NRC Research Press

ARTICLE

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, TaShauna U. Goldsby, Gordon Fisher, Eric P. Plaisance, Barbara A. Gower, Stephen P. Glasser, and Gary R. Hunter

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 h later) acute bouts of moderate-intensity continuous (MIC) and high-intensity interval (HII) exercise matched for total work. Because of 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, 22 AA and EA women initiated aerobic exercise training 3 times/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: (*i*) in the trained state (TS; 8–16 weeks of aerobic training); (*ii*) ≈22 h after an acute bout of MIC exercise; and (*iii*) ≈22 h 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 versus HII, 128 ± 14; $p = 0.01$) whereas responses (TS, 116 ± 12 versus 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, 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.

Key words: aerobic training, arterial stiffness, endothelial function, health disparity, hypertension, race.

Résumé : L'exercice aérobie abaisse de manière transitoire la pression sanguine. Toutefois, il y a peu d'études qui traitent en même temps de l'évaluation de la pression sanguine et de l'élasticité des petites artères (SAE), un indice de la fonction endothéliale; cette étude est réalisée auprès d'Américaines originaires de l'Afrique (AA) et de l'Europe (EA) le lendemain matin suivant (c.-à-d. ≈22 h plus tard) des séances continues d'exercice d'intensité modérée (MIC) et des séances par intervalles d'intensité élevée (HII) appariées selon le travail total. L'hypothèse est la suivante : à cause des plus importants gradients de stress de cisaillement, HII devrait susciter une plus grande baisse de pression systolique (SBP) que MIC. Après les mesures du niveau de base, 22 AA et EA démarrent leur programme d'entraînement aérobie à raison de 3 fois par semaine. À partir de la huitième semaine, on effectue aléatoirement au cours des huit semaines suivantes trois évaluations de suivi de la pression sanguine de repos et de SAE. Au total, toutes les participantes se sont entraînées durant 16 semaines. Les évaluations de suivi sont : (*i*) dans la condition dite entraînée, TS; 8-16 semaines d'entraînement aérobie, (*ii*) ≈22 h après une séance MIC et (*iii*) ≈22 h après une séance HII. Dans le groupe AA, la séance HII suscite une augmentation significative de SBP (mm Hg) (TS, 121 ± 14 versus HII, 128 ± 14; $p = 0,01$) alors que dans le groupe EA, les adaptations ne diffèrent pas (TS, 116 ± 12 versus HII, 113 ± 9; $p = 0,34$). Après un rajustement pour tenir compte de la race, les variations de SAE (r partielle = −0,533; $p = 0,01$) sont associées aux variations de SBP dues à HII. D'après ces résultats, une séance HII ponctuelle et sans accoutumance suscite des perturbations physiologiques causant une augmentation significative de SBP qui est indépendamment associée à la diminution de SAE chez des femmes AA, mais pas chez des femmes EA. [Traduit par la Rédaction]

Mots-clés : entraînement aérobie, rigidité artérielle, fonction endothéliale, disparité sanitaire, hypertension, race.

Received 28 September 2015. Accepted 21 December 2015.

S.J. Carter.* Department of Human Studies, University of Alabama at Birmingham, Birmingham, AL 35233, USA; Nutrition Obesity Research Center, University of Alabama at Birmingham, Birmingham, AL 35233, USA.
T.U. Goldsby. Nutrition Obesity Research Center, University of Alabama at Birmingham, Birmingham, AL 35233, USA; Office of Energetics, University of Alabama at Birmingham, Birmingham, AL 35233, USA.
G. Fisher and G.R. Hunter. Department of Human Studies, University of Alabama at Birmingham, Birmingham, AL 35233, USA; Department of Nutrition Sciences, University of Alabama at Birmingham, Birmingham, AL 35233, USA.
E.P. Plaisance. Department of Human Studies, University of Alabama at Birmingham, Birmingham, AL 35233, USA.
B.A. Gower. Department of Nutrition Sciences, University of Alabama at Birmingham, Birmingham, AL 35233, USA.
S.P. Glasser. Department of Preventive Medicine, University of Alabama at Birmingham, Birmingham, AL 35233, USA.

Corresponding author: Stephen J. Carter (email: carters@uab.edu).
*Present address: Department of Nutrition Sciences, 1675 University Blvd, Webb Building #248, University of Alabama at Birmingham, Birmingham, AL 35233-2432, USA.

Introduction

While cardiovascular disease 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 cardiovascular disease mortality and morbidity compared to European Americans (EA) (Mensah and Brown 2007). Hypertension is one such morbidity that has remained prevalent among AAs (Egan et al. 2010; Liu et al. 2014). 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 et al. 2007), and peripheral resistance, (Stein et al. 2000) along with reduced vasodilator potential (Ozkor et al. 2014; Pienaar 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 (CDC 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, 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 (Slordahl et al. 2004; Matsuo et al. 2014) 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, 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 (McVeigh et al. 2001; Grey et al. 2003), compared to MIC exercise. However, because of 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 h 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 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 less than one time 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/week on a stationary cycle ergometer. Beginning at week 8, participants were evaluated on three additional occasions (i.e., weeks 12 and 16), each separated by 1 month during the follicular phase of the menstrual cycle (Fig. 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: (*i*) in the "trained state," having completed 8–16 weeks of aerobic training and no exercise in the room calorimeter; (*ii*) ≈22 h after acute MIC exercise on a stationary cycle ergometer at 50% peak VO_2; and (*iii*) ≈22 h 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 h 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", because 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 ≈60% energy as carbohydrates, ≈25% energy as fat, and ≈15% energy as protein. Dietary sodium and the ratio between polyunsaturated and 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 (VO_{2peak}) as determined by the highest level reached in the final stage of exercise. Power output began at 25 W and increased further by 25 W every 2 min 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).

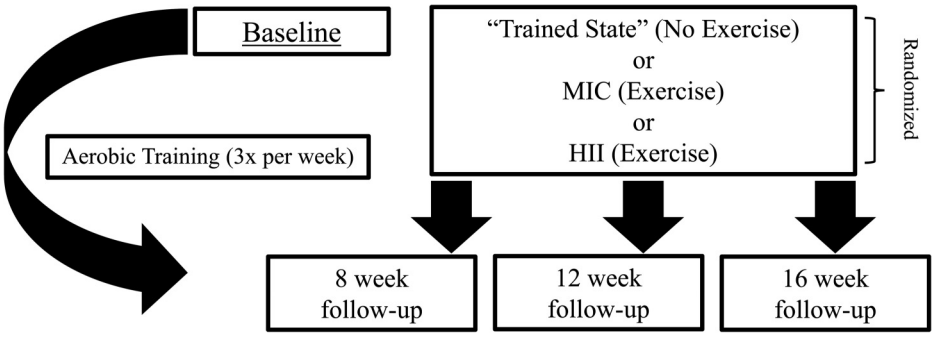
Overnight stay in room calorimeter

Participants entered at 0800 and spent 23 h in a whole-room respiration calorimeter (3.38 m × 2.11 m × 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 VO_{2peak} that corresponded to each follow-up assessment using the appropriate metabolic equation for leg ergometry [VO_2 (mL/(kg · min)) = 2 × (mass (kg) × length (m))/(time (min)) + (3.5 × body mass)] in accordance with the American College of Sports Medicine (Kenny

Fig. 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 h 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 ≈22 h an overnight stay in a room calorimeter. Procedures were conducted under standardized conditions at the same time of day.



et al. 1995). During the MIC exercise, participants cycled continuously for 60 min at an intensity of 50% VO_{2peak}. Fifteen work intervals were performed for 2 min 24 s with rest intervals of 1 min 43 s for the HII exercise. Work was calculated by multiplying 1.66 to the MIC exercise workload, which equated to 84% VO_{2peak}. 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 (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 s analog tracing of the radial waveform digitized at 200 samples/s. As previously described by Cohn and colleagues (Cohn et al. 1995), a beat determination is constructed using a beat-marking algorithm that determines beginning systole, peak systole, onset of diastole, and end diastole during the 30 s 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 min period for warm-up and stretching at each session. During week 1, continuous exercise was maintained for 20 min at ≈67% of maximum heart rate. Over a 16 week period, exercise intensity and duration increased so that by the final week, exercise was consistently maintained for 60 min at ≈80% maximum heart rate. Participants cooled down for 3–5 min, following cessation of exercise.

Twenty-four hour fractionated urinary catecholamines

Twenty-four 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 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 analyses of variance with repeated measures were performed to compare the acute effects of MIC and HII exercise from the trained state. Post hoc 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 ΔSAE (occurring from HII exercise) on Δsystolic blood pressure. Collinearity diagnostics for all variables were within acceptable limits and variable inflation factors for all models were less than 1.04. All data were analyzed with SPSS (v22; IBM Corp., Armonk, NY). 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 versus EA, 67.2 ± 10.0 kg; $p = 0.02$) and had a lower VO_{2peak} (AA, 35.5 ± 3.5 mL/kg FFM/min versus 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 μg/24 h versus EA, 197.4 ± 96.8 μg/24 h; $p = 0.04$) at baseline.

Table 1. Baseline characteristics among participants.

Variables	African American (n = 11)	European American (n = 11)	p-values
Age (years)	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 h)[a,c]	4.8±3.2	5.1±5.0	0.89
NOR (μg/24 h)[b]	32.2±6.4	27.1±9.3	0.32
DOP (μg/24 h)[b]	320.6±70.6	197.4±96.8*	0.04

Note: All values are presented as means ± standard deviation; peak VO_2, peak oxygen uptake; FFM, fat-free mass; SBP, systolic blood pressure; DBP, diastolic blood pressure; SAE, small arterial elasticity; mm Hg, millimetres of mercury; EPI, epinephrine; NOR, norepinephrine; DOP, dopamine. *, Significant difference (p < 0.05) between race at baseline.

[a]African Americans (n = 4).

[b]African Americans (n = 5).

[c]European Americans (n = 7).

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, VO_{2peak}, systolic/diastolic blood pressure, SAE, and catecholamines across the three 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 h after MIC exercise. Similar to the baseline evaluation, there was a significant between-race difference, as AAs had a greater body weight and lower VO_{2peak} 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 h after HII exercise (Fig. 2).

In Table 3, positive associations were found among the changes (i.e., delta, Δ) between the trained state and ≈22 h after HII exercise for race and Δsystolic blood pressure (r = 0.446, p < 0.05). Negative associations were found between Δsystolic blood pressure and ΔSAE (r = –0.539, p < 0.01). A linear regression revealed the independent effects of ΔSAE ≈22 h after HII exercise such that a significant association between Δsystolic blood pressure and ΔSAE 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 Δsystolic blood pressure after adjusting for ΔSAE.

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 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 h 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. Because hypertension is prevalent among AAs (Egan et al. 2010; Liu et al. 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

Table 2. Participant characteristics in the trained state and acute responses to MIC and HII exercise (n = 22).

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

Note: All values are presented as means ± standard deviation; T × 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, millimetres 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.

[a]African Americans (n = 7) and European Americans (n = 7) for catecholamines.

(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 (i.e., 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. Because 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 three follow-up assessments. Generally, aerobic exercise training lowers cardiovascular risk factors though results demonstrating improved arterial compliance and stiffness have been equivocal. Previous work has revealed increased carotid compliance in young, sedentary male

Fig. 2. Resting systolic blood pressure responses after 8–16 weeks of aerobic training (trained state); ≈22 h after moderate-intensity continuous exercise (MIC) and ≈22 h after 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 ± standard error of the mean. ($n = 22$).

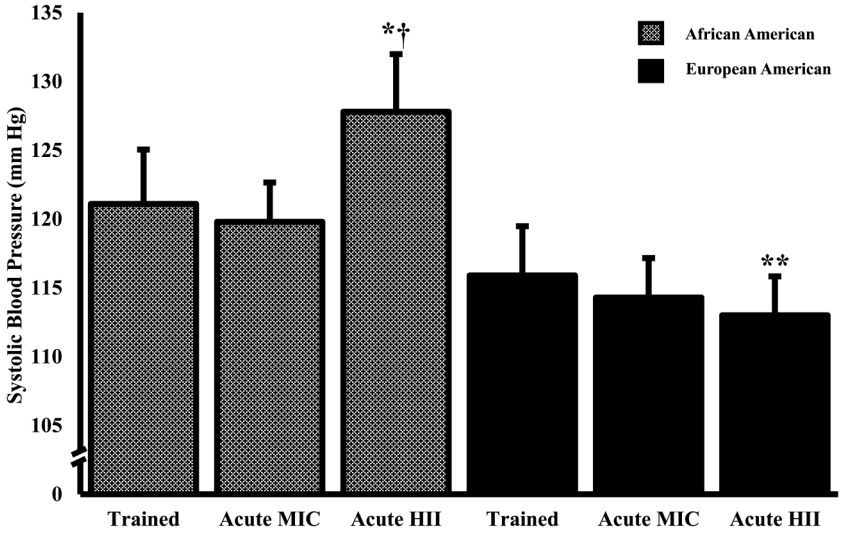


Table 3. Correlations for changes (Δ) among selected variables ≈22 h after an acute bout of high-intensity interval exercise.

Variables	Race	ΔSBP	ΔSAE	ΔEPI[a]	ΔNOR[a]	ΔDOP[a]
Race	—	—	—	—	—	—
ΔSBP	0.446†	—	—	—	—	—
ΔSAE	−0.153	−0.539‡	—	—	—	—
ΔEPI[a]	−0.048	0.322	−0.409	—	—	—
ΔNOR[a]	0.168	−0.067	−0.101	0.357	—	—
ΔDOP[a]	0.101	−0.157	−0.043	0.371	0.836‡	—

Note: 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 (μg/24 h); NOR, norepinephrine (μg/24 h); DOP, dopamine (μg/24 h). †, Significant at $p < 0.05$. ‡, Significant at $p < 0.01$.

[a]African Americans ($n = 7$) and European Americans ($n = 7$) for catecholamines.

participants following 12 weeks of aerobic training at 70%–75% maximum heart rate (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% maximum heart rate) exercise 3 times/week. Discrepancies in training duration, exercise intensity, age or health disparities, as well as the methods used to evaluate arterial compliance and 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 or 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 cardiovascular disease 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 h 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 that 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 h 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 three 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. (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 h 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

Table 4. Model estimation for changes (Δ) in systolic blood pressure ≈22 h 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	—	—	—
ΔSAE (mL/mm Hg·100)	—	—	–1.65	–0.482	–0.533†	0.013
Race	—	—	6.006	0.372	0.437†	0.048

Note: †, Significant at $p < 0.05$. SAE, small arterial elasticity; race, African Americans coded by 1 and European Americans coded by 0.

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. Because follow-up assessments were performed 1 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 three 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 h 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.

Conflict of interest

We declare no conflicts of interest.

Acknowledgements

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, R.M., Caprarola, M., Vaccaro, P., Adams, R.G., Blakely, R., et al. 1999. Aerobic exercise attenuates blood pressure reactivity to cold pressor test in normotensive, young adult African-American women. Ethn. Dis. **9**(1): 104–110. PMID:10355479.

Campia, U., Choucair, W.K., Bryant, M.B., Waclawiw, M.A., Cardillo, C., and Panza, J.A. 2002. Reduced endothelium-dependent and -independent dilation of conductance arteries in African Americans. J. Am. Coll. Cardiol. **40**(4): 754–760. doi:10.1016/S0735-1097(02)02015-6. PMID:12204507.

Centers for Disease Control and Prevention (CDC). 2011. Vital signs: prevalence, treatment, and control of hypertension–United States, 1999–2002 and 2005–2008. MMWR Morb. Mortal Wkly. Rep. **60**(4): 103–108. PMID:21293325.

Chase, N.L., Sui, X., Lee, D.-C., and Blair, S.N. 2009. The association of cardiorespiratory fitness and physical activity with incidence of hypertension in men. Am. J. Hypertens. **22**(4): 417–424. doi:10.1038/ajh.2009.6. PMID:19197248.

Chaturvedi, N., Bulpitt, C.J., Leggetter, S., Schiff, R., Nihoyannopoulos, P., Strain, W.D., et al. 2004. Ethnic differences in vascular stiffness and relations to hypertensive target organ damage. J. Hypertens. **22**(9): 1731–1737. doi:10.1097/00004872-200409000-00017. PMID:15311101.

Ciolac, E.G., Bocchi, E.A., Bortolotto, L.A., Carvalho, V.O., Greve, J.M., and Guimarães, G.V. 2010. 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. **33**(8): 836–843. PMID:20448634.

Ciolac, E.G., Bocchi, E.A., Greve, J.M., and Guimarães, G.V. 2011. 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. **18**(6): 824–830. PMID:21450597.

Cohn, J.N., Finkelstein, S., McVeigh, G., Morgan, D., LeMay, L., Robinson, J., and Mock, J. 1995. Noninvasive pulse wave analysis for the early detection of vascular disease. Hypertension, **26**(3): 503–508. doi:10.1161/01.HYP.26.3.503. PMID:7649589.

Cornelissen, V.A., and Fagard, R.H. 2005. Effects of endurance training on blood pressure, blood pressure-regulating mechanisms, and cardiovascular risk factors. Hypertension, **46**(4): 667–675. doi:10.1161/01.HYP.0000184225.05629.51. PMID:16157788.

Cushman, W.C., Reda, D.J., Perry, H.M., Williams, D., Abdellatif, M., and Materson, B.J. 2000. 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. **160**(6): 825–831. PMID:10737282.

Egan, B.M., Zhao, Y., and Axon, R.N. 2010. US trends in prevalence, awareness, treatment, and control of hypertension, 1988-2008. JAMA, **303**(20): 2043–2050. doi:10.1001/jama.2010.650. PMID:20501926.

Ferrier, K.E., Waddell, T.K., Gatzka, C.D., Cameron, J.D., Dart, A.M., and Kingwell, B.A. 2001. Aerobic exercise training does not modify large-artery compliance in isolated systolic hypertension. Hypertension, **38**(2): 222–226. doi:10.1161/01.HYP.38.2.222. PMID:11509480.

Flack, J.M. 1995. The epidemiology of hypertension and related conditions in the African-American population. J. Natl. Med. Assoc. **87**(8 Suppl): 606–609. PMID:7674352.

Ford, E.S., and Capewell, S. 2011. Proportion of the decline in cardiovascular mortality disease due to prevention versus treatment: public health versus clinical care. Annu. Rev. Public Health, **32**: 5–22. doi:10.1146/annurev-publhealth-031210-101211. PMID:21417752.

Go, A.S., Mozaffarian, D., Roger, V.L., Benjamin, E.J., Berry, J.D., Blaha, M.J., et al. 2014. Heart disease and stroke statistics–2014 update: a report from the American Heart Association. Circulation, **129**(3): e28–e292. doi:10.1161/01.cir.0000441139.02102.80. PMID:24352519.

Grey, E., Bratteli, C., Glasser, S.P., Alinder, C., Finkelstein, S.M., Lindgren, B.R., and Cohn, J.N. 2003. Reduced small artery but not large artery elasticity is an independent risk marker for cardiovascular events. Am. J. Hypertens. **16**(4): 265–269. doi:10.1016/S0895-7061(02)03271-5. PMID:12670741.

Harris, J.A., and Benedict, F.G. 1918. A biometric study of human basal metabolism. Proc. Natl. Acad. Sci. U.S.A. **4**(12): 370–373. doi:10.1073/pnas.4.12.370. PMID:16576330.

Haskell, W.L., Lee, I.M., Pate, R.R., Powell, K.E., Blair, S.N., Franklin, B.A., et al. 2007. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. Circulation, **116**(9): 1081–1093. doi:10.1161/CIRCULATIONAHA.107.185649. PMID:17671237.

Heffernan, K.S., Jae, S.Y., and Fernhall, B. 2007. Racial differences in arterial stiffness after exercise in young men. Am. J. Hypertens. **20**(8): 840–845. doi:10.1016/j.amjhyper.2007.03.015. PMID:17679030.

Hertz, R.P., Unger, A.N., Cornell, J.A., and Saunders, E. 2005. Racial disparities in hypertension prevalence, awareness, and management. Arch. Intern. Med. **165**(18): 2098–2104. doi:10.1001/archinte.165.18.2098. PMID:16216999.

Hunter, G., and McCarthy, J. 1983. Pressor response associated with high-intensity anaerobic training. Physician Sports Med. **11**(4): 151–162.

Kakiyama, T., Sugawara, J., Murakami, H., Maeda, S., Kuno, S., and Matsuda, M. 2005. Effects of short-term endurance training on aortic distensibility in young males. Med. Sci. Sports Exerc. **37**(2): 267–271. doi:10.1249/01.MSS.0000152733.12578.5A. PMID:15692323.

Kenny, L., Humphrey, R., Bryant, C., Mahler, D., Froelicher, V., Miller, N., and York, T. 1995. ACSM's guidelines for exercise testing and prescription.

Lemogoum, D., Van Bortel, L., Van den Abeele, W., Ciarka, A., Degaute, J.P., van de Borne, P., and Leeman, M. 2004. Effect of beta-adrenergic stimulation on pulse wave velocity in black and white subjects. J. Hypertens. **22**(12): 2349–2353. doi:10.1097/00004872-200412000-00017. PMID:15614029.

Liu, X., Tsilimingras, D., and Paul, T.K. 2014. Prevalence and changes of untreated isolated systolic hypertension among non-Hispanic black adults in the United States. Hypertens. Res. **37**(7): 685–691. doi:10.1038/hr.2014.58. PMID:24621464.

Madden, K.M. 2010. Continuous vs. interval exercise training in hypertensive subjects. Hypertens. Res. **33**(6): 544–545. PMID:20431596.

Matsuo, T., Saotome, K., Seino, S., Shimojo, N., Matsushita, A., Iemitsu, M., et al. 2014. Effects of a low-volume aerobic-type interval exercise on VO2max and cardiac mass. Med. Sci. Sports Exerc. **46**(1): 42–50. doi:10.1249/MSS.0b013e3182a38da8. PMID:23846165.

McVeigh, G.E., Allen, P.B., Morgan, D.R., Hanratty, C.G., and Silke, B. 2001. Nitric oxide modulation of blood vessel tone identified by arterial waveform analysis. Clin. Sci. (Lond.), **100**(4): 387–393. doi:10.1042/CS20000227.

Mensah, G.A., and Brown, D.W. 2007. An overview of cardiovascular disease burden in the United States. Health Aff. (Millwood), **26**(1): 38–48. PMID:17211012.

Ozkor, M.A., Rahman, A.M., Murrow, J.R., Kavtaradze, N., Lin, J., Manatunga, A., et al. 2014. Differences in vascular nitric oxide and endothelium-derived hyperpolarizing factor bioavailability in blacks and whites. Arterioscler. Thromb. Vasc. Biol. **34**(6): 1320–1327. doi:10.1161/ATVBAHA.113.303136. PMID:24675657.

Pescatello, L.S., and Kulikowich, J.M. 2001. The aftereffects of dynamic exercise on ambulatory blood pressure. Med. Sci. Sports Exerc. **33**(11): 1855–1861. doi:10.1097/00005768-200111000-00009. PMID:11689735.

Phillips, S.A., Mahmoud, A.M., Brown, M.D., and Haus, J.M. 2015. Exercise interventions and peripheral arterial function: implications for cardio-metabolic disease. Prog. Cardiovasc. Dis. **57**(5): 521–534. doi:10.1016/j.pcad.2014.12.005. PMID:25529367.

Pienaar, P.R., Micklesfield, L.K., Gill, J.M., Shore, A.C., Gooding, K.M., Levitt, N.S., and Lambert, E.V. 2014. Ethnic differences in microvascular function in apparently healthy South African men and women. Exp. Physiol. **99**(7): 985–994. doi:10.1113/expphysiol.2014.078519. PMID:24803528.

Slordahl, S.A., Madslien, V.O., Stoylen, A., Kjos, A., Helgerud, J., and Wisloff, U. 2004. Atrioventricular plane displacement in untrained and trained females. Med. Sci. Sports Exerc. **36**(11): 1871–1875. doi:10.1249/01.MSS.0000145444.01292.3D. PMID:15514500.

Stein, C.M., Lang, C.C., Singh, I., He, H.B., and Wood, A.J. 2000. Increased vascular adrenergic vasoconstriction and decreased vasodilation in blacks. Additive mechanisms leading to enhanced vascular reactivity. Hypertension, **36**(6): 945–951. PMID:11116105.

Strisciuglio, T., De Luca, S., Capuano, E., Luciano, R., Niglio, T., Trimarco, B., and Galasso, G. 2014. Endothelial dysfunction: its clinical value and methods of assessment. Curr. Atheroscler. Rep. **16**(6): 417-014-0417-1. doi:10.1007/s11883-014-0417-1. PMID:24764181.

Swift, D.L., Johannsen, N.M., Lavie, C.J., Earnest, C.P., Johnson, W.D., Blair, S.N., et al. 2013. Racial differences in the response of cardiorespiratory fitness to aerobic exercise training in Caucasian and African American postmenopausal women. J. Appl. Physiol. (1985), **114**(10): 1375–1382. doi:10.1152/japplphysiol.01077.2012. PMID:23471944.

Tjonna, A.E., Lee, S.J., Rognmo, O., Stolen, T.O., Bye, A., Haram, P.M., et al. 2008. Aerobic interval training versus continuous moderate exercise as a treatment for the metabolic syndrome: a pilot study. Circulation, **118**(4): 346–354. doi:10.1161/CIRCULATIONAHA.108.772822. PMID:18606913.

Wisloff, U., Stoylen, A., Loennechen, J.P., Bruvold, M., Rognmo, O., Haram, P.M., et al. 2007. Superior cardiovascular effect of aerobic interval training versus moderate continuous training in heart failure patients: a randomized study. Circulation, **115**(24): 3086–3094. doi:10.1161/CIRCULATIONAHA.106.675041. PMID:17548726.

Yan, H., Ranadive, S.M., Heffernan, K.S., Lane, A.D., Kappus, R.M., Cook, M.D., et al. 2014. Hemodynamic and arterial stiffness differences between African-Americans and Caucasians after maximal exercise. Am. J. Physiol. Heart Circ. Physiol. **306**(1): H60–H68. doi:10.1152/ajpheart.00710.2013. PMID:24186094.