 0.86$) and lower fasting TAG concentrations (75.8 ± 28.0 vs. 114.5 ± 56.0 mg/dL; $P = 0.001$, $d = 0.87$) compared with white females. In addition, Black females had significantly higher ERI values compared with white females (29.8 ± 6.4 vs. 24.1 ± 3.1 bpm·mLO₂⁻¹·min⁻¹; $P = 0.004$, $d = 1.13$) as displayed in Figure 2. Importantly, the time point during recovery where the ERI was determined was not significantly different between the two groups (Black females, 169.5 ± 34.0 sec vs. White females, 157.5 ± 22.6 sec; $P = 0.260$, $d = 0.42$). Notably, the medical history included parental report of hypertension. Only four of the white adolescent females had a parent report history of hypertension. Of the black adolescents, 16 had at least one parent report a history of hypertension. There was no significant difference in ERI between the black adolescent females who had parental reported history of hypertension compared with those with no parental history of hypertension (History, $N = 16$, 30.5 ± 4.1 vs. No History, $N = 33$, 29.5 ± 7.3 bpm·mLO₂⁻¹·min⁻¹; $P = 0.590$, $d = 0.17$).

Pearson product-moment correlations between ERI and health variables in both black and white females are presented in Table II. As shown in Figure 3, black females displayed a significant relationship between ERI and VO_{2FFM}. No significant relationships were observed between ERI and the health variables ($P = 0.374$) in white females.

A multiple linear regression analysis was performed on black females to determine relative contributions of various health characteristics to ERI, as displayed in Table III. The independent variables tested were %FAT, HOMA-IR, fasting insulin, fasting TAG, VO_{2FFM}, and SBP. In the black females, 27.0% ($P = 0.037$) of the ERI variance was explained for by the independent variables with significant contributions from %FAT (10.0%) and VO_{2FFM} (9.6%).

Additionally, semi-partial correlations, presented in Table III, describe the unique contribution of variables to ERI when variance was controlled. A multiple linear regression was not performed on the white females due to the low subject number and lack of statistical power.

DISCUSSION

In the US, adolescent obesity is more common in black individuals compared with white individuals, particularly black females (22). More specifically, 14.5% of white adolescent females were categorized as obese, compared with 29.2% of their black counterparts (22). Overactivation of the SNS is a common hallmark of obesity, often implicated as a link between excess adiposity and cardiovascular disease risk, especially in black adults (23).

Various studies have shown ethnic differences in ANS function in adult (24–27) and healthy youth (10, 28–30) populations; however, little is known in regards to ANS function and ethnic differences in obese adolescents

Findings from the current study revealed a significant inverse relationship between ERI and VO_{2FFM} in the black females only. Previous results have suggested similar findings associated with impaired ANS function in a group of 304 bi-racial non-obese male and female adolescents (10). Gutin et al calculated ANS balance from heart rate variability variables during resting conditions with subjects in a supine position, prior to completing a graded treadmill test to exhaustion for the assessment of cardiovascular fitness (10). The black adolescents had more favorable heart rate variability profiles, marked by higher PNS activity, compared with the white adolescents. Higher cardiovascular fitness was significantly associated with more favorable PNS function in black females but was not significantly related to PNS activity in white females. Interestingly, the impact of adiposity on ANS function revealed a detrimental effect of PNS and SNS activity in the black females only, despite more favorable heart rate variability indices. Supporting the present implications in obese adolescents, these findings may suggest that as weight gain ensues and cardiovascular fitness deteriorates, cardiac ANS function is compromised to a greater extent in black female adolescents than white adolescents.

Autonomic function in adolescence has been shown to be mediated by several metabolic factors including body composition (31), insulin resistance (IR) (8), leptin (13), SBP (4) and cardiovascular fitness (10). Given this, a multiple regression was used to adjust for potential covariates of ERI including %FAT, HOMA-IR, fasting insulin, fasting TAG, VO_{2FFM} , and SBP. The results of the present study suggest that only %FAT and VO_{2FFM} were able to explain a significant independent portion of the ERI in the black females. Our findings are similar to those of Abate et al, who used microneurographic recordings to explore relationships between adiposity and SNS function in normotensive black and white adults of similar age and BMI (32). Sympathetic nervous system activity was significantly correlated with BMI in both black and white females. Regional SNS modulation has been shown to vary in magnitude with various organ systems in an obese population (33). Therefore, the different techniques used to assess SNS function between our study and Abate et al. could explain the lack of agreement in findings for the white females.

Several hypotheses have been proposed to explain the over-expression of SNS activity observed in obesity, including the presence of IR, which has been shown to cause higher SNS activity in obese adolescents when compared with non-IR obese controls (8). HOMA-IR, a method used to quantify IR, was not related to ERI in either group. Previous findings have suggested that HOMA-IR was associated with ANS dysfunction, as defined by exercise recovery kinetics, in a group of young individuals with a wide range of ERI values (9 to 34 bpm/(mL $O_2 \cdot kg^{-1} \cdot min^{-1}$)) (11). Yeckel et al evaluated the relationship between ERI and HOMA-IR in three groups whose fitness levels were significantly different, accounting for a large variance in ERI among the various groups (11). Although results within the current study suggest that black females had a significantly higher ERI than their white counterparts, both groups had higher ERI values than previously reported in a non-obese

population. Without a larger variance in ERI, there may have been a limited ability to assess any relationships with HOMA-IR.

There are a number of factors that may have influenced the ANS function measures observed within this study. This study did not have a lean control group, therefore we are unable to evaluate any independent effect of obesity on sympathetic activity. Interestingly, although neither group independently expressed a significant relationship of ERI to percent body fat, when all the participants were combined, there was a weak positive relationship between ERI and percent body fat ($r = 0.292$, $P = 0.23$). The racial differences in SNS activity observed in our study, as indicated by an ERI, are further supported by Arensman et al, who demonstrated that healthy black male children have increased systemic vascular resistance following exercise as compared with their white counterpart (34). In addition, it has been suggested that young black women have greater orthostatic tolerance compared with white women, most likely attributed to greater SNS activity and vascular stiffness (35). It is plausible that the standing exercise protocol within the current study may have contributed to the differences observed in ERI between black and white female adolescents. Furthermore, the ERI has only been validated during a high-carbohydrate meal-induced thermogenesis. Validation studies against standard measurements of sympathetic activity are warranted prior to using the ERI as a clinical tool. Additionally, this study did not include the measurement of variables that have been shown to impact sympathetic activity, such as leptin and obstructive sleep apnea. Nevertheless, the impact of leptin within the current study is thought to be minimal due to increased leptin resistance in individuals with greater than 30% body fat (36). Lastly, the current population of pubertal adolescents continue to experience individual cycle irregularity thus making it difficult to control for menstrual phase, which along with Tanner staging has been shown to impact ANS function (37, 38). Notably, the physical examination within the current study included an optional standardized assessment of pubertal development via Tanner staging. There was no significant difference in Tanner stage between the obese adolescent female groups (Black females, $N = 38$, 3.8 ± 0.7 vs. White females).

Elevated SNS activity in adolescence plays a pivotal role in the development of hypertension, cardiovascular disease, and type II diabetes later in life (39). Therefore, based upon the results from the current study, it is plausible that black obese female adolescents may be at a higher risk for the development of disease later in life. Additionally, %FAT and VO_{2FFM} were significant independent predictors of the variance in SNS activity in the black females, highlighting the importance of physical training aimed at improving VO_{2max} coupled with appropriate changes in diet. Together, these lifestyle modification changes may elicit more favorable changes in ANS function in black adolescent obese females.

Acknowledgments

Supported by Virginia Premier Health Plan, Inc, Children's Hospital Foundation, the National Institutes of Health CTSA (K23-HD053742 [to E.W.] and UL1TR000058 [to V.U.]). The authors declare no conflicts of interest.

Abbreviations

BMI	body mass index
ERI	exercise recovery index
HOMA-IR	homeostasis model assessment of insulin resistance
VO₂FFM	oxygen consumption per fat free mass
SBP	systolic blood pressure
%FAT	percent body fat
ANS	autonomic nervous system
PNS	parasympathetic activity
HR	heart rate
VO₂	oxygen consumption
TAG	triglyceride

References

1. Snitker S, Macdonald I, Ravussin E, Astrup A. The sympathetic nervous system and obesity: role in aetiology and treatment. *Obes Rev.* 2000; 1:5–15. [PubMed: 12119646]
2. Davy KP, Hall JE. Obesity and hypertension: two epidemics or one? *Am J Physiol Regul Integr Comp Physiol.* 2004; 286:R803–R813. [PubMed: 15068965]
3. Rabbone I, Bobbio A, Rabbia F, Bertello MC, Ignaccollo MG, Saglio E, et al. Early cardiovascular autonomic dysfunction, beta cell function and insulin resistance in obese adolescents. *Acta Biomed.* 2009; 80:29–35. [PubMed: 19705617]
4. Guizar JM, Ahuatzin R, Amador N, Sanchez G, Romer G. Heart autonomic functions in overweight adolescents. *Indian Pediatr.* 2005; 42:464–9. [PubMed: 15923693]
5. Nagai N, Matsumoto T, Kita H, Moritani T. Autonomic nervous system activity and the state of and development of obesity in Japanese school children. *Obes Res.* 2003; 11:25–32. [PubMed: 12529482]
6. Rabbia F, Silke B, Conterno A, Grosso T, De Vito B, Rabbone I, et al. Assessment of cardiac autonomic modulation during adolescent obesity. *Obes Res.* 2003; 11:541–48. [PubMed: 12690083]
7. Riva P, Martini G, Rabbia F, Milan A, Faglieri C, Chiandussi L, et al. Obesity and autonomic function in adolescence. *Clin Exp Hypertens.* 2001; 23:57–67. [PubMed: 11270589]
8. Tascilar ME, Yokusoglu M, Boyraz M, Baysan O, Koz C, Dundaroz R. Cardiac autonomic functions in obese children. *J Clin Res Pediatr Endocrinol.* 2011; 3:60–4. [PubMed: 21750633]
9. Vanderlei LC, Pastre CM, Freitas IF Junior, Godoy M. Analysis of cardiac autonomic modulation in obese and eutrophic children. *Clinics.* 2010; 65:789–92. [PubMed: 20835556]
10. Gutin B, Howe C, Johnson MH, Humphries MC, Snieder H, Barbeau P. Heart rate variability in adolescents: relations to physical activity, fitness, and adiposity. *Med Sci Sports Exerc.* 2005; 37:1856–63. [PubMed: 16286853]
11. Yeckel CW, Gulanski B, Zgorski ML, Dziura J, Parish R, Sherwin RS. Simple exercise recovery index for sympathetic overactivity is linked to insulin resistance. *Med Sci Sports Exerc.* 2009; 41:505–15. [PubMed: 19204601]
12. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ.* 2000; 320:1240–3. [PubMed: 10797032]

13. Kaufman CL, Kaiser DR, Steinberger J, Kelly AS, Dengel DR. Relationships of cardiac autonomic function with metabolic abnormalities in childhood obesity. *Obesity*. 2007; 15:1164–71. [PubMed: 17495192]
14. Casabiell X, Pineiro V, Peino R, Lage M, Camina J, Gallego R, et al. Gender differences in both spontaneous and stimulated leptin secretion by human omental adipose tissue in vitro: dexamethasone and estradiol stimulate leptin release in women, but not in men. *J Clin Endocrinol Metab*. 1998; 83:2149–2155. [PubMed: 9626154]
15. Wabitsch M, Blum WF, Muche R, Braun M, Hube F, Rascher W, et al. Contribution of androgens to the gender difference in leptin production in obese children and adolescents. *J Clin Invest*. 1997; 100:808–813. [PubMed: 9259579]
16. Haynes WG, Morgan DA, Walsh SA, Mark AL, Sivitz WI. Receptor-mediated regional sympathetic nerve activation by leptin. *J Clin Invest*. 1997; 100:270–8. [PubMed: 9218503]
17. Sallis JF, Buono MJ, Roby JJ, Micale FG, Nelson JA. Seven-day recall and other physical activity self-reports in children and adolescents. *Med Sci Sports Exerc*. 1993; 25:99–108. [PubMed: 8423762]
18. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem*. 1972; 18:499–502. [PubMed: 4337382]
19. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and β -cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*. 1985; 28:412–19. [PubMed: 3899825]
20. Franco RL, Bowen MK, Arena R, Privett SH, Acevedo EO, Wickham EP, et al. Sex Differences in Pulmonary Oxygen Uptake Kinetics in Obese Adolescents. *J Pediatr*. 2014; 165:1161–5. [PubMed: 25241180]
21. Imai K, Sato H, Hori M, Kusuoka H, Ozaki H, Yokoyama H, et al. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. *J Am Coll Cardiol*. 1994; 24:1529–35. [PubMed: 7930286]
22. gden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index of US children and adolescents, 2007–2008. *JAMA*. 2010; 303:242–49. [PubMed: 20071470]
23. Eslami P, Tuck M. The role of sympathetic nervous system in linking obesity with hypertension in white versus black Americans. *Curr Hypertens Rep*. 2003; 5:269–72. [PubMed: 12724061]
24. Abbas A, Szczepaniak LS, Tuncel M, McGavock JM, Huet B, Fadel PJ, et al. Adiposity-independent sympathetic activity in black men. *J Appl Physiol*. 2010; 108:1613–18. [PubMed: 20299621]
25. Choi J, Hong S, Nelesen R, Bardwell WA, Natarajan L, Schubert C, et al. Age and ethnicity differences in short-term heart-rate variability. *Psychosom Med*. 2006; 68:421–26. [PubMed: 16738074]
26. Liao D, Barnes RW, Chambless LE, Simpson RJ, Sorlie P, Heiss G. Age, race, and sex differences in autonomic cardiac functions measured by spectral analysis of heart rate variability—The ARIC study. *Atherosclerosis Risk in Communities*. *Am J Cardiol*. 1995; 76:906–12. [PubMed: 7484830]
27. Weyer C, Pratley RE, Snitker S, Spraul M, Ravussin E, Tatarami PA. Ethnic differences in insulinemia and sympathetic nervous system tone as links between obesity and blood pressure. *Hypertension*. 2000; 36:531–37. [PubMed: 11040231]
28. Faulkner MS, Hathaway D, Tolley B. Cardiovascular autonomic function in healthy adolescents. *Heart Lung*. 2003; 32:10–22. [PubMed: 12571544]
29. Urbina EM, Bao W, Pickoff AS, Berenson GS. Ethnic (black-white) contrasts in heart rate variability during cardiovascular reactivity testing in male adolescents with high and low blood pressure: the Bogalusa Heart Study. *Am J Hypertension*. 1998; 11:196–202.
30. Wang X, Thayer JF, Treiber F, Snieder H. Ethnic differences and heritability of heart rate variability in African- and European American youth. *Am J Cardiol*. 2005; 96:1166–72. [PubMed: 16214458]
31. Li Z, Snieder H, Su S, Ding X, Thayer JF, Treiber FA, et al. A longitudinal study in youth of heart rate variability at rest and in response to stress. *Int J Psychophysiol*. 2009; 73:212–17. [PubMed: 19285108]

32. Abate NI, Mansour YH, Tuncel M, Arbique D, Chavoshan B, Kizilbash A, et al. Overweight and sympathetic overactivity in black Americans. *Hypertension*. 2001; 38:379–83. [PubMed: 11566908]
33. Davy KP, Orr JS. Sympathetic nervous system behavior in human obesity. *Neurosci Biobehav Rev*. 2009; 33:116–24. [PubMed: 18602694]
34. Arensman FW, Treiber FA, Gruber MP, Strong WB. Exercise-induced differences in cardiac output, blood pressure, and systemic vascular resistance in a healthy biracial population of 10-year-old boys. *Am J Dis Child*. 1989; 143:212–6. [PubMed: 2916494]
35. Hinds K, Stachenfeld NS. Greater orthostatic tolerance in young black compared with white women. *Hypertension*. 2010; 56:75–81. [PubMed: 20458005]
36. Matsumoto T, Miyatsuji A, Miyawaki T, Yanagimoto Y, Moritani T. Potential association between endogenous leptin and sympatho-vagal activities in young obese Japanese women. *Am J Hum Biol*. 2003; 15:8–15. [PubMed: 12552573]
37. Tanaka H, Borres M, Thulesius O, Tamai H, Ericson MO, Lindblad LE. Blood pressure and cardiovascular autonomic function in healthy children and adolescents. *J Pediatr*. 2000; 137:63–67. [PubMed: 10891823]
38. McKinley PS, King AR, Shapiro PA, Slavov I, Fang Y, Chen IS, et al. The impact of menstrual cycle phase on cardiac autonomic regulation. *Psychophysiology*. 2009; 46:904–11. [PubMed: 19386049]
39. Srinivasan SR, Myers L, Berenson GS. Rate of change in adiposity and its relationship to concomitant changes in cardiovascular risk variables among biracial (black-white) children and young adults: The Bogalusa Heart Study. *Metabolism*. 2001; 50:299–305. [PubMed: 11230782]

Figure 1.
Representative curve of ERI plateau

* $P = 0.004$; Box plot line is median value, box is divided as upper and lower quartiles.

Figure 2.
Exercise Recovery Data between Groups

Figure 3.
Relationship between ERI and VO_{2FFM} in Black Adolescent Females

Table 1

Subject characteristics.

Variable	Black (N = 49)	White (N = 12)
Age (years)	13.7 ± 1.7	13.3 ± 2.1
Height (cm)	163.5 ± 7.7	160.5 ± 9.8
Weight (kg)	102.4 ± 19.5	90.8 ± 19.3
BMI (kg/m ²)	38.2 ± 6.2	35.1 ± 5.4
Percent Fat (%FAT)	52.1 ± 4.6	49.9 ± 5.1
Fat Mass (kg)	53.9 ± 14.4	46.1 ± 14.2
Lean Mass (kg)	48.4 ± 6.2	44.7 ± 5.8
Fitness (VO _{2Peak} [mLO ₂ /kg/min])	24.08 ± 4.4	26.6 ± 3.8
Fitness (VO _{2Peak} [L/min])	2.4 ± 0.4	2.4 ± 0.3
Fitness (VO _{2FFM} [mLO ₂ /kg(FFM)/min])	50.0 ± 7.4	54.0 ± 6.4
HR _{max} (bpm)	190.1 ± 10.4	192.6 ± 9.7
Fasting Glucose (mmol/L)	4.7 ± 0.4	4.7 ± 0.2
Fasting Insulin (μU/mL)	19.1 ± 15.4	16.1 ± 10.9
HOMA-IR [†]	4.0 ± 3.4	3.4 ± 2.4
Fasting TAG (mg/dL)	75.8 ± 28.0*	114.5 ± 56.0
Total Cholesterol (mg/dL)	155.8 ± 36.7	158.2 ± 30.0
LDL Cholesterol (mg/dL)	79.72 ± 36.0	92.13 ± 20.3
HDL Cholesterol (mg/dL)	43.1 ± 12.4	41.67 ± 8.8
Systolic Blood Pressure (mmHg)	119.2 ± 9.7*	111.0 ± 9.3
Diastolic Blood Pressure (mmHg)	64.6 ± 5.4	61.9 ± 6.7
HRR-2 (bpm)	48.6 ± 10.8	52.8 ± 8.1
ERI (bpm·mLO ₂ ·kg ⁻¹ ·min ⁻¹)	29.8 ± 6.4*	24.1 ± 3.1

* $P < 0.05$, Black vs. White.

Values are mean ± SD.

[†] Black (N = 47).

BMI, Body Mass Index; VO_{2Peak}, Maximal Oxygen Consumption; VO_{2FFM}, Maximal Oxygen Consumption per kg Fat-Free Mass; HR, Heart Rate; HOMA-IR, Homeostasis Model Assessment for Insulin Resistance; TAG, Triglycerides; LDL, Low Density Lipoprotein; HDL, High Density Lipoprotein; HRR-2, Heart Rate Recovery at 2 min post exercise; ERI, Exercise Recovery Index.

Table 2

Pearson Product-Moment Correlations to ERI

Variable Coefficients (r)	Black	White
Percent Fat (%FAT)	0.275	0.095
logHOMA-IR	0.162	0.215
Fasting Insulin (μ U/mL)	0.102	0.094
Fasting TAG (mg/dL)	0.154	0.282
Systolic Blood Pressure (mmHg)	-0.138	0.204
CVF (VO_{2FFM} [mL/kg(FFM)/min])	-0.347*	0.026

* $P < 0.05$; ERI, Exercise Recovery Index; HOMA-IR, Homeostasis Model Assessment for Insulin Resistance; TAG, Triglycerides; CVF, Cardiovascular Fitness; VO_{2FFM} , Maximal Oxygen Consumption per kg Fat-Free Mass

Table IIIMultiple Linear-Regression for ERI as the Dependent Variable Black Females ($R^2 = 0.273$, $P = 0.037$)

Variable Coefficient (β)	Standardized	Semi-Partial (r)
Percent Fat (%FAT)*	0.360	0.326
logHOMA-IR	0.291	0.159
Fasting Insulin (μ U/mL)	-0.134	-0.075
Fasting TAG (mg/dL)	0.001	0.001
Systolic Blood Pressure (mmHg)	-0.230	-0.214
CVF (VO_{2FFM} [mL O_2 /kg(FFM)/min])*	0.313	-0.310