Published in final edited form as:

Appl Physiol Nutr Metab. 2016 May ; 41(5): 484–490. doi:10.1139/apnm-2015-0512.

Systolic blood pressure response after high-intensity interval exercise is independently related to decreased small arterial elasticity in normotensive African American women

Stephen J. Carter, PhD^{1,3}, TaShauna U. Goldsby, PhD^{3,4}, Gordon Fisher, PhD^{1,2}, Eric P. Plaisance, PhD¹, Barbara A. Gower, PhD², Stephen P. Glasser, MD⁵, and Gary R. Hunter, PhD^{1,2}

TaShauna U. Goldsby: tgoldsby@uab.edu; Gordon Fisher: grdnfs@uab.edu; Eric P. Plaisance: plaise@uab.edu; Barbara A. Gower: bgower@uab.edu; Stephen P. Glasser: sglasser@uab.edu; Gary R. Hunter: ghunter@uab.edu

¹Department of Human Studies, University of Alabama at Birmingham; Birmingham, Alabama, 35233, USA.

²Department of Nutrition Sciences, University of Alabama at Birmingham; Birmingham, Alabama, 35233, USA.

³Nutrition Obesity Research Center, University of Alabama at Birmingham; Birmingham, Alabama, 35233, USA.

⁴Office of Energetics, University of Alabama at Birmingham; Birmingham, Alabama, 35233, USA.

⁵Department of Preventive Medicine, University of Alabama at Birmingham; Birmingham, Alabama, 35233, USA.

Abstract

Aerobic exercise transiently lowers blood pressure. However, limited research has concurrently evaluated blood pressure and small arterial elasticity (SAE), an index of endothelial function, among African American (AA) and European American (EA) women the morning after (i.e., ≈ 22 hours later) acute bouts of moderate-intensity continuous (MIC) and high-intensity interval (HII) exercise matched for total work. Due to greater gradients of shear stress, it was hypothesized that HII exercise would elicit a greater reduction in systolic blood pressure (SBP) compared to MIC exercise. After baseline, twenty-two AA and EA women initiated aerobic exercise training 3x/week. Beginning at week 8, three follow-up assessments were conducted over the next 8 weeks at random to measure resting blood pressure and SAE. In total all participants completed 16 weeks of training. Follow-up evaluations were made: 1) in the Trained State (TS; 8–16 weeks of aerobic training); 2) ≈ 22 hours after an acute bout of MIC exercise; and 3) ≈ 22 hours after an acute bout of HII exercise. Among AAs, the acute bout of HII exercise incited a significant increase in SBP (mm Hg) (TS, 121 ± 14 vs. HII, 128 ± 14 ; $p=0.01$) whereas responses (TS, 116 ± 12 vs. HII, 113 ± 9 ; $p=0.34$) did not differ in EAs. After adjusting for race, changes in SAE were associated (partial $r=-0.533$; $p=0.01$) with changes in SBP following HII exercise. These data demonstrate an acute,

Corresponding Author: Stephen J. Carter, Ph.D., Department of Nutrition Sciences, 1675 University Blvd, Webb Building #248, University of Alabama at Birmingham, Birmingham, Alabama 35233-2432, Tel: 205.975.0269; Fax: 205.975.8040, carters@uab.edu.

Disclosures. We declare no conflicts of interest.

unaccustomed bout of HII exercise produces physiological perturbations resulting in a significant increase in SBP that are independently associated with decreased SAE among AA women, but not EA women.

Keywords

aerobic training; arterial stiffness; endothelial function; health disparity; hypertension; race

INTRODUCTION

While cardiovascular disease (CVD) mortality in the United States has steadily declined over recent decades (Ford and Capewell 2011), it remains a pervasive health crisis throughout modern society. Despite this notable shift, African Americans (AA) continue to exhibit disproportionately higher CVD mortality and morbidity compared to European Americans (EA) (Mensah and Brown 2007). Hypertension is one such morbidity that has remained prevalent among AAs (Liu, Tsilimingras and Paul 2014; Egan, Zhao and Axon 2010). Compared to EAs, hypertension in AAs is characterized by earlier onset and higher mean blood pressures, (Hertz et al. 2005, Go et al. 2014) both of which contribute to an increased burden of hypertension-related health complications (Flack 1995). Furthermore, the collective influence of increased arterial stiffness, (Heffernan, Jae and Fernhall 2007) and peripheral resistance, (Stein et al. 2000) along with reduced vasodilator potential (Pienaar et al. 2014; Ozkor et al. 2014) may directly heighten the risk for end-organ damage in AAs (Chaturvedi et al. 2004). Though hypertension is considered a modifiable risk factor, (Centers for Disease Control and Prevention 2011) research has shown that AAs and EAs respond differentially to antihypertensive interventions (Cushman et al. 2000). Taken together, a singular approach to exercise training may contribute to divergent cardiovascular-related responses among AAs and EAs. Nevertheless, higher levels of physical activity and cardiorespiratory fitness are believed to lower the risk of hypertension, (Chase et al. 2009) thus underscoring the importance of habitual exercise.

Although many studies have demonstrated the efficacy of exercise training to improve various parameters of arterial health, (Ciolac et al. 2010; Ciolac et al. 2011) a degree of uncertainty exists with regard to optimal exercise dose and intensity. Moderate-intensity continuous (MIC) exercise has been traditionally recommended for hypertension prevention and treatment (Haskell et al. 2007). Alternatively, high-intensity interval (HII) exercise has become an attractive option, due in-part to the oscillating periods of vigorous activity followed by rest and overall time-savings. Research has also shown greater improvements in cardiorespiratory fitness with HII exercise compared to MIC exercise in both healthy (Matsuo et al. 2014; Slordahl et al. 2004) and clinical populations (Tjonna et al. 2008). Wisloff and colleagues (Wisloff et al. 2007) previously showed that HII exercise significantly improved endothelial function whereas MIC exercise did not. It was believed that HII exercise may have produced greater gradients of shear stress, and thus conferring superior cellular adaptations. Accordingly, it seems reasonable that HII exercise may be a more effective modality to transiently lower blood pressure and enhance small arterial elasticity (SAE), shown to be a reliable index of endothelial function (Grey et al. 2003;

McVeigh et al. 2001), compared to MIC exercise. However, due to the known cardiovascular-related differences between AAs and EAs further research is needed.

Therefore, the purpose of this study was to evaluate resting blood pressure and SAE by local pulse contour analysis the morning after (i.e., ≈ 22 hours later) a single exercise session of MIC and HII, matched for total work, in aerobically trained AA and EA women. It was hypothesized that HII exercise would produce a greater reduction in blood pressure among participants, independent of race, and these changes would be associated with improved SAE.

METHODS

Study Participants

Twenty-two AA ($n = 11$) and EA ($n = 11$) women between 20–40 years of age volunteered to for this study. Participants reported normal menstrual cycles and were not taking oral contraceptives or any medications known to influence blood pressure, glucose and/or lipid metabolism. Additional inclusion criteria were: i) normotensive; ii) non-smoker; iii) sedentary as defined by participating in any exercise-related activities $< 1\times$ per week; and iv) normoglycemic as evaluated by postprandial glucose response to a 75-g oral glucose tolerance test. All participants provided written informed consent prior to inclusion. Study procedures were approved by the Institutional Review Board at the University of Alabama at Birmingham and conformed to the guidelines set forth by the Declaration of Helsinki.

Procedures

After initial screening and baseline measures, all participants completed 16 weeks of supervised, aerobic training $3\times$ per week on a stationary cycle ergometer. Beginning at week 8, participants were evaluated on 3 additional occasions (i.e., weeks 12 and 16), each separated by one month during the follicular phase of the menstrual cycle (Figure 1). In random order under standardized conditions (i.e., same time of day, 0700), after an overnight stay in a room calorimeter, follow-up treatments were: 1) in the “Trained State,” having completed 8–16 weeks of aerobic training and no exercise in the room calorimeter; 2) ≈ 22 hours after acute MIC exercise on a stationary cycle ergometer at 50% peak VO_2 ; and 3) ≈ 22 hours after acute HII exercise on a stationary cycle ergometer at 84% peak VO_2 . Before the Trained State assessment, participants abstained from any exercise and/or vigorous physical activity 48 hours prior to testing for the purpose of attenuating the residual effects of any previous exercise. Hence, the purpose was to compare the acute physiological responses of MIC and HII exercise to the ‘new trained baseline,’ since participants had been habitually exercise training for a minimum of 8 weeks. Food was provided to participants on the 2 days preceding testing procedures while initial energy needs were estimated using the Harris-Benedict equation (Harris and Benedict 1918). Diets were prepared by the local Clinical Research Unit consisting of $\approx 60\%$ energy as carbohydrates, $\approx 25\%$ energy as fat, and $\approx 15\%$ energy as protein. Dietary sodium and the ratio between polyunsaturated: saturated fats were held constant to reduce the potential confounding effects of thermic processes.

Peak Aerobic Capacity

After an initial warm-up, participants completed a graded exercise test on a stationary cycle ergometer to measure peak oxygen uptake ($\text{VO}_{2\text{peak}}$) as determined by as the highest level reached in the final stage of exercise. Power output began at 25 watts and increased further by 25 watts every 2 minutes until participants reached volitional exhaustion. Oxygen uptake, ventilation, and respiratory exchange ratio were determined by indirect calorimetry using a MAX-II metabolic cart (Physio-Dyne Instrument Company, Quogue, NY). Heart rate was continuously monitored by Polar® Vantage XL heart rate monitors (Polar Beat, Port Washington, NY).

Overnight Stay in Room Calorimeter

Participants entered at 0800 and spent 23 hours in a whole-room respiration calorimeter ($3.38 \times 2.11 \times 2.58$ m). Oxygen uptake and carbon dioxide production were measured continuously with a magnetopneumatic differential oxygen analyzer (Magnos 4G; Hartmann & Braun, Frankfurt, Germany) and NDIR industrial photometer differential carbon dioxide analyzer (Uras 3G, Hartman & Braun). The calorimeter was calibrated before each participant entered the chamber. The zero calibration was carried out concurrently for both analyzers. The full scale was set at 0–2% for the oxygen analyzer and 0–1% for the carbon dioxide analyzer. The following day participants were awakened at 0630 and physiological measurements (i.e., blood pressure and SAE) were conducted shortly thereafter.

Acute Exercise Sessions in Room Calorimeter

Workload for the MIC exercise was calculated from the $\text{VO}_{2\text{peak}}$ that corresponded to each follow-up assessment using the appropriate metabolic equation for leg ergometry [VO_2 (mL/kg/min) = $2 \times (\text{kg} \times \text{m} \times \text{min}^{-1}) + (3.5 \times \text{body mass})$] in accordance with the American College of Sports Medicine (Kenny et al. 1995). During the MIC exercise, participants cycled continuously for 60 minutes at an intensity of 50% $\text{VO}_{2\text{peak}}$. Fifteen work intervals were performed for 2 minutes 24 seconds with rest intervals of 1 min 43 seconds for the HII exercise. Work was calculated by multiplying 1.66 to the MIC exercise workload with equated to 84% $\text{VO}_{2\text{peak}}$. Total work was identical among the two exercise bouts. Work load was controlled outside the room calorimeter using a Collins electronically braked cycle ergometer (Warren E. Collins, Braintree, MA).

Body Composition

Total and regional body composition (i.e., fat mass and lean mass) was determined by dual-energy X-ray absorptiometry (DEXA; iDXA, GE-Lunar, Madison, WI). Participants wore light clothing and remained supine in compliance with normal testing procedures. Scans were analyzed with ADULT software, LUNAR-DPX-L version 1.33 (GE Medical Systems Lunar).

Resting Blood Pressure and Small Arterial Elasticity

Blood pressure and SAE measures were performed using non-invasive, local pulse contour analysis (HDI/Pulse Wave TM CR-2000, Hypertension Diagnostics Inc., Eagan, MN). Pulse contour analyses from the radial artery are based on a modified Windkessel model, allowing

for the evaluation of microcirculatory vessels (Cohn et al. 1995). In short, participants were seated and remained quiet during the testing procedure. An adult-sized blood pressure cuff was placed around the non-dominant arm. After palpating the radial pulse, a solid-state pressure transducer was fastened over the radial artery of the dominant arm. The sensor was adjusted as needed to achieve the highest relative signal strength. All assessments were performed in triplicate and averaged for analysis. Arterial elasticity is determined by gathering and analyzing a 30-second analog tracing of the radial waveform digitized at 200 samples/sec. As previously described by Cohn and colleagues (Cohn et al. 1995), a beat determination is constructed using a beat-marking algorithm which determines beginning systole, peak systole, onset of diastole, and end diastole during the 30-second period. The beat is incorporated into a parameter estimating algorithm (Hypertension Diagnostics Inc., Eagan, MN) and a modified Windkessel model is used to determine SAE.

Supervised Aerobic Exercise Training

Continuous exercise on a cycle ergometer was performed 3 days per week, beginning with a 3–5 minutes period for warm-up/stretching at each session. During week 1, continuous exercise was maintained for 20 minutes at a maximum heart rate (MHR) of $\approx 67\%$. Over a 16 week period, exercise intensity and duration increased so that by the final week, exercise was consistently maintained for 60 minutes at $\approx 80\%$ MHR. Participants cooled down for 3–5 minutes, following cessation of exercise.

24-hour Fractionated Urinary Catecholamines

24-hour urine samples were treated with hydrochloric acid and glutathione upon collection, and stored at -20°C until assayed. Epinephrine, norepinephrine, and dopamine were measured by high performance liquid chromatography.

Statistical Analyses

Means and standard deviations were calculated then dichotomized by race. Independent t -tests were used to compare differences between races where appropriate. Two (time) by two (race) analysis of variance (ANOVA) with repeated measures for treatment were used to compare differences in variables of interest at baseline and after 8–16 weeks of aerobic training (i.e., Trained State). Separate ANOVAs with repeated measures were performed to compare the acute effects of MIC and HII exercise from the Trained State. Posthoc analyses were performed as needed. Comparisons of change (deltas; Δ) were made from selected variables by determining the differences between HII or MIC and the Trained State. Bivariate correlation analyses were performed to examine relationships in the Trained State among variables and also the deltas following HII exercise. Based on the results of simple correlations, a multiple regression model was used to evaluate the independent effects of race and SAE (occurring from HII exercise) on systolic blood pressure. Collinearity diagnostics for all variables were within acceptable limits and variable inflation factors for all models were < 1.04 . All data were analyzed with SPSS (v22; SPSS, Inc., Chicago, IL, USA). Statistical significance was accepted when p -value ≤ 0.05 for all analyses.

RESULTS

Baseline Assessment

As shown in Table 1, baseline analyses revealed participants were of similar age, height, and body fat percent. Additionally, no between-race differences were found for systolic/diastolic blood pressure, SAE, epinephrine, or norepinephrine. However, AAs weighed more (AA, 81.3 ± 14.9 kg vs. EA, 67.2 ± 10.0 kg; $p = 0.02$) and had a lower $\text{VO}_{2\text{peak}}$ (AA, 35.5 ± 3.5 mL/kg FFM/min vs. EA, 44.6 ± 5.8 mL/kg FFM/min; $p < 0.001$) compared to EAs. On the other hand, dopamine was significantly greater in AAs (AA, 320.6 ± 70.6 $\mu\text{g}/24$ hrs vs. EA, 197.4 ± 96.8 $\mu\text{g}/24$ hrs; $p = 0.04$) at baseline.

Follow-Up Assessments

All participants completed 16 weeks of exercise training. The acute bouts of continuous (MIC) and interval (HII) exercise were well-tolerated and without adverse events. Table 2 contains comparisons of variables of interest including body fat percent, $\text{VO}_{2\text{peak}}$, systolic/diastolic blood pressure, SAE, and catecholamines across the 3 follow-up assessments. No treatment effect (suggesting no change among follow-up assessments) was found for any variable with the exception of SAE. Post hoc analyses indicated a significant increase in SAE ≈ 22 hours after MIC exercise. Similar to the baseline evaluation, there was a significant between-race difference, as AAs had a greater body weight and lower $\text{VO}_{2\text{peak}}$ compared to EAs. However, no treatment by race interactions were observed for any variable apart from systolic blood pressure which, in AAs, was significantly increased ≈ 22 hours after HII exercise.

In Table 3, positive associations were found among the changes [i.e., delta ()] between the Trained State and ≈ 22 hours after HII exercise for race and systolic blood pressure ($r = 0.446$, $p < 0.05$). Negative associations were found between systolic blood pressure and SAE ($r = -0.539$, $p < 0.01$). A linear regression revealed the independent effects of SAE ≈ 22 hours after HII exercise such that a significant association between systolic blood pressure and SAE persisted ($r = -0.533$, $p = 0.013$) after adjusting for race (Table 4). Additionally, race was significantly related ($r = 0.437$, $p = 0.048$) to systolic blood pressure after adjusting for SAE.

DISCUSSION

Though systolic blood pressure and SAE were not different among AA and EA women in the Trained State, after aerobically training for 8–16 weeks, an acute/unaccustomed bout of HII resulted in divergent cardiovascular-related responses between races. Contrary to our hypothesis, resting systolic blood pressure was significantly increased in AAs compared to EAs ≈ 22 hours after HII exercise and these changes were independently associated with a decrease in SAE. As evidenced by the elevated systolic blood pressures, it is possible that the AA women may have been overstressed and not fully recovered from the HII exercise. Since hypertension is prevalent among AAs, (Egan, Zhao and Axon 2010; Liu, Tsilimingras and Paul 2014) these results suggest that an unaccustomed bout of HII exercise may not be the ideal mode of exercise for AA women trying to improve cardiovascular health outcomes.

While aerobic exercise is known to favorably lower blood pressure, these effects are most significant in hypertensive patients (Pescatello and Kulikowich 2001). A meta-analysis by Cornelissen and Fagard (Cornelissen and Fagard 2005) showed that aerobic exercise profoundly lowers systolic and diastolic blood pressure in hypertensive patients by 6.9 and 4.9 mm Hg, respectively, whereas normotensive patients tend to exhibit modest decreases (e.g., 2.4 and 1.6 mm Hg). Similarly, we found that aerobic exercise training 3 times/week for 16 weeks had a minimal effect on resting blood pressure in either cohort of AA or EA women. Since our participants were normotensive at baseline, we reasoned that the training stimulus used in our design may not have been of sufficient volume to elicit a blood pressure-lowering effect.

At high pressure gradients collagen fibers strengthen the arterial wall, whereas, elastin fibers provide elasticity at lower pressures. In the present study, SAE was not significantly different between races at baseline or among the 3 follow-up assessments. Generally, aerobic exercise training lowers cardiovascular risk factors though results demonstrating improved arterial compliance/stiffness have been equivocal. Previous work has revealed increased carotid compliance in young, sedentary male participants following 12 weeks of aerobic training at 70–75% MHR (Kakiyama et al. 2005). On the other hand, Ferrier and colleagues (Ferrier et al. 2001) found that arterial stiffness was resistant to change in elderly hypertensive participants following 8 weeks moderate-intensity (i.e., 65% MHR) exercise 3× per week. Discrepancies in training duration, exercise intensity, age/health disparities, as well as, the methods used to evaluate arterial compliance/stiffness in the previously described studies make it difficult to reconcile the conflicting results. In the present study we can only speculate about why SAE did not improve following our exercise intervention. Though regular aerobic exercise incites a pulsatile ‘stretch’ (i.e., mechanical distension) of the arterial wall (Madden 2010), participants not presenting any co-morbidities may require a greater training volume/frequency to elicit favorable vessel modifications.

While endothelial function was not directly measured, local pulse contour analysis used in the present study provides an assessment of SAE, and thus, an index of microcirculation and endothelial function (McVeigh et al. 2001; Grey et al. 2003). Endothelial nitric oxide synthase is constitutively expressed in the endothelium and obligatory for vascular homeostasis. Though CVD risk factors are known to adversely affect nitric oxide (NO) bioavailability, (Strisciuglio et al. 2014) research has consistently shown improvement in endothelial function with exercise training (Phillips et al. 2015). However, in the present study SAE was significantly decreased in AAs ≈22 hours following an acute bout of HII exercise compared to MIC exercise. Previous work by Campia and colleagues (Campia et al. 2002) demonstrated that AAs have lower microcirculatory NO bioactivity and vasodilator responsiveness to NO-donors. Moreover, there is evidence that AAs tend to exhibit cardiovascular hyper-reactivity to stress, such that blood pressure responses are markedly increased in the presence of a physiological sympathoexcitation (Bond et al. 1999) which aligns with the results of the present study.

Research has suggested both heightened α_1 -adrenergic receptor sensitivity and reduced β -adrenergic sensitivity may contribute to the exaggerated cardiovascular hyper-reactivity (Lemogoum et al. 2004) among AAs. However, in the present study it is possible the

observed increase in resting systolic blood pressure witnessed in AAs (≈ 22 hrs after HII exercise) may have been transient and abated with rest. Hunter and McCarthy (Hunter and McCarthy 1983) previously showed that 8 weeks of intense anaerobic training significantly increased resting systolic blood pressure in competitive cyclists. In that study, daily training volume was drastically reduced if resting systolic blood pressure was $+10$ mm Hg above baseline. A total of 40 reduced-training days occurred during that study, of which, elevated resting systolic blood pressure was alleviated (i.e., lowered) the subsequent day in all but 3 instances. The results of that study reveal that an elevated resting blood pressure may be a product of intense training and quickly restored with rest.

More recently, Yan et al. (Yan et al. 2014) showed that young AAs and EAs have differential responses following maximal exercise in central blood pressure and central stiffness. Importantly, no differences in either measure were seen at rest, thus highlighting the utility of maximal exercise to induce race-related cardiovascular differences. Likewise, we have shown that blood pressure and SAE were not different among AAs and EAs in the Trained State, though differences emerged ≈ 22 hours after HII exercise. Clearly an acute, unaccustomed bout of HII exercise can produce physiological perturbations so that differences in vascular properties (i.e., SAE) may emerge among AA and EA women.

Discrepancies in peak oxygen uptake at baseline and in response to aerobic training have previously been observed among AA and EA women (Swift et al. 2013). We found that AA women had a lower VO_{2peak} at baseline and after aerobic training. While high cardiorespiratory fitness is a known protectant against cardiovascular disease, the exact mechanisms responsible are not fully understood. Certainly autonomic tone and endothelial function may act to moderate disease progression. However, we did not find a relationship between VO_{2peak} and the changes in systolic blood pressure or SAE following HII exercise.

Several limitations are present in this study. Our study was limited to an indirect assessment of endothelial function, thus, use of flow-mediated dilation should be considered. Given the absence of a control group, it is difficult to determine causality between observed changes and influence of exercise training. Since follow-up assessments were performed one month apart we cannot rule out the possibility of day-to-day or measurement variation. However, all procedures were conducted under standardized conditions controlled for diet, time of day, and phase of the menstrual cycle. Importantly though, VO_{2peak} was not different across the 3 follow-up assessments, indicating no biasing of results due to variance in aerobic fitness. Because all participants had been habitually training, we evaluated the responses to HII exercise from the perspective of a 'new trained baseline' (i.e., Trained State). Nevertheless, our findings extend the breadth of knowledge concerning the differential effects of MIC and HII exercise on resting blood pressure among participants of different races.

In conclusion, the present study shows that AA women had an increased systolic blood pressure response ≈ 22 hours after an acute, unaccustomed bout of HII exercise compared to EAs and these changes were independently associated with a decrease in SAE. It is important to emphasize that this observation may have been transient and abated shortly thereafter. Future work should evaluate if extended (i.e., >16 weeks) HII exercise leads to more robust improvements in blood pressure and SAE among AA and EA participants.

Acknowledgments

We would like to recognize David R. Bryan and Brandon L. Kane for their commitment and respective contributions. The authors also wish to express their appreciation to the participants for their willingness to complete this investigation. This investigation was supported by following grants from the National Institutes of Health: R01DK049779, P30DK056336, P60DK079626, and UL1RR025777.

REFERENCES

- Bond V, Mills RM, Caprarola M, Vaccaro P, Adams RG, Blakely R, Roltsch M, Hatfield B, Davis GC, Franks BD, Fairfax J, Banks M. Aerobic exercise attenuates blood pressure reactivity to cold pressor test in normotensive, young adult African-American women. *Ethn Dis.* 1999; 9(1):104–110. [PubMed: 10355479]
- Campia U, Choucair WK, Bryant MB, Wacławiw MA, Cardillo C, Panza JA. Reduced endothelium-dependent and -independent dilation of conductance arteries in African Americans. *J Am Coll Cardiol.* 2002; 40(4):754–760. [PubMed: 12204507]
- Centers for Disease Control and Prevention (CDC). Vital signs: prevalence, treatment, and control of hypertension--United States, 1999–2002 and 2005–2008. *MMWR Morb Mortal Wkly Rep.* 2011; 60(4):103–108. [PubMed: 21293325]
- Chase NL, Sui X, Lee DC, Blair SN. The association of cardiorespiratory fitness and physical activity with incidence of hypertension in men. *Am J Hypertens.* 2009; 22(4):417–424. [PubMed: 19197248]
- Chaturvedi N, Bulpitt CJ, Leggetter S, Schiff R, Nihoyannopoulos P, Strain WD, Shore AC, Rajkumar C. Ethnic differences in vascular stiffness and relations to hypertensive target organ damage. *J Hypertens.* 2004; 22(9):1731–1737. [PubMed: 15311101]
- Ciolac EG, Bocchi EA, Bortolotto LA, Carvalho VO, Greve JM, Guimaraes GV. Effects of high-intensity aerobic interval training vs. moderate exercise on hemodynamic, metabolic and neuro-humoral abnormalities of young normotensive women at high familial risk for hypertension. *Hypertens Res.* 2010; 33(8):836–843. [PubMed: 20448634]
- Ciolac EG, Bocchi EA, Greve JM, Guimaraes GV. Heart rate response to exercise and cardiorespiratory fitness of young women at high familial risk for hypertension: effects of interval vs continuous training. *Eur J Cardiovasc Prev Rehabil.* 2011; 18(6):824–830. [PubMed: 21450597]
- Cohn JN, Finkelstein S, McVeigh G, Morgan D, LeMay L, Robinson J, Mock J. Noninvasive pulse wave analysis for the early detection of vascular disease. *Hypertension.* 1995; 26(3):503–508. [PubMed: 7649589]
- Cornelissen VA, Fagard RH. Effects of endurance training on blood pressure, blood pressure-regulating mechanisms, and cardiovascular risk factors. *Hypertension.* 2005; 46(4):667–675. [PubMed: 16157788]
- Cushman WC, Reda DJ, Perry HM, Williams D, Abdellatif M, Materson BJ. Regional and racial differences in response to antihypertensive medication use in a randomized controlled trial of men with hypertension in the United States. Department of Veterans Affairs Cooperative Study Group on Antihypertensive Agents. *Arch Intern Med.* 2000; 160(6):825–831. [PubMed: 10737282]
- Egan BM, Zhao Y, Axon RN. US trends in prevalence, awareness, treatment, and control of hypertension, 1988–2008. *JAMA.* 2010; 303(20):2043–2050. [PubMed: 20501926]
- Ferrier KE, Waddell TK, Gatzka CD, Cameron JD, Dart AM, Kingwell BA. Aerobic exercise training does not modify large-artery compliance in isolated systolic hypertension. *Hypertension.* 2001; 38(2):222–226. [PubMed: 11509480]
- Flack JM. The epidemiology of hypertension and related conditions in the African-American population. *J Natl Med Assoc.* 1995; 87(8 Suppl):606–609. [PubMed: 7674352]
- Ford ES, Capewell S. Proportion of the decline in cardiovascular mortality disease due to prevention versus treatment: public health versus clinical care. *Annu Rev Public Health.* 2011; 32:5–22. [PubMed: 21417752]
- Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, Dai S, Ford ES, Fox CS, Franco S, Fullerton HJ, Gillespie C, Hailpern SM, Heit JA, Howard VJ, Huffman MD, Judd SE, Kissela BM, Kittner SJ, Lackland DT, Lichtman JH, Lisabeth LD, Mackey RH, Magid DJ, Marcus GM,

- Marelli A, Matchar DB, McGuire DK, Mohler ER 3rd, Moy CS, Mussolino ME, Neumar RW, Nichol G, Pandey DK, Paynter NP, Reeves MJ, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Wong ND, Woo D, Turner MB. American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics-2014 update: a report from the American Heart Association. *Circulation*. 2014; 129(3):e28–e292. [PubMed: 24352519]
- Grey E, Bratteli C, Glasser SP, Alinder C, Finkelstein SM, Lindgren BR, Cohn JN. Reduced small artery but not large artery elasticity is an independent risk marker for cardiovascular events. *Am J Hypertens*. 2003; 16(4):265–269. [PubMed: 12670741]
- Harris JA, Benedict FG. A Biometric Study of Human Basal Metabolism. *Proc Natl Acad Sci U.S.A.* 1918; 4(12):370–373. [PubMed: 16576330]
- Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, Macera CA, Heath GW, Thompson PD, Bauman A. American College of Sports Medicine and American Heart Association. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation*. 2007; 116(9):1081–1093. [PubMed: 17671237]
- Heffernan KS, Jae SY, Fernhall B. Racial differences in arterial stiffness after exercise in young men. *Am J Hypertens*. 2007; 20(8):840–845. [PubMed: 17679030]
- Hertz RP, Unger AN, Cornell JA, Saunders E. Racial disparities in hypertension prevalence, awareness, and management. *Arch Intern Med*. 2005; 165(18):2098–2104. [PubMed: 16216999]
- Hunter G, McCarthy J. Pressor response associated with high-intensity anaerobic training. *Physician Sports Med*. 1983; 11(4):151–162.
- Kakiyama T, Sugawara J, Murakami H, Maeda S, Kuno S, Matsuda M. Effects of short-term endurance training on aortic distensibility in young males. *Med Sci Sports Exerc*. 2005; 37(2):267–271. [PubMed: 15692323]
- Kenny L, Humphrey R, Bryant C, Mahler D, Froelicher V, Miller N, York T. ACSM's guidelines for exercise testing and prescription. 1995
- Lemogoum D, Van Bortel L, Van den Abeele W, Ciarka A, Degaute JP, van de Borne P, Leeman M. Effect of beta-adrenergic stimulation on pulse wave velocity in black and white subjects. *J Hypertens*. 2004; 22(12):2349–2353. [PubMed: 15614029]
- Liu X, Tsilimingras D, Paul TK. Prevalence and changes of untreated isolated systolic hypertension among non-Hispanic black adults in the United States. *Hypertens Res*. 2014; 37(7):685–691. [PubMed: 24621464]
- Madden KM. Continuous vs. interval exercise training in hypertensive subjects. *Hypertens. Res*. 2010; 33(6):544–545. [PubMed: 20431596]
- Matsuo T, Saotome K, Seino S, Shimojo N, Matsushita A, Iemitsu M, Ohshima H, Tanaka K, Mukai C. Effects of a low-volume aerobic-type interval exercise on VO₂max and cardiac mass. *Med Sci Sports Exerc*. 2014; 46(1):42–50. [PubMed: 23846165]
- McVeigh GE, Allen PB, Morgan DR, Hanratty CG, Silke B. Nitric oxide modulation of blood vessel tone identified by arterial waveform analysis. *Clin Sci (Lond)*. 2001; 100(4):387–393. [PubMed: 11256976]
- Mensah GA, Brown DW. An overview of cardiovascular disease burden in the United States. *Health Aff (Millwood)*. 2007; 26(1):38–48. [PubMed: 17211012]
- Ozkor MA, Rahman AM, Murrow JR, Kavtaradze N, Lin J, Manatunga A, Hayek S, Quyyumi AA. Differences in vascular nitric oxide and endothelium-derived hyperpolarizing factor bioavailability in blacks and whites. *Arterioscler Thromb Vasc Biol*. 2014; 34(6):1320–1327. [PubMed: 24675657]
- Pescatello LS, Kulikowich JM. The aftereffects of dynamic exercise on ambulatory blood pressure. *Med Sci Sports Exerc*. 2001; 33(11):1855–1861. [PubMed: 11689735]
- Phillips SA, Mahmoud AM, Brown MD, Haus JM. Exercise interventions and peripheral arterial function: implications for cardio-metabolic disease. *Prog Cardiovasc Dis*. 2015; 57(5):521–534. [PubMed: 25529367]
- Pienaar PR, Micklesfield LK, Gill JM, Shore AC, Gooding KM, Levitt NS, Lambert EV. Ethnic differences in microvascular function in apparently healthy South African men and women. *Exp Physiol*. 2014; 99(7):985–994. [PubMed: 24803528]

- Stordahl SA, Madslien VO, Stoylen A, Kjos A, Helgerud J, Wisloff U. Atrioventricular plane displacement in untrained and trained females. *Med Sci Sports Exerc.* 2004; 36(11):1871–1875. [PubMed: 15514500]
- Stein CM, Lang CC, Singh I, He HB, Wood AJ. Increased vascular adrenergic vasoconstriction and decreased vasodilation in blacks. Additive mechanisms leading to enhanced vascular reactivity. *Hypertension.* 2000; 36(6):945–951. [PubMed: 11116105]
- Strisciuglio T, De Luca S, Capuano E, Luciano R, Niglio T, Trimarco B, Galasso G. Endothelial dysfunction: its clinical value and methods of assessment. *Curr Atheroscler Rep.* 2014; 16(6): 417-014-0417-1. [PubMed: 24764181]
- Swift DL, Johannsen NM, Lavie CJ, Earnest CP, Johnson WD, Blair SN, Church TS, Newton RL Jr. Racial differences in the response of cardiorespiratory fitness to aerobic exercise training in Caucasian and African American postmenopausal women. *J Appl Physiol.* 2013; 1985; 114(10): 1375–1382. [PubMed: 23471944]
- Tjonna AE, Lee SJ, Rognmo O, Stolen TO, Bye A, Haram PM, Loennechen JP, Al-Share QY, Skogvoll E, Stordahl SA, Kemi OJ, Najjar SM, Wisloff U. Aerobic interval training versus continuous moderate exercise as a treatment for the metabolic syndrome: a pilot study. *Circulation.* 2008; 118(4):346–354. [PubMed: 18606913]
- Wisloff U, Stoylen A, Loennechen JP, Bruvold M, Rognmo O, Haram PM, Tjonna AE, Helgerud J, Stordahl SA, Lee SJ, Videm V, Bye A, Smith GL, Najjar SM, Ellingsen O, Skjaerpe T. Superior cardiovascular effect of aerobic interval training versus moderate continuous training in heart failure patients: a randomized study. *Circulation.* 2007; 115(24):3086–3094. [PubMed: 17548726]
- Yan H, Ranadive SM, Heffernan KS, Lane AD, Kappus RM, Cook MD, Wu PT, Sun P, Harvey IS, Woods JA, Wilund KR, Fernhall B. Hemodynamic and arterial stiffness differences between African-Americans and Caucasians after maximal exercise. *Am J Physiol Heart Circ Physiol.* 2014; 306(1):H60–H68. [PubMed: 24186094]

Figure 1. Study schema

Following baseline evaluations, all participants completed at least 8 weeks of supervised, aerobic exercise training before further assessments were made at 12 and 16 weeks. At least one 1 month separated follow-up assessments to ensure measures corresponded to the follicular phase of the menstrual cycle. Participants continued to exercise train until completing 16 weeks. All follow-up assessments were randomized. Before evaluations were made in the “Trained State,” participants abstained from any exercise (No Exercise) 48 hours prior to testing for the purpose of attenuating the acute effects of exercise. Measures of moderate-intensity continuous exercise (MIC) and high-intensity interval exercise (HII) were completed $\approx$ 22 hours an overnight stay in a room calorimeter. Procedures were conducted under standardized conditions at the same time of day.

Figure 2.

Resting systolic blood pressure responses after 8–16 weeks of aerobic training (Trained State); ≈ 22 hours after a bout of moderate-intensity continuous exercise (MIC) and ≈ 22 hours after a bout of high-intensity interval exercise (HII). *Significant difference ($p < 0.05$) between Trained State and acute HII exercise in African Americans. †Significant difference ($p < 0.05$) between acute MIC exercise and acute HII exercise in African Americans. **Significant difference ($p < 0.01$) between African American and European American women after acute HII exercise. Data presented as means $\pm$ s.e.m. ($n = 22$).

Table 1

Baseline characteristics among participants.

Variables	African American (<i>n</i> = 11)	European American (<i>n</i> = 11)	<i>p</i> -values
Age (yrs)	32 ± 5	32 ± 7	0.98
Height (cm)	166 ± 5	165 ± 5	0.81
Body weight (kg)	81.3 ± 14.9	67.2 ± 10.0 *	0.02
Body fat (%)	37.0 ± 5.9	37.4 ± 6.4	0.86
Peak VO ₂ (mL/kg FFM/min)	35.5 ± 3.5	44.6 ± 5.8 *	< 0.001
SBP (mm Hg)	120 ± 13	111 ± 10	0.08
DBP (mm Hg)	73 ± 10	67 ± 7	0.08
SAE (mL/mm Hg·100)	6.9 ± 2.6	7.9 ± 2.4	0.35
EPI (μg/24 hrs) ^{a, c}	4.8 ± 3.2	5.1 ± 5.0	0.89
NOR (μg/24 hrs) ^b	32.2 ± 6.4	27.1 ± 9.3	0.32
DOP (μg/24 hrs) ^b	320.6 ± 70.6	197.4 ± 96.8 *	0.04

All values are presented as means ± SD; Peak VO₂, peak oxygen uptake; FFM, fat-free mass; SBP, systolic blood pressure; DBP, diastolic blood pressure; SAE, small arterial elasticity; mm Hg, millimeters of mercury. EPI, epinephrine; NOR, norepinephrine; DOP, dopamine.

* Significant difference ($p < 0.05$) between race at baseline.

^a denotes African Americans ($n = 4$);

^b denotes African Americans ($n = 5$);

^c denotes European Americans ($n = 7$).

Table 2Participant characteristics in the Trained State and acute responses to MIC and HII exercise ($n = 22$).

Variables	African Americans	European Americans	Treatment	<i>p</i> -values	
				Race	T × R
Body weight (kg)					
Trained State	82.0 ± 16.3	67.1 ± 9.6			
MIC	81.5 ± 15.2	67.4 ± 10.1	0.31	0.02	0.26
HII	80.9 ± 15.0	67.2 ± 9.6			
Body fat (%)					
Trained State	37.6 ± 6.6	37.0 ± 5.5			
MIC	37.8 ± 6.5	36.8 ± 5.5	0.15	0.78	0.28
HII	37.3 ± 6.5	36.8 ± 5.5			
Peak VO ₂ (mL/kg FFM/min)					
Trained State	36.9 ± 4.7	47.0 ± 6.0			
MIC	37.6 ± 6.2	47.6 ± 6.8	0.75	0.001	0.95
HII	36.9 ± 4.9	47.6 ± 7.0			
SBP (mm Hg)					
Trained State	121 ± 14	116 ± 12			
MIC	119 ± 10	114 ± 10	0.44	0.09	0.04
HII	128 ± 14 [‡]	113 ± 9			
DBP (mm Hg)					
Trained State	74 ± 13	69 ± 8			
MIC	73 ± 8	70 ± 7	0.94	0.14	0.15
HII	75 ± 9	68 ± 6			
SAE (mL/mmHg·100)					
Trained State	7.0 ± 2.3	6.7 ± 2.4			
MIC	8.3 ± 3.0 [‡]	7.3 ± 2.2	0.05	0.71	0.31
HII	6.4 ± 2.9	6.8 ± 1.6			
Epinephrine (μg/24 hrs) ^a					
Trained State	4.7 ± 3.8	3.4 ± 1.8			
MIC	5.1 ± 2.3	3.6 ± 3.4	0.60	0.56	0.71
HII	5.1 ± 5.1	5.7 ± 5.6			
Norepinephrine (μg/24 hrs) ^a					
Trained State	27.9 ± 10.9	30.3 ± 14.7			
MIC	27.6 ± 11.7	30.9 ± 10.9	0.60	0.80	0.78
HII	33.0 ± 14.3	31.6 ± 14.5			
Dopamine (μg/24 hrs) ^a					
Trained State	317.1 ± 164.2	283.7 ± 225.5			
MIC	286.9 ± 174.2	230.9 ± 114.5	0.49	0.41	0.72
HII	307.3 ± 140.5	210.9 ± 119.0			

All values are presented as means $\pm$ SD; T $\times$ R, treatment by race interaction; MIC, moderate-intensity continuous exercise; HII, high-intensity interval exercise; SBP, systolic blood pressure; DBP, diastolic blood pressure; SAE, small arterial elasticity; FFM, fat-free mass; mm Hg, millimeters of mercury.

[†]Significant difference ($p < 0.05$) after HII exercise compared to Trained State and after MIC exercise among African Americans.

[‡]Significant difference ($p < 0.05$) after MIC exercise compared to Trained State and after HII exercise.

^adenotes African Americans ($n = 7$) and European Americans ($n = 7$) for catecholamines.

Table 3

Correlations for changes () among selected variables ≈ 22 hours after an acute bout of high-intensity interval exercise.

Variables	Race	SBP	SAE	EPI ^a	NOR ^a	DOP ^a
Race	--	--	--	--	--	--
SBP	0.446 [‡]	--	--	--	--	--
SAE	-0.153	-0.539 [‡]	--	--	--	--
EPI ^a	-0.048	0.322	-0.409	--	--	--
NOR ^a	0.168	-0.067	-0.101	0.357	--	--
DOP ^a	0.101	-0.157	-0.043	0.371	0.836 [‡]	--

[‡]Significant at $p < 0.01$;

[‡]Significant at $p < 0.05$. Race, African Americans coded by 1 and European Americans coded by 0. SBP, systolic blood pressure (mm Hg); SAE, small arterial elasticity (mL/mm Hg-100); EPI, epinephrine ($\mu\text{g}/24$ hr); NOR, norepinephrine ($\mu\text{g}/24$ hr); DOP, dopamine ($\mu\text{g}/24$ hr).

^adenotes African Americans ($n = 7$) and European Americans ($n = 7$) for catecholamines.

Table 4

Model estimation for changes () in systolic blood pressure ≈ 22 hours following an acute bout of high-intensity interval exercise ($n = 22$).

	Model R	R ²	Slope	Standardized β	Partial r	p -value
Intercept	0.65	0.43	-2.756			
SAE (mL/mm Hg·100)			-1.65	-0.482	-0.533 [†]	0.013
Race			6.006	0.372	0.437 [†]	0.048

[†] Significant at $p < 0.05$. SAE, small arterial elasticity; Race, African Americans coded by 1 and European Americans coded by 0.