Racial Differences in Aortic Stiffness in Children

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Objective To investigate racial differences in central blood pressure and vascular structure/function as subclinical markers of atherosclerotic cardiovascular disease in children.

Study design This cross-sectional study recruited 54 African American children (18 female, 36 male; age 10.5 ± 0.9 years) and 54 white children (27 female, 26 male; age 10.8 ± 0.9 years) from the Syracuse City community as part of the Environmental Exposures and Child Health Outcomes study. Participants underwent blood lipid and vascular testing on 2 separate days. Carotid artery intima-media thickness and aortic stiffness were measured by ultrasonography and carotid-femoral pulse wave velocity, respectively. Blood pressure was assessed at the brachial artery and estimated in the carotid artery using applanation tonometry.

Results African American children had significantly higher pulse wave velocity (4.8 ± 0.8 m/s) compared with white children (4.2 ± 0.7 m/s; $P < .05$), which remained significant after adjustment for confounding variables including socioeconomic status. African American children had significantly higher intima-media thickness (African American 0.41 ± 0.06, white 0.39 ± 0.05 mm), and carotid systolic blood pressure (African American 106 ± 11, white 102 ± 8 mm Hg; $P < .05$) compared with white children, although these racial differences were no longer present after covariate adjustments for height.

Conclusions Racial differences in aortic stiffness are present in childhood. Our findings suggest that racial differences in subclinical cardiovascular disease occur earlier than previously recognized. *(J Pediatr 2017;180:62-7).*

Cardiovascular disease (CVD) morbidity and mortality is greater in African Americans compared with their white American counterparts.[1] African Americans appear to develop elevated blood pressure (BP)[2] and related sequela like heart failure[3] earlier in life compared with white Americans, and racial differences in BP have been documented in young adults.[4] Racial differences in the prevalence of elevated BP and overall hemodynamic burden in children has not been consistently noted,[5,6] and this may be related to the manner in which hemodynamic load is assessed.

BP is traditionally measured in the brachial artery, but this may not accurately mirror load placed on the central vasculature and heart.[7] Methods have been devised to noninvasively estimate central BP, which holds greater prognostic capability than conventional BP; central BP is more predictive of vascular target organ damage, cardiovascular morbidity, and cardiovascular mortality than brachial BP among adults.[7,8] Assessment of central BP may be particularly useful for the assessment of CVD risk in African Americans[9,10] as young African Americans may have higher central BP compared with white Americans despite comparable brachial BP.[11,12] Racial differences in central BP may be the result of early vascular aging among African Americans.[11,12] Large (central) artery stiffness increases with advancing age owing to numerous structural and functional changes.[13] Young and middle-aged African American adults have higher aortic stiffness than their age-matched white counterparts.[4,14,15] These racial differences in aortic stiffness are important to note because aortic stiffness is a predictor of CVD risk later in life[12,16] and is associated with hypertensive target organ damage in African Americans.[17]

The purpose of this study was to investigate racial differences in central hemodynamic burden and vascular structure/function as subclinical markers of atherosclerotic CVD in children. The first specific aim was to investigate racial differences in central BP among African American children and white children. It was hypothesized that African American children would exhibit higher central BP compared with their white counterparts. The second specific aim was to explore racial differences in aortic stiffness and carotid intima-media thickness (IMT) as measures of early vascular aging and vascular target organ damage.[18,19] We hypothesized that aortic stiffness and carotid IMT would be higher in African American children compared with white children.

ADD/ADHD	Attention-deficit disorder/attention-deficit-hyperactivity disorder
BMI	Body mass index
BP	Blood pressure
CVD	Cardiovascular disease
DBP	Diastolic BP
IMT	Intima-media thickness
MAP	Mean arterial pressure
PWV	Pulse wave velocity
SES	Socioeconomic status
SBP	Systolic BP

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Methods

A total of 108 children (54 African American [18 female], 53 white [27 female]) from the Syracuse City community participated in this study as part of the Environmental Exposures and Child Health Outcomes study, a larger, ongoing investigation. Descriptive characteristics are displayed in **Table I**. Participants' race was identified by parents as African American or white. All participants were English speaking, between 9 and 12 years of age, had a body mass >18 kg, and were free from any serious medical/developmental disabilities that would prevent them from participating in the study, as assessed by a health history questionnaire filled out by the guardian. Participants had no medical history of cancer, diagnosed hypertension, stroke, thyroid disease, pancreatitis, neuralgia, or diabetes. Medication use in our cohort included drugs for asthma (n = 30), respiratory allergies (n = 17), and attention-deficit disorder/attention-deficit-hyperactivity disorder (ADD/ADHD; n = 8). This study was approved by the institutional review board of Syracuse University and State University of New York-Upstate University. All guardians and participants gave written consent and assent, respectively, before enrollment.

All participants underwent testing on 2 separate days (day 1, fasted blood lipids; day 2, descriptive measures and vascular assessment). Visits were separated by approximately 2 weeks. All visits occurred between 8:00 a.m. and 12:00 p.m. Participants were instructed to arrive in a fasted state and avoid taking vasoactive medications (ie, medication for respiratory allergies, ADD/ADHD) on test days. Vascular measures were assessed in the supine position following a 5-minute supine rest period in a dimly lit, temperature-controlled laboratory.

Total cholesterol, high-density lipoprotein, low-density lipoprotein, triglycerides, and glucose levels were measured from venous blood using an automated point-of-care blood lipid analysis device (Cholestech LDX, Alere, Waltham, Massachusetts). Participants were classified as having dyslipidemia according to the guidelines established by the Expert Panel on Cardiovascular Health and Risk Reduction in Children and Adolescents.[20]

Table I. Participant characteristics (mean ± SD)

	African American (n = 54)	White (n = 53)
Age (y)	10.5 ± 0.9	10.8 ± 0.9
Male n (%)	36 (67)	26 (48)
Female n (%)	18 (33)	27 (52)
Hypertension n (%)	14 (26)	11 (21)
Dyslipidemia n (%)	5 (9)	9 (17)
Asthma n (%)	18 (33)	12 (23)
ADD/ADHD n (%)	8 (15)	5 (9)
Height (cm)*	148 ± 8	144 ± 9
Weight (kg)*	49 ± 16	42 ± 12
BMI (kg/m^2)	22.1 ± 5.6	20.1 ± 5.2
Pubertal development	2.65 ± 0.89	2.47 ± 0.82
Z score SES*	–0.26 ± 0.69	0.35 ± 0.86
Total cholesterol (mg/dL)	153 ± 23	159 ± 25
HDL cholesterol (mg/dL)	56 ± 13	54 ± 14
Triglycerides (mg/dL)	64 ± 27	74 ± 39
Glucose (mg/dL)	91 ± 7	90 ± 6

HDL, high-density lipoprotein.
*Significant group difference ($P < .05$).

Height and weight were assessed using a mechanical physician scale and stadiometer (Detecto, Webb City, Missouri). Body mass index (BMI) was calculated as weight (kg)/height (m)^2. Socioeconomic status (SES) score was calculated based on parental occupation, income, and education data. Level of parental education and parental occupation were scored using a 1-7 scale and 1-9 scale, respectively.[21] Parental income was based on a 1-10 scale and adjusted by dividing income by the square root of the number of people living in the household.[22] Occupation, income, and data from each of these 3 indices were averaged across parents for each participant when data from both parents was available. All income, education, and occupation data were finally converted into a z score. Participants also reported subjective pubertal development using Peterson's Pubertal Development Scale[23] (scale from 1-5; 1 = prepubertal, 2 = early puberty, 3 = midpuberty, 4 = late puberty, 5 = postpubertal).

The common carotid artery was imaged below the carotid bulb using ultrasound (ProSound α7; Aloka, Tokyo, Japan) and a 7.5-10.0 MHz linear-array probe. Common carotid artery IMT was assessed using a longitudinal view of the artery with both near wall and far wall lumen-IMT boundaries clearly visible. IMT was measured as the distance from the lumen-intima interface to the media-adventitia interface across a 5 mm region of interest via semiautomated digital calipers during diastole (indicated by the R-wave from simultaneous electrocardiogram gating).

Applanation tonometry (AtCor Medical, Sydney, Australia) was used to acquire pressure waves at 2 sites on the right side of the body (carotid-femoral). The distances from the suprasternal notch to the femoral and carotid arteries were measured using a standard tape measure to the nearest mm. Consistent waveforms were collected over 10 seconds from the carotid and femoral arteries using a high-fidelity tonometer. Carotid-femoral pulse wave velocity (PWV) was calculated using the distance (femoral-carotid, to account for different directions of pressure wave transmission) and the measured time delay between the proximal/distal waveforms and peak R wave from simultaneous electrocardiogram gating and taken as a measure of aortic stiffness.

Brachial systolic BP (SBP), brachial diastolic BP (DBP), and mean arterial pressure (MAP) were measured on the nondominant arm using a validated, automated oscillometric sphygmomanometer (Mobil-O-Graph, I.E.M., Stolberg, Germany).[24] Pressures were taken in duplicate and averaged. If values were different by more than 5 mm Hg, a third measure was obtained and the average of the 2 closest measures was used for subsequent analyses. Carotid pressure waveforms obtained during the measurement of PWV (described above) were ensemble averaged to a single waveform for determination of carotid SBP. Carotid waveforms were calibrated to brachial MAP and DBP. MAP and pulse pressure were calculated as 1/3 SBP + 2/3 DBP and SBP-DBP, respectively. Resting brachial BP values were categorized as hypertensive if they were ≥95th percentile for height, in accordance with pediatric hypertension guidelines.[25]

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