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Journal Title: American Journal of Cardiology
Volume: Volume 121, Number 5
Publisher: Elsevier: 12 months | 2018-03-01, Pages 564-569
Type of Work: Article | Post-print: After Peer Review
Publisher DOI: 10.1016/j.amjcard.2017.11.031
Permanent URL: https://pid.emory.edu/ark:/25593/tntpc

Final published version: http://dx.doi.org/10.1016/j.amjcard.2017.11.031

Copyright information:

HHS Public Access

Author manuscript

Am J Cardiol. Author manuscript; available in PMC 2019 March 01.

Published in final edited form as:

Am J Cardiol. 2018 March 01; 121(5): 564–569. doi:10.1016/j.amjcard.2017.11.031.

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Abstract

The pooled cohort atherosclerotic cardiovascular (ASCVD) risk calculator is designed to improve cardiovascular risk estimation compared to the Framingham risk score, particularly in blacks. While the ASCVD risk score better predicts mortality and incident cardiovascular disease in blacks, less is known about its performance for subclinical vascular disease measures, including arterial stiffness and carotid intima-media thickness. We sought to determine if the ASCVD risk score better identifies subclinical vascular disease in blacks compared to the Framingham risk score. We calculated both the Framingham and ASCVD cohort risk scores in 1231 subjects (mean age 53 years, 59% female, 37% black) without known cardiovascular disease and measured the extent of arterial stiffness, as determined by pulse wave velocity (PWV), central pulse pressure (CPP) and central augmentation index (CAIx), and subclinical atherosclerosis, as determined by carotid-IMT (C-IMT). Compared to whites, blacks had higher CAIx [23.9 ± 10.2 vs. 22.1 ± 9.6%, $p = 0.004$], CPP [36.4 ± 10.5 vs. 34.9 ± 9.8 mmHg, $p = 0.014$], PWV [7.6 ± 1.5 vs. 7.3 ± 1.3 m/s, $p = 0.004$] and C-IMT [0.67 ± 0.10 vs. 0.65 ± 0.10 mm, $p = 0.005$]. In a multivariable analysis including race and Framingham risk score, race remained an independent predictor of all measures of subclinical vascular disease; however, models with race and the ASCVD risk score showed that

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Disclosures:
None

race was not an independent predictor of subclinical vascular disease. In conclusion, greater subclinical vascular disease in blacks was not estimated by the Framingham risk score. The new ASCVD risk score provided a better estimate of racial differences in vascular function and structure.

Keywords

Risk assessment; vascular stiffness; carotid intima-media thickness; health status disparities

Introduction

Despite an overall trend toward decreasing morbidity and mortality from cardiovascular disease (CVD) over the last decade in the United States, blacks continue to suffer from higher CVD burden and mortality.[1] Racial differences in the severity of subclinical vascular disease are well documented and may partially explain differences in CVD risk between black and white individuals.[2] Although the Framingham Risk Score (FRS) has traditionally been used to quantify coronary heart disease risk, there have been concerns regarding its generalizability for risk prediction in non-white ethnicities.[3] The Atherosclerotic Cardiovascular Disease (ASCVD) risk calculator was developed using pooled community-based population cohorts that included a significant number of blacks, and incorporates race (black, white or other) in the calculation of the 10-year ASCVD risk.[4] The ASCVD risk calculator has been validated for prediction of clinical events in more diverse cohorts;[5] however, its utility in identifying subclinical vascular disease is unknown. The aim of this study was to investigate whether the ASCVD risk score, compared to the FRS risk score, better identifies subclinical vascular disease in blacks versus whites.

Methods

Self-identified black and white residents of metropolitan Atlanta, age 40 to 78 years (n=1231) and without a known history of cardiovascular disease, were recruited to the Morehouse and Emory Team Up to Eliminate Health Disparities (META-Health) Study or to the Emory-Georgia Tech Center for Health Discovery and Well-Being (CHDWB) cohort study between March 2005 and October 2012. Details of the META-Health cohort[6] and CHDWB cohort[7] have been previously described, and full details of each are included in the Appendix (Supplemental Methods). Informed consent was obtained from all participants and the study was approved by the Emory University and Morehouse School of Medicine Institutional Review Boards.

Detailed demographic and anthropometric data included a detailed personal and family history. Blood pressure was measured with a sphygmomanometer following five minutes of rest and based on the average of three readings measured five minutes apart with cuff sized appropriately for the subject. Height and weight were measured and body mass index (BMI) calculated as weight in kilograms divided by height in meters (kg/m^2). History of hypertension, diabetes, and dyslipidemia were defined by subject self-report or current use of medications. Smoking history was defined as “current” using standardized questionnaires.

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

The FRS score was calculated using the following variables: age (continuous, in years), sex (male vs female), history of smoking (yes vs no), total cholesterol (continuous, in mg/dL), HDL-C (continuous, in mg/dL), systolic blood pressure (continuous, in mm Hg) and treatment for hypertension (yes vs no).[8] The ASCVD risk score was calculated using the each of the variables for the FRS score, with the addition of race (white vs black vs other) and history of diabetes (yes vs no).[4] Risk scores were further divided into “Low” and “High” risk based on established cutoffs; specifically, individuals with FRS < 10% or ASCVD < 7.5% were considered “Low” risk.[9, 10]

Arterial stiffness and wave reflections were measured in the supine position (Sphygmocor, Atcor Medical, Australia) as described previously.[2] Briefly, high-fidelity sequential pressure waveforms were recorded using a handheld tonometer over the radial artery pressure and application of a generalized transfer function allowed for calculation of the central (aortic) pressure, the degree of pressure augmentation resulting from reflected waves from the periphery, and computation of the central augmentation index (CAIx = Augmented Pressure/ Total Central Pulse Pressure). Central pulse pressure (CPP) is the difference between aortic systolic and diastolic pressures. As CAIx varies with heart rate, a CAIx standardized to a heart rate of 75 bpm was calculated in 1159 participants. There were no significant differences in the racial composition between participants with and without CAIx measurements.

Arterial stiffness, estimated as carotid and femoral pulse wave velocity (PWV) was measured using pressure waveforms at carotid and femoral arterial sites using tonometry and electrocardiogram gating as previously described.[2] Velocity (m/s) was calculated by measuring the time interval between the R-waves at each site divided by the distance. PWV was available in 960 participants. There was a higher percentage of blacks (30% vs. 16%, $p<0.001$) in the 271 participants missing PWV measurements. Reproducibility studies in our laboratory on consecutive days demonstrated a coefficient of variation of 20.8% and 3.8% for CAIx and PWV, respectively.

Common carotid artery intima-media thickness was measured in 581 participants with Bmode ultrasonography operating at 7 MHz using standardized techniques as described previously.[11] Longitudinal images of the distal common carotid arteries, proximal to the carotid bulb were obtained using multiple scanning angles at a standardized depth of 4 cm. Images were stored digitally, and measurements were made off-line using semi-automated computerized analytical software (Carotid Tools, MIA Inc., Iowa City, Iowa) by two observers blinded to the test results. Average values of the C-IMT of each of the four segments of the distal 1.0 cm of both common carotid arteries were used as the C-IMT values for each subject. There was a higher percentage of blacks (69% vs. 43%, $p<0.001$) in the 650 participants missing C-IMT measurements. The inter-observer variability for C-IMT was 0.03 ± 0.02 mm.

Continuous variables are presented as mean ± standard deviations (SD) when normally distributed, and as median [interquartile range] when skewed, or as proportions for categorical variables. Continuous variables were assessed for normality using the Kolmogorov-Smirnov criterion. Analysis for CAIx was adjusted for height, and analysis for C-IMT was adjusted for gender. Differences between blacks and whites were compared using the chi-square test for categorical variables, and independent t-tests or Wilcoxon rank sum tests for continuous variables. Racial differences in subclinical vascular disease were compared for “Low” and “High” risk subgroups of FRS and ASCVD, as previously defined. Univariate associations between measures of subclinical vascular disease and predictors were assessed using Spearman rank-sum correlation coefficients. Generalized linear models with race and FRS or ASCVD were performed. Two-tailed P-value ≤ 0.05 was considered statistically significant. All analyses were performed using SAS 9.4 (Cary, NC).

Results

Detailed demographic and clinical characteristics of the study cohort and differences between black and white subjects are presented in Table 1. Compared to whites, blacks were younger and were more likely to have a history of smoking, hypertension and diabetes. Blacks had higher BMI and blood pressure, but lower levels of triglycerides and fasting glucose than whites. While there was no significant racial difference in FRS, blacks had a higher median ASCVD risk score compared to whites (Table 1). The distribution of black and white individuals above and below the median FRS risk score was similar, but more black individuals had an ASCVD risk score greater than the median compared to whites; similar findings were observed using the clinically-relevant cut-offs for each risk score (Supplemental Table 1).

In univariate analyses, blacks had higher PWV, sex- and height-adjusted CAIx, CPP, and sex-adjusted C-IMT compared to whites (Table 1). PWV was positively correlated with age, black race, diabetes, hypertension, blood pressure, BMI, triglycerides and fasting glucose levels, and negatively correlated with female sex and HDL-C (Table 2). CAIx was positively correlated with age, female sex, smoking, hypertension, blood pressure, total cholesterol, HDL-C and triglyceride levels. CPP was positively correlated with black race and BMI. C-IMT positively correlated with age, black race, diabetes, hypertension, blood pressure, BMI, LDL-C, triglycerides and fasting glucose levels, and negatively correlated with female sex and HDL-C (Table 2).

Multivariable analyses were performed to determine if race remained a significantly associated with subclinical disease, after adjustment for risk score (Table 3). In a model that included race, study cohort and FRS, race remained an independent predictor of CAIx CPP, PWV and C-IMT; however, in a model that included race, study cohort and ASCVD risk, race was no longer a significant predictor of any measure of subclinical vascular disease (Table 3).

After categorizing black and white individuals into “Low” and “High” risk groups based on clinically-relevant risk score cut-offs for both the FRS and ASCVD score, there were no racial differences in subclinical vascular disease for “High” risk blacks and whites using

either the FRS or ASCVD risk scores (Table 4). However, racial differences were present in the "Low" FRS risk group, with blacks having statistically significantly greater CPP, PWV, C-IMT and CAIx. Conversely, in the "Low" ASCVD risk group, only PWV was significantly greater in blacks compared to whites (Table 4).

Discussion

In this biracial community-based sample, we demonstrate greater levels of subclinical vascular disease in blacks compared to whites. Despite the increased prevalence of CVD risk factors and subclinical vascular disease in blacks, there was no significant difference in the mean FRS between the races. In contrast, the ASCVD risk score was significantly higher in blacks, and a greater proportion of black individuals were classified as "high risk" using both median- and clinically-relevant cutoff of the ASCVD score. Whereas race remained an independent risk factor for subclinical vascular disease after adjusting for FRS, it was no longer an independent predictor after adjusting for the ASCVD score, suggesting that the ASCVD risk score is a better estimate of subclinical vascular disease than the FRS in blacks. Furthermore, FRS underestimated disease burden in "low risk" blacks, resulting in racial differences in subclinical vascular disease for those with the FRS < 10%. However, when using a clinically-relevant "low risk" cutoff for ASCVD (< 7.5%), the racial differences in subclinical vascular disease were attenuated.

Subclinical vascular disease, measured as arterial stiffness and thickness, is a proxy for underlying pre-clinical vascular disease and is predictive of future adverse cardiovascular events and mortality.[12–16] The Bogalusa Heart Study, conducted in a biracial cohort of 517 younger participants found a linear relationship between FRS and C-IMT in both blacks and whites.[17] In 2,287 Japanese participants, PWV was found to be significantly associated with FRS,[18] and similar relationships have been reported with between CAIx and FRS.[19, 20] Although findings have been robust, few of these studies have included a representative sample of black subjects.

The ASCVD risk score has shown excellent promise in addressing the generalizability concerns associated with the FRS and has demonstrated good risk prediction for hard events in diverse populations.[5] A recent analysis of 5,300 black subjects from the Jackson Heart Study found that the ASCVD risk calculator performed as well as traditional CVD risk models that include additional assessment of B-type natriuretic peptide and ankle-brachial index.[21] And while validation of the ASCVD risk calculator in black populations has been reassuring, few studies have assessed the association of ASCVD risk with subclinical vascular disease. This may be especially relevant given the stark racial differences seen in subclinical disease, with black individuals having greater hypertension-related disease compared to whites that is not captured with traditional risk scoring.[22, 23] The ASCVD risk score, although not developed for assessment of subclinical risk, may help identify individuals at earlier time points who could benefit from initiation or intensification of medical therapy. A recent study by Shah et al, in the Jackson Heart Study showed that statin eligibility, as determined by ASCVD risk score, better captured black individuals with non-zero coronary calcium who were at greater risk for CVD events, specifically those individuals with baseline low-to-intermediate risk.[24] Our study further supports the notion

that ASCVD risk score provides utility in identifying patients with subclinical disease and specifically improves risk assessment in blacks compared to the FRS.

Strengths of our study include its large sample size and good representation of young, female, and black subjects free from prevalent CVD. In addition, ours is the first study to examine the association between the most commonly used cardiovascular risk scores in the United States and multiple markers of subclinical vascular disease. Finally, to the best of our knowledge, we are the first to demonstrate the relationship between the ASCVD risk calculator and markers of arterial stiffness in a biracial cohort. Limitations include the cross-sectional design of our study, which precludes any statement of causality. Because not all individuals underwent each vascular study, there is the possibility of selection bias. Furthermore, while subclinical vascular disease is surrogate of future risk, we do not have long-term follow-up data in this cohort. However, multiple studies have demonstrated the relationship between the CVD risk calculators, subclinical cardiovascular disease, and future cardiovascular events.[8, 12, 14, 16]

In conclusion, we have demonstrated increased prevalence of abnormal wave reflections, arterial stiffness, and arterial wall thickness in blacks compared to whites in a large community-based sample. However, cardiovascular risk assessed by the FRS was similar in blacks in whites. In contrast, the ASCVD risk scores were higher in blacks and the calculator appropriately accounted for racial differences in both vascular function and structure, specifically within the low-risk category of subjects. Thus, the benefit of the ASCVD risk calculator in not underestimating CVD risk in blacks also extends to measures of subclinical vascular disease. Use of the ASCVD score rather than the FRS is likely to enable better prediction of subclinical and clinical CVD risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We wish to thank all the participants of the META-Health and Predictive Health studies who contributed their time and effort for this project.

Grant Support:

NIH/NHLBI 1 U01 HL079156-01 (Quyyumi) and 1 U01 HL79214-01 (Gibbons). Further support was provided by the Emory Heart and Vascular Center and grants from the National Institutes of Health (UL1 RR025008 from the Clinical and Translational Science Award program, R01 HL089650 and R01 HL113451) as well as the American Heart Association (AHA) Strategically Focused Research Network on Disparities (Grant 0000031288).

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Table 1

Clinical characteristics of subjects, stratified by race

Variables	All (N=1231)	Black (N=452)	White (N=779)	P-value
Age (years)	53 ± 7	51 ± 7	53 ± 7	<0.001
Women	730 (59%)	289 (64%)	441 (57%)	0.012
Smoker	173 (14%)	103 (23%)	70 (9%)	<0.001
Diabetes mellitus	107 (9%)	58 (13%)	49 (6%)	<0.001
Hypertension	470 (38%)	212 (47%)	258 (33%)	<0.001
Systolic BP (mmHg)	122 ± 16	125 ± 18	121 ± 15	<0.001
Diastolic BP (mmHg)	78 ± 11	80 ± 12	76 ± 11	<0.001
Body mass index (kg/m^2)	27 [24–32]	30 [26–35]	26 [23–30]	<0.001
Total Cholesterol (mg/dL)	199 ± 38	197 ± 39	200 ± 37	0.25
LDL Cholesterol (mg/dL)	116 ± 32	117 ± 35	115 ± 31	0.49
HDL Cholesterol (mg/dL)	62 ± 18	62 ± 17	61 ± 19	0.58
Triglycerides (mg/dL)	93 [67–128]	85 [63–113]	99 [70–136]	<0.001
Fasting glucose (mg/dL)	88 [83–95]	87 [82–95]	89 [84–95]	0.006
Framingham Risk Score (%)	6.0 [3.4–10.1]	6.2 [3.5–10.1]	5.9 [3.4–10.1]	0.47
Pooled Cohort Risk Score (%)	2.9 [1.2–6.3]	4.1 [1.6–7.4]	2.5 [1.1–5.5]	<0.001
Central augmentation index[a] (%)	22.8 ± 9.9	23.9 ± 10.2	22.1 ± 9.6	0.004
Central pulse pressure (mmHg)	35.5 ± 10.1	36.4 ± 10.5	34.9 ± 9.8	0.014
Pulse wave velocity (m/s)	7.4 ± 1.4	7.6 ± 1.5	7.3 ± 1.3	0.004
Carotid intima-media thickness[b] (mm)	0.66 ± 0.10	0.67 ± 0.10	0.65 ± 0.10	0.005

Values are number (% prevalence) for categorical variables, mean ± SD for normal continuous variables and median [IQR] for non-normal continuous variables.

Abbreviations: BP = blood pressure, LDL = low-density lipoprotein, HDL = high-density lipoprotein

[a] Adjusted for sex and height

[b] Adjusted for sex

Table 2

Correlations between traditional cardiovascular disease risk factors and measures of vascular function and structure

Variable	Central augmentation index	Central pulse pressure	Pulse wave velocity	Carotid intima-media thickness
Age	0.26 (<0.001)	0.33 (<0.001)	0.19 (<0.001)	0.50 (<0.001)
Women	0.18 (<0.001)	0.21 (<0.001)	−0.19 (<0.001)	−0.16 (<0.001)
Black	0.06 (0.06)	0.07 (0.02)	0.09 (0.01)	0.10 (0.02)
Smoker	0.14(<0.001)	0.01(0.87)	0.03 (0.35)	0.01 (0.79)
Diabetes mellitus	−0.03 (0.28)	0.05 (0.08)	0.10 (0.003)	0.16 (<0.001)
Hypertension	0.16 (<0.001)	0.22 (<0.001)	0.25 (<0.001)	0.22 (<0.001)
Systolic BP (mmHg)	0.25 (<0.001)	0.50 (<0.001)	0.34 (<0.001)	0.32 (<0.001)
Diastolic BP (mmHg)	0.24 (<0.001)	0.05 (0.10)	0.29 (<0.001)	0.20 (<0.001)
Body mass index (kg/m^2)	−0.04 (0.25)	0.17 (<0.001)	0.10 (0.003)	0.34 (<0.001)
Total Cholesterol (mg/dL)	0.12 (<0.001)	0.07 (0.02)	0.008 (0.82)	0.08 (0.08)
LDL Cholesterol (mg/dL)	0.06 (0.06)	0.04 (0.18)	0.021 (0.52)	0.09 (0.04)
HDL Cholesterol (mg/dL)	0.07 (0.02)	0.04 (0.15)	−0.11 (<0.001)	−0.11 (0.01)
Triglycerides (mg/dL)	0.10 (0.002)	0.07 (0.02)	0.11 (<0.001)	0.21 (<0.001)
Fasting glucose (mg/dL)	−0.04 (0.22)	0.05 (0.10)	0.10 (0.002)	0.23 (<0.001)
Framingham Risk Score	0.19 (<0.001)	0.26 (<0.001)	0.33 (<0.001)	0.52 (<0.001)
Pooled Cohort Risk Score	0.20 (<0.001)	0.25 (<0.001)	0.33 (<0.001)	0.55 (<0.001)

Values are Spearman rank-sum correlation coefficients (p-value). Partial correlations controlling for height (central augmentation index) and sex (carotid intima-media thickness).

Abbreviations: BP = blood pressure, LDL = low-density lipoprotein, HDL = high-density lipoprotein

Table 3

Associations between race, cardiovascular risk scores and measures of vascular function and structure

	CAIx[a]		CPP		PWV		C-IMT	
	β (SE)	P-value	β (SE)	P-value	β (SE)	P-value	β (SE)	P-value
Black race	1.11 (0.54)	0.040	1.53 (0.61)	0.013	0.25 (0.09)	0.007	0.03 (0.01)	0.003
FRS	0.21 (0.03)	<0.001	0.36 (0.04)	<0.001	0.05 (0.01)	<0.001	0.01 (0.00)	<0.001
Black race	0.79 (0.54)	0.15	0.98 (0.62)	0.12	0.17 (0.09)	0.07	0.02 (0.01)	0.09
ASCVD	0.30 (0.05)	<0.001	0.50 (0.06)	<0.001	0.08 (0.01)	<0.001	0.01 (0.00)	<0.001

Abbreviations: CAIx = central augmentation index; CPP = central pulse pressure; PWV = pulse wave velocity; C-IMT = carotid intima-media thickness; SE = standard error; FRS = Framingham risk score; ASCVD = pooled cohort risk score BP = blood pressure, LDL = low-density lipoprotein, HDL = high-density lipoprotein

[a]Model is additionally adjusted for height

Table 4

Comparison of racial differences in measures of vascular function and structure

		Low Risk				High Risk			
		FRS < 10%		ASCVD < 7.5%		FRS ≥ 10%		ASCVD ≥ 7.5%	
		Value (SD)	P-value	Value (SD)	P-value	Value (SD)	P-value	Value (SD)	P-value
CAIx (%)	Black	24.0 (10.0)	0.049	23.9 (10.1)	0.13	24.1 (10.8)	0.22	24.4 (10.7)	0.43
	White	22.0 (10.2)		22.0 (10.0)		22.6 (8.0)		22.9 (7.7)	
CPP (mmHg)	Black	35.3 (9.4)	0.016	35.2 (9.3)	0.08	40.2 (12.6)	0.20	40.5 (12.8)	0.31
	White	33.6 (8.9)		34.0 (9.2)		38.3 (11.0)		38.8 (11.1)	
PWV (m/s)	Black	7.4 (1.4)	<0.001	7.4 (1.4)	0.003	8.1 (1.6)	0.72	8.0 (1.7)	0.65
	White	7.1 (1.1)		7.2 (1.2)		8.0 (1.5)		8.1 (1.6)	
C-IMT (mm)	Black	0.66 (0.09)	0.008	0.65 (0.10)	0.10	0.73 (0.13)	0.88	0.74 (0.12)	0.46
	White	0.63 (0.09)		0.64 (0.09)		0.74 (0.10)		0.76 (0.08)	

Abbreviations: CAIx = central augmentation index; CPP = central pulse pressure; PWV = pulse wave velocity; C-IMT = carotid intima-media thickness; FRS = Framingham risk score; ASCVD = pooled cohort risk score