Racial Differences in Arterial Stiffness After Exercise in Young Men

Kevin S. Heffernan, Sae Young Jae, and Bo Fernhall

**Background:** African-American men have a higher prevalence of hypertension compared with their white American counterparts. African-American men also develop high blood pressure sooner, and this may manifest as early as their second decade of life. One consequence or potential mechanism for the underlying racial differences in hypertension is detrimental alterations in arterial mechanical function. African Americans have higher arterial stiffness than white Americans, and this is directly related to hypertensive target-organ damage. Central aortic stiffness is currently recognized as an independent predictor of stroke and other negative cardiovascular outcomes, and, indeed, African-Americans have a higher incidence of both stroke and coronary heart disease when compared with white Americans. Although both high blood pressure and aging contribute to increases in arterial stiffness, this phenomenon was reported in normotensive African-American men as young as 21 years of age.

Maximal aerobic exercise testing can be used to differentiate physiologic responses between groups that may not be apparent at rest. This form of exercise was also shown to acutely reduce peripheral muscular artery stiffness. African-American men have reduced vasodilation and heightened vasoconstriction in response to various forms of adrenergic stimulation. Given that African-American men have greater tonic arterial stiffness and blunted vasodilatation in response to adrenergic stimulation, reductions in stiffness after aerobic exercise may also be attenuated.

The purpose of this study was to examine arterial stiffness after a maximal aerobic exercise test in young African-American and white American men. We hypothesized that reductions in peripheral arterial stiffness after exercise would be attenuated in African-American men.

**Methods**

**Subjects**

Twenty-four young, healthy men (12 African American and 12 white) volunteered for this study. None reported the use of any medication known to affect heart rate or blood pressure. All subjects were free of cardiovascular, metabolic, endocrine, and pulmonary diseases.

**Methods**

**Subjects**

Twenty-four young, healthy men (12 African American and 12 white) volunteered for this study. None reported the use of any medication known to affect heart rate or blood pressure. All subjects were free of cardiovascular, metabolic, endocrine, and pulmonary diseases.
metabolic, renal, or respiratory disease as self-reported via a medical history questionnaire, and none smoked. Subjects were self-defined as African American if they reported that both parents were of African descent. African-American and white men were matched for physical activity, as self-reported on a questionnaire. All subjects were recruited from the local university student population between August 2005 and October 2006. The response rate was similar between African-American and white participants. All subjects gave written consent. This study was approved by the Institutional Review Board of the University of Illinois at Urbana-Champaign.

Study Design
All participants were at least 3 h postprandial and did not consume caffeine or exercise for 24 h before testing. After written consent had been provided, body anthropometrics were obtained. Participants were then required to rest in supine position for 10 min in a temperature-controlled room. Resting blood pressure and central and peripheral pulse-wave velocity (PWV) measures were made at rest and at 15 min and 30 min after maximal cycling exercise. All measurements were carried out at the same time of day to reduce the potential effects of diurnal variations in physiologic parameters.

Anthropometrics
Height and weight were measured using a stadiometer (to the nearest 0.5 cm) and a beam balance platform scale, respectively. Body mass index (BMI) was calculated as: weight (kg) divided by height (m) squared. Waist circumference was measured using a tape measure at the level of the umbilicus.

Brachial-Artery Blood-Pressure Assessment
Resting blood pressure was measured via sphygmomanometry with subjects in supine position in accordance with established guidelines, using a standing mercury-column manometer, stethoscope, and blood-pressure cuff. Measurements were made in duplicate, and the average value was recorded for data analysis. If the two values differed by >5 mm Hg, a third measure was taken, and the closest two of the three recorded values were used for subsequent analysis. The same experienced technician conducted all auscultatory blood-pressure measurements in all subjects.

Regional Arterial Stiffness and Pulse-Wave Velocity
A high-fidelity strain-gauge transducer (Millar Instruments, Houston, TX) was used to obtain a pressure waveform for a 10-sec epoch from: (1) the right common carotid artery and the right femoral artery, and (2) the right femoral artery and the ipsilateral superior dorsalis pedis artery. Distances from the carotid-artery sampling site to the femoral artery, carotid artery to the suprasternal notch, and femoral artery to the superior dorsalis pedis artery were measured as straight lines with a tape measure. The distance from the carotid artery to the sternal notch was then subtracted from the carotid-femoral segment length to account for differences in the direction of pulse-wave propagation. The peak of an in-phase R wave, as attained from sequential electrocardiogram (ECG) monitoring (CM5 configuration) was used as a timing marker. The ECG recordings were also used to obtain the resting and recovery heart rate. The PWV was calculated from the distances between measurement points and the measured time delay (Δt) between proximal and distal foot waveforms, using a validated and reproducible algorithm. This technique was previously shown to be highly reproducible. In our laboratory, the test-retest repeatability for resting PWV, calculated on 2 separate days 1 week apart, is >0.90. The intraclass correlation coefficient (a measure of internal consistency) for postmaximal exercise PWV in our laboratory, calculated on 2 separate days, is also very high.

Maximal Aerobic Capacity
Peak oxygen consumption (VO2 peak) was assessed using a graded cycle ergometry protocol. After a brief warm-up consisting of cycling against no resistance (0 W) for 2 min, subjects began cycling at 50 W. Every 2 min, the exercise intensity was increased by 30 W until volitional fatigue was reached. A Polar Heart Rate Monitor was used to measure heart rate once per minute during the protocol (Polar Electro, Inc., Woodbury, NY). Ratings of perceived exertion (RPE) were also assessed once per stage. Expired gases were analyzed using a Quark b2 breath-by-breath metabolic system (Cosmed, Rome, Italy). The test was terminated when three of the following four criteria were present: (1) a final RPE score of ≥17 on the Borg scale (scale of 6 to 20), (2) a respiratory exchange ratio (RER) >1.15, (3) no change in heart rate (HR) with a change in workload, or (4) a “plateau” (increase of no more than 150 mL) in oxygen uptake with an increase in workload.

Statistics
All data are reported as mean ± SEM. A priori significance was set at alpha < .05. Data analysis was performed using the Statistical Package for the Social Sciences (SPSS, version 12.0.1; SPSS, Inc., Chicago, IL). Significant differences for descriptive variables (Table 1) between African-American and white men were assessed by one-way analysis of variance (ANOVA). When assessing group differences in resting central and peripheral PWV, an analysis of covariance (ANCOVA) was also performed, with potential confounders (HR and mean arterial pressure [MAP]) entered as covariables.

To assess group differences after exercise in PWV, ANCOVA was performed at both the 15-min and 30-min time points. The PWV at the desired time point (15 min or 30 min) was entered as the dependent variable, race as the
fixed factor, and resting PWV as the covariate. This process was then repeated with systolic blood pressure (SBP), diastolic blood pressure (DBP), MAP, and HR.

A 2 × 3 (2 groups × 3 time points) ANCOVA with repeated measures (covarying for HR and MAP) was also used to assess differences in central and peripheral PWV over time. A 2 × 3 (2 groups × 3 time points) ANOVA with repeated measures was used to assess differences in all other dependent variables over time. Whenever significant interactions were detected, Fisher’s least significant difference was used for post hoc multiple comparisons.

Results

All subject characteristics are presented in Table 1. There were no group differences in age, height, weight, waist circumference, BMI, body surface area, exercise test time, VO2peak, SBP, DBP, and resting peripheral PWV (P > 0.05). African-American men had a significantly higher resting central PWV compared with their white peers. This relationship prevailed after adjusting for MAP and HR (P < .05).

According to ANCOVA, there were racial differences in SBP at 15 min after exercise (P < .05) after covarying for resting SBP. There were no racial differences in SBP at 30 min after exercise after covarying for resting SBP. According to ANCOVA, there were also racial differences in peripheral PWV at 15 min after exercise and 30 min after exercise (P < .05) after covarying for resting peripheral PWV. There were no racial differences in central PWV, MAP, DBP, and HR at 15 min after exercise or 30 min after exercise (P < .05) after covarying for resting central PWV, resting MAP, resting DBP, and resting HR, respectively.

Table 2 presents results from the repeated-measures ANOVA before and after acute exercise. A group-by-time interaction was detected for SBP, because SBP was significantly elevated above resting values in white men 15 min after exercise, while there was no change in SBP in African-American men at any time point (P < .05). The SBP returned to resting values 30 min after exercise in white men. The DBP decreased similarly 15 min after exercise in both groups and returned to pre-exercise values by 30 min after exercise. Both groups experienced a similar drop in MAP 15 min after exercise (P < .05) that returned to resting values by 30 min after exercise. Heart rate remained above resting values in both groups at both 15 min (P < .05) and 30 min (P < .05) after exercise.

As shown in Fig. 1 (top), repeated-measures ANCOVA revealed no change in central PWV in either group after exercise. Thus, the group effect seen at rest remained at all time points after exercise. As shown in Fig. 1 (bottom), a group-by-time interaction was detected for peripheral PWV, because there were significant reductions in white men at 15 min and 30 min after exercise, with no change in African-American men (P < .05).

### Table 1. Subjects’ characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>African American (n = 12)</th>
<th>White (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>22 ± 1</td>
<td>22 ± 1</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>178 ± 2</td>
<td>180 ± 2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>88 ± 3</td>
<td>93 ± 4</td>
</tr>
<tr>
<td>Body surface area</td>
<td>2.0 ± 0.05</td>
<td>2.1 ± 0.04</td>
</tr>
<tr>
<td>Body mass index</td>
<td>27.3 ± 1.2</td>
<td>28.7 ± 1.0</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>87.8 ± 3.5</td>
<td>87.8 ± 1.7</td>
</tr>
<tr>
<td>Central PWV (m/sec)</td>
<td>7.3 ± 0.3</td>
<td>5.7 ± 0.3*</td>
</tr>
<tr>
<td>Peripheral PWV (m/sec)</td>
<td>8.2 ± 0.4</td>
<td>8.4 ± 0.4</td>
</tr>
<tr>
<td>Exercise test time (min)</td>
<td>12.9 ± 0.5</td>
<td>14.3 ± 0.8</td>
</tr>
<tr>
<td>VO2peak (ml/kg/min)</td>
<td>32.5 ± 1.1</td>
<td>34.6 ± 1.0</td>
</tr>
</tbody>
</table>

PWV = pulse-wave velocity; VO2peak = peak oxygen consumption. * Significant group difference (P < .05).

### Table 2. Brachial blood pressures and heart rate before and after exercise

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before exercise</th>
<th>Post 1</th>
<th>Post 2</th>
<th>P for interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>125 ± 3</td>
<td>122 ± 2</td>
<td>122 ± 2</td>
<td>0.016</td>
</tr>
<tr>
<td>White</td>
<td>123 ± 3</td>
<td>128 ± 2*</td>
<td>121 ± 2</td>
<td></td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>80 ± 2</td>
<td>71 ± 3*</td>
<td>79 ± 1</td>
<td>NS</td>
</tr>
<tr>
<td>White</td>
<td>77 ± 2</td>
<td>64 ± 3*</td>
<td>80 ± 1</td>
<td></td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>94 ± 2</td>
<td>87 ± 2*</td>
<td>92 ± 1</td>
<td>NS</td>
</tr>
<tr>
<td>White</td>
<td>92 ± 2</td>
<td>84 ± 2*</td>
<td>92 ± 1</td>
<td></td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>60 ± 3</td>
<td>93 ± 3*</td>
<td>76 ± 3*</td>
<td>NS</td>
</tr>
<tr>
<td>White</td>
<td>61 ± 3</td>
<td>96 ± 3*</td>
<td>83 ± 3*</td>
<td></td>
</tr>
</tbody>
</table>

SBP = systolic blood pressure; MAP = mean arterial pressure; NS = not significant; DBP = diastolic blood pressure.

* Significantly different from values before exercise (P < .05).
The main finding of the present investigation is of a racial difference in the peripheral arterial response to acute exercise. Our hypothesis was supported, because young African-American men had an attenuated reduction in peripheral artery stiffness compared with a group of white peers of similar age, resting blood pressure and HR, BMI, and cardiorespiratory fitness. To our knowledge, this is the first study to report a racial difference in arterial stiffness in response to exercise.

African-American men have reduced vasodilatation and heightened vasoconstriction in response to various forms of adrenergic stimulation. Infusion of isoproterenol was shown to decrease arterial stiffness in white but not black participants, suggesting an attenuated relaxation of large arteries in response to beta-2 adrenoreceptor stimulation, and our findings after exercise support this contention. This may be mediated by reduced beta-2 adrenoreceptor sensitivity in African-American men. The vasodilatory capacity of resistance arteries was shown to be attenuated in young (22 year old) African-American men, suggesting early onset of microvascular dysfunction. Forearm blood flow is significantly attenuated in young black versus white men after separate infusions of isoproterenol, methacholine, acetylcholine, bradykinin, and sodium nitroprusside, signifying a global impairment of endothelial-dependent and endothelial-independent vasodilation. Given that there were no differences in peripheral PWV at rest, it is likely that our present findings reflect racial differences in peripheral vascular functional properties. Impaired vascular smooth muscle relaxation after acute exercise may contribute to an attenuated reduction in vessel-wall stiffness of peripheral muscular arteries, thus altering vessel-wall load-bearing properties.

There were no group differences in resting peripheral artery stiffness in the present study. Chaturvedi et al noted increased peripheral PWV in older (40 to 64 years) African Caribbeans compared with white Europeans. However, this was completely attributable to higher resting and ambulatory blood pressure in the African-Caribbean group. Our findings would suggest that young, normotensive African-American men exhibit similar resting peripheral arterial stiffness compared with their white peers.

Our finding of increased resting central PWV is consistent with previous reports of lower vascular compliance and greater vascular stiffness (the inverse of compliance) in African-American compared with white men. Ferreira et al noted greater aortic stiffness in young normotensive (mean age, 24 years) and hypertensive (mean age, 28 years) black men compared with their white counterparts. Din-Dzietham et al also noted that carotid artery compliance (measured with carotid ultrasonography) was lower in African Americans. Reduced arterial compliance was reported in normotensive African-American males as young as 21 years of age. African-American boys as young as 12 years of age exhibit higher pulse pressure, a surrogate marker of arterial stiffness, compared with their young white peers. Our data support these previous findings, showing that young, normotensive African-American men exhibit increased resting central arterial stiffness compared with their white peers, and this may likely be mediated via structural differences in vessel-wall composition. Large central elastic artery stiffness does not appear to be affected by maximal aerobic exercise. Moreover, the central arterial response to acute exercise does not seem to vary with race.

While the central and peripheral arterial trees are not detached, only central aortic stiffness was found to be associated with negative health outcomes. There are no such studies linking peripheral artery stiffness with morbidity and mortality. Pannier et al examined both central (aortic) and peripheral (brachial and femoral) artery stiffness in patients with end-stage renal disease and discovered that only central artery stiffness was a strong and independent predictor of cardiovascular mortality. In patients with type 2 diabetes, peripheral artery stiffness was associated with the peripheral circulation. Whether peripheral stiffness after exercise may hold prognostic significance in apparently healthy populations remains to be elucidated. Recovery of other physiologic parameters after exercise testing, such as HR, was associated
with an inflammatory state and atherosclerosis. Future research is warranted to examine the predictive significance of peripheral arterial responsiveness to acute exercise testing.

Resting PWV may be influenced by several factors, including HR and blood pressure. Given that similar reductions in MAP and similar increases in HR occurred in both groups after exercise, whereas only the white group exhibited a significant reduction in peripheral PWV, changes in peripheral PWV in the present study likely reflect racial differences in vessel-wall properties after exercise and are not secondary to alterations in these other factors. We noted a slight increase in SBP in the white cohort 15 min after exercise, despite a large reduction in peripheral PWV. Although resting brachial pressures are strong correlates of resting PWV, it appears that postexercise brachial pressures are not related to postexercise PWV. Naka et al concluded that although alterations in exercise brachial pressures are not related to postexercise factors. We noted a slight increase in SBP in the white group after exercise, whereas only the white group exhibited a significant reduction in peripheral PWV, both groups after exercise, whereas only the white group exhibited a significant reduction in peripheral PWV.

The strength of the present study lay in having subjects matched for aerobic fitness. A strong inverse relationship exists between cardiorespiratory fitness and arterial stiffness. Thus, fitness level may be a strong confounder when comparing young individuals of potentially varying activity levels. According to the present findings, it appears that potential differences in cardiorespiratory fitness status may not explain racial differences in arterial-wall properties.

A limitation of the present study lay in not measuring and controlling for other cardiovascular comorbidities. Indeed, factors such as hypercholesterolemia, blood glucose levels, and central adiposity were shown to affect arterial stiffness. It is possible that differences between groups may have been mediated by preexisting conditions. We noted no group differences in waist circumference, an index of central adiposity. According to BMI, our participants were overweight. Thus, we are limited in our ability to extrapolate our findings to a normal-weight population. Finally, we did not control for socioeconomic status. However, all subjects were of a similar level of education, insofar as all participants were currently enrolled university students.

In conclusion, young African-American men have greater resting central artery stiffness and an attenuated reduction in peripheral artery stiffness after acute exercise when compared with white peers of similar age, resting blood pressure, BMI, and cardiorespiratory fitness. A standard, graded exercise test may be used in young men as a noninvasive perturbation to delineate racial differences in peripheral arterial-wall properties that may not be apparent at rest. Future studies are needed to examine the potential mechanisms and clinical relevance of these differences in vascular structure or function.

References


