

Racial Differences Exist in Cardiovascular Parasympathetic Modulation Following Maximal Exercise

Michael R. Esco, Ph.D.

Michele S. Olson, Ph.D.

Henry N. Williford, Ed.D.

*Auburn University Montgomery, Montgomery, Alabama
Department of Physical Education and Exercise Science*

KEY WORDS: African American, Heart rate variability, Heart rate recovery, Exercise, Autonomic

ABSTRACT

The purpose of this study was to determine the difference in heart rate recovery (HRR) and heart rate variability (HRV) after a maximal treadmill test between white and black men. Sixty white ($n = 30$) and black ($n = 30$) college-age men volunteered as study subjects. All subjects performed a maximal graded exercise test on a treadmill. HRR was determined from the difference between HR recorded at maximal exercise and the HR recorded at 1-minute (HRR1) and 2-minutes (HRR2) post-exercise. HRV was analyzed in the supine position 5 minutes before and between 25 to 30 minutes after the exercise protocol. The HRV was examined as the standard deviation of all successful R-R intervals (SDNN), normalized high frequency power (HFnu), and normalized low frequency to high frequency ratio (LFnu:HFnu). No difference was found in pre-exercise HRV or HRR1. However, racial differences were revealed in the following parameters: HRR 2 = 39.93 ± 8.87 beats.min⁻¹ in whites and 46.16 ± 8.95 beats.min⁻¹ in blacks ($p < .01$), post-exercise SDNN = 35.55 ± 16.58 ms in whites

and 48.37 ± 22.51 ms in blacks ($p < .05$), post-exercise HFnu = 16.60 ± 12.24 ms² in whites and 27.62 ± 15.52 ms² in blacks ($p < .01$), and post-exercise LFnu:HFnu = 9.48 ± 6.51 in whites and 4.85 ± 5.01 in blacks ($p < .01$). The results of this study suggest that college-age black men have a faster parasympathetic rebound following maximal exercise compared to young white men.

INTRODUCTION

At the onset of exercise, cardiac output rises with progressively graded work rates because of a prompt decrease in parasympathetic activity followed by an increase in sympathetic outflow.^{1,2} After exercise, heart rate quickly begins to return towards resting values. This is primarily due to parasympathetic reactivation followed by diminishing sympathetic outflow.³ The assessment of heart rate recovery (HRR) immediately after exercise has become a useful method for analyzing cardiac autonomic modulation.⁴ Lower/abnormal HRR is widely accepted as a predictor of premature mortality independently of other markers of cardiovascular disease.^{4,5}

Heart rate variability (HRV) can be defined as the oscillations that occur between adjacent QRS complexes, or more specifically the distance between successive R to

Table 1: Descriptive Statistics

| | Whites (n = 30) | Blacks (n = 30) |
|----------------------------------|------------------------|------------------------|
| Age (yrs) | 22.30 ± 3.14 | 23.20 ± 3.23 |
| Height (cm) | 181.78 ± 6.74 | 182.39 ± 8.42 |
| Weight (kg) | 82.30 ± 10.22 | 85.67 ± 11.02 |
| BMI (kg/m ²) | 25.00 ± 2.34 | 25.70 ± 3.00 |
| BF% | 10.20 ± 4.40 | 9.91 ± 5.00 |
| VO ₂ max (ml/kg/min)* | 48.13 ± 7.99 | 42.60 ± 6.47 |
| SBP (mmHg) | 122.73 ± 10.45 | 123.30 ± 8.90 |
| DBP (mmHg)* | 71.13 ± 8.06 | 77.73 ± 8.06 |
| RHR (beats/min) | 60.53 ± 7.60 | 60.70 ± 9.26 |

All values are reported as means ± standard deviations. BMI = body mass index, BF% = body fat percentage, SBP = systolic blood pressure, DBP = diastolic blood pressure, RHR = resting heart rate, * racial differences exist, $p < 0.05$.

R intervals, as derived from an electrocardiogram (ECG). Examining HRV is also a valuable procedure for analyzing autonomic influence.⁶ Depressed HRV has been linked to abnormal cardiovascular autonomic modulation and is a valuable predictor of fatal and non-fatal cardiovascular events in clinical and asymptomatic populations.⁷⁻⁹

Relative to white individuals, blacks suffer from greater cardiovascular disorders and death at any age and have higher rates of hypertension, which can manifest itself in early ages.^{10,11,12,13} Individuals who are at higher prevalence of cardiovascular disease and hypertension have been shown to have a dysfunctional autonomic nervous system.^{4,5,7,14} Therefore, it has been suggested that differences exist in cardiovascular autonomic modulation between whites and blacks populations.^{15,16,17}

While differences have been revealed between white and black individuals in HRV and HRR, the extent of these differences have yet to be fully elucidated. Some studies suggest that whites have a superior HRV profile at rest compared to blacks, while others report either opposite findings or no difference between the two races.^{15,16,18,19, 17,20,21,22,23} In addition, few studies are available to suggest a racial difference in post-exercise cardiovascular autonomic function.^{24,25} The data regarding

the effects of race on cardiac autonomic control are mixed and more research in this area is needed. Furthermore, there are no studies available to address a racial difference in post-exercise HRV. The primary aim of this investigation was to determine the difference in heart rate recovery (HRR) and heart rate variability (HRV) after a maximal treadmill test between white and black college-age men.

SUBJECTS AND METHODS

Subjects

Sixty white (n = 30) and black (n = 30) men participated in this study. Each subject provided informed consent in writing and the study was approved by the Institutional Review Board for Human Participants. Descriptive statistics are represented in Table 1. Subjects reported ethnicity/race as either Non-hispanic/White or Non-hispanic/Black over three generations. All participants were non-smokers and had no history or clinical signs of cardiovascular or pulmonary diseases. Subjects were excluded if they were hypertensive (i.e., blood pressure > 140/90 mmHg), currently taking any prescribed or over-the-counter medications, currently smoked or had quit smoking within 6 months, and/or displayed abnormal ECG patterns. All data was collected for each subject on one visit to the lab during one of two 2-hour time slots: 7:00 to 9:00am

or 9:00 to 11:00am on any day of the week. The subjects were instructed to not consume alcoholic beverages or sympathomimetic agents 24 hours before the test and to not eat at least 3 hours before the test.

Anthropometric Variables

Height was measured with a wall mounted stadiometer (SECA) and rounded to the nearest 0.1 cm. Body weight was measured with a digital scale (TANITA BWB-800A) and rounded to the nearest 0.1 kg. Body mass index (BMI) was calculated as height divided by weight squared (kg/m²). Body fat percentage (BF%) was estimated via the 7-site skinfold method as described by written standards.²⁶

Maximal Graded Exercise Test

All subjects performed a maximal graded exercise test on a Parker Treadmill (Parker Co., Opelika, AL). The Bruce protocol was employed during each exercise test. The protocol incorporated a series of 3-minute stages with progressively increased workloads (i.e., speed and grade) until the subjects met the criteria for VO₂max which was considered to be achieved if two of the four following criteria occur: a plateau in VO₂ with increasing work rate; RER > 1.10; heart rate within 10 beats of age predicted maximum (220 – age); or volitional fatigue. During the test, an Applied Electrochemistry (AM-ETEK, Pittsburg, PA) metabolic analyzer was used to determine the concentration of expired gases (oxygen and carbon dioxide) via a continuous manner at the mouth with a pneumotach. All data was recorded on a personal computer (PC) every 30-seconds using Turbofit 5.06 software (VACUMED, Ventura, CA). Once the subjects achieved VO₂max, a 3-minute cool-down period was allowed. During this time, the speed was decreased to 2.5 mph at a 1.5% grade.

Heart rate recovery

Heart rate was examined before, during, and after the maximal exercise test with an ECG. A modified lead II configuration using three Ag/AgCl electrodes (BIOPAC ES509) was used for the ECG recordings. The electrodes were interfaced with a Biopac MP100

data acquisition system (Goletta, CA). All data was stored in a designated PC for analysis. The ECG sampling frequency was set at 1000 Hz.

To examine HRR, the heart rate that corresponded to VO₂max (i.e., the maximal heart rate[MHR]), the heart rate at 1-minute cool-down (i.e., HR1) and the heart rate at 2-minutes cool-down (HR2) were recorded. HRR was determined from the difference between MHR and the HR recorded at 1-minute (HRR1) and 2-minutes (HRR2) post-exercise.

Heart rate variability

Before exercise, each subject assumed a supine position. The subjects remained in this position for 10 minutes prior to the exercise test. Following the cool-down period of exercise, subjects once again assumed a supine position for 30 minutes. During the pre- and post-exercise ECG recordings, all external stimuli (e.g., external noise) were excluded and the laboratory was dimly lit and climate controlled.

Heart rate variability was analyzed during the last 5 minutes of the pre-exercise ECG recording. After exercise, HRV was again assessed at 25 to 30 minutes post-exercise. During each 5-minute ECG recording that was used for HRV analysis, breathing was paced with a metronome at 12 breaths.min⁻¹. Each 5-minute ECG recording was visually inspected. Any ectopic/non-sinus beats were removed and replaced by the adjacent normal R-R interval. If three or more ectopic beats were found within any ECG segment, the reading was excluded from analysis.

Heart rate variability was analyzed in the time and frequency domains with the use of Nevrokard HRV software (version 10.2; MediaStar, Lubljana, Slovenia). For time domain analysis, the standard deviation of all successful R-R intervals (SDNN) was recorded. For the frequency domain method, a power spectral analysis was completed on the ECG by applying a Hanning window and a fast Fourier transformation to the R-R intervals. In the frequency domain, HRV

Table 2. Pre- and post-exercise HRV parameters between groups.

| | Whites (n = 30) | Blacks (n = 30) |
|----------------------------|-----------------|-----------------|
| Pre-exercise | | |
| SDNN (ms) | 97.47 ± 40.38 | 84.63 ± 38.19 |
| HFnu (ms ²) | 34.79 ± 13.50 | 42.20 ± 16.47 |
| LF:HF | 2.43 ± 1.97 | 1.68 ± 1.37 |
| Post-exercise | | |
| SDNN (ms)* | 35.55 ± 16.58 | 48.37 ± 22.51 |
| HFnu (ms ²)* * | 16.60 ± 12.24 | 27.62 ± 15.52 |
| LF:HF** | 9.48 ± 6.51 | 4.85 ± 5.01 |

was separated into high frequency (HF)
*All values are reported as means ± standard deviations. SDNN = standard deviation of all R-to-R intervals, HFnu = normalized high frequency power, LF:HF = low frequency to high frequency power ratio. Racial differences exist: *p < .05, **p < .01.*

power (0.15-0.40 Hz) and low frequency (LF) power (0.04 – 0.15 Hz). Both of these values were normalized (HFnu, LFnu) to account for the influence total power of the entire wave and the very low frequency (VLF) band (0.0033 – 0.04 Hz). Both HFnu and the ratio of LFnu to HFnu (i.e., LFnu:HFnu) were recorded and analyzed during the pre- and post-exercise 5-minute intervals.

Statistical Analysis

All statistical analysis was completed using SPSS version 16.0. A 2 (group) by 3 (time – MHR, HR1, HR2) repeated measures analysis of variance (ANOVA) was utilized to determine a group by time effect for the change in HR after exercise. A Bonferroni Post hoc analysis was used as a follow-up to further examine group differences in the post-exercise heart rate values. Repeated measures ANOVA procedures were also utilized to determine significant differences between the mean values of HRR1 and HRR2 as well as significant differences between the pre- and post-exercise HRV parameters between the two groups. Follow-up analysis of covariance (ANCOVA) procedures were utilized to determine if any significant differences in the HRR or HRV parameters remained when controlling for potential covariates. A priori statistical significance for all tests was set at $p < 0.05$.

RESULTS

All subjects that agreed to participate completed the testing procedures. Table 1 represents descriptive characteristics. The white subjects had significantly lower DBP and significantly higher VO₂max ($p < 0.05$, refer to Table 1). A racial difference in the change in HR after exercise was revealed (Figure 1; $p < 0.05$). During the 2-minute recovery period, blacks had a faster trend for recovery compared to whites. There was no significant difference in MHR and HR1 between the white (MHR = 190.53 ± 9.01, HR1 = 170.70 ± 11.91) and black (MHR = 187.67 ± 8.65, HR1 = 166.87 ± 11.59) subjects. However, the white subjects had significantly higher HR2 compared to the black subjects (150.43 ± 13.89 versus 141.73 ± 13.48, respectively, $p < 0.01$). Furthermore, there was not a difference in HRR1 (18.63 ± 6.27 beats/min for whites and 20.73 ± 6.17 beats/min for blacks), but the white subjects had lower HRR2 compared to the black subjects (39.93 ± 8.87 and 46.16 /- 8.95 beats/min, respectively, $p < 0.01$, Figure 2).

There were no significant differences revealed for the resting HRV parameters between the black and white subjects (Table 2). However, during the post-exercise period, the white subjects had significantly lower SDNN ($p < 0.05$) and HFnu ($p < 0.01$) and significantly higher LFnu:HFnu ($p < 0.01$) compared to the blacks (Table 2 and

Figure 1 - Heart rate responses post-maximal exercise between groups

*Significantly different between groups, $p < 0.01$.

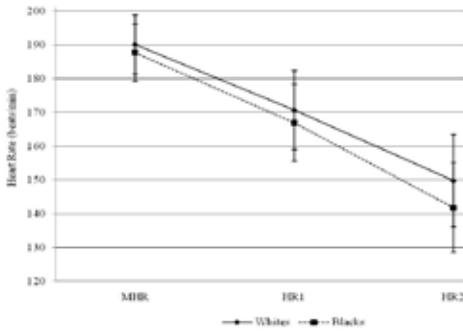


Figure 3 – SDNN (ms) values pre- and post-exercise for white and black subjects.

*Significantly different between groups, $p < 0.05$.

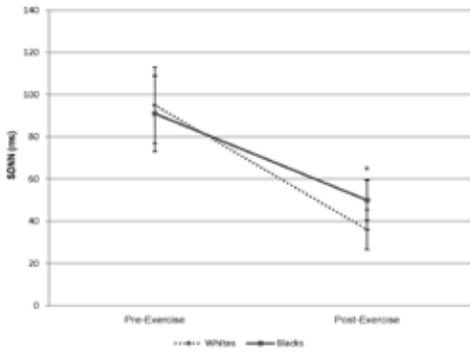


Figure 2 – HRR1 and HRR2 values for white and black subjects.

*Significantly different between groups, $p < 0.01$.

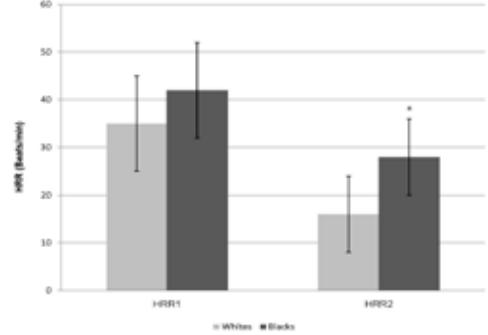


Figure 4 - HFnu values pre- and post-exercise for white and black subjects

*Significantly different between groups, $p < 0.01$.

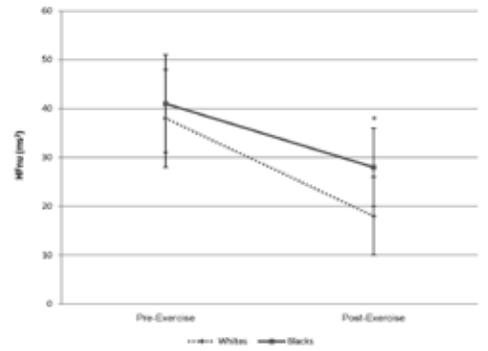
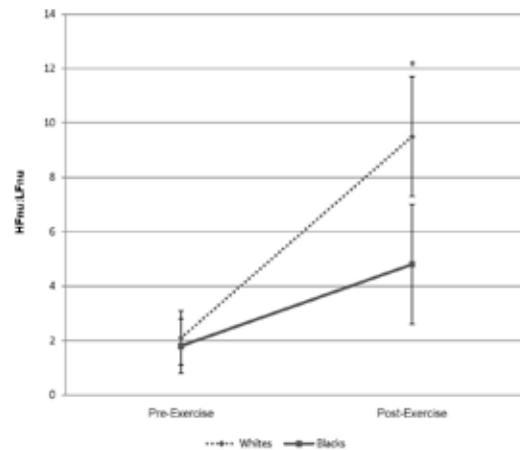


Figure 5 - LFnu:HFnu values pre- and post-exercise for white and black subjects

*Significantly different between groups, $p < 0.01$.



Figures 3 - 5). An ANCOVA procedure was utilized to control for the differences in the potential confounders (i.e., VO₂max and DBP). After controlling for these covariates, the racial differences remained for HR₂ ($p < 0.001$), HRR₂ ($p < 0.001$), and post-exercise SDNN ($p < 0.05$), HFnu ($p < 0.05$), and LFnu:HFnu ($p < 0.05$).

DISCUSSION

The major finding of this investigation was that college-age black men had faster HRR after maximal exercise compared to the white men. The black men also exhibited significantly higher SDNN and HFnu and significantly lower LF:HF ratio during the 25 to 30 minutes post-exercise HRV recording. Similar findings have been previously reported. In a large study, black men had greater HRR compared to white men and women.²⁴ In addition, Heffernan et al.²⁵ revealed that resistance training induced improvements in HRR were maintained after 2 weeks of detraining in young black men but returned to baseline in the white men. These findings are indicative of a faster parasympathetic rebound following maximal exercise in black versus white men.

The results of this study may be linked to the racial differences found in parasympathetic-sympathetic balance in response to various physiological stressors. It has also been reported elsewhere that blacks have a faster return of heart rate immediately after exercise.²⁴ In addition, compared to whites, blacks have been shown to have lower cardiovascular response after a head-up tilt test.²⁷ While it has been suggested that young blacks have higher resting HRV, they have also been shown to have higher HRV in response to isometric handgrip, cold pressor test, and the valsalva maneuver.¹⁶ However, blacks have an increase in total peripheral resistance during exercise.²⁸ Black children have also been shown to have an increase in peripheral resistance in response to a cold face stimulus.²⁹ In addition, hypertensive black adults had greater vascular tone than hypertensive whites in response to a mental arithmetic task, a simulated public speak-

ing task, and a forehead cold pressor test.³⁰ Thus, it appears that racial differences exist in adrenergic response to stress, with whites having more pronounced sympathetic stimulation to the heart, and blacks having higher sympathetic activation of peripheral vascular resistance.

The study results may also be related to racial differences found in the pressor reflex. When the pressor reflex is activated during exercise, marked increases in heart rate and arterial pressure occur.³¹ However, when the pressor reflex is activated during post-exercise recovery there is a loss of central command, an increase in parasympathetic activity to the heart, and a decrease in heart rate.³¹ Young black men have been shown to have an increased pressor response during dynamic exercise compared to age-matched white men.²⁸ Perhaps the pressor reflex is activated to a greater degree during recovery in the black subjects which would result in an increase parasympathetic activity during this time (i.e., a faster HRR, higher SDNN and HFnu, and lower LFnu:HFnu ratio). The authors can only speculate the possibility of this relationship, because the pressor reflex was not analyzed in this study.

Analyses of HRV and HRR have grown in popularity as non-invasive tools to assess cardiac autonomic function.^{3,4,6} Both have valuable clinical implications. For example, a number of large studies suggest that delayed HRR and blunted HRV are independent predictors of premature mortality and sudden cardiac death.^{3,4,6-9} On average, blacks are at increased risk of premature cardiovascular disorders and sudden death compared to whites.^{10,12} In addition, blacks are shown to have higher rates of hypertension at earlier ages and lower cardiovascular fitness levels compared to whites, which are both linked to lower HRR and HRV.^{3,12-14,32,33} The blacks in this study had higher resting blood pressures and lower aerobic fitness values, despite having superior cardiac-autonomic profiles. Thus, it is quite difficult to consider these paradoxical findings in relation to clinical significance. However, it has

been suggested that the higher prevalence of hypertension in blacks may be related to issues within the peripheral vessels (e.g., vascular resistance) rather than cardiac-autonomic control.^{16,19,33}

There are some limitations with the present investigation that concern the chosen sample. This study was limited to only black and white college-aged men. Perhaps the difference in resting HRV between the racial groups is dependent on age, with HRV being greater in younger blacks but lower in older blacks compared to age-matched whites.^{15,21} To determine if similar findings exist in HRR and post-exercise HRV, future research involving older subjects is needed. These findings cannot be extrapolated to different races, women, or subjects of older ages. Also, overall physical activity has been shown to exert an influence on autonomic influence of the heart.²⁴ Daily physical activity was not accounted for in this study. Last, due to the nature of this study, a causal relationship for the findings cannot be determined.

In conclusion, black men appear to have a faster HRR and a superior HRV after maximal exercise which indicates a quicker parasympathetic rebound compared to white men. A complete explanation for these findings cannot be fully clarified with this investigation. However, these findings do show the importance of accounting for racial characteristics of participants when studying the heart rate recovery pattern post-maximal exercise.

REFERENCES

1. Bruerer HM, Skyschally A, Schulz R, Martin C, Wehr M, Heusch G. Heart rate variability and circulating catecholamine concentrations during steady state exercise in healthy volunteers. *Br Heart J* 1993; 70: 144 – 149.
2. Goldsmith RL, Bloomfeld DM, Rosenwinkel ET. Exercise and autonomic function. *Coron Artery Dis* 2000; 11: 129 – 135.
3. Imai K, Sato H, Hori M, Kusuoka H, Ozaki H, Yokoyama H, Takeda H, Inoue M, Kamada T. 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 – 1535.
4. 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: 1351 – 1357.
5. Jouven X, Empana JP, Schwartz PJ, Desnos M, Courbon D, Ducimetiere P. Heart-rate profile during exercise as a predictor of sudden death. *N Engl J Med* 2005; 352: 1951 – 1958.
6. Malik M. Task Force of the European Society of Cardiology and The North American Society of Pacing and Electrophysiology. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. *Circulation* 1996; 93: 1043 – 1065.
7. Kiviniemi AM, Tulppo MP, Wichterle D, Hautala AJ, Tiinanen S, Seppanen T, Makikallio TH, Huikuri HV. Novel spectral indexes of heart rate variability as predictors of sudden and non-sudden cardiac death after an acute myocardial infarction. *Ann Med* 2007; 39: 54 – 62.
8. Ponikowski P, Anker SD, Chua TP, Szelemej R, Piepoli M, Adamopoulos S, Webb-Peploe K, Harrington D, Banasiak W, Wrabec K, Coats AJ. Depressed heart rate variability as an independent predictor of death in chronic congestive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. *Am J Cardiol* 1997; 79: 1645 – 1650.
9. Tsuji H, Larson MG, Venditti FJ. Impact of reduced heart rate variability on risk of cardiac events. The Framingham Heart Study. *Circulation* 1996; 94: 2850 – 2855.
10. Budoff MJ, Nasir K, Mao S, Tseng PH, Chau A, Liu ST, Flores F, Blumenthal RS. Ethnic differences of the presence and severity of coronary atherosclerosis. *Atherosclerosis* 2003; 187: 343 – 350.
11. Gillium RF. Trends in acute myocardial infarction and coronary heart disease in the United States. *J Am Coll Cardiol* 1993; 23: 1233 – 1237.
12. Kramer H, Han C, Post W, Goff D, Diez-Rouz A, Cooper R, Jinagouda S, Shea S. Racial/ethnic differences in hypertension and hypertension treatment and control in the multi-ethnic study of atherosclerosis (MESA). *Am J Hypertens* 2003; 17: 963 – 970.
13. Stein C, Lang C, Hong-Guang X, Wood A. Hypertension in black people: Study of specific genotypes and phenotypes will provide a greater understanding of inter individual and inter-ethnic variability in blood pressure regulation than studies based on race. *Pharmacogenetics* 2001; 11: 95 – 110.
14. Neumann SA, Jennings JR, Muldoon MF, Manuck SB. White-coat hypertension and autonomic nervous system dysregulation. *Am J Hypertens* 2005; 18: 584 – 588.
15. Gutin B, Howe CA, 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 – 1863.
16. Urbina EM, Bao W, Pickoff AS, Berenson GS. Ethnic (black-white) contrast in heart rate variability during cardiovascular reactivity testing in male adolescents with high and low blood pressure. *Am*

- J Hypertens* 1998; 11: 196 – 202.
17. Zion AS, Bond V, Adams RG, Williams D, Fullilove RE, Sloan RP, Bartels MN, Downey JA, De Meersman RE. Low arterial compliance in young African American males. *Am J Physiol Heart Circ Physiol* 2003; 265: H457 – H462.
 18. Liao D, Barnes RW, Chambless LE, Simpson RJ, Sorlie P, Heiss G. Age, race, and sex differences in autonomic cardiac function measured by spectral analysis of heart rate variability – the ARIC study. *Am J Cardiol* 1995; 76: 906 – 912.
 19. 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 – 1172.
 20. Choi J, Hong S, Nelsen R, Bardwell WA, Natarajan L, Schubert C, Dimsdale JE. Age and ethnicity differences in short-term heart-rate variability. *Psychosom Med* 2006; 68: 421 – 426.
 21. Lampert R, Ichovics J, Horwitz R, Forrester L. Depressed autonomic nervous system function in African American and individuals of lower social class: A potential mechanism of race-and class-related disparities in health outcomes. *Am Heart J* 2005; 150: 153 – 160.
 22. Sloan RP, Huang MH, McCreath H, Sidney S, Liu K, Dale-Williams O, Seeman T. Cardiac autonomic control and the effects of age, race, and sex: the CARDIA. *Auton Neurosci* 2008; 139: 78-85.
 23. Franke WD, Lee K, Buchanan DB, Hernandez JP. Blacks and whites differ in responses, but not tolerance, to orthostatic stress. *Clin Auton Res* 2004; 14: 19 – 25.
 24. Carnethon MR, Jacobs DR, Sidney S, Sternfeld B, Gidding SS, Shoushtari C, Liu K. A longitudinal study of physical activity and heart rate recovery: CARDIA 1987-1993. *Med Sci Sports Exerc* 2005; 37: 606 – 612.
 25. Heffernan KS, Jae SY, Vieira VJ, Iwamoto GA, Wilund KR, Woods JA, Fernhall B. C-reactive protein and cardiac vagal activity following resistance exercise training in young African American and white men. *Am J Physiol Regul Integr Comp Physiol* 2009; 296: R1098 – R1105.
 26. American College of Sports Medicine. ACSM's Guidelines for Exercise Testing and Prescription. 7th ed. 2007; Philadelphia (PA): Lippincott Williams and Williams.
 27. Venter CP, Joubert PH, Strydom WJ. The relevance of ethnic differences in hemodynamic responses to the head-up tilt manoeuvre to clinical pharmacological investigations. *J Cardiovasc Pharmacol* 1985; 7: 1009 – 1010.
 28. Walker AJ, Bassett DR, Duey WJ, Howley ET, Bond V, Torok DJ, Mancuso P. Cardiovascular and plasma catecholamine responses to exercise in blacks and whites. *Hypertension* 1992; 20: 542 – 548.
 29. Treiber FA, Musante L, Braden D, Arensman F, Strong WB, Levy M, Leverett S. Racial differences in hemodynamic responses to the cold face stimulus in children and adults. *Psychosom Med* 1990; 52: 286 – 296.
 30. Sherwood A, May CW, Siegel WC, Blumenthal JA. Ethnic differences in hemodynamic responses to stress in hypertensive men and women. *Am J Hypertens* 1995; 8: 552 – 557.
 31. O'Leary. Autonomic mechanisms of muscle metaboreflex control of heart rate. *J Appl Physiol* 1993; 74: 1748 – 1754.
 32. Gillium RF. Sudden cardiac death in Hispanic Americans and African Americans. *Am J Public Health* 1997; 87: 1461 – 1466.
 33. Lavie CJ, Kuruvanka T, Milani RV, Prasad A, Ventura HO. Exercise capacity in adult African Americans referred for exercise stress testing. *Chest* 2004; 126: 1962 – 1968.