



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

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ABSTRACT

Few studies have investigated the association of socioeconomic status (SES) and coronary artery calcification (CAC) and only one study has examined African Americans separately from Caucasians, despite empirical evidence suggesting that blacks have equivalent or lower CAC, relative to whites. We tested the hypotheses that lower childhood SES and lower average education, occupation, and income and change in SES (slope) in adulthood are related to risk of CAC in blacks and whites in the US CARDIA study. Parental education and occupation were measured at study entry (Year 0 in 1985–1986) and participant education, occupation, and household income were evaluated multiple times throughout a 20 year follow-up period at four sites in the United States. CAC was measured at Year 20 in 3138 (45% black) participants in CARDIA; 19% had CAC. Latent growth models and multivariate logistic regression analyses adjusted for the major risk factors for CAC. Multivariate models showed that lower paternal education in blacks and lower maternal occupational status in the full sample and in whites were related to higher risk of any CAC, independent of adult SES. Lower average adult education, occupation, and income were related to higher risk of any CAC, with the effects primarily in blacks. Our results are the first to show that SES, measured retrospectively and prospectively in multiple ways, is related to CAC, and the first to document the effects primarily in blacks.

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Introduction

A large literature documents the association between socioeconomic status (SES) and cardiovascular disease (CVD) risk factors, morbidity, and mortality (Kaplan & Keil, 1993). SES is usually measured by educational attainment, occupational status/prestige, or family income at one point in time, usually in mid- or late-life. Exposure to low SES early in life may be particularly critical (Ben-Shlomo & Kuh, 2002; Kuh, Hardy, Langenberg, Richards, & Wadsworth, 2002; Matthews et al., 2002; Smith, Hart, Blane, Gillis, & Hawthorne, 1997). However, change in SES across the life course may also be a predictor of subsequent CVD morbidity and mortality, perhaps because of the costs of adaptation to varying life circumstances or because of early exposures in life associated with low SES, setting the stage for a trajectory of high CVD risk (Hart, Smith, & Blane, 1998; McDonough, Duncan, Williams, & House, 1997).

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Coronary artery calcification (CAC) is a noninvasive measure of the calcified component of atherosclerotic plaque in the coronary arteries and it is strongly related to the extent of atherosclerosis and predicts clinical coronary events (Budoff et al., 2006; Greenland et al., 2007; Pletcher, Tice, Pignone, & Browner, 2004). Studies suggest race differences in the prevalence of CAC, with lower levels of CAC in blacks than whites (Bild et al., 2005; Lee, O'Malley, Feuerstein, & Taylor, 2003; Newman et al., 2002; Tang et al., 1995). It is speculated that lower CAC in blacks, despite their higher rates of CVD events, is due to race differences in pathophysiology, e.g., blacks having more unstable plaque, leading to rupture; higher levels of inflammation; and more endothelial stress (Bild et al., 2005; Nazmi & Victora, 2007; Newman et al., 2002).

Few studies have investigated the influence of SES on CAC, especially in multiethnic samples. In the Pittsburgh Healthy Women Study, largely composed of Caucasians, lower educational attainment was associated with extent of CAC (Gallo, Matthews, Kuller, Sutton-Tyrrell, & Edmundowicz, 2001). In a sample of healthy British patients in their thirties, being in the manual social class and having left full time education prior to age 19 were

associated with higher prevalence of CAC (Colhoun, Rubens, Underwood, & Fuller, 2000). In asymptomatic patients and community samples from southern California, educational level and income were unrelated to presence or severity of CAC (Kop et al., 2005). However, these samples were skewed toward higher status and white participants. In the Multi-Ethnic Study of Atherosclerosis (MESA), lower educational attainment was related to CAC in whites, and lower income was related to CAC in blacks. These associations were substantially reduced by adjusting for traditional CVD risk factors (Diez Roux et al., 2005). In the Coronary Artery Risk Development in Young Adults (CARDIA) study of black and white men and women, educational attainment was concurrently related to prevalence of any CAC at the year 15 follow-up when participants were 33–45 years of age (Yan et al., 2006); neither race nor gender stratified analyses or tests for race by gender interactions were presented. No studies have reported how prospectively measured SES across young adulthood through mid-life, measured as cumulative exposure or change in SES, and childhood SES is related to future CAC. That SES over the life span may determine CAC is important to consider because of potential implications for timing of prevention and intervention treatments.

In the present article, we tested the influence of education, occupation, and family income measured prospectively and periodically across 20 years on CAC measured at year 20, in contrast to the prior CARDIA analyses based on concurrently measured education and CAC at year 15 (Yan et al., 2006). In addition, we tested the relationship of retrospectively collected measures of childhood SES based on parental education and occupation. Specifically, we hypothesized that the cumulative exposure to lower SES in adulthood, i.e., average education, income, and occupational prestige, would be associated with higher risk of prevalent CAC at Year 20 because atherosclerosis is a progressive disease, usually beginning in adolescence and young adulthood. We also anticipated that a decline in SES or a smaller increase in SES over the follow-up period measured as a slope of SES would be associated with higher risk of having CAC. We tested whether lower childhood SES based on maternal and paternal education and occupation influenced risk of CAC, independent of adult SES. We adjusted for standard CVD risk factors to evaluate whether they accounted for any obtained associations between SES and CAC. Finally, we examined whether race influenced the observed associations because of the differential pattern of CAC by race.

Methods

Participants

The CARDIA study is a prospective, multi-center study of the natural history of cardiovascular risk development. In 1985–1986, 5115 black and white men and women, 18–30 years old, were recruited and examined at Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California. Participants were recruited to achieve a balance at each site by race (black, white), sex, education (high school degree or less vs. more than a high school degree), and age (18–24 years, 25–30 years). The sites' institutional review boards approved this study with all procedures followed in accordance with institutional guidelines. All subjects gave informed consent. More detailed descriptions of the sampling plan and initial cohort characteristics are available elsewhere (Friedman et al., 1988).

Participants were reexamined in Years 2, 5, 7, 10, 15, and 20 years after baseline, with re-examination rates among surviving cohort members of 91%, 86%, 81%, 79%, 74%, and 69% respectively.

At Year 20, 3138 (547 black men, 863 black women, 807 white men, and 921 white women) or 61% participated in the protocol to measure CAC. Compared to those who did not participate in the year 20 exam (2005–6), those who did participate were (at baseline) younger (0.8 years), better educated (0.7 years), more likely to be female, white, and either a current or never smoker (relative to former smoker), and had higher low density lipoprotein cholesterol (LDL-C; 2.4 mg/dl) and higher systolic blood pressure (SBP; 1.1 mmHg). We based our analyses on the SES data collected in Year 5 through Year 20 because family income was assessed starting in Year 5, education was not completed for many of the younger cohort (i.e., 18–24 year-olds at study entry), and changes from Year 5 through Year 20 in occupation and income would reflect changes in SES as opposed to finishing one's education and reaping the benefits of improving education on income and occupational rank.

Traditional risk factors

Blood pressure was measured at each examination on the right arm with the participant seated and following a 5-min rest. Three measurements were taken at 1-min intervals, with the latter two averaged. SBP and diastolic blood pressure (DBP) were recorded as Phase I and Phase V Korotkoff sounds. Standardized questionnaires were used to collect self-report data on diagnosis and treatment of hypertension, diabetes, and other chronic conditions.

Body weight in light clothing was measured to the nearest 0.5 pound using a balance beam scale. Height without shoes was measured to the nearest 0.5 cm using a vertically mounted centimeter ruler and a metal carpenter's square. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Smoking status was categorized as currently smoking at least five cigarettes per week, yes/no. A fasting blood draw was conducted and total cholesterol, triglycerides, and high density lipoprotein cholesterol (HDL-C) were measured (Warnick, 1986) and LDL-C was calculated by the Friedewald equation (Friedewald, Levy, & Frederickson, 1972).

Socioeconomic status

Participant SES

At each evaluation, participants were asked about their education in years and highest degree obtained. Years of education were used in the analysis. We used average education in years from Years 5 through 20. Slope in education was not used in analyses because few participants obtained more education between Years 5 and 20. At each evaluation, participants were asked about their employment status, current position, type of work, and most important duties of the position. Responses were used to classify individuals' occupations according to the 1990 US Census Bureau 3-digit occupation codes. These, in turn, were used to assign a socioeconomic index (SEI) score according to Stevens and Cho (Stevens & Cho, 1985). Briefly, the SEI scores are predicted "prestige ratings" derived from a regression of the estimated percentage of US adults who would rate the prestige of a given occupation as good or excellent on the proportions of those in the occupation in 1970 who had at least 1 year of college and whose annual personal income was \$10,000 or more (approximately the median). Accordingly, the SEI has a theoretical range of 0–100%, though the actual scores range from 14.0% to 90.5%. The Year 20 occupation data have not been coded so participant occupational data were based on Years 5 through 15. We used SEI at Year 5 and thereafter, average across Years 5–15, and slope in SEI as predictor variables. Total family income was measured in years 5, 7, 10, 15, and 20. Categories were <\$5000; \$5000–11,999;

\$12,000–\$15,999; \$16,000–\$24,999; \$25,000–\$34,999; \$35,000–\$49,000; \$50,000–\$74,999; \$75,000 + for all years except Years 15 and 20. At Years 15 and 20, the categories were the same except that the last category was divided into \$75,000–\$99,999 and \$100,000+. Midpoints were used for most income categories, with the open-ended top category being assigned a value 50% above its lower boundary. The consumer price index was used to adjust each year's income values for inflation. Income was further adjusted for household size, dividing by the square root of the number of household members, after which a cube root transformation was applied to reduce the skewness of the distributions (Schwartz, 1985). Predictor variables were income at each available year, average income, and slope.

Childhood SES

At study entry (Year 0), participants were asked about the number of years of education of their father and mother (or "responsible male and female adult") and their highest degree obtained, as well as the parents' employment status (job title and type of work) when the participants were children. Mothers' and fathers' education and occupation were coded in the same manner as those of the participants. We did not construct an overall life course SES measure because the parental SES measures were retrospectively collected whereas the participant SES measures were prospectively collected; a substantial minority of participants did not know their parents' education and occupation; and more measures of adult SES were available, which proportionally would overweight a life course measure.

Coronary calcification

Coronary calcification was measured at the Year 20 examination by computed tomography (CT) of the chest. Electron beam CT (Imatron C-150, GE Imatron, San Francisco, California) or multi-detector CT scanners (GE Lightspeed, General Electronic Company, Fairfield, Connecticut and Siemens VZ, Siemens AG, Munich, Germany) obtained contiguous 2.5- to 3 mm-thick transverse images from the root of the aorta to the apex of the heart in 2 sequential electrocardiogram-gated scans. Scan data were transmitted electronically to the CARDIA CT Reading Center, where a trained technician examined each image and identified potential foci of CAC using specially developed image-processing software. An expert investigator reviewed and adjudicated all discordant scan pairs. A total calcium score was computed by summing the Agatston score of calcified lesions within each artery (left main, left circumflex, left anterior descending, and right) and across all arteries. We used presence/absence of CAC in the present analyses because of the distributions. All readers were blinded to participant characteristics and to image data from the other paired scan. Details of the scanning protocol are published, demonstrating high between- and within-reader reproducibility (Carr et al., 2005).

Statistical analysis

All analyses predicted a single outcome variable, presence/absence of CAC at the Year 20 exam. Age, sex, and race were covariates in initial models. In the full multivariate models, BMI, blood pressure (systolic, diastolic and self-reported anti-hypertensive medications), LDL-C, and smoking status at Year 20 were added to examine whether obtained effects were statistically accounted for by standard risk factors. For all SES analyses, excluding trajectories or slopes, we used logistic regression predicting CAC presence/absence from SES and the covariates in the full sample, followed by a model that estimated race-specific

effects of SES and tested their difference (i.e., race by SES interaction) within the multivariable models. Parallel analyses estimating separate effects for males and females revealed very little evidence of a gender by SES interaction and are not presented. When the race by SES interaction was significant, we further tested for a gender by SES interaction within race. Significant results for parental SES were followed by adjustments for participant's average SES.

To estimate the relationship between the average annual rate of change in SES and CAC, we estimated a two-equation model where the first equation was a latent growth curve model in which participants' SES trajectories – the slope from a linear regression of their repeated measures of adult SES on the CARDIA exam year – were treated as a random (i.e., latent) factor, and the second equation was a logistic regression predicting CAC from this factor and the covariates. The M-Plus software (Version 4.1) was used to estimate this two-equation model (Muthen & Muthen, 1998). SAS, version 9.2, was used for all other analyses. Relationships of occupational status and income to CAC were estimated in separate equations. In the tables and text, we present 1/standardized odds ratio (sOR) so that an estimate >1.0 represents the increase in risk associated with a one standard deviation decrease in SES.

Results

Sample characteristics

Overall, 18.6% of participants had some calcification, with the highest proportion being among white men, followed by black men (Table 1). On average their blood pressure and LDL-C levels were in the normal range, although 17% were on anti-hypertensive medication. On average the sample was overweight, with the highest BMI among black females. About 20% were current smokers and nearly 20% ex-smokers.

The sample was relatively well educated and had well educated parents. Due to their age at Year 5 (23–35 years), there was little change in education between Years 5 and 20, but occupational prestige and household income increased substantially from Years 5 to 15 or 20 respectively. There were no differences by race in change in SES across the follow-up.

Maternal and paternal education was positively associated with participant education, and maternal and paternal occupational prestige was positively associated with participant occupational prestige (Table 2). Participant education was highly and positively correlated with both their occupational prestige and household income. By and large, the correlations among SES measures were similar in blacks and whites.

Influence of SES on calcification

Parental Education and Occupation during Participants' Childhood

In the full sample, lower paternal (sOR = 1.14, 95% CI: 1.02–1.27) and maternal education (sOR = 1.12, CI: 1.01–1.24) in years were related to higher risk of CAC in initial models adjusted for age, sex, and race. In full multivariate models, adding BMI, blood pressure and blood pressure medications, LDL-C, and smoking status, only among blacks was lower paternal education related to higher risk of CAC (Table 3).

Gender-specific analyses showed that the effect was due to the black women. Adjustments for average adult education plus all covariates showed the same pattern of results, i.e., only among blacks was lower parental education related to higher risk of CAC (sOR = 1.24, CI: 1.01–1.53), race by paternal education interaction term, $P = .04$.

Table 1
Characteristics of sample participating at year 20 (2005–2006) in calcification protocol, CARDIA Study.

	Black		White		Full sample (N = 3138)
	Male (N = 547)	Female (N = 863)	Male (N = 807)	Female (N = 921)	
Mean (SD) Age (yrs.)	44.6 (4.0)	44.6 (3.8)	45.7 (3.4)	45.9 (3.4)	45.3 (3.6)
<i>Parental SES</i>					
Mean (SD) education in years:					
Father	11.7 (3.3)	11.6 (3.4)	14.5 (3.4)	14.4 (3.5)	13.3 (3.7)
Mother	12.5 (2.6)	12.3 (2.7)	13.9 (2.7)	13.6 (2.8)	13.2 (2.8)
Mean (SD) occupational prestige in SEI categories:					
Father	32.0 (17.4)	30.0 (16.8)	48.7 (21.7)	48.7 (22.2)	41.5 (22.0)
Mother	33.6 (18.4)	31.7 (17.4)	44.1 (19.0)	41.8 (18.4)	37.8 (19.0)
<i>Participant SES</i>					
Mean (SD) education in years:					
Year 5	13.7 (2.1)	13.8 (2.0)	15.3 (2.6)	15.3 (2.4)	14.6 (2.4)
Year 20	13.9 (2.2)	14.2 (2.3)	15.8 (2.7)	15.9 (2.4)	15.0 (2.6)
Change (years 5–20)	0.24 (1.12)	0.52 (1.52)	0.45 (1.31)	0.58 (1.39)	0.47 (1.37)
Average (years 5–20)	13.8 (2.1)	14.0 (2.0)	15.6 (2.6)	15.6 (2.3)	14.8 (2.4)
Mean (SD) occupational prestige in SEI categories:					
Year 5	33.1 (17.6)	35.3 (16.7)	47.8 (21.7)	45.4 (18.9)	41.3 (19.9)
Year 15	37.0 (19.2)	41.1 (18.2)	50.7 (20.3)	50.8 (18.2)	45.9 (19.8)
Change (years 5–15)	4.43 (17.6)	6.04 (17.9)	2.57 (17.6)	5.43 (20.15)	4.64 (18.54)
Average (years 5–15)	34.5 (15.5)	36.8 (14.1)	48.9 (19.2)	47.7 (15.4)	42.7 (17.3)
Mean (SD) household income ^a :					
Year 5	26.0 (17.3)	22.4 (15.9)	38.6 (22.1)	37.3 (20.9)	31.8 (20.7)
Year 20	36.7 (26.3)	31.1 (23.8)	52.7 (27.3)	48.9 (26.8)	42.9 (27.5)
Change (years 5–20)	11.9 (23.0)	9.56 (22.1)	14.2 (26.5)	11.0 (26.0)	11.9 (24.7)
Average (years 5–20)	30.1 (18.2)	26.1 (16.4)	44.9 (20.0)	42.5 (19.1)	36.5 (20.2)
No. (%) any calcification	130 (23.8)	94 (10.9)	264 (32.7)	94 (10.2)	582 (18.6)
No. (%) current smokers	160 (29.4)	191 (22.3)	132 (16.6)	116 (12.7)	599 (19.2)
Mean (SD) Systolic Blood Pressure (mm/Hg)	122 (14)	118 (17)	117 (12)	109 (12)	116 (15)
Mean (SD) diastolic blood pressure (mm/Hg)	75.4 (10.7)	75.2 (11.5)	71.6 (9.9)	67.5 (10.1)	72.1 (11.0)
No. (%) blood pressure medications	102 (18.6)	246 (28.5)	112 (13.9)	78 (8.5)	538 (17.1)
Mean (SD) low-density lipoprotein (mg/dl)	116 (34)	111 (29)	120 (31)	107 (27)	113 (30)
No. (%) LDL ≥ 130 mg/dl	148 (27.1)	184 (21.3)	277 (34.3)	184 (20.0)	793 (25.3)
Mean (SD) body mass index (kg/m ²)	28.9 (5.5)	32.0 (7.3)	28.5 (6.0)	27.2 (6.5)	29.1 (6.7)
No. (%) normal	138 (25.3)	160 (18.6)	196 (24.3)	412 (44.8)	906 (28.9)
No. (%) overweight	195 (35.8)	221 (25.7)	383 (47.5)	264 (28.7)	1063 (33.9)
No. (%) obese	212 (38.9)	479 (55.7)	228 (28.2)	244 (26.5)	1163 (37.1)

CARDIA - Coronary Artery Risk Development in Young Adults; SES- Socioeconomic Status; SEI - Socioeconomic Index; LDL - Low-density Lipoprotein.

^a Income adjusted for consumer price index (dollars in 2000) and household size.

In the full sample, paternal occupational status (in 2612 fathers who had an occupation) was unrelated to CAC; lower maternal occupational status (in the 2129 whose mothers had an occupation) was related to higher risk of CAC (sOR = 1.23, CI: 1.08–1.39) in age, sex, and race-adjusted model. The interaction with race was

nonsignificant; the association between maternal occupation and CAC was apparent in whites in multivariate models (Table 3). Adjustments for average adult occupational status plus all covariates showed the same pattern of results for maternal occupational status in the full sample (sOR = 1.14, CI: 1.01–1.32); the effects were

Table 2
Correlations among socioeconomic status measures in blacks and whites separately (1985–2006), CARDIA study.

(N)	Paternal education (2639)	Maternal education (2907)	Paternal occupational prestige (2612)	Maternal occupational prestige (2129)	Average education (3138)	Average occupational prestige (3084)	Average household income ^a (3136)
<i>Education</i>							
Paternal		0.51	0.55	0.37	0.23	0.17	0.19
Maternal	0.60		0.35	0.60	0.28	0.18	0.19
<i>Occupational prestige</i>							
Paternal	0.69	0.45		0.34	0.29	0.21	0.21
Maternal	0.44	0.64	0.42		0.30	0.21	0.22
<i>Participant:</i>							
<i>Education</i>							
Average	0.35	0.30	0.33	0.21		0.70	0.55
<i>Occupational prestige</i>							
Average ^a	0.27	0.21	0.28	0.19	0.70		0.53
<i>Household income</i>							
Average	0.20	0.16	0.19	0.14	0.43	0.50	

CARDIA - Coronary Artery Risk Development in Young Adults.

All correlations are statistically significant at $P < 0.01$, unless noted otherwise, * $0.01 > P > 0.05$, ** $P \geq 0.05$.^a Correlations above the diagonal are for blacks and below the diagonal for whites; income adjusted for consumer price index (dollars in 2000) and household size, and then transformed to the cube root.

Table 3
Standardized odds ratios (95% Confidence Interval) for 1 standard deviation lower parental socioeconomic status (SES) measured in 1985–1986 from multivariate models predicting coronary calcification in 2005–2006 in CARDIA^a

SES Measure	Full Sample	Blacks	Whites	P-value for interaction of race × SES
Fathers' education (Yrs) (N = 2612)	1.06 (0.94–1.19)	1.26 (1.03–1.55) ^b	0.98 (0.85–1.13)	.05
Mothers' education (Yrs) (N = 2878)	1.05 (0.94–1.17)	1.11 (0.93–1.32)	1.03 (0.90–1.17)	>.20
Fathers' occupation status (SEI units; N = 2586)	1.03 (0.91–1.16)	1.09 (0.85–1.40)	1.01 (0.88–1.15)	>.20
Mothers' occupation status (SEI units; N = 2107)	1.16 (1.02–1.33)	1.12 (0.91–1.38)	1.20 (1.01–1.42)	>.20

^a Adjusted for age, sex, race, year 20 BMI, blood pressure, blood pressure medications, LDL-C, and smoking status.

^b Due to black women.

significant only in whites in full multivariate adjustment plus adult occupational status.

Participant education

Lower education at each assessment (sORs = 1.29–1.35, P s < .0001) and lower average education (sOR = 1.32, CI: 1.20–1.46) were related to CAC in Year 20 in initial models. In full multivariate analyses, lower education at all exams after Year 5 (P = 0.08 for Year 5) and lower average education were related to higher risk of CAC. Race-stratified analyses showed the effects were present only among blacks, and not among whites (Table 4).

Participant occupational status

Lower occupational status at Years 5, 7, 10, and 15 (sORs = 1.18 to 1.28, all P s < .002), and lower average occupational status (sOR = 1.26; CI: 1.14–1.40) were related to higher risk of CAC in initial models. In multivariate analyses, occupational status at Years 10 and 15 and average occupational status remained significant predictors. Full multivariate analyses showed that the occupational status effect was significant for blacks on average (Table 4) and at Year 7, Year 10 (black women only), and Year 15. Differences in individuals' average annual rate of change in SEI were unrelated to CAC risk.

Participant income

Lower household income, adjusted for household size and consumer price index, at all years, and lower average household income were related to higher risk of CAC in initial models, sORs = 1.16–1.25, all P s < .005. Full multivariate analyses showed that lower household income was related to higher risk of CAC in the full sample only at Years 5 and 20. However, lower average

income (Table 4) and lower income at each year were related to higher risk of CAC in blacks, sORs = 1.20–1.38, all P s < .05. Slope of income was unrelated in multivariate analyses.

Discussion

The objectives of this study were to test the association of adult SES prospectively measured across 15 years and of childhood SES retrospectively measured with presence of CAC and to examine whether the associations varied by ethnicity. Our findings showed that in the full CARDIA sample, lower average years of educational attainment and lower average level of occupational prestige were associated, and lower average income tended to be associated with higher risk of CAC. These effects were independent of the major determinants of CAC, including age, race, sex, and Year 20 BMI, blood pressure, blood pressure medications, LDL-C, and smoking status. The results, however, were primarily obtained in black men and women. Black men and women at elevated risk for having CAC at Year 20 had lower educational attainment at all years and averaged across Years 5 through 20; lower occupational status at years 7 through 15, and averaged across Years 5 through 15; and lower household income at each year from 5 through 20 and lower average income. Blacks, especially women at elevated risk for having CAC had fathers with lower paternal education, independent of their own adult educational level. We found no evidence that increasing or decreasing SES (as estimated by slope) across the duration of the study was related to risk for CAC, independent of average SES. Our results are the first to show that SES, measured retrospectively and prospectively in multiple ways, is related to CAC, and the first to document the effects primarily in blacks.

Table 4
Standardized odds ratios (95% Confidence Interval) for 1 standard deviation decrease in adult socioeconomic status (SES) measured in 1990–2006 from multivariate models predicting coronary calcification in 2005–2006 in CARDIA.^a

SES measure	Full sample (N = 3102)	Blacks (N = 1393)	Whites (N = 1709)	P-Value for interaction of race × SES
Education				
Year 5	1.11 (0.99–1.24)	1.29 (1.05–1.58)	1.05 (0.92–1.19)	.09
Year 20	1.13 (1.01–1.26)	1.38 (1.14–1.67)	1.03 (0.91–1.18)	.01
Average	1.13 (1.02–1.26)	1.36 (1.12–1.65)	1.05 (0.93–1.20)	.03
Occupation				
Year 5	1.06 (0.95–1.18)	1.21 (0.98–1.49)	1.01 (0.89–1.15)	.14
Year 15	1.12 (1.00–1.25)	1.37 (1.13–1.66)	1.01 (0.88–1.16)	.01
Average	1.12 (1.00–1.25)	1.39 (1.14–1.71)	1.03 (0.91–1.17)	.01
Slope from Year 5 to Year 15	1.12 (0.94–1.34)	1.27 (0.97–1.66)	1.05 (0.84–1.31)	>.20
Household income ^b				
Year 5	1.14 (1.01–1.28)	1.37 (1.14–1.66)	1.01 (0.87–1.17)	.01
Year 20	1.12 (1.00–1.24)	1.31 (1.12–1.54)	0.97 (0.84–1.12)	.006
Average	1.11 (0.99–1.24)	1.30 (1.10–1.53)	0.96 (0.83–1.12)	.008
Slope from Year 5 to Year 20	1.06 (0.93–1.23)	1.21 (0.97–1.51)	0.98 (0.82–1.17)	.14

^a Adjusted for age, sex, race, year 20 BMI, blood pressure, blood pressure medications, LDL-C, and smoking status.

^b Adjusted for household size and consumer price index.

Lower maternal occupational prestige was related to higher risk of CAC in the full sample and in whites. These findings are consistent with other epidemiological analyses showing that retrospective measures of childhood SES predict later risk for clinical coronary events, although the effects became weaker in those analyses that adjusted for adult SES (Galobardes, Lynch, & Davey, 2004; Galobardes, Smith, & Lynch, 2006). Childhood SES was not tied to a specific age in our study and there is some evidence that childhood SES at various ages matters (Chen, Matthews, & Boyce, 2002). In consequence, it still remains unclear whether SES at a critical period during development – e.g., during infancy or early adolescence – may be critical for the influence of SES to begin. It is also unknown precisely how childhood SES exerts its influence, whether it be through early exposure to poor parental health behaviors, ineffective parenting, or material deprivation (Repetti, Taylor, & Seeman, 2002; Troxel & Matthews, 2004). In CARDIA, lower childhood SES was related to adult levels of risk factors through poor psychosocial functioning as an adult (Lehman, Taylor, Kiefe, & Seeman, 2005).

Although we anticipated that race differences in the influence of SES on CAC would emerge, we did not expect that adult SES would have largely null effects in whites, although southern California samples skewed to higher SES did not reveal associations of CAC with education or income (Rozanski et al., 2011). Our analyses adjusted for a substantial number of important risk factors for CAC. Perhaps the CAC risk associated with low SES indices are more strongly estimated by standard risk factors in whites than in blacks. However, we have no evidence that this is the case, and CAC was predictive of clinical events in all ethnic groups in MESA (Detrano et al., 2008). Another possibility is related to the “weathering hypothesis”, i.e. that blacks are exposed to not only greater economic adversity but also greater social adversity and in combination blacks age more rapidly (Geronimus, Hicken, Keene, & Bound, 2006). In other words, unmeasured social adversity may exacerbate the effects of low economic status in blacks to a greater extent than in whites. It is well established that whites have greater wealth than blacks, so even when whites are currently un- or under-employed, they have more resources to fall back on than do blacks (Williams & Collins, 2001). Thus, adult SES measured by occupational status and income may be a better marker of living conditions in blacks than whites. Finally, poorer blacks live in poorer residential areas, with the attendant exposures to pollution, crime, and crowding, whereas whites live in neighborhoods that vary in neighborhood-level SES measures, e.g., proportion of census tract residents with high school education or more (Sampson & Wilson, 1995). Thus, risk associated with being lower class in whites may be offset by exposure to a higher neighborhood-level SES environment. It is noteworthy, however, that rates of neighborhood unemployment were related to higher CAC, independent of individual indicators of SES in the Heinz Nixdorf Recall Study (Dragano et al., 2009).

Various indicators of subclinical cardiovascular disease are only modestly correlated, e.g. in MESA, common carotid intima media thickness (IMT) is correlated $r = 0.33$ with CAC (Jacobs Jr., & Crow, 2007). Nonetheless, it is informative to consider SES in relation to measures of subclinical cardiovascular disease, other than CAC. In MESA (Lemelin et al., 2009), using multivariate analyses, lower adult SES (based on a combined index of education, income, and wealth) was associated with lower carotid IMT in black men and higher carotid IMT in white men; and higher neighborhood poverty was associated with higher carotid IMT in black women but not in white women. In the Atherosclerosis Risk in Communities Study (ARIC), the lower the cumulative SES across the life span (based on childhood, young adulthood, and older adulthood) the higher the concurrent IMT in men but not in women (Carson et al., 2007), whereas progression of IMT was greater among lower income

whites and higher income blacks (Ranjit et al., 2006). Taken together, the IMT findings and our results demonstrate the complexity of associations of SES and measures of subclinical CVD, and highlight the importance of considering race and gender in understanding the influence of SES on risk for subclinical and clinical CVD.

In summary, lower SES based on average education, occupation, and household income prospectively measured across 15 years was a significant predictor of higher risk of any CAC. The findings were particularly striking in blacks. Improving occupational status through increasing opportunities, improving job training, and lowering barriers to achieving high status positions in blacks may constitute important public health interventions. Independent of adult SES, lower childhood SES was related to higher risk of CAC. No support was found for change in SES in adulthood being related to CAC. This paper is the first to report associations of SES prospectively measured in relation to CAC in black adults.

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