Published in final edited form as:

J Hypertens. 2019 June ; 37(6): 1262–1268. doi:10.1097/HJH.0000000000002049.

Effect of acute maximal exercise on vasodilatory function and arterial stiffness in African Americans and Caucasians

Elizabeth C. SCHROEDER¹, Sushant M. RANADIVE², Huimin YAN³, Abbi D. LANE-CORDOVA⁴, Rebecca M. KAPPUS⁵, Marc D. COOK⁶, and Bo FERNHALL¹

¹Department of Kinesiology and Nutrition, University of Illinois at Chicago, Chicago, IL

²Department of Kinesiology, University of Maryland, College Park, MD

³Department of Exercise and Health Sciences, University of Massachusetts Boston, Boston, MA

⁴Department of Exercise Science, University of South Carolina at Columbia, Columbia, SC

⁵Department of Health and Exercise Science, Appalachian State University, Boone, NC

⁶Department of Human Performance and Leisure Studies, North Carolina A&T State University, Greensboro, NC

Abstract

African-Americans (AA) are at increased risk of cardiovascular disease compared to Caucasians (CA), potentially due to greater arterial stiffness and reduced vasodilatory capacity. Racial differences also exist in arterial stiffness and blood pressure following maximal aerobic exercise; AA do not exhibit central post exercise blood pressure reductions. Whether impaired vasodilatory function contributes to the lack of blood pressure response is unknown.

Purpose: To evaluate vasodilatory function, arterial stiffness and hemodynamics following a maximal aerobic exercise test in young, healthy AA and CA adults.

Methods: Twenty-seven AA and 35 CA adults completed measures at baseline, 15- and 30-minutes after a maximal exercise test. Measures included vasodilatory capacity of forearm resistance arteries, central pulse wave velocity (PWV), and carotid artery stiffness (β).

Results: Forearm reactive hyperemia was greater in CA but increased similarly following exercise in both groups ($p < 0.05$). Carotid β -stiffness increased at 15- and 30-min ($p = 0.03$) in both groups, but PWV controlled for mean arterial pressure decreased after maximal exercise ($p = 0.03$). CA exhibited reductions in systolic and mean pressure, whereas no changes were seen for AA (interaction effects: $p < 0.05$).

Corresponding Author: Elizabeth C. Schroeder, eschro7@uic.edu; Address: Integrative Physiology Laboratory, University of Illinois at Chicago, 1640 W. Roosevelt Road, Chicago, IL 60608.

Previous presentation: Part of this work was previously presented as a poster at the American College of Sports Medicine Annual Meeting 2004; Baseline data has been previously published (Reference #13)

Conflict of Interest: NONE

Supplemental Digital Content

Supplemental Digital Content 1. Tables of matched group analyses. PDF

Conclusions: AA and CA had similar decreases in PWV, increases in β -stiffness, and increases in vasodilatory function following maximal exercise. AA, however, did not display reductions in blood pressure and had overall lower vasodilatory function in comparison to CA. Our results suggest AA exhibit similar vasodilatory function changes following aerobic exercise as CA, and therefore vasodilatory function likely does not explain the lack of blood pressure response in AA.

Keywords

African American; Vasodilatory capacity; Arterial stiffness; Exercise

Introduction

African Americans (AA) have a higher prevalence of cardiovascular disease (CVD) and hypertension in comparison to Caucasians (CA) [1]. Even at a young age, AA display greater arterial stiffness and reduced vasodilatory capacity of conduit and resistance vessels than CA [2,3]. Subclinical arterial dysfunction may contribute to the elevation in CVD risk, aside from traditional risk factors. Indeed, elevated arterial stiffness is linked to CVD risk [4] and impaired endothelial function is linked with CVD risk factors [5,6]. Thus, characterizing vascular dysfunction in AA may help reveal potential therapeutic targets to attenuate CVD risk burden in this population.

Acute maximal exercise is one physiological perturbation that can reveal underlying vascular dysfunction not apparent at rest. Racial differences have been revealed in arterial stiffness following maximal aerobic exercise, although not consistently. One study has reported that CA exhibit a reduction in central stiffness, whereas AA increase central stiffness, following maximal exercise [7]. Furthermore, racial differences have been noted in the central and peripheral pulse wave velocity response to maximal exercise [8]. Despite reporting no change in central stiffness, Heffernan et al. reported greater reductions in peripheral pulse wave velocity in CA than AA following maximal exercise, indicating that different segments of the arterial tree may not all respond similarly [8]. Importantly, the effects of exercise on central arterial stiffness may vary between the aorta and carotid arteries and between racial groups [9]. It is currently unknown whether aortic and carotid stiffness respond similarly in AA compared to CA, but, remains of interest considering each offers independent insight in CVD risk [10].

Racial differences in post-exercise central arterial stiffness may also relate to exercise-induced changes in blood pressure. Interestingly, AA do not exhibit central post exercise blood pressure reduction despite having similar resting values and brachial blood pressure responses [7]. The lack of central blood pressure response could be attributable to impaired peripheral vasodilation, thus preventing the reductions in blood pressure from occurring. To our knowledge, no studies have investigated racial differences in vasodilatory function following acute maximal aerobic exercise. Considering the beneficial effect of moderate- and high-intensity aerobic exercise on vasodilatory function [11] and inclusion of higher-intensity exercise in national guidelines for physical activity [12], it is important to understand whether AA experience differential responses to an acute bout of maximal aerobic exercise.

The purpose of this study was to evaluate vasodilatory function, central (carotid, aortic) arterial stiffness and hemodynamics following a maximal aerobic exercise test in young, healthy AA and CA adults. We hypothesized AA would 1) present a reduced vasodilatory response to exercise, 2) display greater increases in carotid stiffness and smaller reductions in aortic stiffness and 3) not exhibit blood pressure reductions compared to CA following maximal aerobic exercise.

Methods

Subjects

We performed an analysis of secondary outcomes from a previously published longitudinal exercise training intervention on racial differences in the arterial and hemodynamic response to aerobic training [13]. This study utilizes a cross-sectional analysis of the first testing day of the longitudinal study and was approved by the Institutional Review Board at the University of Illinois at Urbana-Champaign. Healthy African Americans and Caucasians between the ages of 18 and 35 were recruited for participation in this study. Participants were sedentary (not meeting physical activity guidelines [12]), free of CVD or respiratory disease, not taking any medication on a regular basis other than oral contraceptives, and were non-smokers. Other exclusion criteria have previously been described [13]. Women were tested during the early follicular phase of their menstrual cycle or during the placebo phase if they were on oral contraceptives. Prior to the testing session, participants were asked to fast for a minimum of 4 hours; refrain from physical activity, caffeine, or alcohol for 24 hours; and avoid anti-inflammatory medications for at least 3 days.

Study Design

Participants initially rested in the supine position for 15 minutes in a temperature-controlled room for baseline measures of hemodynamics and vascular function. Following baseline measures, a maximal exercise test was completed. Participants returned to the supine position post-exercise for recovery measures. Blood pressure, arterial stiffness, and augmentation index were assessed at 15 and 30 minutes after exercise [8]. Forearm resistance artery function was analyzed at 30 minutes only due to time constraints to complete all measures at 15 and 30 minutes.

Measurements

Anthropometrics—Height and weight were measured using a standard stadiometer and scale, respectively. Body mass index (BMI) was calculated as weight (kg) divided by height (m) squared.

Forearm Resistance Artery Function—Vasodilatory function of the forearm resistance arteries was assessed using strain-gauge plethysmography (EC-6, DE Hokanson, Inc, Bellevue, WA), as previously described [13]. In brief, a strain gauge was placed at the widest area of the forearm and resting forearm blood flow (FBF) was assessed. Immediately following, a 5-minute upper arm occlusion and last 1-minute wrist occlusion were performed. Changes in forearm volume were measured after rapid release of the upper arm cuff. Thirteen readings following cuff release were taken in 15 second cycles. Peak FBF was

recorded as the highest reading. Reactive hyperemia (RH) was determined as peak FBF minus resting FBF. Area under the curve (AUC) was used as a measure of total RH by plotting all 13 measures against time. Forearm vascular conductance (FVC) was determined as $\text{FBF}/\text{MAP} \times 100$. FBF is expressed as $\text{ml}^{-1} \times 100^{-1}$ ml of forearm tissue.

Central Pulse Wave Velocity—A high-fidelity strain-gauge transducer (Millar Instruments, Houston, TX) was used to obtain pressure waveforms from the left common carotid artery and left femoral artery for subsequent calculation of pulse wave velocity (PWV) (SphygmoCor, AtCor Medical, Sydney, Australia). Distances from the carotid artery to femoral artery and carotid artery to suprasternal notch were recorded. The distance from the carotid artery to sternal notch was subtracted from the carotid-femoral segment to adjust for differences in direction of the pulse wave. PWV was calculated using the distance between measurement points and the measured time delay between the proximal and distal foot waveforms. All measures were made in duplicate and the mean value used for analysis. In addition, PWV was normalized to brachial mean arterial pressure (MAP) for further analyses (PWV/MAP).

Carotid Artery Stiffness—Longitudinal carotid artery images were obtained 1 to 2 centimeters proximal to the bifurcation via ultrasonography (Aloka Alpha-10, Tokyo, Japan) with a 7.5 MHz linear array probe. Arterial compliance and β -stiffness index were determined using an automated wall detection echo-tracking software system. Measures were calibrated to carotid systolic and diastolic blood pressure obtained simultaneously from the contralateral carotid artery via applanation tonometry.

Brachial Blood Pressure—Brachial blood pressure was measured using an automated oscillometric device (HEM-907XL, Omron Corporation, Japan). All measures were repeated and the average of two values was recorded and used for analysis. If values differed by >5 mmHg, a third measurement was obtained and the two closest values used in analysis.

Pulse Wave Analysis—Radial artery pressure waveforms were obtained from a 10-second epoch using applanation tonometry (SphygmoCor, AtCor Medical, Sydney, Australia). Using a validated generalized transfer function, a central aortic pressure waveform was reconstructed to estimate aortic systolic (SBP) and diastolic blood pressure (DBP). Augmentation index (AIx) was determined as the difference between the first and second systolic peaks expressed as the percentage of central pulse pressure. Heart rate is known to influence AIx, therefore values were normalized to a heart rate of 75 bpm (AIx75).

Maximal Exercise Test—A graded protocol on a cycle ergometer was used to determine peak oxygen consumption, as previously described [8]. Participants started at 50W and wattage was increased every 2 minutes by 30W until volitional exhaustion. The test was terminated and considered a maximal effort when meeting 3 of the following criteria: 1) inability to maintain a 60 rpm pedal rate; 2) a respiratory exchange ratio (RER) >1.10 ; 3) achieving a heart rate (HR) within 10 bpm of age-predicted HR; 4) a final rating of perceived exertion of ≥ 17 on the Borg scale.

Statistical Analysis

All data are reported as mean and standard deviations. Normality was assessed with visual inspection and analyzed with the Kolmogorov-Smirnov test. Data were log transformed where necessary and reported as raw means for interpretation. Descriptive characteristics were tested for differences with independent t-tests for continuous variables and chi-square tests for categorical variables. Hemodynamics and vascular function were analyzed using two-tailed repeated measures analysis of variance (group x time) to determine if differential responses occurred following maximal aerobic exercise. When significant main effects were determined, post-hoc analysis with the Bonferroni adjustment were applied.

All analyses included sex as a covariate due to the much larger number of males in the CA group compared to AA group, likely influencing the differences in other baseline characteristics. In addition to statistically adjusting for sex, further analyses were conducted on a sample where groups were matched for sex, age, body mass index, and cardiorespiratory fitness to control for the differences in descriptive characteristics between groups (Supplemental Digital Content, Table 1 and 2).

Data analyses were performed using SPSS version 24 (IBM Corporation, Armonk, NY) and an *a priori* significance was set at $\alpha < 0.05$.

Results

Descriptive Characteristics

Out of the 83 participants in the study, 62 participants (27 AA, 11 male; 35 CA, 20 male) were included in our final analysis. Participants were excluded due to invalid max testing ($n=12$), a BMI > 40 ($n=3$), or incomplete vasodilatory function data ($n=6$). Descriptive characteristics are presented in Table 1. AA and CA were similar in age, weight, and BMI, but differed in height. CA had a greater $\text{VO}_{2\text{peak}}$ and HR_{peak} compared to AA, despite similar RER_{peak} . In our matched sample analysis, all differences in descriptive characteristics were removed except HR_{peak} (Supplementary Table 1).

Resistance Artery Function Following Maximal Exercise

In our full sample, resting FBF increased post-exercise in both AA (2.4 ± 1.1 to $2.7 \pm 1.1 \text{ ml}^{-1} \cdot 100^{-1} \text{ ml tissue}$) and CA (3.1 ± 1.7 to $3.4 \pm 1.4 \text{ ml}^{-1} \cdot 100^{-1} \text{ ml tissue}$) ($p=0.01$). Peak FVC following the release of the occlusion was higher in CA ($p=0.04$, Figure 1) but increased following exercise in both groups ($p=0.02$). Similar group differences were seen for AUC and peak blood flow ($p<0.05$, Figure 1), in which both parameters were increased following exercise in both groups ($p<0.05$). There were no interaction effects for any resistance artery function parameters, indicating a similar increase following maximal exercise in AA and CA. Importantly, our matched analysis also reported no interaction effects for any resistance artery function parameters ($p>0.05$, Supplementary Table 2). Post-exercise increases in FBF, peak blood flow, and AUC were still evident, as well as group differences in AUC ($p<0.05$). An exercise effect was also now noted for reactive hyperemia, which increased following exercise ($p<0.05$). The matched analysis, however, did remove all

effects on FVC and group differences were no longer evident in peak blood flow ($p>0.05$).

Hemodynamic Response to Maximal Exercise

Brachial and aortic SBP were reduced in CA following exercise but did not change in AA (interaction effect: $p<0.05$, Table 2). There was no change in brachial DBP in CA or AA following exercise ($p>0.05$). MAP was reduced in CA at 30-min compared to baseline and 15-min post, but MAP was maintained in AA (interaction effect: $p=0.04$). The effects of maximal exercise on blood pressure outcomes were not altered in our matched sample (Supplementary Table 2).

Local arterial stiffness of the carotid artery changed following exercise. In both groups, β -stiffness index increased at 15- and 30-min ($p=0.02$), whereas arterial compliance was reduced at 15-min ($p<0.01$, Table 2). Interestingly, central arterial stiffness, assessed by PWV, did not change following maximal exercise ($p>0.05$). However, after controlling for MAP, there was a reduction in PWV in the full sample ($p=0.04$), but not in the matched sample.

AA had higher AIx than CA ($p<0.01$, Table 2). Both groups showed a reduction in AIx following maximal exercise at 15- and 30-min ($p=0.01$). However, heart rate was elevated at both 15- and 30-min following exercise ($p<0.01$) and was higher in CA ($p=0.03$, in full sample only). After controlling for heart rate at 75 bpm (AIx@75), both the group and time effect disappeared ($p>0.05$).

Discussion

The main findings of the present investigation were 1) although AA had lower resistance artery function overall, in comparison to CA, resistance artery function responded similarly to maximal aerobic exercise in both AA and CA and 2) local and central arterial stiffness responded differently to maximal exercise in both groups depending on where arterial stiffness was measured, with increases in carotid stiffness and reductions in central pulse wave velocity after controlling for mean arterial pressure. Furthermore, our study supports previous literature showing only CA experience post-exercise blood pressure reductions following a maximal exercise bout.

Following exercise there is a biphasic response in endothelial function, in which immediately following exercise endothelial function is reduced and then returns to baseline or improves when measured at least 30 minutes following [14]. This response is highly dependent on the duration and intensity of the exercise bout, however, in line with this hypothesis, vasodilatory function was improved following the acute exercise bout in this study. Alternatively, contrary to our hypothesis, this response was not different between AA and CA.

At rest, AA have reduced vasodilatory capacity of resistance arteries when compared with CA [2]. Furthermore, vasodilatory capacity is attenuated in young AA compared to CA in response to several vasoactive drugs, indicative of a global impairment in vasodilation [15–17]. The reduced vasodilatory capacity in AA has been attributed to reduced levels of nitric oxide bioavailability and increased oxidative stress [18]. However, the stimulus of acute

high-intensity aerobic exercise did not result in further racial differences in our study. This finding indicates that with maximal aerobic exercise, both CA and AA were able to increase vasodilatory capacity to a similar extent. However, the vasodilatory capacity of AA was still lower than that of CA.

Our study did not confirm racial differences in central or local arterial stiffness, as has previously been reported at rest and following exercise in young AA and CA [2,8,19]. With maximal exercise, local carotid stiffness was increased at 15- and 30-minutes post, whereas central PWV did not change unless we accounted for mean arterial pressure. After controlling for arterial pressure, the expected reduction in PWV following aerobic exercise occurred in the full sample [20]. However, we did not see the expected reduction in our matched analyses. This may have been a function of the smaller sample size in the matched analyses. Our findings add to the conflicting literature on racial differences in central stiffness after maximal exercise. Others have reported central pulse wave velocity increased in AA and decreased in CA following maximal aerobic exercise [7], but has also been reported to not change in either race [8]. Future studies with larger samples sizes and standardized exercise protocols are needed to better address this discrepancy.

Interestingly, we observed divergent responses between carotid and aortic stiffness in both races. Beta stiffness index is considered a blood pressure independent measure [21], whereas pulse wave velocity is blood pressure dependent [22,23]. However, controlling pulse wave velocity for blood pressure still lead to the opposite response of that observed for carotid beta stiffness index. Indeed, it appears that the different arterial segments respond differently to acute maximal exercise. It is possible that the different segments of the arterial tree responds differently to increased sympathetic stimulation (detectable due to the elevated heart rate at 15- and 30-minutes), or, the wall composition may respond differently to the change in exercise hemodynamics. Differential responses have been previously reported following submaximal aerobic exercise with increases in aortic stiffness, with no changes in carotid stiffness [24]. However, the increase in carotid stiffness in this study may be a protective mechanism to dampen pulsatile pressure and flow transmission to the brain, as maximal aerobic exercise would create a much larger pressure stimulus than submaximal exercise. Further research is needed to investigate why disparate responses occur.

In line with previous literature, AA in the present study did not exhibit reductions in blood pressure. We show a reduction in central and peripheral systolic blood pressure as well as mean arterial pressure in CA following the maximal exercise stimulus, with no change in AA. One previous study has reported greater reductions in central systolic and mean arterial pressure in CA compared with AA men following maximal aerobic exercise at 30 minutes [7]. However, to our knowledge, there are no other studies reporting racial differences at 30 minutes-post maximal exercise in brachial blood pressures [7,8]. Racial differences in brachial blood pressures have been reported following moderate intensity aerobic exercise in which young AA women did not change blood pressure [25] or increased systolic blood pressure [26]. Taken together, results suggest that blood pressure reduction does not occur in AA. Our results suggest that this is not due to impaired vasodilatory responsiveness to the exercise bout.

Sex played a significant role as a covariate in our analyses and this is an important finding of the current study. Sex differences are increasingly important in cardiovascular physiology and have been previously suggested in the hemodynamic and arterial stiffness response to exercise between African-Americans and Caucasians [7]. Future research with adequate power should explore these potential differential responses in vasodilatory function.

Limitations to this study should be noted. This study is cross-sectional and utilized young and healthy adults. Therefore these results may not be directly applicable to an older population or individuals with disease. Furthermore, due to time constraints of measures, we were unable to capture measures immediately, i.e. 0–5 minutes, following exercise, or, measure vasodilatory function at the immediate or 15-minutes post-exercise point to see if the biphasic response was evident in both CA and AA. Additionally, our methodology does not provide information on potential mechanisms.

Conclusion

In conclusion, AA and CA had similar decreases in central arterial stiffness, increases in local arterial stiffness, and increases in resistance artery vasodilatory function following maximal aerobic exercise. AA, however, did not display reductions in blood pressure after maximal exercise and had lower vasodilatory function in comparison to CA. Our results suggest AA exhibit similar vasodilatory responses following maximal aerobic exercise as CA, and therefore vasodilatory function likely does not explain the lack of post-exercise blood pressure reductions in AA.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Funding:

National Institute of Health - NHLBI 1R01HL093249-01A1

References

1. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, et al. Heart Disease and Stroke Statistics-2017 Update: A Report from the American Heart Association. *Circulation* 2017; 135:e146–e603. [PubMed: 28122885]
2. Heffernan KS, Jae SY, Wilund KR, Woods JA, Fernhall B. Racial differences in central blood pressure and vascular function in young men. *Am J Physiol Heart Circ Physiol* 2008; 295:2380–2387.
3. Campia U, Choucair WK, Bryant MB, Waclawiw MA, Cardillo C, Panza JA. Reduced endothelium-dependent and -independent dilation of conductance arteries in African Americans. *J Am Coll Cardiol* 2002; 40:754–760. [PubMed: 12204507]
4. Townsend RR, Wilkinson IB, Schiffrin EL, Avolio AP, Chirinos JA, Cockcroft JR, et al. Recommendations for Improving and Standardizing Vascular Research on Arterial Stiffness: A Scientific Statement from the American Heart Association. *Hypertension* 2015; 66:698–722. [PubMed: 26160955]
5. Brunner H, Cockcroft JR, Deanfield J, Donald A, Ferrannini E, Halcox J, et al. Endothelial function and dysfunction. Part II: Association with cardiovascular risk factors and diseases. A statement by

the Working Group on Endothelins and Endothelial Factors of the European Society of Hypertension. *J Hypertens* 2005; 23:233–246. [PubMed: 15662207]

6. Particone F, Ceravolo R, Pujia A, Ventura G, Iacopino S, Scozzafava A, et al. Prognostic significance of endothelial dysfunction in hypertensive patients. *Circulation* 2001; 104:191–196. [PubMed: 11447085]
7. Yan H, Ranadive SM, Heffernan KS, Lane AD, Kappus RM, Cook MD, et al. Hemodynamic and arterial stiffness differences between African-Americans and Caucasians after maximal exercise. *AJP Hear Circ Physiol* 2014; 306:H60–H68.
8. Heffernan KS, Jae SY, Fernhall B. Racial differences in arterial stiffness after exercise in young men. *Am J Hypertens* 2007; 20:840–845. [PubMed: 17679030]
9. Melo X, Fernhall B, Santos DA, Pinto R, Pimenta NM, Sardinha LB, et al. The acute effect of maximal exercise on central and peripheral arterial stiffness indices and hemodynamics in children and adults. *Appl Physiol Nutr Metab* 2016; 41:266–276. [PubMed: 26842667]
10. van Sloten TT, Sedaghat S, Laurent S, London GM, Pannier B, Ikram MA, et al. Carotid stiffness is associated with incident stroke: A systematic review and individual participant data meta-analysis. *J Am Coll Cardiol* 2015; 66:2116–2125. [PubMed: 26541923]
11. Siasos G, Athanasiou D, Terzis G, Stasinaki A, Oikonomou E, Tsitkanou S, et al. Acute effects of different types of aerobic exercise on endothelial function and arterial stiffness. *Eur J Prev Cardiol* 2016; 23:1565–1572. [PubMed: 27121699]
12. United States. Dept. of Health and Human Services. 2008 Physical Activity Guidelines for Americans. ; 2008.
13. Ranadive SM, Yan H, Lane AD, Kappus RM, Cook MD, Sun P, et al. Aerobic exercise training and arterial changes in African-Americans versus Caucasians. *Med Sci Sport Exerc* 2016; 48:90–97.
14. Dawson EA, Green DJ, Timothy Cable N, Thijssen DHJ. Effects of acute exercise on flow-mediated dilatation in healthy humans. *J Appl Physiol* 2013; 115:1589–1598. [PubMed: 24030665]
15. Lang CC, Stein CM, Brown RM, Deegan R, Nelson R, He HB, et al. Attenuation of isoproterenol-mediated vasodilatation in blacks. *N Engl J Med* 1995; 333:155–160. [PubMed: 7791817]
16. Stein CM, Lang CC, Nelson R, Brown M, Wood AJJ. Vasodilation in black Americans: Attenuated nitric oxide-mediated responses. *Clin Pharmacol Ther* 1997; 62:436–443. [PubMed: 9357395]
17. Ozkor MA, Rahman AM, Murrow JR, Kavtaradze N, Lin J, Manatunga A, et al. Differences in vascular nitric oxide and endothelium-derived hyperpolarizing factor bioavailability in blacks and whites. *Arterioscler Thromb Vasc Biol* 2014; 34:1320–1327. [PubMed: 24675657]
18. Kalinowski L, Dobrucki IT, Malinski T. Race-specific differences in endothelial function: Predisposition of African Americans to vascular diseases. *Circulation* 2004; 109:2511–2517. [PubMed: 15159296]
19. Baldo MP, Cunha RS, Ribeiro ALP, Lotufo PA, Chor D, Barreto SM, et al. Racial differences in arterial stiffness are mainly determined by blood pressure levels: Results from the ELSA-Brasil Study. *J Am Heart Assoc* 2017; 6:e005477. [PubMed: 28637779]
20. Mutter AF, Cooke AB, Saleh O, Gomez Y-H, Daskalopoulou SS. A systematic review on the effect of acute aerobic exercise on arterial stiffness reveals a differential response in the upper and lower arterial segments. *Hypertens Res* 2017; 40:146–172. [PubMed: 27733765]
21. Kawasaki T, Sasayama S, Yagi SI, Asakawa T, Hirai T. Non-invasive assessment of the age related changes in stiffness of major branches of the human arteries. *Cardiovasc Res* 1987; 21:678–687. [PubMed: 3328650]
22. Fitch RM, Vergona R, Sullivan ME, Wang YX. Nitric oxide synthase inhibition increases aortic stiffness measured by pulse wave velocity in rats. *Cardiovasc Res* 2001; 51:351–358. [PubMed: 11470475]
23. Nichols WW, O'Rourke MF, Vlachopoulos C. McDonald's Blood Flow in Arteries: Theoretical, Experimental, and Clinical Principles. 6th ed. Boca Raton, FL: CRC Press; 2011.
24. Lefferts WK, DeBlois JP, Receno CN, Barreira TV, Brutsaert TD, Carhart RL, et al. Effects of acute aerobic exercise on arterial stiffness and cerebrovascular pulsatility in adults with and without hypertension. *J Hypertens* 2018; :1. [PubMed: 29076922]

25. Enweze L, Oke LM, Thompson T, Obisesan TO, Blakely R, Adams RG, et al. Acute exercise and postexercise blood pressure in African American women. *Ethn Dis* 2007; 17:664–8. [PubMed: 18072376]
26. Pescatello LS, Bairos L, VanHeest JL, Maresh CM, Rodriguez NR, Moyna NM, et al. Postexercise hypotension differs between white and black women. *Am Heart J* 2003; 145:364–370. [PubMed: 12595857]

Figure 1. Change in resistance artery function following maximal exercise. No significant interaction effects were observed. *Group effect, $p < 0.05$; ^Time effect, $p < 0.05$.

Table 1.

Descriptive Characteristics

	African-American (n = 27)	Caucasian (n = 35)	p-value
Age, years	25 (4)	24 (4)	0.52
Sex, male/female	11/16	20/15	0.20
Height, cm	167.2 (9.0)	172.6 (9.5)	0.03
Weight, kg	75.3 (17.2)	74 (15.1)	0.75
BMI, kg/m ²	26.8 (5.0)	24.7 (4)	0.07
VO ₂ peak, ml/kg/min	30.6 (6.7)	36.8 (8.3)	<0.01
HRpeak, bpm	180 (13)	189 (10)	<0.01
RERpeak, (L/min)/(L/min)	1.20 (0.08)	1.19 (0.07)	0.84

All data presented as mean (SD).

BMI: body mass index; HR: heart rate; RER: respiratory exchange ratio

Table 2.

Hemodynamic response to maximal exercise.

		n	Baseline	15-min	30-min	Time	Group	Interaction
β -stiffness, a.u. *	AA	26	5.8 (2.4)	6.2 (2.0)	5.8 (2.4)	0.02^{ab}	0.99	0.31
	CA	32	5.4 (1.3)	6.7 (2.2)	6.0 (1.7)			
Arterial Compliance, mm ² /kPa *	AA	26	1.17 (0.37)	1.00 (0.36)	1.15 (0.31)	0.00^a	0.74	0.74
	CA	32	1.16 (0.31)	0.96 (0.42)	1.09 (0.31)			
Central PWV, m/s *	AA	24	5.8 (1.1)	5.7 (1.2)	5.8 (1.1)	0.07	0.59	0.54
	CA	32	5.9 (1.1)	5.7 (0.9)	5.6 (0.9)			
PWV/MAP, m/s•mmHg ⁻¹	AA	23	0.069 (0.01)	0.064 (0.01)	0.068 (0.01)	0.04	0.99	0.58
	CA	32	0.068 (0.01)	0.067 (0.01)	0.067 (0.01)			
Aortic SBP, mmHg	AA	24	104 (9)	105 (11)	104 (12)	0.12	0.04	0.02
	CA	32	103 (9)	101 (10)	99 (10) ^b			
Brachial SBP, mmHg	AA	26	120 (12)	124 (15)	122 (14)	0.01	0.16	0.00
	CA	34	123 (11)	120 (12)	116 (13) ^{bc}			
Brachial DBP, mmHg	AA	26	71 (8)	74 (8)	73 (10)	0.27	0.11	0.59
	CA	34	69 (9)	70 (11)	69 (9)			
Brachial MAP, mmHg	AA	26	88 (8)	90 (10)	89 (10)	0.05	0.11	0.04
	CA	34	87 (9)	87 (11)	84 (9) ^{bc}			
Heart rate, bpm	AA	24	63 (9)	81 (11)	77 (11)	0.00^{abc}	0.03	0.25
	CA	31	65 (10)	88 (10)	81 (9)			
AIx, %	AA	24	9.9 (13.1)	-0.8 (15.9)	0.0 (13.4)	0.00^{ab}	0.03	0.75
	CA	31	2.1 (11.6)	-7.4 (9.1)	-8.3 (11.2)			
AIx@75, %	AA	24	3.9 (13.3)	2.0 (14.8)	0.8 (11.7)	0.16	0.19	0.52
	CA	31	-2.6 (11.1)	-0.9 (8.5)	-4.5 (10.9)			

All data reported as mean (SD) and controlled for sex. AIx: augmentation index; a.u.: arbitrary unit; DBP: diastolic blood pressure; MAP: mean arterial pressure; PWV: pulse wave velocity; SBP: systolic blood pressure

^a15-min different from baseline, p<0.05;

^b30-min different from baseline, p<0.05;

^c30-min different from 15-min, p<0.05

* Analysis on log transformed data