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Racial Differences in Arterial Stiffness and Microcirculatory Function Between Black and White Americans

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Background—Compared with whites, black Americans suffer from a disproportionate burden of cardiovascular disease (CVD). We hypothesized that racial differences in the prevalence of CVD could be attributed, in part, to impaired vascular function in blacks after adjustment for differences in risk factor burden.

Methods and Results—We assessed vascular function in 385 black and 470 white subjects (mean age, 48±11 years; 45% male). Using digital pulse amplitude tonometry (EndoPAT) we estimated the reactive hyperemia index (RHI), a measure of microvascular endothelial function, and peripheral augmentation index (PAT-AIx). Central augmentation index (C-AIx) and pulse-wave velocity (PWV) were measured as indices of wave reflections and arterial stiffness, respectively, using applanation tonometry (Sphygmocor). Compared with whites, blacks had lower RHI (2.1±0.6 versus 2.3±0.6, P<0.001), greater arterial wave reflections assessed as both PAT-AIx (20.4±21.5 versus 17.0±22.4, P=0.01) and C-AIx (20.8±12.3 versus 17.5±13.3, P=0.001), and greater arterial stiffness, measured as PWV (7.4±1.6 versus 7.1±1.6 m/s, P=0.001). After adjustment for traditional CVD risk factors, black race remained a significant predictor of lower RHI and higher PAT-AIx and C-AIx (all P<0.001) in all subjects and of higher PWV in men (P=0.01). Furthermore, these associations persisted in a subgroup analysis of “healthy” individuals free of CVD risk factors.

Conclusion—Black race is associated with impaired microvascular vasodilatory function, and greater large arterial wave reflections and stiffness. Because impairment in these vascular indices may be associated with worse long-term outcomes, they may represent underlying mechanisms for the increased CVD risk in blacks. (J Am Heart Assoc. 2013;2:e002154 doi: 10.1161/JAHA.112.002154)

Key Words: arterial stiffness • racial disparities • reactive hyperemia

尽管近期几十年来，美国心血管疾病（CVD）的总体死亡率呈下降趋势，但黑人与白人的CVD死亡率和发病率仍然存在显著差异。这些观察结果主要由少数传统CVD危险因素所致。

**背景**——与白人相比，黑人美国人在心血管疾病（CVD）方面的负担不成比例。我们假设CVD的发病率在黑人中可能部分归因于动脉僵硬度和微循环功能的差异。

**方法和结果**——我们对385名黑人和470名白人（平均年龄48±11岁；45%男性）进行了血管功能评估。使用数字脉冲幅度调制技术（EndoPAT）来估计反应性血流指数（RHI），这是一个评估微血管内皮功能的指标，以及脉冲波速度（PWV）作为波反射和动脉僵硬度的指标，分别使用应用内皮调制技术（Sphygmocor）。与白人相比，黑人的RHI更低（2.1±0.6 vs. 2.3±0.6，P<0.001），较大的动脉波反射表现在PAT-AIx（20.4±21.5 vs. 17.0±22.4，P=0.01）和C-AIx（20.8±12.3 vs. 17.5±13.3，P=0.001），以及更大的动脉僵硬度，测量为PWV（7.4±1.6 vs. 7.1±1.6 m/s，P=0.001）。在调整传统CVD危险因素后，黑人种族仍然是较低的RHI和更高的PAT-AIx和C-AIx（所有P<0.001）在所有受试者中，以及在男性中较高的PWV。此外，这些关联在“健康”个体中（即没有CVD危险因素）仍然存在。

**结论**——黑人种族与微循环血管舒张功能障碍相关，且动脉波反射和僵硬度更大。这些血管指标的损害可能与更差的长期结果有关，它们可能代表了增加CVD风险的内在机制。

**关键词**：动脉僵硬度 • 种族差异 • 反应性血流指数

[Deceased.]

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The article was prepared while Dr. Gibbons was employed at Morehouse School of Medicine. The opinion expressed in this article are the author’s own and do not reflect the view of the National Institutes of Health, the Department of Health and Human Services, or the United States Government.

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Arterial stiffening is also associated with CVD risk factors and may worsen hypertension through a feed-forward mechanism, with chronic elevations in blood pressure leading to arterial wall thickening and compensatory remodeling, adversely affecting the internal elastic properties of the vessel wall. Noninvasive and reproducible techniques allow estimation of central aortic pressures and stiffness. Aortic pulse-wave velocity (PWV), an estimate of the speed of the pressure wave traveling along the aorta, is regarded as a direct measure of large artery stiffness. The augmentation index (Alx), a composite measure of the magnitude of arterial wave reflections and systemic arterial stiffness, increases as PWV increases. Impaired arterial elastic properties, measured as the aortic Alx and/or PWV, are increasingly recognized as independent predictors of incident CVD events (myocardial infarction, stroke, revascularization), as well as all-cause mortality.

Prior studies examining the relationship between race and vascular function have often been restricted to relatively small cohorts using invasive and/or cumbersome techniques to assess arterial health. Blacks were found to have impaired endothelium-dependent and -independent vasodilation compared with whites. Moreover, endothelial cells of blacks appear to generate more oxidant stress, leading to enhanced nitric oxide (NO) inactivation. This diminished response to endogenous and exogenous NO in blacks may partly account for the more severe hypertension in this population. In addition, blacks appeared to have greater arterial stiffness, although many of these analyses did not fully account for differences in risk factor burden.

Methods

Study Sample

Self-identified black and white residents of metropolitan Atlanta, aged 20 to 70 years (n=929) without a history of preexisting CVD, were recruited from March 2005 to October 2009 to come to either the Emory or Morehouse Schools of Medicine for evaluation. Detailed information on demographics and anthropometrics was collected. Blood pressure was measured with a sphygmomanometer after 5 minutes of rest and was based on the average of the final 2 of 3 readings measured 5 minutes apart. Height and weight were measured, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m²). History of diabetes and hypertension was defined by participant self-report, or use of antidiabetic or antihypertensive medications. Smoking history, obtained using standardized questionnaires, was defined as current or never/former (no cigarettes within the past 30 days). Pregnant women, participants with history of myocardial infarction or stroke, and those with acute illnesses were excluded. The study was approved by the Emory University and Morehouse School of Medicine Institutional Review Committees. Informed consent was obtained from all participants.

Blood Specimens

Participants were instructed to fast and to refrain from smoking for 12 hours before the study visit. Venous blood was collected in sodium heparin tubes. Serum levels of total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides, and glucose were measured by spectrophotometry.

Pulse Volume Analysis

Digital pulse amplitude tonometry (PAT) was used to measure pulse volume amplitude (PVA) in the tip of the index finger, with participants resting in the supine position in a quiet, temperature-controlled environment set at 22°C after an overnight fast (Endo-PAT, Itamar-Medical, Israel). Full details of the probe technology and the basis of measurements are available elsewhere. PVA was analyzed at rest and during reactive hyperemia, which was elicited by the release of an upper arm blood pressure cuff inflated to suprasystolic pressure for 5 minutes. The reactive hyperemia index (RHI) was calculated as the ratio of the post- to preocclusion PVA of the tested arm, divided by the post- to preocclusion ratio of the control arm (the average PVA over a 1-minute interval starting 1-minute after cuff deflation divided by the average PVA measured for 1 minute before cuff inflation [baseline]).

Indices of arterial stiffness and wave reflections were estimated in the supine position after an overnight fast.
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**Results**

**Subject Characteristics**

Demographic and clinical characteristics of the study participants are presented in Table 1. The mean age was 48±11 years, 55% were female, and 45% were black. Compared with whites, blacks were younger, were more likely to have a history of hypertension, diabetes, and smoking, and were less likely to be college graduates. Blacks had higher BMI and mean arterial pressure and lower levels of triglycerides than whites.

**Clinical Correlates of PAT and Arterial Stiffness**

Table 2 depicts the associations of traditional CVD risk factors with indices of vascular function, after adjusting for race, sex, age, smoking, history of hypertension or diabetes, BMI, mean arterial pressure, lipids, and glucose. RH1 positively correlated with age and LDL-C, but was negatively correlated with total/HDL cholesterol. FR1 was negatively correlated with smoking, BMI, and total/HDL cholesterol. PAT-Alx and CAIx positively correlated with female sex, age, mean arterial pressure, but were negatively correlated with total/HDL cholesterol. PAT-Alx also positively correlated with smoking and trended toward correlation with history of hypertension (P=0.06), but was negatively correlated with history of diabetes and BMI. CAIx also positively correlated with history...

**Statistical Methods**

Study variables are described as the mean±standard deviation (SD) for normally distributed continuous variables, median (interquartile range) for skewed continuous variables, or proportions for categorical variables. RH1, FR1, PAT-Alx, CAIx, and PWV were examined as continuous variables. All continuous variables were first tested for normality using the Kolmogorov–Smirnov criterion. Groups were compared using the chi-square test for categorical outcomes and t tests or Wilcoxon rank sum tests for continuous outcomes.

Multivariable linear regression models were constructed to examine the association of race with indices of vascular function after adjusting for risk factors. Risk factors were selected on the basis of their known association with vascular function and included age, sex, socioeconomic status (level of education), history of hypertension, history of diabetes, smoking status, BMI, mean arterial pressure, triglycerides, LDL-C, ratio of total/HDL cholesterol, and glucose. Predefined subgroup analyses were performed in participants who were free of traditional risk factors for CVD. All tests of statistical significance were 2-tailed, and P<0.05 was considered significant. Statistical analyses were performed using SPSS, Inc, v19.0 (Chicago, IL).

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**Figure 1.** Digital pulse amplitude in a participant with a pulse amplitude tonometry (PAT) ratio in the highest tertile (A) and in a different participant with a PAT ratio in the lowest tertile (B). In the arm undergoing hyperemia (top tracing, A and B), baseline pulse amplitude is recorded. Flow is occluded in both participants during cuff inflation; subsequently, flow rises rapidly after cuff release in the participant with a high response (A) but not in the participant with a low response (B) during the hyperemic period. In the contralateral control finger (bottom tracing, A and B), flow continues throughout, and pulse amplitude undergoes minimal change. Adapted with permission from Hamburg et al.8

using the SphygmoCor device (Atcor Medical, Australia), which records sequential high-quality pressure waveforms at peripheral pulse sites using a high-fidelity tonometer. Full details of the probe technology and basis of measurements are available elsewhere.24

Pulse-wave analysis was performed on the basis of acquisition of radial artery pressure waveforms with application of a generalized transfer function to derive the central aortic pressure waveform, from which estimates of central pulse pressure (CPP) are generated. The central augmentation index (CAIx) is a function of the degree of pressure augmentation (AP) secondary to reflected waves from the periphery (ΔP/total CPP). CAIx standardized to a heart rate of 75 bpm was calculated and used for the purposes of this study.25

Pulse-wave velocity (PWV) measured between carotid and femoral arteries is a regional assessment of aortic stiffness and is the gold standard index of arterial stiffness.24 Pressure waveforms at the carotid and femoral arterial sites were acquired using tonometry and electrocardiographic gating. Velocity (distance/time in meters/second) was calculated using the “foot-to-foot” method, measuring the interval between the R wave on the ECG and the foot of the recorded pressure waveform at each site, whereas distance between the sites was measured manually by the operator. Data on PWV were available for 577 participants. Compared with the total population, there was a higher percentage of blacks (56% versus 40%, P<0.001) and women (64% versus 52%, P=0.001) in the 278 participants who were missing data on PWV. Reproducibility studies in our laboratory on consecutive days have demonstrated a coefficient of variation of 20.3% and 3.8% for CAIx and PWV, respectively.
of hypertension, triglycerides, and LDL-C. PWV positively correlated with age, history of diabetes, and mean arterial pressure, but was negatively correlated with LDL-C.

Race-Related Differences in Microvascular Function

RHI was significantly lower in blacks than whites (Table 3). Furthermore, blacks were more likely to have RHI <1.67 (21.4% versus 13.5%, \( P = 0.003 \)), a value that has previously been associated with coronary endothelial dysfunction. \(^{11}\)

Baseline digital pulse volume amplitude (PVA) was also significantly lower in blacks compared with whites (Table 3). Although the peak fingertip hyperemic response was similar in blacks and whites, hyperemia during recovery from the peak response remained significantly lower in blacks at all times (\( P \leq 0.01 \); Figure 2A). After stratifying by sex, the findings were similar; white women had the highest hyperemic response, whereas black men had the lowest (Figure 2B). After multivariable adjustment for age, sex, history of hypertension, history of diabetes, smoking status, BMI, mean arterial pressure, triglycerides, LDL-C, ratio of total/HDL cholesterol, and glucose, black race remained an independent predictor of lower RHI (\( \beta = -0.169, P < 0.001 \)) and lower baseline PVA (\( \beta = -0.143, P < 0.001 \)).

Race-Related Differences in Wave Reflections and Arterial Stiffness

As shown in Table 3, blacks had higher PAT-AIx, CAIx, and PWV compared with whites. After adjusting for risk factors, black race remained an independent predictor of higher PAT-AIx (\( \beta = 0.127, P < 0.001 \)) and CAIx (\( \beta = 0.170, P < 0.001 \)). In multivariable models stratified by sex, black race remained an independent predictor of higher PWV in men (\( \beta = 0.153, P = 0.02 \)) but not women (\( \beta = 0.037, P = 0.60 \)), although the formal test for race \( \times \) sex interaction was not significant (\( P = 0.40 \)).

Subgroup Analysis in Low-Risk Participants

For this analysis, participants were excluded for history of hypertension or diabetes, current smoking, measured SBP \( \geq 140 \) or DBP \( \geq 90 \) mm Hg, BMI \( \geq 30 \) kg/m\(^2\), or fasting glucose \( \geq 100 \) mg/dL. In the 390 participants who were free of these traditional risk factors for CVD, blacks (n=123) had lower RHI

<table>
<thead>
<tr>
<th>Table 1. Subject Characteristics by Racial Group</th>
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<tbody>
<tr>
<td>Age, y</td>
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<tr>
<td>Female, %</td>
</tr>
<tr>
<td>History of hypertension, %</td>
</tr>
<tr>
<td>History of diabetes, %</td>
</tr>
<tr>
<td>Current smoking, %</td>
</tr>
<tr>
<td>Education*</td>
</tr>
<tr>
<td>High school or GED</td>
</tr>
<tr>
<td>Some college</td>
</tr>
<tr>
<td>College graduate</td>
</tr>
<tr>
<td>Body mass index, kg/m(^2)</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
</tr>
<tr>
<td>Total/HDL-C</td>
</tr>
<tr>
<td>Triglycerides, mg/dL</td>
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<tr>
<td>LDL-C, mg/dL</td>
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<tr>
<td>Glucose, mg/dL</td>
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<tr>
<td>Framingham risk, %</td>
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<tr>
<td>Low</td>
</tr>
<tr>
<td>Intermediate</td>
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<tr>
<td>High</td>
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</tbody>
</table>

Values shown are mean±SD, median (interquartile range), or n (%). GED indicates General Educational Development; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SD, standard deviation. \( P \) values are for black–white comparison.

*Data on level of education available for n=653 participants.

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Mechanisms underlying the observed ethnic differences in vascular function may involve both NO-dependent and -independent mechanisms. NO is tonically released from endothelial cells and is essential to the maintenance of vasodilator tone and homeostasis, which are adversely affected by CVD risk factors. Decreased NO bioavailability can lead to vascular remodeling in experimental models, affecting both large elastic arteries and smaller resistance vessels.

Discussion

In this biracial community-based sample, we demonstrate that black race is associated with impaired vascular function compared with whites. Specifically, blacks had (1) reduced microvascular endothelial function measured as RHI; (2) abnormal arterial wave reflections, measured as higher PAT-AIx and CAIx; and (3) greater arterial stiffness, measured as higher PWV, even after adjustment for traditional CVD risk factors. Importantly, these differences were present even in the subgroup of participants who were completely free of conventional CVD risk factors.

Table 3. Measures of Vascular Function by Racial Group Adjusted for CVD Risk Factors

<table>
<thead>
<tr>
<th>Race</th>
<th>Baseline PVA</th>
<th>RHI</th>
<th>fRHI</th>
<th>PAT-AIx</th>
<th>CAIx</th>
<th>PWV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blacks</td>
<td>275±6**</td>
<td>2.1±0.04**</td>
<td>0.8±0.01</td>
<td>21.4±1.1**</td>
<td>21.2±0.6**</td>
<td>7.3±0.1***</td>
</tr>
<tr>
<td>Whites</td>
<td>328±6</td>
<td>2.3±0.03</td>
<td>0.8±0.01</td>
<td>15.7±1.0</td>
<td>16.6±0.6</td>
<td>7.1±0.1</td>
</tr>
</tbody>
</table>

Values are mean±standard error. Values are adjusted for race, sex, age, smoking, history of hypertension or diabetes, BMI, mean arterial pressure, lipids, and glucose. CVD indicates cardiovascular disease; PVA, pulse volume amplitude; RHI, reactive hyperemia index; fRHI, Framingham reactive hyperemia index; PAT-AIx, peripheral augmentation index; CAIx, central augmentation index; PWV, pulse-wave velocity; BMI, body mass index.

*P<0.01, **P<0.001 when compared with whites.

Values shown are in occluded arm.
The pulsatile arterial tonometer is programmed to estimate the peripheral augmentation index (PAT-AIx) using an algorithm that transforms the digital signal. In the largest investigation to date, our study shows that PAT-AIx is not only higher in blacks but is also associated with other conventional risk factors, as recently reported in smaller studies.}

Race-independent associations between CVD risk factors and indices of digital and central vascular reactivity have been previously investigated. Comparable to our findings, in the largely white Framingham cohort, RHI was directly associated with age and inversely associated with total/HDL cholesterol. In addition, we calculated fRHI, an index that had a better correlation with risk factors in the Framingham study, and found similar results. In contrast to RHI, we found strong associations between other traditional CVD risk factors and indices of arterial stiffness and wave reflections. These findings illustrate that the impact of risk factors on microvascular function in the digital vascular bed differs considerably from that on central arterial stiffness, and yet both these measures are abnormal in blacks. Although other factors such as socioeconomic class and physical activity may contribute to the observed differences, adjustment for these factors did not diminish the racial differences in PAT hyperemia measured in a recent study.

Understanding racial differences in endothelial function and arterial stiffness can help to identify patients at high risk for adverse outcomes including renal disease, myocardial infarction, and stroke. Vascular function measures have the advantage of integrating the time-dependent effects of risk factors over the life span, as well as the modulatory influences of psychosocial factors and racial differences in genetic susceptibility. The striking abnormalities we observed in microvascular tone and arterial elasticity in blacks without any CVD risk factors illustrate that elements beyond conventional risk factors are important. Future studies should investigate whether incorporation of vascular function into new risk prediction models for CVD provides more accurate assessment of risk in blacks.

The strengths of our study include a large sample size of an unselected community-based population with good representation of young, female, and black subjects. In addition, our cohort was large enough to allow the selection of a subgroup that lacked traditional risk factors for CVD, illustrating that elements beyond conventional risk factors are important. Future studies should investigate whether incorporation of vascular function into new risk prediction models for CVD provides more accurate assessment of risk in blacks.

Figure 2. A, Pulse amplitude response shown for the hyperemic finger and control finger in whites and blacks. Peak hyperemia occurred at 90 seconds in both groups. Blacks had lower responses from peak hyperemia throughout in both fingers (P≤0.01). Values are means. The minimum and maximum SEs were 0.02 to 0.06. B, Pulse amplitude response shown for the hyperemic finger and control finger in whites and blacks stratified by sex. Blacks had lower responses from peak hyperemia throughout in both fingers. Values are means. The minimum and maximum SEs were 0.02 to 0.09. PAT indicates pulse amplitude tonometry; SE, standard error of the mean.
Limitations include the cross-sectional design of our study, which precludes establishment of a causal relationship between risk factors and digital vascular function or arterial stiffness. Although we accounted for antihypertensive and statin medication use (<12% of our study population reported use of these medications), it is uncertain whether treatment duration and intensity influenced the vascular parameters. It is recognized that socioeconomic factors may mediate racial differences in CVD risk and that a low level of education has been associated with greater arterial stiffness. However, adding level of education in our sample did not significantly alter our findings. PWV was higher in both black men and women in the healthy subgroup, but only in men with risk factors. This may be because of technical aspects such as abdominal obesity and large bust size in women that can make distance measurements imprecise and affect PWV measurements.

In conclusion, we have demonstrated lower digital reactive hyperemia and increased arterial wave reflections and arterial stiffness in blacks compared with whites in a community-based sample. These findings demonstrate that blacks have worse vascular function compared with whites independent of differences in CVD risk factor prevalence. Because impaired RHI and indices of arterial elasticity have been associated with worse long-term outcomes, these measures may be useful tools for monitoring risk in blacks who have higher CVD risk compared with whites and for following response to therapy in this higher-risk population.

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Disclosure

None.

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