Life Course Socioeconomic Position and Subclinical Disease: The Jackson Heart Study

Bradley Deere, MD[1,2]; Michael Griswold, PhD[2,3,4]; Seth Lirette, MS[2]; Ervin Fox, MD, MPH[3,4]; Mario Sims, PhD[3,4]

Objectives: African Americans experience higher rates of cardiovascular disease (CVD) and lower childhood and adult socioeconomic position (SEP). Research that examines the associations of multiple measures of SEP with subclinical CVD markers among African Americans is limited.

Methods: Data from the Jackson Heart Study (JHS) were used to examine cross-sectional associations of childhood SEP and adult SEP with subclinical markers among 4,756 African American participants (mean age 54, 64% female), adjusting for age, health behaviors and CVD risk factors. Subclinical markers included prevalent left ventricular hypertrophy (LVH), peripheral artery disease (PAD), coronary artery calcification (CAC), and carotid intima-media thickness (CIMT).

Results: The prevalence of LVH, PAD and CAC was 7%, 6% and 45%, respectively. The mean CIMT was .72 ± .17 mm. In fully-adjusted models, having a college education was inversely associated with PAD (OR, .27; 95% CI .13,.56) and CIMT (β=-29.7, P<.01). Income was inversely associated with LVH after adjustment for health behaviors (OR, .49 95% CI .25,.96), though associations attenuated in the fully-adjusted model. Measures of childhood SEP (material resources and mother's education) were not consistently associated with subclinical disease measures other than a positive association between material resources and CIMT.

Conclusions: Subclinical disease markers were patterned by adult SEP measures among African Americans. *Ethn Dis*. 2016;26(3);355-362; doi:10.18865/ed.26.3.355

Introduction

Cardiovascular disease (CVD) is a major contributor to morbidity and mortality in the United States.[1] Subclinical disease markers are a prominent risk factor for CVD risk.[2] Left ventricular hypertrophy (LVH) has been shown to be strongly predictive of CVD outcomes including coronary heart disease (CHD), myocardial infarction (MI), sudden cardiac death, stroke, and congestive heart failure.[3,4] Persons with ankle brachial index (ABI) defined peripheral arterial disease (PAD) are approximately 1.5 to 2 times more likely to have clinical CVD events than those without.[5,6] Additionally, evidence has shown that both coronary artery calcification (CAC) and carotid-intima media thickness (CIMT) are predictive of incident CHD, MI, and stroke.[7]

African Americans have a higher prevalence of subclinical CVD than other subpopulations, which places them at greater risk for CVD.[8] Socioeconomic position (SEP) has been linked to CVD disparities, in that persons with low SEP have been shown to have greater CVD prevalence and risk than persons with higher SEP.[9] African Americans are more represented among the least educated, lowest income and low-skilled occupations than their White counterparts,[10,11] which suggests their SEP may explain some of the racial disparity in CVD.[1,12,13] Childhood and adult SEP independently and jointly contribute to an increased prevalence of CVD risk factors, subclinical disease, and CVD disparities.[14-21] Prior work has also shown that lower cumulative life course SEP was associated with increased prevalence of subclinical CVD[17,22-24] among relatively smaller or non-African American samples.

Examining the associations of life-course SEP with subclinical disease among African Americans is important given that African American men and women experience a greater burden of CVD risk factors that

Keywords: Subclinical Cardiovascular Disease; Socioeconomic Position; African American

[1]University of Mississippi Medical Center School of Medicine
[2]Center of Biostatistics and Bioinformatics, University of Mississippi Medical Center School of Medicine
[3]Department of Medicine, University of Mississippi Medical Center School of Medicine
[4]Jackson Heart Study, Coordinating Center

Address correspondence to Bradley Deere, 2500 N. State Street; Jackson, MS 39216; 601.984.5610; bradleydeere@gmail.com

likely develop at early ages.[25] Studies of mostly White populations have shown that early exposure to CVD risk factors may contribute to the development of subclinical CVD as an adolescent, young adult, and adult.[19-21,23,24,26] However, it remains uncertain whether the childhood social and economic circumstances of African American adults are related to greater prevalence of subclinical CVD.

We hypothesized that lower SEP will be associated with higher prevalence of subclinical CVD markers after adjusting for health behaviors and CVD risk factors.

The aim of our study was to examine the social patterning of subclinical disease among a large cohort of African Americans using multiple measures of life-course and adult SEP. We hypothesized that lower SEP will be associated with higher prevalence of subclinical CVD markers after adjusting for health behaviors and CVD risk factors.

Methods

Study Population

The JHS is a population-based, prospective epidemiologic study of CVD among African Americans. The JHS cohort included 5,301 men and women between the ages of 21 and 95 at the baseline examination (2000-2004) living in the tri-county Jackson, Mississippi Metropolitan Statistical Area (MSA) and include Hinds, Madison and Rankin counties. Participants were geographically representative of the Jackson MSA African American population distribution.[27] Participants were recruited from the following sources: 1) 31% from prior Jackson participants of the Atherosclerosis Risk in Communities (ARIC) Study who were recruited randomly from the drivers' license registry; 2) 17% were recruited randomly from a commercially available list (AccuData Integrated Marketing, Fort Myers, FL) of all residents aged 35-84 years in the Jackson MSA; 3) 30% of the participants were volunteers, aged 35-84 years, who were representative of the overall African American population in Jackson MSA in terms of age, sex, and socioeconomic characteristics; 4) 22% were family members of participants (to allow for genetic studies) who reported having at least two siblings and four other first-degree relatives ≥ 21 years old and lived in the tri-county area. All data in the current analyses were drawn from the baseline exam except CAC, which was available from exam 2 (2005-2008) when participants underwent multi-detector CT scanning. Further details of variable measurement and collection and the overall design of the JHS protocol have been described elsewhere.[28,29] This study was approved by the institutional review boards of University of Mississippi Medical Center, Jackson State University, and Tougaloo College. All participants provided informed consent.

Subclinical CVD Markers

Cross-sectional subclinical disease outcomes included LVH, PAD, CAC, and CIMT. Participants underwent a standardized 2-dimensional and M-mode echocardiographic examination. According to recommendations from the American Society of Echocardiography,[30] the presence of LVH was defined as left ventricular mass index of ≥ 51 g/m$^{2.7}$. Presence of PAD was defined as an ABI <.90, which is 95% sensitive and 90% specific for PAD. Agatston scores were used for measuring CAC, with CAC >0 defining presence of any CAC. Common CIMT was quantified as the maximum likelihood estimate of average right and left common carotid far wall intima-media thickness and was obtained using B-mode ultrasonography. All studies were read according to a standardized protocol.[29]

SEP Measures

Childhood SEP was represented by childhood material resources and mother's educational attainment. Material resources included seven household assets: electricity, car, indoor plumbing, air conditioning, refrigerator, telephone, and television. These variables were summarized into an index ranging from 0 to 7, with higher values reflecting greater childhood SEP, and divided into tertiles (low, medium, high) to determine if there were threshold effects. Mother's educational attainment was represented by an ordinal variable with three categories: less than high school, high school graduate to some college, and college graduate and above. Adult SEP was measured by participant educational attainment and family

income. Educational attainment was represented by an ordinal variable with the same categories as the mother's educational attainment. Family income was an ordinal variable consisting of three categories: <$25,000, $25,000-$49,999, and ≥$50,000. P for trend was calculated for each predictor variable to test for the linear trend across each SEP measure.

Covariates

Covariates included age and sex. Health behaviors included current smoking status, alcohol consumption (drinks/week), physical activity, and percentage of calories from fat. Cigarette smoking status was classified as current, former, and never. Alcohol consumption was measured by number of drinks per week. Physical activity was measured as a sum of four index scores (active living, work/occupational, home life, and sport) from the JHS physical activity instrument. Diet, assessed using a validated 158 item food frequency questionnaire, was measured by percent calories from fat per day. Body mass index (BMI, kg/m^2) was derived from in-clinic weight and height measurements using standardized procedures. Biological risk factors included hypertension, type 2 diabetes, hypercholesterolemia (HCL) and hypertriglyceridemia (HTR) and were assessed using standard guidelines and laboratory techniques.[27,28]

Statistical Analysis

Participants with known cardiovascular disease (n=545) were excluded from this study leaving a total of 4756 participants to examine subclinical disease. Exclusions were also made due to missing data for childhood material resources (386), mother's education (1,247), education (99), and income (835). Due to the high missing data for the father's education variable (40%), we used mother's education as a measure of childhood SEP. The missing data were due to missing responses. The following complete data for each subclinical measure were retained for the regression analysis: LVH (3,114 – measured on a subset of participants at baseline), PAD (4,026), CAC (2,661 – measured at examination 2), and CIMT (4,524). We presented descriptive characteristics of the sample by subclinical disease status, where Pearson Chi-Square assessed statistical significance for categorical variables and ANOVA assessed statistical significance for continuous measures. Multivariable logistic regression was used to estimate adjusted odds ratios for binary outcomes (LVH and PAD). Poisson regression was used to estimate adjusted prevalence ratios (PR) for CAC given the high prevalence of CAC (45%) in our sample.[31] Linear regression was used to estimate adjusted associations with continuous outcomes (CIMT). In each regression method, we examined multiple adjustment models that included demographics, health behaviors, and CVD risk factors. Model 1 adjusted for age, sex, and each SEP measure. Model 2 adjusted for variables in model 1 plus health behaviors (alcohol consumption, cigarette use, physical activity, and percent calories from fat). Model 3 adjusted for variables in model 2 plus CVD risk factors (hypertension, BMI, diabetes status, HCL, HTR). Linear trends across levels of SEP measures were tested. Effect modification by sex was investigated by including interaction terms between sex and SEP measures in the regression models. Likelihood ratio tests were used to examine statistical support for sex-related heterogeneity of associations. Statistical analyses were performed using SAS version 9.3 (SAS Institute Inc, Cary, NC) and STATA version 13 (StataCorp LP, College Station, TX).

Results

Study Population

Table 1 provides a description of the sample by subclinical disease. The median age was 54 years and 64% were women. The prevalence of LVH, PAD, and CAC was 7%, 6%, and 45%, respectively and the mean CIMT was .72 ± .17. Tests of unadjusted differences in subclinical disease markers across SEP measures are reported in Table 2. Twenty-three percent of participants had a mother who had a college degree or higher and 23% of participants were in the upper tertile of childhood material resources. Forty-one percent of participants had a college degree or higher and 36% of participants had a family income $50,000 or more. Some inverse social patterning is seen in childhood SEP and PAD and CIMT, mother's education and each subclinical measure, and adult income and LVH, PAD and CIMT (P<.001).

Associations of Subclinical Disease by Childhood SEP and Adult SEP

Table *3* shows odds ratios (OR) for LVH and PAD, prevalence ratios (PR) for CAC, and mean differences

Table 1. Sample characteristics by subclinical disease in the Jackson Heart Study

	Total	LVH (n=3114)			PAD (n=4026)			CAC (n=2661)			CIMT[a] (n=4524)	
	N=4766(%)	No	Yes	P	No	Yes	P	None	Any	P	mean(SD)	P
Women	3059(64%)	1839(64%)	155(70%)	.05	2420(64%)	155(68%)	.193	1010(69%)	731(61%)	<.001	.70(.17)	<.001
Men	1697(36%)	1054(36%)	66(30%)		1378(36%)	73(32%)	.193	449(31%)	471(39%)		.75(.2)	
Age (mean)	53.93(12.78)	51.08(12.44)	58.95(10.86)	<.001	53.41(12.31)	61.34(13.85)	<.001	50.03(9.64)	59.39(10.05)	<.001		
Current smoker	594(13%)	340(12%)	37(17%)	.022	456(12%)	40(18%)	<.001	108(7%)	156(13%)	<.001	.72(.17)	<.001
Alcohol drinking per week	3.50(7.65)	3.30(7.32)	1.69(2.98)	.057	3.65(7.88)	3.44(10.23)	.815	2.91(7.71)	3.50(7.04)	.176		
Total physical activity score	8.48(2.57)	8.81(2.49)	7.64(2.54)	<.001	8.63(2.52)	7.16(2.47)	<.001	9.05(2.35)	8.17(2.54)	<.001		
% Calories from fat	35.16(6.90)	35.42(6.86)	34.28(7.84)	.020	35.20(6.90)	34.54(6.98)	.175	36.15(6.53)	34.80(7.08)	<.001		
BMI (mean)	31.77(7.29)	30.98(6.73)	36.14(8.20)	<.001	31.46(6.84)	29.76(6.87)	<.001	31.28(6.59)	31.57(6.42)	.260		
<18.5	22(0%)	10(0%)	1(0%)		15(0%)	3(1%)		4(0%)	6(0%)		.67(.13)	
18.5-24.9	674(14%)	459(16%)	12(5%)	<.001	546(14%)	49(21%)	<.001	212(15%)	140(12%)	.132	.7(.17)	<.001
25-29.9	1525(32%)	999(34%)	43(19%)		1249(33%)	82(36%)		495(34%)	416(35%)		.73(.19)	
≥30	2547(53%)	1429(49%)	165(75%)		1988(52%)	94(41%)		748(51%)	640(53%)		.72(.18)	
Hypertension	2815(60%)	1503(52%)	190(86%)	<.001	2184(58%)	169(74%)	<.001	706(49%)	865(73%)	<.001	.76(.19)	<.001
Type 2 diabetes	782(17%)	384(14%)	65(31%)	<.001	572(15%)	59(26%)	<.001	123(9%)	256(22%)	<.001	.79(.21)	<.001
Hypercholesterolemia	1337(31%)	743(28%)	76(39%)	.001	1060(30%)	104(50%)	<.001	313(23%)	463(42%)	<.001	.76(.18)	<.001
Hypertriglyceridemia	288(7%)	155(6%)	7(4%)	.233	233(7%)	14(7%)	.902	46(4%)	97(10%)	<.001	.74(.19)	.004

Values in the table represent N (%) for categorical variables and mean (SD) otherwise; P for unadjusted differences are from Pearson's chi-squared tests for categorical variables and t-tests for continuous variables.
SEP, socioeconomic position; LVH, left ventricular hypertrophy; PAD, peripheral arterial disease; CAC, coronary artery calcification; CIMT, carotid intima media thickness; SD, standard deviation; BMI, body mass index.
Characteristics and CVD risk factor data by subclinical disease from the 5,301 participants enrolled in the Jackson Heart Study.
a. μm.

(β) for CIMT by childhood and adult SEP across adjusted models. Childhood material resources were positively associated with CIMT in the age and sex adjusted model, (β=22.9; P<.03). These associations were strengthened after adjustment for health behaviors and CVD risk factors. Associations between mother's education level and subclinical CVD were not statistically significant.

Adult SEP, as measured by education, was associated with select subclinical disease markers. Having a college education or more was associated with a 33% reduction in the odds of prevalent LVH compared with those with less than a high school education (OR .67, 95% CI .45, .99). This association attenuated after adjustment for health behaviors and CVD risk factors. Having greater education was inversely associated with PAD in the fully adjusted model (OR.27, 95% CI .13, .56) with a significant linear trend (P<.001). Additionally, greater education was inversely associated with CIMT in the fully-adjusted model (β= -29.7; P<.01). Tests for linear relationship showed that the patterns were generally graded in the PAD and CIMT models, suggesting a dose-response relationship.

Adult SEP, as measured by family income, was inversely associated with LVH and PAD. Having a family income of >$50,000 was associated with a 50% reduction in the odds of LVH when compared with participants who earned <$25,000 (OR .49, 95% CI .25,.96). The strength of this association weakened after adjusting for CVD risk factors. Greater family income (>$50,000 vs <$25,000) was also associated with a near 50% reduction in the odds of PAD after adjustment for age and sex (OR .54, 95% CI .36,.81) (P for trend = .01). SEP measures were not statistically significant predictors of subclinical CVD markers across sex; thus, we did not estimate sex-stratified models.

Discussion

We examined the social patterning of subclinical disease markers in a large cohort of African Americans. LVH and PAD prevalence were lower

Table 2. Socioeconomic position by subclinical disease in the Jackson Heart Study

	Total	LVH (n=3114)			PAD (n=4026)			CAC (n=2661)			CIMT[a] (n=4524)	
	N=4766(%)	No	Yes	P	No	Yes	P	None	Any	P	mean(SD)	P
Childhood SEP												
Material resources	3.90(2.54)	4.4(2.4)	3.5(2.5)	<.001	4.11(2.5)	2.93(2.5)	<.001	4.65(2.25)	3.28(2.47)	<.001		
Low	1371(31%)	677(25%)	73(37%)		1034(30%)	107(51%)		269(19%)	480(42%)		.8(.19)	
Medium	2022(46%)	1272(47%)	96(48%)	<.001	1668(48%)	77(37%)	<.001	755(54%)	528(47%)	<.001	.71(.17)	<.001
High	987(23%)	740(28%)	31(16%)		798(23%)	25(12%)		374(27%)	127(11%)		.63(.14)	
Mother's education												
< High school	1815(52%)	1049(47%)	102(65%)		1445(51%)	96(62%)		584(49%)	544(61%)		.74(.18)	
High school graduate to some college	906(26%)	608(27%)	33(21%)	<.001	731(26%)	33(21%)	.019	329(27%)	197(22%)	<.001	.69(.16)	.001
College graduate	798(23%)	564(25%)	22(14%)		662(23%)	25(16%)		284(24%)	149(17%)		.66(.16)	
Adult SEP												
Education												
< High school	872(19%)	412(14%)	59(27%)		609(16%)	89(40%)		137(10%)	239(20%)		.81(.21)	
High school graduate to some college	1884(40%)	1155(41%)	85(40%)	.001	1544(41%)	63(28%)	<.001	572(40%)	450(38%)	<.001	.70(.17)	<.001
College graduate	1911(41%)	1282(45%)	71(33%)		1576(42%)	73(32%)		723(50%)	493(42%)		.7(.16)	
Income												
<$25,000	1425(36%)	757(32%)	90(49%)		1059(34%)	91(48%)		315(26%)	331(33%)		.74(.2)	
$25,000-49,999	1094(28%)	673(28%)	47(26%)	.001	878(28%)	58(31%)	.001	357(29%)	295(29%)	<.001	.7(.17)	<.001
≥$50,000	1412(36%)	961(40%)	47(26%)		1199(38%)	40(21%)		539(45%)	380(38%)		.7(.16)	

Values in the table represent N (%) for categorical variables and mean (SD) otherwise; P for unadjusted differences are from Pearson's chi-squared tests for categorical variables and t-tests for continuous variables.
SEP, socioeconomic position; LVH, left ventricular hypertrophy; PAD, peripheral arterial disease; CAC, coronary artery calcification; CIMT, carotid intima media thickness; SD, standard deviation; BMI, body mass index.
a. μm.

among those with more education relative to those with less education, with associations persisting after risk factor adjustment in the PAD models. The associations of education with CIMT were inversely significant in fully-adjusted models. LVH and PAD prevalence were also significantly higher in poor-income than affluent-income groups, with associations remaining significant after risk factor adjustment, particularly in the LVH models. Other than a positive association of childhood material resources and CIMT, subclinical markers were not consistently patterned by childhood SEP.

Adult educational attainment was the most robust SEP predictor of subclinical disease. This is consistent with prior studies showing similar relationships between life-course SEP and subclinical markers, CAC and CIMT. However, many of these studies used limited SEP measures, subclinical markers, or relatively smaller or non-African American sample.[2,17,22-24,32] Adult family income also significantly predicted LVH and PAD after adjustment for age and sex. In another recently published study, investigators found that prevalent diabetes was significantly higher in lower vs high SEP African Americans with associations persisting after risk factor adjustment, which is similar to the relationships between LVH with income and PAD with education.[13] Educational attainment is a more stable measure of social class than income and appears to be more consistently predictive of

LVH and PAD prevalence were lower among those with more education relative to those with less education, with associations persisting after risk factor adjustment in the PAD models.

Table 3. Associations of measures of socioeconomic position (SEP) and subclinical disease in the Jackson Heart Study

	LVH (OR, 95% CI)			PAD (OR, 95% CI)			CAC (PR, 95% CI)			CIMT (β coefficient, SE) [d]		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Childhood SEP												
Material Resources (Referent: Low)												
Medium	1.53[a] (1.05,2.22)	.84 (.42,1.69)	.95 (.42,2.16)	.81 (.57,1.16)	.72 (.38,1.40)	.74 (.36,1.54)	1.05 (.91,1.21)	1.02 (.80,1.30)	.95 (.73,1.24)	12.9 (6.3)[a]	21.9 (9.9)[a]	20.6 (10.4)[a]
High	1.71 (.97,3.02)	1.43 (.55,3.72)	1.29 (.42,3.93)	.97 (.55,1.73)	.81 (.32,2.04)	.75 (.27,2.11)	.93 (.73,1.18)	1.01 (.71,1.45)	.94 (.63,1.40)	22.9 (8.9)[b]	29.9 (12.7)[b]	31.5 (13.4)[a]
P for trend	.07	.36	.78	.44	.62	.73	.43	.98	.93	.03	.05	.06
Mother's Education (Referent: < HS)												
HS graduate to some college	.83 (.54,1.26)	.67 (.31,1.43)	.75 (.32,1.76)	.95 (.63,1.45)	.89 (.45,1.78)	1.14 (.53,2.45)	.98 (.83,1.16)	.94 (.72,1.21)	.94 (.77,1.15)	6.49 (6.2)	.32 (8.7)	-3.35 (9.0)
≥College 4 years	0.71 (.43,1.16)	.56 (.25,1.30)	.56 (.21,1.46)	.90 (.56,1.44)	.56 (.25,1.23)	.75 (.33,1.72)	.95 (.79,1.15)	1.05 (.82,1.35)	1.06 (.89,1.28)	-5.07 (6.7)	-8.28 (8.7)	-6.14 (9.1)
P for trend	.34	.32	.47	.90	.35	.66	.88	.75	.77	.26	.55	.79
Adult SEP												
Education (Referent: < HS)												
HS graduate to some college	.88 (.61,1.29)	.64 (.30,1.36)	.84 (.32,2.15)	.43[c] (.30,.62)	.45[a] (.23,.87)	.28[c] (.13,0.58)	1.04 (.88,1.22)	1.02 (.77,1.36)	1.00 (.73,1.38)	-20.8 (6.7)[b]	-24.0 (11.0)[a]	-37.8 (12.0)[b]
≥College 4 years	.67[a] (.45,.99)	.53 (.25,1.12)	.70 (.28,1.74)	.49[c] (.35,.70)	.36[b] (.18,.71)	.27[c] (.13,0.56)	.98 (.83,1.15)	.98 (.74,1.30)	.98 (.72,1.35)	-27.7 (6.7)[c]	-20.0 (10.9)	-29.7 (11.9)[a]
P for trend	.10	.25	.72	<.001	.01	<.001	.65	.93	.98	<.001	.093	.007
Family Income (Referent: < $25K)												
$25,000-49,999	.72 (.49,1.04)	.87 (.45,1.67)	1.02 (.47,2.23)	.97 (.68,1.38)	.81 (.41,1.60)	.78 (.37,1.66)	1.04 (.89,1.22)	.99 (.75,1.30)	.90 (.67,1.23)	-9.54 (6.3)	-9.07 (9.6)	-3.81 (9.9)
≥$50,000	.54[b] (.37,.79)	.49[a] (.25,.96)	0.65 (.30,1.44)	.54[b] (.36,.81)	.57 (.29,1.10)	.71 (.35,1.45)	.97 (.83,1.13)	1.02 (.79,1.32)	1.02 (.77,1.35)	-11.4 (6.0)	.46 (8.8)	6.6 (9.2)
P for trend	.01	.09	.44	.01	.24	.63	.64	.96	.65	.13	.48	.47

SEP, socioeconomic position; LVH, left ventricular hypertrophy; PAD, peripheral arterial disease; CAC, coronary artery calcification; CIMT, carotid intima media thickness; OR, odds ratio; PR, prevalence ratio; CI, confidence interval; SE, standard error; HS, high school.
Model 1 adjusts for age and sex; model 2 adjusts for model 1 plus health behaviors (alcohol, smoking, physical activity, and calories from fat); model 3 adjusts for model 2 plus CVD risk factors (BMI, hypertension, diabetes, hypercholesterolemia, hypertriglyceridemia).
a. P<.05.
b. P<.01.
c. P<.001.
d. μm.

subclinical disease in our sample.[11]

Prior studies have demonstrated that childhood SEP affects the risks of CVD in adulthood. These studies typically included young adult populations and examined CIMT prevalence or progression.[33-37] Our study consisted of middle-aged adults who were asked to recall their socioeconomic conditions in childhood, which may have affected our non-significant findings for childhood material resources. Also, findings for mother's education may suggest that health behaviors and CVD risk factors could play a mediating role in the association.

Overall, our sample showed a relatively lower or similar prevalence of LVH, PAD, and CIMT when compared with other cohort studies. Prevalent CAC in the JHS (45%, n=1,202) was similar to MESA findings among African Americans (52.1%, n=845).[38,39] Given the overall increased prevalence, especially CAC, identifying modifiable risk factors such as SEP has implications toward macro-level structural changes and public health policies.

A major strength of this study is the use of the JHS cohort, which allowed for the use of multiple mea-

sures of subclinical CVD while accounting for behavioral and CVD risk factors not commonly available in many studies. The use of multiple measures of SEP improves the likelihood of capturing social and economic conditions that may exert adverse effects on health earlier in life.

Being restricted to a single site, Jackson, MS, may limit the ability to generalize these results to other geographic regions. Other limitations may include the relatively high SEP sample drawn from a moderately racially segregated metropolitan area. Areas with more variation in SEP and/or different demographic and environmental characteristics could result in different associations of SEP with subclinical CVD markers. Additionally, other predictors of SEP (eg, individual wealth, current occupation, neighborhood SEP) may show stronger associations with subclinical CVD markers.

In conclusion, PAD and CIMT were strongly patterned by education and LVH was patterned by income among African Americans. No consistent social patterning was observed for CAC and adult SEP measures, as well as for subclinical disease and childhood SEP measures. Higher adult SEP may provide a protective buffer against intermediate phenotypes of CVD, particularly, LVH, PAD, and CIMT. These results show that methods to prevent LVH, PAD and increased CIMT among least educated and low-income African Americans need to be explored. This study also demonstrates that important disparities in subclinical disease between African Americans of different social classes exist in this heterogeneous sample.

Acknowledgments

The authors thank the Jackson Heart Study team (University of Mississippi Medical Center, Jackson State University and Tougaloo College) and participants for their long-term commitment that continues to improve our understanding of the epidemiology of cardiovascular and other chronic diseases. The authors take responsibility for the contents of the article and have no disclosures. Funding Sources: The JHS is supported by contracts HHSN268201300046C, HHSN268201300047C, HHSN268201300048C, HHSN268201300049C, HHSN268201300050C from the National Heart, Lung, and Blood Institute (NHLBI) and the National Institute on Minority Health and Health Disparities (NIMHD). The senior author is supported by the grants P60MD002249 and U54MD008176 from the NIMHD.

Author Contributions

Research concept and design: Deere, Lirette, Sims; Acquisition of data: Lirette; Data analysis and interpretation: Deere, Griswold, Lirette, Fox, Sims; Manuscript draft: Deere, Griswold, Lirette, Fox, Sims; Statistical expertise: Griswold, Lirette, Fox; Administrative: Deere, Sims; Supervision: Fox, Sims

Conflict of Interest

No conflicts of interest.

References

1. Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. 1993;88(4 Pt 1):1973-1998. http://dx.doi.org/10.1161/01.CIR.88.4.1973. PMID:8403348.
2. Carson AP, Rose KM, Catellier DJ, et al. Cumulative socioeconomic status across the life course and subclinical atherosclerosis. *Ann Epidemiol*. 2007;17(4):296-303. http://dx.doi.org/10.1016/j.annepidem.2006.07.009. PMID:17027292.
3. Desai CS, Ning H, Lloyd-Jones DM. Competing cardiovascular outcomes associated with electrocardiographic left ventricular hypertrophy: the Atherosclerosis Risk in Communities Study. *Heart*. 2012;98(4):330-334. http://dx.doi.org/10.1136/heartjnl-2011-300819. PMID:22139711.
4. Fox ER, Alnabhan N, Penman AD, et al. Echocardiographic left ventricular mass index predicts incident stroke in African Americans: Atherosclerosis Risk in Communities (ARIC) Study. *Stroke*. 2007;38(10):2686-2691. http://dx.doi.org/10.1161/STROKEAHA.107.485425. PMID:17761924.
5. Heald CL, Fowkes FG, Murray GD, Price JF; Ankle Brachial Index Collaboration. Risk of mortality and cardiovascular disease associated with the ankle-brachial index: systematic review. *Atherosclerosis*. 2006;189(1):61-69. http://dx.doi.org/10.1016/j.atherosclerosis.2006.03.011. PMID:16620828.
6. Weatherley BD, Nelson JJ, Heiss G, et al. The association of the ankle-brachial index with incident coronary heart disease: the Atherosclerosis Risk In Communities (ARIC) study, 1987-2001. *BMC Cardiovasc Disord*. 2007;7(1):3. http://dx.doi.org/10.1186/1471-2261-7-3. PMID:17227586.
7. Peters SAE, den Ruijter HM, Bots ML, Moons KGM. Improvements in risk stratification for the occurrence of cardiovascular disease by imaging subclinical atherosclerosis: a systematic review. *Heart*. 2012;98(3):177-184. http://dx.doi.org/10.1136/heartjnl-2011-300747. PMID:22095617.
8. Rosamond W, Flegal K, Friday G, et al; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics--2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*. 2007;115(5):e69-e171. http://dx.doi.org/10.1161/CIRCULATIONAHA.106.179918. PMID:17194875.
9. Gebreab SY, Diez-Roux AV, Hickson DA, et al. The contribution of stress to the social patterning of clinical and subclinical CVD risk factors in African Americans: the Jackson Heart Study. *Soc Sci Med*. 2012;75(9):1697-1707. http://dx.doi.org/10.1016/j.socscimed.2012.06.003. PMID:22841454.
10. Braveman PA, Cubbin C, Egerter S, et al. Socioeconomic status in health research: one size does not fit all. *JAMA*. 2005;294(22):2879-2888. http://dx.doi.org/10.1001/jama.294.22.2879. PMID:16352796.
11. Winkleby MA, Jatulis DE, Frank E, Fortmann SP. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *Am J Public Health*. 1992;82(6):816-820. http://dx.doi.org/10.2105/AJPH.82.6.816. PMID:1585961.
12. Kaplan GA. Going back to understand the future: socioeconomic position and survival after myocardial infarction. *Ann Intern Med*. 2006;144(2):137-139. http://dx.doi.org/10.7326/0003-4819-144-2-200601170-00012. PMID:16418414.
13. Sims M, Diez Roux AV, Boykin S, et al. The socioeconomic gradient of diabetes prevalence, awareness, treatment, and control among African Americans in the Jackson Heart Study. *Ann Epidemiol*. 2011;21(12):892-898. http://dx.doi.org/10.1016/j.annepi-

dem.2011.05.006. PMID:21737303.
14. Lynch J, Kaplan GA, Salonen R, Cohen RD, Salonen JT. Socioeconomic status and carotid atherosclerosis. *Circulation*. 1995;92(7):1786-1792. http://dx.doi.org/10.1161/01.CIR.92.7.1786. PMID:7671362.
15. Diez-Roux AV, Nieto FJ, Tyroler HA, Crum LD, Szklo M. Social inequalities and atherosclerosis. The atherosclerosis risk in communities study. *Am J Epidemiol*. 1995;141(10):960-972. PMID:7741126.
16. Lynch J, Kaplan GA, Salonen R, Salonen JT. Socioeconomic status and progression of carotid atherosclerosis. Prospective evidence from the Kuopio Ischemic Heart Disease Risk Factor Study. *Arterioscler Thromb Vasc Biol*. 1997;17(3):513-519. http://dx.doi.org/10.1161/01.ATV.17.3.513. PMID:9102170.
17. Rosvall M, Ostergren PO, Hedblad B, Isacsson SO, Janzon L, Berglund G. Life-course perspective on socioeconomic differences in carotid atherosclerosis. *Arterioscler Thromb Vasc Biol*. 2002;22(10):1704-1711. http://dx.doi.org/10.1161/01.ATV.0000032006.75577.24. PMID:12377753.
18. Nordstrom CK, Diez Roux AV, Jackson SA, Gardin JM; Cardiovascular Health Study. The association of personal and neighborhood socioeconomic indicators with subclinical cardiovascular disease in an elderly cohort. The cardiovascular health study. *Soc Sci Med*. 2004;59(10):2139-2147. http://dx.doi.org/10.1016/j.socscimed.2004.03.017. PMID:15351479.
19. Pollitt RA, Rose KM, Kaufman JS. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: a systematic review. *BMC Public Health*. 2005;5(1):7. http://dx.doi.org/10.1186/1471-2458-5-7. PMID:15661071.
20. Galobardes B, Smith GD, Lynch JW. Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. *Ann Epidemiol*. 2006;16(2):91-104. http://dx.doi.org/10.1016/j.annepidem.2005.06.053. PMID:16257232.
21. Murray ET, Mishra GD, Kuh D, Guralnik J, Black S, Hardy R. Life course models of socioeconomic position and cardiovascular risk factors: 1946 birth cohort. *Ann Epidemiol*. 2011;21(8):589-597. http://dx.doi.org/10.1016/j.annepidem.2011.04.005. PMID:21737047.
22. Matthews KA, Schwartz JE, Cohen S. Indices of socioeconomic position across the life course as predictors of coronary calcification in black and white men and women: coronary artery risk development in young adults study. *Soc Sci Med*. 2011;73(5):768-774. http://dx.doi.org/10.1016/j.socscimed.2011.06.017. PMID:21820224.
23. Lemelin ET, Diez Roux AV, Franklin TG, et al. Life-course socioeconomic positions and subclinical atherosclerosis in the multi-ethnic study of atherosclerosis. *Soc Sci Med*. 2009;68(3):444-451. http://dx.doi.org/10.1016/j.socscimed.2008.10.038. PMID:19081660.
24. Nash SD, Cruickshanks KJ, Klein R, et al. Socioeconomic status and subclinical atherosclerosis in older adults. *Prev Med*. 2011;52(3-4):208-212. PMID:21195728.
25. Lloyd-Jones D, Adams RJ, Brown TM, et al; WRITING GROUP MEMBERS; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics--2010 update: a report from the American Heart Association. *Circulation*. 2010;121(7):e46-e215. http://dx.doi.org/10.1161/CIRCULATIONAHA.109.192667. PMID:20019324.
26. Lutsey PL, Diez Roux AV, Jacobs DR Jr, et al. Associations of acculturation and socioeconomic status with subclinical cardiovascular disease in the multi-ethnic study of atherosclerosis. *Am J Public Health*. 2008;98(11):1963-1970. http://dx.doi.org/10.2105/AJPH.2007.123844. PMID:18511718.
27. Hickson DA, Waller LA, Gebreab SY, et al. Geographic representation of the jackson heart study cohort to the African-American population in Jackson, Mississippi. *Am J Epidemiol*. 2011;173(1):110-117. http://dx.doi.org/10.1093/aje/kwq317. PMID:21076050.
28. Taylor HA, Jr., Wilson JG, Jones DW, et al. Toward resolution of cardiovascular health disparities in African Americans: design and methods of the Jackson Heart Study. *Ethn Dis.* 2005;15(4 Suppl 6):S6-4-17.
29. Carpenter MA, Crow R, Steffes M, et al. Laboratory, reading center, and coordinating center data management methods in the Jackson Heart Study. *Am J Med Sci*. 2004;328(3):131-144. http://dx.doi.org/10.1097/00000441-200409000-00001. PMID:15367870.
30. Lang RM, Bierig M, Devereux RB, et al; Chamber Quantification Writing Group; American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr*. 2005;18(12):1440-1463. http://dx.doi.org/10.1016/j.echo.2005.10.005. PMID:16376782.
31. Spiegelman D, Hertzmark E. Easy SAS calculations for risk or prevalence ratios and differences. *Am J Epidemiol*. 2005;162(3):199-200. http://dx.doi.org/10.1093/aje/kwi188. PMID:15987728.
32. Rooks RN, Simonsick EM, Klesges LM, Newman AB, Ayonayon HN, Harris TB. Racial disparities in health care access and cardiovascular disease indicators in Black and White older adults in the Health ABC Study. *J Aging Health*. 2008;20(6):599-614. http://dx.doi.org/10.1177/0898264308321023. PMID:18625758.
33. Galobardes B, Davey Smith G, Jeffreys M, McCarron P. Childhood socioeconomic circumstances predict specific causes of death in adulthood: the Glasgow student cohort study. *J Epidemiol Community Health*. 2006;60(6):527-529. http://dx.doi.org/10.1136/jech.2005.044727. PMID:16698985.
34. Thurston RC, Matthews KA. Racial and socioeconomic disparities in arterial stiffness and intima media thickness among adolescents. *Soc Sci Med*. 2009;68(5):807-813. http://dx.doi.org/10.1016/j.socscimed.2008.12.029. PMID:19147264.
35. Kestilä P, Magnussen CG, Viikari JS, et al. Socioeconomic status, cardiovascular risk factors, and subclinical atherosclerosis in young adults: the cardiovascular risk in Young Finns Study. *Arterioscler Thromb Vasc Biol*. 2012;32(3):815-821. http://dx.doi.org/10.1161/ATVBAHA.111.241182. PMID:22223734.
36. Li S, Chen W, Srinivasan SR, et al. Childhood cardiovascular risk factors and carotid vascular changes in adulthood: the Bogalusa Heart Study. *JAMA*. 2003;290(17):2271-2276. http://dx.doi.org/10.1001/jama.290.17.2271. PMID:14600185.
37. Raitakari OT, Juonala M, Kähönen M, et al. Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: the Cardiovascular Risk in Young Finns Study. *JAMA*. 2003;290(17):2277-2283. http://dx.doi.org/10.1001/jama.290.17.2277. PMID:14600186.
38. Bild DE, Detrano R, Peterson D, et al. Ethnic differences in coronary calcification: the Multi-Ethnic Study of Atherosclerosis (MESA). *Circulation*. 2005;111(10):1313-1320. http://dx.doi.org/10.1161/01.CIR.0000157730.94423.4B. PMID:15769774.
39. Manolio TA, Arnold AM, Post W, et al. Ethnic differences in the relationship of carotid atherosclerosis to coronary calcification: the Multi-Ethnic Study of Atherosclerosis. *Atherosclerosis*. 2008;197(1):132-138. http://dx.doi.org/10.1016/j.atherosclerosis.2007.02.030. PMID:17412347.