American Journal of Epidemiology
Vol. 183, No. 5
DOI: 10.1093/aje/kwv253
Advance Access publication:
February 8, 2016

Original Contribution

The Association of Socioeconomic Status With Subclinical Myocardial Damage, Incident Cardiovascular Events, and Mortality in the ARIC Study

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Initially submitted May 1, 2015; accepted for publication September 9, 2015.

The association between socioeconomic status (SES) and subclinical cardiovascular disease is not well understood. Using data from the Atherosclerosis Risk in Communities Study, we sought to evaluate the cross-sectional and prospective associations of SES, measured by annual income and educational level, with elevated high-sensitivity cardiac troponin T (hs-cTnT) concentrations (≥14 ng/L) using Poisson and multinomial logistic regressions, respectively. We used Cox proportional hazard models to compare the risks of coronary heart disease, heart failure, and mortality according to SES, stratified by baseline hs-cTnT concentration. Our study baseline was 1990–1992, with follow-up through 2011. We found an independent association between SES and hs-cTnT. When comparing participants in the lowest educational level group to those in the highest, the adjusted prevalence ratios for elevated hs-cTnT were 1.36 (95% confidence interval: 1.05, 1.75) overall, 1.83 (95% confidence interval: 1.23, 2.71) in blacks, and 1.05 (95% confidence interval: 0.73, 1.52) in whites (*P* for interaction = 0.08). Among participants with non-elevated hs-cTnT concentrations, when comparing those in the lowest income groups to those in the highest, the adjusted hazard ratios were strongest for heart failure and death. Having elevated baseline hs-cTnT doubled the risk of heart failure and death. Persons with low SES and elevated hs-cTnT concentrations have the greatest risk of cardiovascular events, which suggests that this group should be aggressively targeted for cardiovascular risk reduction.

high-sensitivity cardiac troponin T; racial health disparities; social determinants of health; socioeconomic status

Abbreviations: hs-cTnT, high-sensitivity cardiac troponin T; SES, socioeconomic status.

There are well-established associations between socioeconomic status (SES) and the risks of cardiovascular disease (1–16) and all-cause mortality (1, 3, 17, 18). The observed associations are not fully explained by health-related behaviors, lifestyle factors, or traditional cardiovascular risk factors (1, 3, 5, 7, 9–15, 17). Further, there is some evidence that the magnitude of the associations of SES with cardiovascular disease subtypes differ. The literature suggests that the independent associations of SES with cardiovascular outcomes and mortality at least partially reflect the influence of chronic stressors in daily life, which disproportionately affect persons in low SES groups. These chronic stressors might result in maladaptive physiologic coping mechanisms and chronic elevations in blood pressure and inflammation that can lead to physiologic injury of the vasculature and myocardium (19–21).

Data on SES as a risk factor for subclinical measures of cardiovascular disease, such as carotid intima-media thickness (22–25) and coronary artery calcification (26–28), have been less consistent than results from studies of hard clinical events, with some studies reporting significant independent associations (25, 27, 28) and others showing no association after adjustment for cardiovascular risk factors (22–24, 26). Additionally, associations of SES with dyslipidemia have been weak or nonsignificant (1, 3, 16, 29, 30), whereas the associations of SES with hypertension are quite robust (1, 3, 4, 11, 22, 31). These disparate findings suggest that the etiology underlying the role of SES in cardiovascular risk is heterogeneous and that nonatherosclerotic mechanisms may play an important role (19, 20).

Novel highly sensitive assays for cardiac troponin T (hs-cTnT) can detect cardiac troponin T at levels roughly

10 times lower than those detected using conventional assays (32). There is growing evidence that elevated hs-cTnT in asymptomatic populations with no history of cardiovascular disease indicates the presence of chronic subclinical myocardial damage (32–36). Recent studies have demonstrated that very low, previously undetectable, levels of hs-cTnT are prevalent in a large portion of the general population and are associated with significant cardiovascular risk, particularly heart failure and death (33–36). The association of SES with hs-cTnT is uncharacterized but could provide clues about the process by which SES leads to elevated cardiovascular risk. Thus, the objectives of the present study were 1) to determine whether SES is cross-sectionally and prospectively associated with chronic subclinical myocardial damage, as measured by hs-cTnT level, and 2) to evaluate the subsequent risk of cardiovascular events by SES among persons with and without evidence of chronic subclinical myocardial damage at baseline. Given the complex nature of SES as a multidimensional and dynamic exposure, prior research has suggested the utility of using more than 1 measure as a marker of SES (37–40). Thus, in this present study, we separately examined associations with annual income and educational level. Further, because results from prior studies have suggested substantial racial differences in hs-cTnT concentrations (41, 42) and differences by race in the associations of SES with health outcomes (26, 37, 43–46), we tested for possible effect modification by race. We hypothesized that the association of SES with hs-cTnT might be stronger among blacks. We also tested for potential interactions by age, given our a prior hypothesis that the independent association of SES with cardiovascular outcomes might be weaker in older individuals because of the greater burden and importance of competing clinical risk factors in this age group compared with younger individuals.

METHODS

Study population

The Atherosclerosis Risk in Communities (ARIC) Study is a community-based prospective cohort study of 15,792 participants sampled from 4 US communities: Washington County, Maryland; Forsyth County, North Carolina; Jackson, Mississippi; and the suburbs of Minneapolis, Minnesota. The study began in 1987 when investigators recruited participants who were 45–64 years of age. Three additional follow-up visits were conducted (once every 3 years), and a fifth visit was recently completed between 2011 and 2013. Institutional review boards at each of the 4 sites approved the study, and written informed consent was obtained from all participants at each study visit.

Hs-cTnT was first measured at visit 2 (1990–1992), which served as the baseline for the present study. There were 14,348 individuals who attended visit 2, and of these, 13,425 had valid measurements of hs-cTnT. We excluded individuals with prevalent coronary heart disease, including self-reported and silent myocardial infarction detected by electrocardiogram ($n = 769$), prevalent stroke ($n = 191$), prevalent heart failure ($n = 470$), or missing data on variables of interest ($n = 481$), as well as those from other racial groups ($n = 36$) and blacks from Minnesota and Maryland ($n = 37$), leaving 11,441 participants included in the final study population (Web Figure 1, available at http://aje.oxfordjournals.org/).

Socioeconomic status

SES was characterized using annual household income and lifetime educational level measured at visit 1 (1987–1989). Annual family income was categorized as low (<$16,000; $n = 2,149$), mid-level ($16,000–$34,999; $n = 3,619$), high (≥$35,000; $n = 5,238$), or not reported ($n = 435$). Participants in the last category were not included in our study because of the small sample size (23). These values reflect income in 1987–1989 as opposed to the present day. Lifetime educational level was defined as the highest grade or year of school completed, divided into 3 categories: low (less than 12th grade education; $n = 2,298$), mid-level (12th grade completion, general education diploma, vocational school graduate, or some college; $n = 4,799$), and high (college, graduate, or professional degree; $n = 4,344$). Using both income and educational level allows us to more comprehensively capture the cumulative results of SES on cardiovascular health over the life course in both black and white participants (15, 22, 26, 47–50).

Outcomes

Hs-cTnT concentrations were measured 6 years apart, using the same novel highly sensitive assay (Elecsys Troponin T, Roche Diagnostics, Indianapolis, Indiana). Hs-cTnT was measured in serum specimens from visit 2 (collected in 1990–1992) and stored at −80° until time of assay in 2012–2013 at the University of Minnesota, using a Roche Elecys 2010 Analyzer (Roche Diagnostics). Hs-cTnT was measured in plasma samples from visit 4 (collected in 1996–1998) using a Cobas e411 analyzer (Roche Diagnostics) at Baylor College of Medicine in 2011. Prior data demonstrated high correlation and no significant bias when comparing the measurements conducted at the 2 time points in the different laboratories (51). For the purposes of these analyses, we used Roche's threshold of 5 ng/L as the lower limit of detection (52, 53) and categorized hs-cTnT concentrations into 3 groups: <5 ng/L (undetectable), 5–13 ng/L (detectable), and ≥14 ng/L (elevated). The manufacturer reported 14 ng/L as the 99th percentile for a healthy reference population (35, 53, 54).

Cardiovascular events were ascertained via active surveillance of all participants in the study. Hospitalizations were identified through surveillance of hospitals within the study communities and reported by participants or their proxies during annual telephone calls. Study personnel abstracted potential cardiovascular events from hospital records. Coronary heart disease events were adjudicated by an endpoints committee and defined as definite or probable myocardial infarction or death from coronary heart disease. Heart failure was defined as hospitalization with *International Classification of Diseases, Ninth Revision* code 428, and events from 2005 were adjudicated. All-cause mortality was determined by death surveillance using hospital discharge records, coroner reports, the National Death Index, and next-of-kin interviews.

Covariates

All covariates were measured at visit 2 (1990–1992) unless otherwise indicated. Body mass index was calculated as measured weight in kilograms divided by height in meters squared (55). Lipid parameters were assessed using standard

Table 1. Baseline Characteristics by Category of High-Sensitivity Cardiac Troponin T Level at Visit 2, Atherosclerosis Risk in Communities Study, 1990–1992

Characteristic	Overall Study Population ($n = 11,441$)		Undetectable hs-cTnT (<5 ng/L) ($n = 7,585$)		Detectable hs-cTnT (5–13 ng/L) ($n = 3,418$)		Elevated hs-cTnT (≥14 ng/L) ($n = 438$)		*P* Value[a]
	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	Mean (SD)	%	
Age, years	56.7 (5.7)		55.7 (5.5)		58.4 (5.7)		60.0 (5.4)		<0.001
Female sex		57.3		66.3		40.2		28.2	<0.001
Black race		24.7		21.0		25.4		42.7	<0.001
Body mass index[b]									<0.001
Obese		28.2		24.7		30.5		41.4	<0.001
Overweight		39.9		39.9		42.2		35.8	0.016
Normal weight or underweight		31.9		35.4		27.3		23.2	<0.001
SBP, mm Hg	121.0 (18.6)		118.4 (17.0)		124.5 (19.1)		132.6 (24.7)		<0.001
DBP, mm Hg	72.2 (10.2)		71.3 (9.7)		73.3 (10.6)		74.0 (12.2)		<0.001
Hypertension		28.0		23.3		33.5		48.7	<0.001
Diabetes		13.3		9.6		17.0		34.7	<0.001
HbA1c, %	5.7 (1.1)		5.6 (0.9)		5.9 (1.3)		6.6 (2.0)		<0.001
Left ventricular hypertrophy		2.2		1.2		3.3		8.5	<0.001
Total cholesterol, mg/dL	209.6 (39.1)		209.5 (38.2)		209.6 (39.1)		211.8 (49.3)		0.486
HDL cholesterol, mg/dL	50.3 (16.8)		51.8 (17.0)		47.4 (15.9)		45.4 (17.4)		<0.001
eGFR <60 mL/min/1.73 m^2		1.5		0.7		1.9		12.9	<0.001
Physical activity index	2.4 (0.8)		2.4 (0.8)		2.5 (0.8)		2.3 (0.8)		<0.001
Smoking status									<0.001
Current		21.8		24.0		17.0		21.7	<0.001
Former		36.8		34.6		41.0		43.2	<0.001
Never		41.5		41.4		42.0		35.1	0.052
Drinking status									<0.001
Current		57.5		59.6		56.2		43.4	<0.001
Former		19.6		18.0		20.6		33.9	<0.001
Never		22.8		22.4		23.2		22.7	0.540
Educational level[c]									<0.001
Low		20.1		17.4		22.9		36.3	<0.001
Mid-level		42.0		44.3		38.4		32.7	<0.001
High		38.0		38.3		38.8		31.0	0.002
Income per year									<0.001
<$16,000		18.8		16.7		19.5		30.8	<0.001
$16,000–$34,999		31.6		31.3		31.9		32.0	0.762
≥$35,000		45.8		48.5		44.6		31.7	<0.001
Unknown		3.8		3.5		4.1		5.5	0.037

Abbreviations: DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; HbA1c, glycated hemoglobin; HDL, high-density lipoprotein; hs-cTnT, high-sensitivity cardiac troponin T; SBP, systolic blood pressure; SD, standard deviation.

[a] *P* values for the differences between hs-cTnT groups for continuous variables were obtained using an analysis of variance *F*-test. *P* values for different proportions between hs-cTnT groups for categorical/binary variables were obtained by χ^2 test.

[b] Weight (kg)/height $(m)^2$.

[c] High educational level was defined as a lifetime educational attainment of college, graduate, or professional degree; mid-level education, lifetime educational attainment of 12th grade completion, general education diploma, or vocational training; and low education, lifetime educational attainment of less than 12th grade.

Table 2. Cross-Sectional Association Between Baseline Socioeconomic Status and Elevated High-Sensitivity Cardiac Troponin T[a] at Visit 2, Atherosclerosis Risk in Communities Study, 1990–1992

Socioeconomic Status Indicator	Total ($n = 11,441$)				Black Participants ($n = 2,827$)		White Participants ($n = 8,614$)	
	Model 1[b]		Model 2[c]		Model 2[c]		Model 2[c]	
	PR	95% CI	PR	95% CI	PR	95% CI	PR	95% CI
Income[d]								
High	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
Mid-level	1.27	1.00, 1.62	1.07	0.89, 1.47	1.23	0.72, 2.11	1.02	0.76, 1.38
Low	1.74	1.32, 2.29	1.36	1.01, 1.82	1.63	0.96, 2.76	1.09	0.71, 1.68
P for trend[e]	<0.001		0.073		0.081		0.83	
Educational level[f]								
High	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
Mid-level	1.16	0.91, 1.46	1.12	0.88, 1.43	1.54	1.00, 2.38	0.96	0.71, 1.30
Low	1.54	1.21, 1.97	1.36	1.05, 1.75	1.83	1.23, 2.71	1.05	0.73, 1.52
P for trend[e]	<0.001		0.019		0.003		0.86	

Abbreviations: CI, confidence interval; PR, prevalence ratio.

[a] High-sensitivity cardiac troponin T concentration of 14 ng/L or more.

[b] Adjusted for age (years), race and study center (black participants from Jackson, Mississippi; black and white participants from Forsyth County, North Carolina; white participants from Minneapolis, Minnesota; and white participants from Washington County, Maryland), and sex (male or female).

[c] Adjusted for the variables in model 1 and total cholesterol (mg/dL), high-density lipoprotein cholesterol (mg/dL), systolic blood pressure (mm Hg), diastolic blood pressure (mm Hg), current antihypertensive medication use (yes or no), diabetes (yes or no), estimated glomerular filtration rate (mL/min/1.73 m^2), left ventricular hypertrophy (yes or no), body mass index (weight (kg)/height $(m)^2$), physical activity index score, alcohol consumption (current, former, or never), and tobacco use (current, former, or never).

[d] High income was defined as an annual family income greater than or equal to $35,000; mid-level income, annual family income between $16,000 and $34,999; and low income, annual family income less than $16,000.

[e] *P* for trend obtained from Wald test.

[f] High educational level was defined as a lifetime educational attainment of college, graduate, or professional degree; mid-level education, lifetime educational attainment of 12th grade completion, general education diploma, or vocational training; and low education, lifetime educational attainment of less than 12th grade.

procedures (56). For systolic and diastolic blood pressures, the averages of the second and third of 3 measurements were used for this study. Blood pressure medication use was self-reported, as were smoking, alcohol consumption, and physical activity level (measured at visit 1). Diabetes was defined as a self-reported physician diagnosis, current use of glucose-lowering medications, fasting blood glucose level of greater than or equal to 126 mg/dL (7.0 mmol/L), or random blood glucose level higher than 200 mg/dL (11.1 mmol/L). Glomerular filtration rate was estimated from a participant's serum creatinine level, age, sex, and race using the Chronic Kidney Disease Epidemiology Collaboration equation (57). Left ventricular hypertrophy was measured using electrocardiogram and defined using the Cornell criteria (58).

Statistical analysis

Characteristics of the study population were examined by calculating the means for continuous variables and proportions for categorical variables. Poisson regression was used to generate adjusted prevalence ratios for the cross-sectional associations of income and educational level with elevated (≥14 ng/L) versus nonelevated (<14 ng/L) hs-cTnT. Among persons with nonelevated hs-cTnT, we used multinomial logistic regression to generate risk ratios for the association of SES at visit 2 (baseline) with incident elevated hs-cTnT assessed at visit 4 (6-years later). The multinomial regression approach accounts for the cardiovascular events and deaths between visit 2 and visit 4 by allowing us to model these events as possibilities separate from incident and nonincident elevation of hs-cTnT. It is important to model these events as separate outcomes because their risks for incident elevated hs-cTnT would be very different and thus should be distinguished from the reference group that remained free of clinical disease and did not have incident elevated hs-cTnT. Prospective analyses of the association of SES with coronary heart disease, heart failure, and all-cause mortality in persons with and without elevated hs-cTnT levels at baseline were conducted using Cox proportional hazard models with follow-up for events until January 1, 2012. Model 1 was adjusted for age (years), sex (male or female) and race and field center (black participants from Mississippi; black and white participants from North Carolina; white participants from Minneapolis, Minnesota; and white participants from Maryland). Model 2 was additionally adjusted for body mass index, total cholesterol (mg/dL), high-density lipoprotein cholesterol (mg/dL), systolic blood pressure (mm Hg), diastolic blood pressure (mm Hg), use of blood pressure medications (yes or no), diabetes (yes or no), estimated glomerular filtration rate (mL/min/1.73 m^3), left ventricular hypertrophy (yes or no), smoking

Table 3. Prospective Associations Between Baseline Socioeconomic Status and Elevated High-Sensitivity Cardiac Troponin T[a] at Visit 4, Atherosclerosis Risk in Communities Study, 1996–1998

Socioeconomic Status Indicator	Total ($n = 11,003$)				Black Participants ($n = 2,626$)		White Participants ($n = 8,377$)	
	Model 1[b]		Model 2[c]		Model 2[c]		Model 2[c]	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Income[d]								
High	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
Mid-level	1.06	0.84, 1.33	0.96	0.75, 1.22	1.95	0.98, 3.90	0.85	0.65, 1.10
Low	1.07	0.78, 1.47	1.00	0.72, 1.40	2.28	1.12, 4.61	0.77	0.49, 1.21
P for trend[e]	0.27		0.60		0.028		0.33	
Educational level[f]								
High	1.00	Referent	1.00	Referent	1.00	Referent	1.00	Referent
Mid-level	0.95	0.76, 1.19	0.92	0.73, 1.16	1.14	0.65, 2.01	0.87	0.68, 1.13
Low	1.03	0.79, 1.35	0.97	0.73, 1.29	1.61	0.96, 2.70	0.78	0.55, 1.13
P for trend[e]	0.94		0.72		0.070		0.15	

Abbreviations: CI, confidence interval; RR, risk ratio.

[a] High-sensitivity cardiac troponin T concentration of 14 ng/L or more.

[b] Adjusted for age (years), race and study center (black participants from Jackson, Mississippi; black and white participants from Forsyth County, North Carolina; white participants from Minneapolis, Minnesota; and white participants from Washington County, Maryland), and sex (male or female).

[c] Adjusted for the variables in model 1 and total cholesterol (mg/dL), high-density lipoprotein cholesterol (mg/dL), systolic blood pressure (mm Hg), diastolic blood pressure (mm Hg), current antihypertensive medication use (yes or no), diabetes (yes or no), estimated glomerular filtration rate (mL/min/1.73 m^2), left ventricular hypertrophy (yes or no), body mass index (weight (kg)/height $(m)^2$), physical activity index score, alcohol consumption (current, former, or never), and tobacco use (current, former, or never).

[d] High income was defined as an annual family income greater than or equal to $35,000; mid-level income, annual family income between $16,000 and $34,999; and low income, annual family income less than $16,000.

[e] *P* for trend obtained from Wald test.

[f] High educational level was defined as a lifetime educational attainment of college, graduate, or professional degree; mid-level education, lifetime educational attainment of 12th grade completion, general education diploma, or vocational training; and low education, lifetime educational attainment of less than 12th grade.

status (never, former, or current), alcohol consumption (never, former, or current), and physical activity score (range, 0–5) (59). We tested for interaction by age and race and presented race-stratified results. We also assessed model discrimination using Harrell's *C* statistic (60) and improvement in risk classification for the addition of visit 2 hs-cTnT using continuous net-reclassification improvement statistic (61) stratified by categories of income and educational level. We conducted sensitivity analyses in which we examined occupation as an additional indicator of SES.

All statistical analyses were performed using Stata, version 13 (StataCorp LP, College Station, Texas). Statistical significance was defined a priori as a 2-sided $P < 0.05$.

RESULTS

Baseline characteristics

Participants had a median age of 57 years; 57% were female, 25% were black, 3.8% had elevated hs-cTnT levels (≥14 ng/L), and 30% had detectable hs-cTnT levels (5–13 ng/L) at baseline (visit 2). Persons with the highest levels of hs-cTnT had a higher burden of cardiovascular risk factors and lower SES (Table 1). For example, individuals who had elevated hs-cTnT levels at baseline were more likely to be male, black, obese, hypertensive, and diabetic and to have left ventricular hypertrophy than were persons with undetectable hs-cTnT levels (all $P < 0.001$). In addition, 36.5% individuals in the elevated hs-cTnT group had less than a 12th grade education, compared with 23.4% in the detectable group and 17.6% in the undetectable group ($P < 0.001$). Similar to the trend for educational attainment, 31.7% of those with elevated hs-cTnT compared with only 17.3% of those with undetectable hs-cTnT had low income ($P < 0.001$).

Associations of SES with elevated hs-cTnT

Lower SES was significantly associated with cross-sectional elevation of hs-cTnT even after adjustment for study variables. The adjusted demographic prevalence ratios were 1.74 (95% confidence interval: 1.32, 2.29) and 1.54 (95% confidence interval: 1.21, 1.97) for low income and low educational level, compared with high income and high educational level, respectively (Table 2). After adjustment for demographic characteristics, cardiovascular risk factors, and health behaviors, there remained an independent association of income and educational level with elevated hs-cTnT: The prevalence ratios were 1.36 (95% confidence interval: 1.01, 1.82) for income

Table 4. Risk of Incident Coronary Heart Disease, Heart Failure, and Death Over the Study Follow-Up Period, by Socioeconomic Status and High-Sensitivity Cardiac Troponin T Concentration at Baseline, Atherosclerosis Risk in Communities Study, 1990–1992

Socioeconomic Status Indicator and Baseline Troponin Level	Coronary Heart Disease			Heart Failure			Death		
	IR[a]	HR[b]	95% CI	IR[a]	HR[b]	95% CI	IR[a]	HR[b]	95% CI
Income[c]									
hs-cTnT <14 ng/L									
High	5.67	1.00	Referent	6.97	1.00	Referent	12.48	1.00	Referent
Mid-level	5.70	1.01	0.87, 1.17	8.72	1.30	1.14, 1.49	15.62	1.28	1.17, 1.41
Low	7.24	1.30	1.08, 1.56	10.43	1.61	1.38, 1.89	19.32	1.62	1.45, 1.82
hs-cTnT ≥14 ng/L									
High	7.19	1.30	0.85, 1.99	12.82	1.91	1.35, 2.70	22.02	1.81	1.40, 2.35
Mid-level	8.90	1.76	1.22, 2.53	18.81	3.28	2.48, 4.33	25.54	2.42	1.93, 3.03
Low	11.78	2.10	1.50, 2.94	19.70	3.54	2.66, 4.72	29.43	2.91	2.31, 3.66
Educational level[d]									
hs-cTnT <14 ng/L									
High	5.41	1.00	Referent	7.74	1.00	Referent	13.87	1.00	Referent
Mid-level	6.07	1.12	0.96, 1.30	8.16	1.08	0.95, 1.22	15.15	1.11	1.01, 1.21
Low	6.95	1.30	1.09, 1.54	10.28	1.43	1.24, 1.64	17.04	1.26	1.13, 1.40
hs-cTnT ≥14 ng/L									
High	9.96	1.95	1.33, 2.86	14.44	2.03	1.45, 2.84	23.04	1.85	1.43, 2.40
Mid-level	9.00	1.78	1.24, 2.54	17.53	2.63	1.99, 3.49	24.04	1.96	1.57, 2.45
Low	10.19	2.10	1.50, 2.94	19.38	3.08	2.38, 3.99	26.73	2.29	1.86, 2.83

Abbreviations: CI, confidence interval; HR, hazard ratio; hs-cTnT, high-sensitivity cardiac troponin T; IR, incidence rate.

[a] Incidence rate reported per 1,000 person-years.

[b] Adjusted for age (years), race and study center (black participants from Jackson, Mississippi; black and white participants from Forsyth County, North Carolina; white participants from Minneapolis, Minnesota; and white participants from Washington County, Maryland), sex (male or female), total cholesterol (mg/dL), high-density lipoprotein cholesterol (mg/dL), systolic blood pressure (mm Hg), diastolic blood pressure (mm Hg), current antihypertensive medication use (yes or no), diabetes (yes or no), estimated glomerular filtration rate (mL/min/1.73 m^2), left ventricular hypertrophy (yes or no), body mass index (weight (kg)/height $(m)^2$), physical activity index score, alcohol consumption (current, former, or never), and tobacco use (current, former, or never).

[c] High income was defined as an annual family income greater than or equal to $35,000; mid-level income, annual family income between $16,000 and $34,999; and low income, annual family income less than $16,000.

[d] High educational level was defined as a lifetime educational attainment of college, graduate, or professional degree; mid-level education, lifetime educational attainment of 12th grade completion, general education diploma, or vocational training; and low education, lifetime educational attainment of less than 12th grade.

and 1.36 (95% confidence interval: 1.05, 1.75) for education when comparing the low groups to high groups.

Race-stratified results demonstrated that, compared with whites, blacks had a marginally stronger association between SES and elevated hs-cTnT, and in fact this was the only significant association (Table 2). Among blacks, persons with the lowest level of education had a prevalence ratio of 1.83 for elevated baseline hs-cTnT (95% confidence interval: 1.23, 2.71) when compared with those with highest level of education (P for interaction = 0.08). In white participants, the analogous prevalence ratio for elevated baseline hs-cTnT was 1.05 (95% confidence interval: 0.73, 1.52). These results were similar, but interaction by race was not statistically significant when using income as an indicator of SES (P for interaction = 0.33).

There was no association between SES and incident elevated hs-cTnT in the overall sample (Table 3). However, the race-stratified analysis shows an increased risk of elevated hs-cTnT for low-income blacks (risk ratio = 2.28, 95% confidence interval: 1.12, 4.61) but not whites (risk ratio = 0.77, 95% confidence interval: 0.49, 1.21) (P for interaction = 0.20). Additional adjustment for baseline hs-cTnT did not appreciably alter the results. A cross-tabulation of income, educational level, and race is provided in Web Table 1. Sensitivity analyses in which we used occupation demonstrated similar cross-sectional and prospective results that were not statistically significant.

Prospective associations of SES with incident events

The median follow-up for all outcomes was approximately 20 years. During follow-up, there were 1,140 coronary heart disease events (56.8 per 10,000 person-years), 1,628 heart failure events (81.0 per 10,000 person-years), and 3,042

deaths (146.3 per 10,000 person-years). There was a robust, independent association of SES with cardiovascular events, as well as with death (Table 4). The highest risk of events, particularly heart failure and death, was observed among those persons with both low SES and elevated hs-cTnT at baseline.

Compared with the base model, the model that included hs-cTnT significantly improved prediction of clinical outcomes in 10 of the 18 models tested, as measured by the *C* statistic, and resulted in significant loss of predictive accuracy in only 1 model (Web Table 2). In general, the biggest improvements were seen for the outcomes of heart failure and death among all SES groups, but they were particularly pronounced among persons in the low education and low income groups. Formal tests for interaction by age and race were not statistically significant for any outcome; however, race-stratified analyses (Web Table 3) demonstrated slightly stronger associations between SES and incident events in blacks than in whites. Only a small proportion of eligible participants were missing data (10.6%), and our study population was similar to the overall eligible study population at baseline (Web Table 4).

DISCUSSION

In the present community-based cohort study of 11,441 participants free of cardiovascular disease at baseline, we found statistically significant independent cross-sectional associations between SES and subclinical myocardial damage as assessed by elevated hs-cTnT. We found slightly stronger associations in blacks than in whites; in fact, in stratified analyses, the association was only significant in blacks. The prospective association of SES with incident elevated hs-cTnT also was significant in blacks but not whites. In such, our results suggest that SES may contribute to the presence and progression of chronic subclinical myocardial damage, particularly in African-American adults. Our finding is consistent with literature in which investigators noted strong associations between SES and clinical manifestations of microvascular damage, such as hypertension (1, 3, 4, 11, 22, 31) and diabetes (1, 29, 31), but weaker relationships with macrovascular, atherosclerotic markers such as blood lipids, coronary artery calcium, and carotid intima-medial thickness (1, 3, 16, 29, 30). Our results suggest that factors associated with lower SES may contribute to the development of chronic subclinical myocardial damage.

In this cohort with more than 20 years of follow-up, we found robust associations of SES with clinical cardiovascular events. The consistent and strong relationship between SES and incident events not only confirms results from previous studies but also extends the literature by showing stronger relative associations with heart failure and death. It has been these suggested that the associations are mediated by both chronic inflammation and elevations in blood pressure (1–3), resulting in microvascular and structural heart damage. We further observed that elevated baseline hs-cTnT increased the risk of heart failure and mortality by twice as much. This additional risk appears to be slightly stronger in blacks than in whites, although we are limited by sample size in race-stratified analyses. Incidence rates also demonstrated the greater absolute risk of events, particularly heart failure, in blacks. These findings suggest that persons with low SES, those with elevated hs-cTnT at baseline, and particularly blacks should be targeted for aggressive cardiovascular risk reduction.

The finding that SES is independently associated with elevated hs-cTnT concentrations is novel. The mechanisms by which socioeconomic deprivation may influence myocardial damage are not entirely clear, but they might include elevated blood pressure and chronic inflammation (1–3). There was some evidence of possible racial differences in our findings. The clinical manifestations of low SES may be more severe and possibly more poorly recognized and controlled in blacks than in whites because of differences in access to health care (62–66), quality of care (62–67), and medication adherence (62, 63, 67), resulting in more myocardial damage and future excess risk. Our results suggest that hs-cTnT may be a useful biomarker for identifying persons at future risk, particularly for heart failure and death.

This study has several strengths. It is a large, community-based prospective cohort study of more than 11,000 black and white participants with thorough measurement of risk factors and more than 20 years of rigorous surveillance for cardiovascular events. To our knowledge, this is the first study in which the association of SES with subclinical myocardial damage has been examined in an asymptomatic population with no history of cardiovascular disease. However, there are also some limitations to the study. SES is a highly complex measure, and it certainly extends to an individual's social context far beyond their annual family income and education level. Sensitivity analysis in which we examined occupation as an SES indicator showed similar results, but the results were not statistically significant. In addition, the exact mechanisms by which low SES leads to cardiac and vascular damage remain unclear, although our study demonstrates a significant association with elevated hs-cTnT. In the context of our findings and previous research that demonstrated inconsistent and weak associations between SES and atherosclerotic markers, nonatherosclerotic mechanisms may play an important role in how SES is associated with myocardial damage that leads to future events. Further mechanistic research is needed to elucidate the pathophysiologic mechanisms to better target interventions and treatment.

In conclusion, SES was associated with subclinical myocardial damage, and persons with low SES and elevated levels of hs-cTnT were at highest risk of future events and death independent of traditional cardiovascular risk factors and behavioral and lifestyle factors. The additional increased risk of heart failure and death due to elevated baseline hs-cTnT suggests that hs-cTnT may be a useful biomarker for identifying persons at risk of future cardiovascular events.

ACKNOWLEDGMENTS

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This work was supported by the grant R01 DK089174 from the National Institutes of Health/National Institute of Diabetes and Digestive and Kidney Diseases (E.S.). The Atherosclerosis Risk in Communities Study is carried out as a collaborative study supported by National Heart, Lung, and Blood Institute contracts HHSN268201100005C, HHSN 268201100006C, HHSN268201100007C, HHSN2682011 00008C, HHSN268201100009C, HHSN268201100010C, HHSN268201100011C, and HHSN268201100012C. J.M. is supported by the Pollin Cardiovascular Prevention Fellowship. Roche Diagnostics provided reagents and loan of an instrument to conduct the high-sensitivity cardiac troponin T assay.

We thank Yuan Chen for her assistance with statistical analyses.

This research was presented in abstract form at the American Heart Association Epidemiology, Prevention, Lifestyle and Cardiometabolic Health 2015 Scientific Sessions (moderated poster), March 3–6, 2015, Baltimore, Maryland.

Roche had no role in design, analysis or manuscript preparation.

Conflict of interest: R.H. and C.M.B. have received grant support from Roche Diagnostics. R.H. and C.M.B. are coinvestigators on a provisional patent filed by Roche for use of biomarkers in heart failure prediction. C.M.B. and E.S. have served on an advisory board for Roche Diagnostics. The other authors report no conflicts.

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