Ethnic Differences in Arterial Responses and Inflammatory Markers in Afro-Caribbean and Caucasian Subjects

Lalit Kalra, Curtis Rambaran, Philip Chowienczyk, David Goss, Ian Hambleton, James Ritter, Ajay Shah, Rainford Wilks, Terrence Forrester

Objective—Small vessel disease is more common in Afro-Caribbeans than Caucasians. We investigated underlying differences in metabolic, inflammatory, and vascular responses that may predispose Afro-Caribbeans to small vessel pathology.

Methods and Results—Seventy-eight Afro-Caribbeans aged 35–75 years, with no vascular disease or medications, were compared with 82 matched Caucasians for metabolic variables, fasting insulin, interleukin 6, tumor necrosis factor (TNF) α, and cytoplasmic repressor protein levels. Carotid intima media thickness (CIMT) was measured ultrasonographically. Small vessel function was assessed by measuring the absolute change from baseline in the reflectance index (RI) of the digital volume pulse during IV infusion of albuterol (5 μg/min, ΔRI_{alb}) and GTN (5 μg/min, ΔRI_{GTN}). Large artery elasticity was measured as the stiffness index (SI) and derived from the time to pulse wave reflection adjusted for subject height. Afro-Caribbeans had significantly higher diastolic blood pressure (80.3 versus 77.6 mm Hg; P=0.033), fasting insulin (14.0 versus 10.6 μU/mL; P=0.026), TNF-α (6.7 versus 4.3; pg/mL; P=0.001), and interleukin 6 (2.3 versus 1.5 pg/mL; P=0.036) levels compared with Caucasians. CIMT was greater (0.81±0.20 versus 0.75±0.18 mm; P=0.02) and small vessel reactivity attenuated (mean ΔRI_{alb} 6.8±8.0% versus 12.3±8.8%; P<0.0001) in Afro-Caribbeans, but their large artery elasticity (mean index of large artery stiffness 9.9 versus 9.7 m/s; P=0.48) was comparable with Caucasians. CIMT was independently associated with an index of large artery stiffness (β=0.03; P=0.002) in Caucasians but not in Afro-Caribbeans. There were independent relationships among Afro-Caribbean ethnicity, TNF-α, and insulin levels.

Conclusions—Selective impairment of small artery function may contribute to excess small vessel disease in Afro-Caribbeans. (Arterioscler Thromb Vasc Biol. 2005;25:0-0.)

Key Words: PLEASE ■ SUPPLY ■ KEY ■ WORDS ■ XXXX

Epidemiological research has shown that the incidence of small vessel stroke and end-stage renal failure are high in people of African descent compared with those of Caucasian origin.1–4 Although hypertension, diabetes, and other vascular risk factors are more prevalent in people of African descent in the setting of these studies, these differences alone do not explain the disparities between the 2 ethnic groups.5–7 A possible explanation may be that clustering of metabolic risk factors within individuals is more common in people of African descent and contributes to small vessel pathology.8 Other studies suggest that there are ethnic differences in vascular function, which may predispose subjects of African descent to small vessel disease.9,10 Neither of these possibilities has been systematically investigated.

The precise mechanisms contributing to small vessel disease remain unknown. Studies have shown that endothelial dysfunction predicts stroke in patients without significant atheroma11,12 and that patients with small vessel disease have hyperinsulinemia even in the absence of diabetes mellitus or obesity.11 These findings become particularly interesting in the context of studies describing attenuated NO-mediated vascular reactivity in healthy subjects9,14 and greater correlation between insulin resistance and target organ damage in hypertensive Afro-American compared with Caucasian subjects.15 This implies that there may be relationships among metabolic factors, insulin levels, and small vessel impairment in people of African descent, which merit investigation.

We hypothesized that increased metabolic stress and differences in physiological response between large and small arteries predisposed Afro-Caribbean people to small vessel disease. We undertook a matched cohort comparison between healthy Afro-Caribbean and Caucasian subjects who did not...
have hypertension, diabetes mellitus, or hypercholesterolemia to investigate the differences in metabolic variables, inflammatory markers, and measures of small and large artery function.

Methods

Subjects
Ethnicity was self-defined, and only subjects with both parents and all 4 grandparents belonging to the same ethnic group were included. Subjects were recruited from patients registered at a general practice in South London (32,000 patients, 28% Afro-Caribbeans). A random sample of 800 registrants between 35 and 75 years of age was obtained using patient record numbers, and their medical records were screened for inclusion. Subjects were included if they had no history of vascular disease and were not on any medication. Those with a history of stroke, transient ischemic attacks, angina, myocardial infarction, heart failure, peripheral vascular disease, hypertension, diabetes mellitus, or hypercholesterolemia were excluded. Potentially eligible participants attended a research clinic (n=298) where eligibility was confirmed and consent obtained. If a potential subject failed to meet inclusion criteria during baseline assessment (n=109) or declined (n=27), the next person on the list who matched age, gender, and ethnicity characteristics was selected, until all of the age/sex grids were filled for both ethnic groups. The study was approved by the Research Ethics Committee of King’s College Hospital.

Baseline Assessments
Age, gender, ethnic, and vascular risk profiles were recorded in all of the subjects. Blood pressure, body mass index, abdominal girth, and hip-to-waist ratio were measured using validated techniques. The definition used for the metabolic syndrome was based on epidemiological research and required an alteration in ≥3 of 5 components: waist girth >102 cm for men and >88 cm for women, triglycerides ≥1.7 mmol/L or 150 mg/dL, high-density lipoprotein (HDL) cholesterol <1.1 mmol/L or 40 mg/dL in men and <1.3 mmol/L or 50 mg/dL in women, blood pressure >130/85 mm Hg and fasting blood glucose >6.1 mmol/L or 110 mg/dL.16

A blood sample was taken after an overnight fast to assess the biochemical markers of metabolic status (blood glucose total cholesterol, HDL cholesterol, triglycerides, homocysteine, and insulin) and activation of inflammation (interleukin (IL) 6, tumor necrosis factor (TNF) α, and cytoplasmic repressor protein (CRP)).17 Insulin levels were measured using a DSL-1600 insulin radioimmunoassay (Diagnostics Systems Laboratories). Homocysteine was measured by chemiluminescent immunoassay (Bayer Diagnostics). TNF-α and IL-6 levels were measured using monochodid ELISA techniques (R&D Systems). CRP was measured using a high-sensitivity turbidimetric immunoassay (WAKO Chemicals) on a Cobas Mira Analyzer (Roche Diagnostics). An estimate of insulin sensitivity was derived by homeostasis model assessment (HOMA) using the following formula: fasting plasma glucose (mmols/L) × fasting plasma insulin (μU/mL)/22.5.18 Genotyping for β2-adrenoreceptor (AR) polymorphisms was undertaken using techniques validated previously19 to adjust for the confounding effect of differences in the distribution of functionally active polymorphisms when assessing ethnic differences in albuterol (ALB)-mediated vasodilatation.19,20

Carotid Artery Imaging
Carotid arteries were imaged with high-resolution B mode ultrasound (Accuson Sequoia 512) using an 8-MHz linear transducer. Each examination cycle included sequential longitudinal and transverse views of the common carotid artery, the carotid bifurcation, and the internal carotid artery bulb. Settings for deep-gain compensation, preprocessing, persistence, and postprocessing were held constant. All of the ultrasonic examinations were stored digitally for subsequent offline processing by an experienced ultrasonographer masked to patient identity and ethnicity. Carotid intima media thickness (CIMT) was defined as the mean of differences between the blood/intima borderline and the media/adventitia borderline and was measured over the distal 1 cm of the common carotid artery, just proximal to the bulb on the left and right side. The mean intracorrelation coefficient for CIMT readings was 0.95 (95% CI, 0.93–0.97).

Measurement of Arterial Function
Small vessel reactivity and large artery stiffness were measured noninvasively using digital volume pulse photoplethysmography (MicroMedical).21 All of the assessments were performed in the morning in a temperature-controlled (24±1°C) laboratory with subjects resting supine. The digital volume pulse waveform consists of a systolic (first peak) and a diastolic (second peak) component formed by the reflection of the pulse wave predominantly from small muscular arteries (Figure). The amount of pulse wave reflected depends on the tone of these arteries and can be measured as a percentage of the systolic peak, the reflectance index (RI). Small artery function can be assessed by measuring absolute change in RI from baseline (ΔRI) in response to albuterol (partly mediated by the endothelium) and nitroglycerin (endothelium-independent) at doses that do not change the heart rate or blood pressure.24 The peak-to-peak time (PPT) between the systolic and diastolic peaks is determined by subject height and arterial distensibility. An index of large artery stiffness (SI) can be derived from PPT using the following formula: SI (m/s) = height (m)/PPT (s).22

After resting baseline measurements of heart rate, blood pressure (BP), and RI, subjects were given a predetermined dose of albuterol (5 μg/min) and GTN (5 μg/min) for 30 minutes each separated by a washout period of 60 minutes. Heart rate, BP, and RI were monitored at 3-minute intervals. ΔRI was measured as the mean absolute change in RI from baseline between 12 and 21 minutes for ALB (ΔRI_ALB) and between 9 and 12 minutes for GTN (ΔRI_GTN). Preliminary studies to test the reliability of methodology in 20 healthy and 20 hypertensive subjects showed that the mean within-subject variation of ΔRI and ΔPPT for repeated measurements was 4.9% and 27.7 ms, respectively, at these time points. Pair-wise comparisons of observations showed that mean intraobserver variability was 4.5±1.1% for ΔRI and 10.1±4.0 ms for PPT.

Statistical Analysis
Mean SD for ΔRI was 11% in preliminary studies, and 77 subjects in each group gave the study 80% power to detect a 5% difference in ΔRI at the 5% (2-sided) significance level. Data are presented as the mean ± SD and were tested for normality using the Kolmogorov-Smirnov test. Most of the data collected showed a normal distribu-
tion, with the exception of CRP, IL-6, and TNF-α. These data were logarithmically transformed before analysis. Differences in mean values were compared by the t test for continuous variables, and by the χ² test for categorical variables. The outcome measures of interest were ΔRI_ALB (endothelium-dependent small artery function), ΔRI_GTN (endothelium-independent small artery function), SI (large artery function), and CIMT (arterial morphology). The independent effect of each potential predictor (ethnicity, body mass index, waist-to-hip ratio, blood pressure, fasting glucose, insulin levels, HDL cholesterol levels, and smoking status) was assessed after adjusting for the confounding effects of age, gender, baseline levels of RI, heart rate, and β-adrenoceptor polymorphisms in multiple regression models. Additional models were constructed to investigate the interactions between ethnicity and independent risk factors and the independent contribution of ethnicity to metabolic and inflammatory variables.

Results

The baseline characteristics of the 78 Afro-Caribbeans and 82 Caucasians were comparable for the majority of demographic and metabolic variables (Table 1). Afro-Caribbean subjects had higher mean body mass index (28.6 versus 26.8; P = 0.035) and diastolic blood pressure (80.3 versus 77.6 mm Hg; P = 0.033) but were comparable for abdominal girth, systolic blood pressure, blood glucose, HDL cholesterol, and triglyceride levels. There were no differences in the prevalence of the clinically defined metabolic syndrome between the groups. The HOMA score was comparable between the 2 groups. The functionally important Gln27 and Arg16 allele polymorphisms were significantly more frequent in Afro-Caribbean subjects.

Afro-Caribbean subjects had higher fasting insulin levels (14.0 versus 10.6 μU/mL; P = 0.026), TNF-α (6.7 versus 4.3 pg/mL; P = 0.001), and IL-6 (2.3 versus 1.5 pg/mL; P = 0.036) levels (Table 2). The mean CIMT of Afro-Caribbean subjects was greater than that of Caucasian subjects (0.81 versus 0.75 mm; P = 0.02; Table 2). Small vessel reactivity of Afro-Caribbean subjects was less than that of Caucasians for ALB (ΔRI_ALB 6.8 versus 12.3%; P = 0.0001) but not for GTN (ΔRI_GTN 11.9 versus 13.6%; P = 0.06). Mean SI values were comparable between the 2 groups (9.9 versus 9.7 m/s; P = 0.48).

After adjusting for differences in age, hemodynamic parameters, metabolic profile, and frequency of β2 adrenoceptor polymorphisms, CIMT was 0.05 mm greater (95% CI, 0.05–0.35; P = 0.0001) but not for GTN (95% CI, 0.2–4.8) in Afro-Caribbean compared with Caucasian subjects (Table 3). TNF-α levels showed an independent relationship with Afro-Caribbean ethnicity (coefficient 0.21; 95% CI, 0.05–0.35; P = 0.01) and fasting insulin level (coefficient

### Table 1. Baseline Characteristics, β2 Adrenoceptor Polymorphisms, and Vascular Measurements of 78 Afro-Caribbean and 82 Caucasian Subjects

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Afro-Caribbean (n=78)</th>
<th>Caucasian (n=82)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>53.8 (11.2)</td>
<td>54.6 (12.4)</td>
<td>0.66</td>
</tr>
<tr>
<td>Gender, % female</td>
<td>51</td>
<td>48</td>
<td>0.70</td>
</tr>
<tr>
<td>Current smoker, %</td>
<td>19</td>
<td>18</td>
<td>0.85</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>28.5 (5.9)</td>
<td>26.8 (4.3)</td>
<td>0.03</td>
</tr>
<tr>
<td>Waist-to-hip ratio</td>
<td>0.86 (0.06)</td>
<td>0.87 (0.07)</td>
<td>0.19</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>80.3 (7.3)</td>
<td>77.6 (8.4)</td>
<td>0.03</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>131.1 (15.9)</td>
<td>128.01 (14.2)</td>
<td>0.19</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.5 (0.4)</td>
<td>1.6 (0.4)</td>
<td>0.06</td>
</tr>
<tr>
<td>Low-density lipoprotein cholesterol, mmol/L</td>
<td>3.2 (0.7)</td>
<td>3.2 (0.8)</td>
<td>0.99</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>1.1 (0.7)</td>
<td>1.3 (0.7)</td>
<td>0.10</td>
</tr>
<tr>
<td>Fasting glucose, mmol/L</td>
<td>5.4 (1.1)</td>
<td>5.2 (0.8)</td>
<td>0.12</td>
</tr>
<tr>
<td>Metabolic syndrome, %</td>
<td>14 (18)</td>
<td>15 (18)</td>
<td>0.98</td>
</tr>
<tr>
<td>HOMA score</td>
<td>3.0 (0.9)</td>
<td>3.2 (1.5)</td>
<td>0.31</td>
</tr>
</tbody>
</table>

*Codon 27: χ² = 31.759, P < 0.0001; Codon 16: χ² = 31.759, P < 0.0001.

### Table 2. Inflammatory and Vascular Markers in 78 Afro-Caribbean and 82 Caucasian Subjects

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Afro-Caribbean (n=78)</th>
<th>Caucasian (n=82)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammatory markers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting insulin, μU/ml</td>
<td>14.0 (10.9)</td>
<td>10.6 (5.7)</td>
<td>0.03</td>
</tr>
<tr>
<td>TNF-α, pg/mL</td>
<td>6.7 (6.1)</td>
<td>4.3 (3.6)</td>
<td>0.001</td>
</tr>
<tr>
<td>IL-6, pg/mL</td>
<td>2.3 (3.3)</td>
<td>1.5 (0.7)</td>
<td>0.04</td>
</tr>
<tr>
<td>CRP, mg/L</td>
<td>2.5 (2.9)</td>
<td>2.1 (2.2)</td>
<td>0.82</td>
</tr>
<tr>
<td>Homocysteine, μmol/L</td>
<td>12.2 (3.0)</td>
<td>13.3 (6.3)</td>
<td>0.19</td>
</tr>
<tr>
<td>Vascular measurements</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CIMT, mm</td>
<td>0.81 (0.20)</td>
<td>0.75 (0.18)</td>
<td>0.02</td>
</tr>
<tr>
<td>Baseline RI, %</td>
<td>76.2 (8.0)</td>
<td>74.3 (8.8)</td>
<td>0.21</td>
</tr>
<tr>
<td>ΔRI_ALB, %</td>
<td>6.8 (8.0)</td>
<td>12.3 (8.9)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>ΔRI_GTN, %</td>
<td>11.9 (7.9)</td>
<td>13.6 (8.1)</td>
<td>0.058</td>
</tr>
<tr>
<td>SI, m/s</td>
<td>9.9 (2.2)</td>
<td>9.7 (2.4)</td>
<td>0.48</td>
</tr>
</tbody>
</table>
TABLE 3. Multiple Regression Models Evaluating Independent Determinants of Arterial Structure (CIMT), Large Artery Function (SI), and Small Artery Function (ΔRI_{ABL}) in the Combined Sample

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>CIMT (Adjusted R²=0.11)</th>
<th>SI (Adjusted R²=0.23)</th>
<th>ΔRI_{ABL} (Adjusted R²=0.66)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coef</td>
<td>95% CI</td>
<td>P Value</td>
</tr>
<tr>
<td>Age*</td>
<td>3.80</td>
<td>3.22–4.39</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Male gender</td>
<td>0.92</td>
<td>0.43–1.41</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Waist-to-hip ratio†</td>
<td>0.35</td>
<td>0.06–0.64</td>
<td>0.02</td>
</tr>
<tr>
<td>Blood pressure‡</td>
<td>0.79</td>
<td>0.09–1.50</td>
<td>0.03</td>
</tr>
<tr>
<td>Fasting blood glucose§</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CIMT‡</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Current smoker</td>
<td>4.06</td>
<td>0.13–7.98</td>
<td>0.04</td>
</tr>
<tr>
<td>Afro-Caribbean vs Caucasian</td>
<td>5.14</td>
<td>0.87–9.42</td>
<td>0.02</td>
</tr>
</tbody>
</table>

*Regression coefficient describes change for every 5 years in age
†Regression coefficient describes change for every 0.01 unit increase in waist-to-hip ratio.
‡Regression coefficient describes change for every 5 mmHg increase in systolic blood pressure.
§Regression coefficient describes change for every 1 mmol increase in fasting blood glucose.
¶Coefficient adjusted for baseline RI and heart rate.
#CIMT used as an independent variable only for SI and ΔRI_{ABL}; significant association only in Caucasians not in Afro-Caribbeans (P=0.004).

A difference in the SI between the 2 groups was not seen even after adjusting for differences in age and other variables (Table 3). SI increased by 0.3 m/s for every 0.1-mm increase in CIMT (P=0.002), but this effect was seen only in Caucasian and not in Afro-Caribbean subjects (P=0.004).

In addition to Afro-Caribbean ethnicity, increasing age, waist-to-hip ratio, higher blood pressure, and smoking were independently associated with greater CIMT (Table 3). Male gender, waist-to-hip ratio, increasing blood pressure, and smoking were other independent determinants of ΔRI_{ABL}. β₂-Adrenocceptor polymorphisms at codon 27 (β=0.03; P=0.79) or codon 16 (β=0.004; P=0.97) did not have an independent effect on ΔRI_{ABL}. There were no significant interactions between ethnicity and age, gender, blood pressure, obesity, and smoking for increases in CIMT or attenuation of ΔRI_{ABL}. Arterial stiffness was greater in men and smokers and increased with blood pressure (Table 3). There was a significant interaction between Caucasian ethnicity and CIMT for large artery stiffness.

**Discussion**

Afro-Caribbean subjects without clinical features of vascular disease had greater carotid intima media thickness, attenuated endothelium-mediated small vessel reactivity, higher insulin levels, and increased inflammatory markers compared with matched Caucasians. Large vessel stiffness of Afro-Caribbeans was comparable with Caucasians despite greater CIMT, and there was an independent association between artery intima thickness and large artery stiffness in Caucasians but not in Afro-Caribbeans. This suggests that Afro-Caribbean subjects have selective impairment of small artery, but not large artery, function associated with inflammatory activation, which may be responsible for the higher prevalence of small vessel disease in Afro-Caribbean subjects. These findings are consistent with studies showing few atherosclerotic changes in the large arteries of patients with small vessel disease and hyperinsulinaemia in black, but not white, hypertensive subjects with end organ damage.

Increased carotid intima media thickness and attenuated vascular reactivity but better large vessel function have been described in Afro-Caribbeans previously, but this is the first study in which all of these aspects have been examined together in community settings. Explanations for population differences in disease susceptibility must be sought in representative samples of the general population to ensure unbiased and generalizable comparisons. The family practice register is the most accurate and comprehensive register of the general population in the United Kingdom and was used to obtain a representative sample of each ethnic group. The difficulties in measuring vascular function in large community-based studies are well known; this study used noninvasive techniques, which were simple, free of operator error, and validated previously against widely accepted but logistically difficult methods. In addition, experimental design, measurement techniques, and reproducibility of vascular measurements were established in pilot experiments before the study.

Recent literature suggests that arterial walls show different functional and morphological responses to hemodynamic or inflammatory challenges under different conditions and may provide possible explanations for ethnic differences observed in this study. It is likely that increased CIMT in Afro-Caribbean subjects reflects vascular remodeling to accommodate increased circumferential and shear stress on the vessel wall because of higher blood pressures rather than large vessel atherosclerosis. TNF-α and IL-6 are known to block the action of insulin and induce vascular inflammation, and hyperinsulinaemia has been observed in patients with small vessel cerebrovascular disease and in black hypertensives with end organ damage. These observations tie in with the findings in our study and suggest the possibility...
of a clinically significant link among early metabolic disturbances, activation of inflammation, and small vessel pathology in Afro-Caribbean people.

An interesting finding was that HOMA-IR scores of Afro-Caribbeans were comparable with Caucasians despite significantly higher insulin levels. Although this may represent very early stages in the development of insulin resistance, it is more likely that HOMA methods are relatively insensitive, and glucose clamp studies may have produced different results. Another unexpected observation was the lack of difference in CRP levels despite elevated IL-6 and TNF-α levels in Afro-Caribbeans. The relationship among IL-6, raised CRP, and atherosclerosis is well established, but associations between CRP and small vessel disease remain unclear. There is also a possibility that ethnic differences in CRP levels may have confounded CRP comparisons in this study.

A limitation of this study is its cross-sectional design; a longitudinal design would be required to assess the progression of vascular and inflammatory changes and establish their relationship to clinical end points. However, only a few subjects in any healthy cohort will experience stroke or other vascular diseases, and it may not be feasible to undertake measurements with the same rigor in a large number of subjects over time. Inclusion of subjects with vascular risk factors may have magnified the differences between small and large vessel function but would have confounded the effects of ethnicity. Although previous studies have demonstrated the predominance of small arteries and endothelial mechanisms in pulse wave responses measured by digital plethysmography, it is possible that wave reflection may also have occurred from sites other than small arteries, and activation of cAMP pathways in the vascular smooth muscle may have contributed to vasodilation with ALB. Any bias because of these limitations would apply equally to both ethnic groups and does not affect the interpretation of findings. Because there is no reliable method for assessing cerebral vascular beds, findings in the peripheral circulation have been extrapolated to cerebral vascular beds. Such extrapolation is supported by evidence that peripheral vascular measurements are a reliable indicator of more widespread changes in both arterial structure and function.

This study suggests that differences in vessel wall response of different arteries to hemodynamic and inflammatory challenges may contribute to different manifestations of vascular disease in different ethnic groups, which may prove important in responsiveness to preventive or therapeutic interventions. The study also confirms the importance of conventional risk factors, such as age, gender, and high blood pressure in the etiology of vascular disease, which still remain the main targets for detection and prevention of vascular disease in both ethnic groups. Although additional research is needed to define mechanisms underlying ethnic differences in vascular response and develop specific interventions, effective implementation of proven measures, such as blood pressure reduction, cessation of smoking, and rectification of metabolic abnormalities, remains the cornerstone for reducing racial disparity and excess burden of vascular disease in people of African descent.

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