Neighborhood Socioeconomic Status and Adverse Outcomes in Patients With Cardiovascular Disease

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Neighborhood socioeconomic status (nSES) is associated with cardiovascular morbidity and mortality in the general population; however, its effect on high-risk patients with prevalent coronary artery disease (CAD) is unclear. We hypothesized "double jeopardy," whereby the association between nSES and adverse outcomes would be greater in high-risk patients with heart failure (HF) and/or previous myocardial infarction (MI) compared with those without. We followed 3,635 patients (mean age 63.2 years, 42% with HF, 25% with previous MI) with known or suspected CAD over a median of 3.3 years for all-cause death and cardiovascular death or nonfatal MI. Patients were categorized by a composite nSES score, and proportional hazards models were used to determine the association between nSES and outcomes. Cross-product interaction terms for previous MI × nSES and HF × nSES were analyzed. Compared with high nSES patients, low nSES patients had increased risk of all-cause death (hazard ratio [HR] = 1.61; 95% confidence interval [CI] = 1.20, 2.15) and cardiovascular death or MI (subdistribution HR [sHR] = 1.82; 95% CI = 1.30, 2.54). Associations were more pronounced among patients without HF or previous MI. Low nSES patients without HF had a higher risk of all-cause death (HR = 2.27; 95% CI = 1.41, 3.65) compared with those with HF (HR = 1.21; 95% CI = 0.82, 1.77, P interaction = 0.04). Similarly, low nSES patients without previous MI had a higher risk of cardiovascular death or MI (sHR = 2.72; 95% CI = 1.73, 4.28) compared with those with previous MI (sHR = 1.02; 95% CI = 0.58, 1.81, P interaction = 0.02). In conclusion, low nSES was independently associated with all-cause death and cardiovascular death or MI in patients with CAD; however, associations were greater in patients without HF or previous MI compared with those with HF or MI. **(Am J Cardiol 2019;123:284−290)**

Neighborhood socioeconomic status (nSES) is a key social determinant of health,[1] and its association with cardiovascular health in the general population is well established.[2–9] Neighborhood deprivation and low nSES are additionally associated with adverse outcomes in patients with heart failure (HF),[10] stroke,[11] and those hospitalized for acute myocardial infarction (MI)[12]; however, findings are inconsistent across disease type and outcome of interest.[13,14] Most studies suggest a "double jeopardy" hypothesis, whereby risk of adverse events is compounded by the simultaneous presence of low nSES and high-risk clinical cardiovascular disease (CVD), but explicit investigations of the interaction among nSES, CVD, and adverse outcomes are lacking. We sought to investigate the associations between nSES and adverse outcomes in a cohort of patients with prevalent coronary artery disease (CAD), specifically exploring whether the association between nSES and adverse outcomes is modified by high-risk prevalent CVD. We hypothesized that patients living in low nSES areas would have worse outcomes, and that the association of low nSES would be greater among patients with HF or previous MI.

Methods

We studied 3,635 adults, aged 21 years and older enrolled in the Emory Cardiovascular Biobank, a prospective cohort of patients who underwent left heart catheterization for suspected or confirmed CAD in Atlanta, Georgia, between 2004 and 2014. Participants were interviewed to collect demographic characteristics, medical history, medication use, and behavioral habits. Patients with previous cardiac transplantation or under consideration for transplant were excluded. All participants provided written informed consent at the time of enrollment, and the study was approved by the institutional review board at Emory University (Atlanta, Georgia).

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Participants' residential addresses were geocoded by using latitude and longitude coordinates with ArcMap 10.2 (Environmental Systems Research Institute, Inc., Redlands, California) and 2010 US Census TIGER/Line Shapefiles based on the North American Industry Classification. Of patients, 9% were not located in the state of Georgia (n = 463) and were excluded due to incomplete follow-up. Additionally, 19% of patients were unmatched due to missing addresses, use of PO boxes, or were not located by the Geographic Information Systems (GIS) software (n = 967). Compared with patients outside of Georgia or those who were not geocoded, geocoded patients were younger, more likely to be black, more likely to have a history of smoking and hypertension, and less likely to use antihypertensive medications or have a history of revascularization.

Using the geocoded coordinates, data from the 2006 to 2010 United States Census Bureau's American Community Survey (ACS) 5-year estimates were merged with clinical data at the census-tract level for residents living in Georgia, resulting in 3,635 participants who represented 1,114 census tracts across Georgia. The median number of patients per census tract was 2 (interquartile range 1 to 4), and the maximum number of patients from any census tract was 22.

As previously described,[6,15] a composite nSES was determined from 6 census variables: (1) median household income; (2) median value of owner-occupied housing units; (3) percentage of adults ≥25 years of age who have graduated high school; (4) percentage of adults ≥25 years of age who have graduated college; (5) percentage of persons in management, business, science, and arts occupations; and (6) percentage of households with interest, dividend, or rental income. Median household income and median value of housing units were log-transformed due to their skewed distributions, and each variable was standardized and summed together to create an overall *Z* score, which ranged from −12.62 (lowest) to 16.67 (highest). Summary scores were separated by quartiles and categorized into 3 groups: low nSES (quartile 1, nSES score −12.62 to −3.96), middle nSES (quartiles 2 and 3, nSES score −3.96 to 3.34), and high nSES (quartile 4, nSES score 3.34 to 16.67). High nSES served as the referent group and represented patients from neighborhoods with greatest socioeconomic advantage.

Patients enrolled in the Emory Cardiovascular Biobank underwent a detailed baseline evaluation by using standardized self-report questionnaires and medical records review. Age (years), gender (male vs female), race (white vs black), and smoking (current or former vs never) were obtained by self-report. Additionally, medical history was obtained by self-report and confirmed by medical records evaluation and/or medication use for the following conditions: hypertension, diabetes, hyperlipidemia, HF, previous MI, and previous revascularization (either percutaneous coronary intervention or coronary artery bypass grafting). Patients were additionally categorized by the presence or absence of an acute coronary syndrome on presentation for cardiac catheterization. Body mass index (in kg/m^2) and systolic blood pressure (in mm Hg) were measured by trained staff. Routine laboratory data included fasting values of low-density lipoprotein cholesterol, and serum creatinine (mg/dl), which was used to calculate the estimated glomerular filtration rate (in ml/min/1.73 m^2). All participants underwent a detailed medication questionnaire to document use of the following: antihypertensives (angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, and beta blockers), aspirin, clopidogrel, and statins. Obstructive CAD was defined as the presence of ≥50% stenosis at least 1 major epicardial vessel.

Follow-up was conducted by phone, electronic medical record review, social security death index, and state records to identify cardiovascular death or nonfatal MI. Cardiovascular death was defined as death from MI, HF, sudden death, stroke, pulmonary embolization, or as a complication during any cardiovascular-related procedure. MI was defined according to relevant medical history, diagnostic cardiac enzymes, and/or electrocardiogram tracing consistent with myocardial injury. Event adjudication was conducted by 2 independent, board-certified cardiologists blinded to baseline characteristics.

Demographic factors, education, marital status, nSES, clinical risk factors, biomarkers, and outcomes were reported, by nSES group, as n (%) or mean ± standard deviation. Analysis of variance (continuous, normally distributed variables) and the chi-square test (categorical variables) were used to compare baseline clinical characteristics between groups of nSES.

The cumulative incidences of all-cause death and cardiovascular death or MI were plotted as 1-survival for each nSES group. Follow-up time was defined as the time from enrollment until one of the following: death, cardiovascular death, MI, loss to follow-up, or end of follow-up. Cox proportional hazard models were used to determine the association between nSES and all-cause death. We performed competing risk analyses for cardiovascular death or MI by using Fine and Gray's method, treating noncardiovascular death as a competing risk, and stepwise proportional hazards models were used to determine the association between nSES category and outcomes. As previously described, high nSES was used as the reference. There were no violations of the proportional hazards assumption. A robust sandwich estimator was used to account for residual correlation among patients living in the same census tract.[16]

Modeling steps were performed to determine the effect of additional adjustment for levels of risk factors. Sensitivity analysis was performed in a subset of patients with stable CAD and excluded patients with normal coronary arteries (n = 370) and those presenting with ACS (n = 351). Cross-product interaction terms for nSES × previous MI and nSES × HF were specifically tested to determine if the association between low nSES and outcomes differed by severity of prevalent CVD, as these conditions are associated with particularly high rates of adverse outcomes. Further stratified analyses were performed for previous revascularization, ACS on admission, age (<60 years vs ≥60 years), race, gender, aspirin use, and statin use. Statistical significance was defined as $p < 0.05$ (2-sided) for all main effects and interaction terms. SAS version 9.4 (Cary, North Carolina) was used for all analyses.

Results

Overall, the study cohort consisted of 3,635 patients. Low nSES patients were younger, more likely to be female

Table 1
Baseline characteristics of the cohort

Variable	Overall (n = 3,635)	Categories of neighborhood socioeconomic status			
		Low (n = 910)	Middle (n = 1,823)	High (n = 902)	p value
Age, mean ± SD (years)	63.2 ± 12.2	61.8 ± 12.1	62.9 ± 12.1	65.3 ± 12.2	< 0.001
Women	1,309 (36%)	380 (42%)	694 (38%)	235 (26%)	< 0.001
Black	943 (26%)	333 (37%)	556 (31%)	54 (6%)	< 0.001
Married	2,429 (67%)	524 (58%)	1,220 (67%)	685 (76%)	< 0.001
College graduate	1,330 (37%)	200 (22%)	580 (32%)	550 (61%)	< 0.001
Median household income, mean ± SD (in $1,000)	56.8 ± 25.6	33.6 ± 8.3	52.9 ± 12.1	88.1 ± 26.8	< 0.001
Median value of housing units, mean ± SD (in $1,000)	196.7 ± 111.1	113.6 ± 28.0	169.6 ± 34.5	335.5 ± 137.5	< 0.001
Percent of households with interest, dividends, or rental income, mean ± SD (%)	20.5 ± 13.0	10.4 ± 5.3	16.9 ± 7.4	37.8 ± 10.5	< 0.001
Percent of adult residents who completed high school, mean ± SD (%)	85.3 ± 9.2	73.9 ± 6.5	86.3 ± 5.6	94.9 ± 3.0	< 0.001
Percent of adult residents who completed college, mean ± SD (%)	19.3 ± 11.1	8.0 ± 3.2	17.3 ± 5.7	34.5 ± 7.3	< 0.001
Percent of employed residents with executive, managerial, or professional occupation, mean ± SD (%)	37.2 ± 15.4	21.7 ± 6.1	34.5 ± 7.2	58.5 ± 10.0	< 0.001
nSES score, mean ± SD (z-score)	0.0 ± 5.3	−6.1 ± 1.7	−0.6 ± 1.9	7.6 ± 2.9	< 0.001
Current/former smoker	2,493 (69%)	654 (72%)	1,233 (68%)	606 (67%)	0.047
Body mass index, mean ± SD (kg/m^2)	29.8 ± 6.3	30.1 ± 6.2	30.1 ± 6.5	29.0 ± 6.0	< 0.001
Hypertension	2,941 (81%)	779 (86%)	1,476 (81%)	686 (76%)	< 0.001
Systolic blood pressure, mean ± SD (mm Hg)	137.6 ± 21.8	139.8 ± 23.2	137.3 ± 21.5	136.0 ± 21.0	< 0.001
Antihypertensive use	2,877 (79%)	706 (78%)	1,447 (79%)	724 (80%)	0.35
Diabetes mellitus	1,335 (37%)	342 (38%)	712 (39%)	281 (31%)	< 0.001
Hyperlipidemia	2,596 (72%)	633 (70%)	1,289 (71%)	674 (75%)	0.037
Low-density lipoprotein cholesterol, mean ± SD (mg/dL)	94.9 ± 36.3	97.3 ± 37.6	96.2 ± 37.4	90.1 ± 32.3	< 0.001
Prior myocardial infarction	887 (25%)	229 (26%)	443 (25%)	215 (24%)	0.78
History of revascularization	1,914 (53%)	489 (54%)	950 (52%)	475 (53%)	0.73
Obstructive coronary artery disease	2,629 (81%)	662 (81%)	1,314 (81%)	653 (80%)	0.84
Acute coronary syndrome on admission	744 (21%)	170 (19%)	389 (21%)	185 (21%)	0.27
Statin use	2,575 (71%)	606 (67%)	1,293 (71%)	676 (75%)	< 0.001
Aspirin use	2,761 (76%)	651 (72%)	1,402 (77%)	708 (79%)	0.001
Clopidogrel use	1,590 (44%)	415 (46%)	796 (44%)	379 (42%)	0.30
Heart failure	1,403 (42%)	382 (46%)	707 (42%)	314 (37%)	< 0.001
Estimated glomerular filtration rate, mean ± SD (mL/min/1.73 m^2)	72.6 ± 24.8	71.8 ± 27.2	72.8 ± 25.3	73.1 ± 20.8	0.48
All-cause death	610 (17%)	171 (19%)	308 (17%)	131 (15%)	0.015
Cardiovascular death	369 (10%)	112 (12%)	184 (10%)	73 (8%)	0.012
Non-fatal myocardial infarction	188 (5%)	52 (6%)	109 (6%)	27 (3%)	0.003
Cardiovascular death or non-fatal myocardial infarction	487 (13%)	146 (16%)	251 (14%)	90 (10%)	< 0.001

and black, and were less likely to be married or college graduates (Table 1). Additionally, the prevalence of traditional cardiovascular risk factors and HF was higher, whereas the use of statins and aspirin was lower, among lower nSES patients (Table 1).

Over a median follow-up period of 3.3 years (interquartile range 1.6 to 6.4 years), a total of 610 (17%) all-cause deaths, 369 (10%) cardiovascular deaths, and 188 (5%) MIs occurred. The cumulative incidence for all-cause death and cardiovascular death or MI with respect to nSES are shown in Figure 1. There was a stepwise increase in the risk of adverse outcomes with decreasing nSES. Compared with high nSES patients, those in the middle and low nSES groups had increased risk of all-cause death and cardiovascular death or MI (Table 2). After adjustment for demographics, education, marital status, and clinical risk factors, the association between nSES and adverse outcomes was attenuated for middle nSES patients but remained statistically significant for low nSES patients (Table 2). Similar findings were found in a subset of patients with stable CAD (Table 3).

The absolute incidence rates of all-cause death and cardiovascular death or MI were greater in patients with a history of HF or previous MI at all levels of nSES (Table 4). There was a significant interaction between low nSES and HF for the outcome of all-cause death; those without HF had a higher risk for death than those with HF (Figure 2). Similarly, there was a significant interaction between low nSES and previous MI for the outcome of cardiovascular death or MI; those without previous MI had a higher risk for cardiovascular death or MI than those without previous MI (Figure 2).

Sensitivity analyses revealed no significant interactions between low nSES and age (<60 years vs ≥ 60 years), race (white vs black), gender (male vs female) previous revascularization, ACS on admission, aspirin use, or statin use (all P interaction > 0.10).

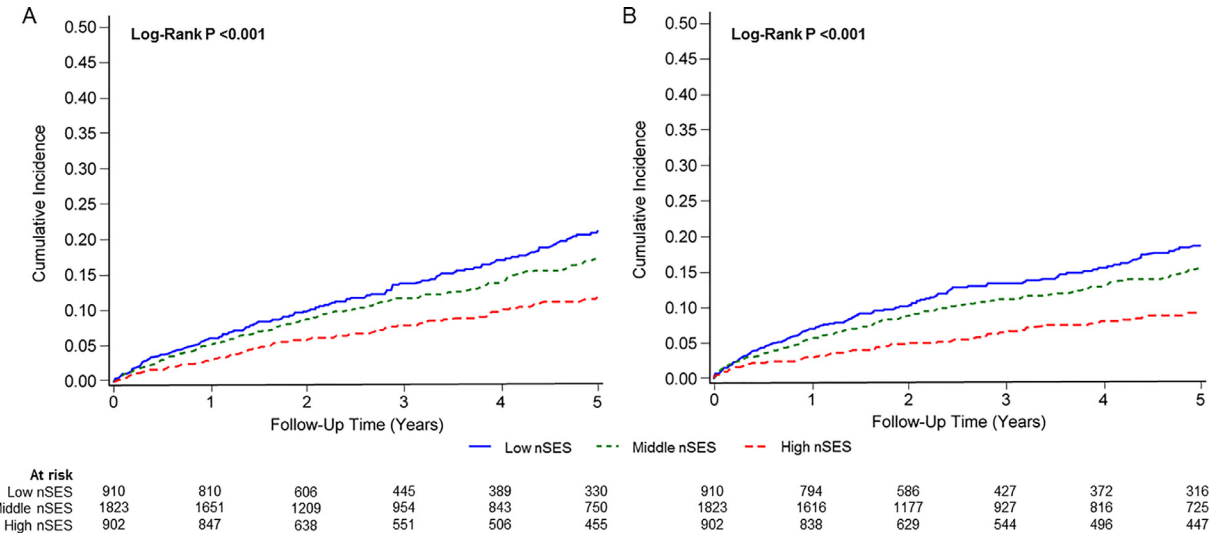

Figure 1. Cumulative incidence plots for adverse events. Unadjusted cumulative incidence curves for *(A)* all-cause mortality and *(B)* cardiovascular death or nonfatal myocardial infarction across categories of neighborhood socioeconomic status (nSES). The lowest quartile of nSES (Q1) is denoted by the *solid blue line*. (Color version of figure is available online.)

Discussion

In this study, we demonstrate that among high-risk patients with CAD, living in census tracts with low compared with high nSES is independently associated with an increased risk of all-cause death and cardiovascular death or MI, irrespective of demographic, clinical, or individual-level SES characteristics. Furthermore, despite a higher absolute adverse event rate in patients with HF or previous MI, the relative association between low nSES and adverse outcomes was greater in those without HF or previous MI. This is in contrast to our expected hypothesis that the combination of low nSES and cardiovascular disease would be associated with worse outcomes and prompts inquiry into the cause of this seeming paradox.

While population studies have shown a consistent association between lower nSES and worse CVD outcomes[3–9]; data from high-risk CV cohorts are varied. In patients with acute events, such as MI or stroke, disparities in outcomes between low and high nSES patients are typically attributed to differences in processes of care.[13,14] Furthermore, these associations between nSES and outcomes are largely attenuated after accounting for individual-level SES and other high-risk clinical morbidities.[5] Although most studies suggest that the combination of low nSES and prevalent CVD result in worse outcomes, few have explicitly sought to answer this question. Surprisingly, in those that have the associations between low nSES and adverse outcomes were either equivalent to[17] or more pronounced[18] in patients without CVD than in those with CVD. Because our study had similarly unexpected findings, further exploration is warranted.

At the individual level, perhaps a history of HF or MI is "protective" in low nSES patients. Previous studies have

Table 2
Association between categories of neighborhood socioeconomic status and incident events

Variable	Unadjusted HR or sHR (95% CI)	Demographic model* HR or sHR (95% CI)	Individual SES† model HR or sHR (95% CI)	Clinical model‡ HR or sHR (95% CI)
All-cause death				
High nSES (n = 910)	Reference	Reference	Reference	Reference
Middle nSES (n = 1,823)	1.32 (1.08-1.62)	1.39 (1.13-1.72)	1.32 (1.07-1.64)	1.23 (0.95-1.60)
Low nSES (n = 902)	1.57 (1.27-1.98)	1.73 (1.37-2.18)	1.58 (1.24-2.02)	1.61 (1.20-2.15)
Linear trend p-value	< 0.001	< 0.001	0.001	0.002
Cardiovascular death or MI				
High nSES (n = 910)	Reference	Reference	Reference	Reference
Middle nSES (n = 1,823)	1.54 (1.22-1.96)	1.52 (1.19-1.94)	1.40 (1.09-1.79)	1.32 (0.97-1.80)
Low nSES (n = 902)	1.91 (1.47-2.48)	1.91 (1.46-2.49)	1.66 (1.26-2.19)	1.82 (1.30-2.54)
Linear trend p-value	< 0.001	< 0.001	0.002	0.002

BMI = body mass index; CAD = coronary artery disease; CI = confidence interval; eGFR = estimated glomerular filtration rate; HR = hazard ratio; LDL-C = low-density lipoprotein cholesterol; MI = myocardial infarction; SBP = systolic blood pressure; SES = socioeconomic status; sHR = subdistribution hazard ratio.

* Model adjusted for age, gender, race, and year of enrollment.

† Model adjusted for demographic model covariates plus individual education and marital status.

‡ Model adjusted for individual SES model covariates plus BMI, smoking history, acute coronary syndrome on admission, previous MI, previous revascularization, heart failure, obstructive CAD, diabetes, dyslipidemia, antihypertensive use, statin use, aspirin use, clopidogrel use, SBP, LDL-C, and eGFR.

Table 3
Association between categories of neighborhood socioeconomic status and incident events, excluding patients presenting with acute coronary syndrome (n = 351) and those with normal coronary arteries (n = 370)

Variable	Unadjusted HR or sHR (95% CI)	Demographic model* HR or sHR (95% CI)	Individual SES† model HR or sHR (95% CI)	Clinical model‡ HR or sHR (95% CI)
All-cause death				
High nSES (n = 677)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Middle nSES (n = 1330)	1.24 (0.99-1.57)	1.35 (1.07-1.72)	1.29 (1.01-1.64)	1.22 (0.92-1.63)
Low nSES (n = 664)	1.45 (1.12-1.89)	1.64 (1.26-2.14)	1.50 (1.14-1.98)	1.52 (1.10-2.10)
Linear trend p-value	0.02	0.001	0.02	0.04
Cardiovascular death or MI				
High nSES (n = 667)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Middle nSES (n = 1330)	1.29 (0.98-1.69)	1.29 (0.97-1.70)	1.19 (0.89-1.58)	1.18 (0.83-1.68)
Low nSES (n = 664)	1.67 (1.25-2.24)	1.70 (1.25-2.30)	1.47 (1.07-2.02)	1.70 (1.17-2.49)
Linear trend p-value	0.003	0.003	0.05	0.01

BMI = body mass index; CAD = coronary artery disease; CI = confidence interval; eGFR = estimated glomerular filtration rate; HR = hazard ratio; LDL-C = low-density lipoprotein cholesterol; MI = myocardial infarction; SBP = systolic blood pressure; SES = socioeconomic status; sHR = subdistribution hazard ratio.

* Model adjusted for age, gender, race, and year of enrollment.

† Model adjusted for demographic model covariates plus individual education and marital status.

‡ Model adjusted for individual SES model covariates plus BMI, smoking history, acute coronary syndrome on admission, previous MI, previous revascularization, heart failure, obstructive CAD, diabetes, dyslipidemia, antihypertensive use, statin use, aspirin use, clopidogrel use, SBP, LDL-C, and eGFR.

Table 4
Absolute and relative incident rates of adverse events among individuals with and without a heart failure or previous myocardial infarction

Variable	Low nSES		Middle nSES		High nSES		IRR_{Low} (95% CI)	IRR_{Mid} (95% CI)
	N	IR	N	IR	N	IR		
All-cause death								
Heart failure								
No	61	3.39	123	2.92	46	1.68	2.01 (1.37-2.95)	1.73 (1.23-2.43)
Yes	94	7.14	169	6.21	79	5.89	1.21 (0.90-1.64)	1.06 (0.81-1.38)
Prior MI								
No	121	4.82	212	3.93	84	2.63	1.83 (1.39-2.42)	1.50 (1.16-1.93)
Yes	48	5.56	89	4.57	47	4.63	1.20 (0.81-1.79)	0.99 (0.69-1.40)
Cardiovascular death or MI								
Heart failure								
No	54	3.00	86	2.04	33	1.21	2.48 (1.61-3.83)	1.69 (1.13-2.52)
Yes	83	6.30	146	5.37	53	3.95	1.60 (1.13-2.25)	1.36 (0.99-1.86)
Prior MI								
No	97	3.86	157	2.91	44	1.38	2.80 (1.96-4.00)	2.11 (1.51-2.95)
Yes	48	5.55	86	4.41	45	4.43	1.25 (0.83-1.88)	0.99 (0.69-1.43)

IR = incidence rate, in events per 100 person-years; IRR_{Low} = incidence rate ratio of low nSES to high nSES; IRR_{Mid} = incidence rate ratio of middle nSES to high nSES; MI = myocardial infarction; N = number of events; nSES = neighborhood socioeconomic status.

shown that low nSES patients generally receive secondary preventive medications and/or procedural intervention less than high nSES patients[19]; however, patients with high-risk cardiovascular histories are more likely to follow-up with providers[20] and be prescribed secondary preventive medication.[21] It is possible that higher healthcare surveillance or treatment in patients with HF or previous MI offsets the lower rates of evidence-based care that low nSES patients typically receive. At the neighborhood level, several hypotheses are possible, and it is still unclear what nSES is a proxy for in ascribing overall cardiovascular risk. The nSES variable is comprised of only 6 variables, primarily relating to the income, wealth, occupation, and education of the census tract at large; however, several other exposures with biomechanistic ties to HF and previous MI follow along nSES gradients and may help explain the excess burden of disease in low nSES patients. Adverse neighborhood and built environment characteristics such as social isolation,[22] violent crime,[23] pollution,[24] and proximity to roadways[25] have all been associated with CVD and cluster in lower nSES areas. Additionally, food access and diet quality are tightly aligned with nSES and cardiovascular health and may be important in determining how nSES adds to the individual risk profile.[26] That these factors would more adversely affect patients without HF or previous MI is unexpected and cannot be readily explained by our data; however, the impact of social support on improved outcomes in high-risk patients with CVD may be especially important.[27] Although social support is associated with improved self-care behaviors in patients with prevalent CVD,[28] neighborhood poverty is associated with lower overall social integration.[29] Overall, low nSES

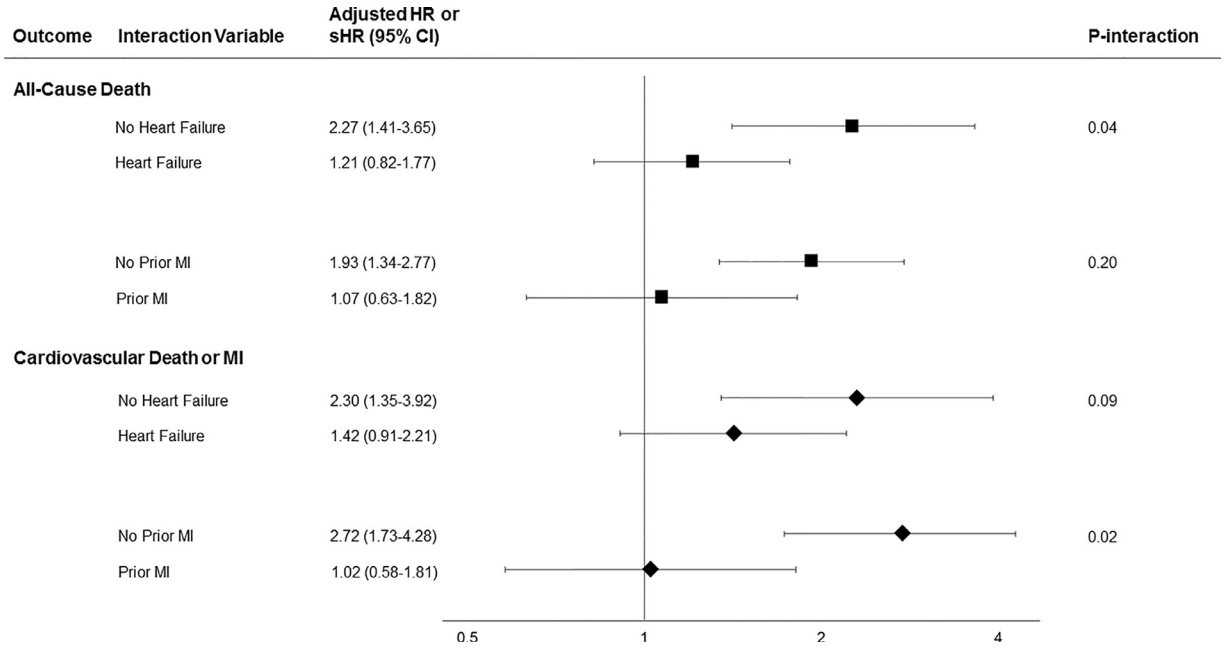

Figure 2. Interaction between low neighborhood SES and cardiovascular disease. Forest plot depicting risk of all-cause death (HR, *squares*) and cardiovascular death or MI (sHR, *diamonds*) for low neighborhood SES patients with and without heart failure or previous MI. Models are adjusted for age, gender, race, year of enrollment, education, marital status, BMI, smoking history, acute coronary syndrome on admission, history of MI, history of revascularization, history of heart failure, prevalent obstructive CAD, hypertension, diabetes, dyslipidemia, antihypertensive use, statin use, aspirin use, clopidogrel use, systolic blood pressure, LDL-C, and eGFR. BMI = body mass index; CAD = coronary artery disease; CI = confidence interval; eGFR = estimated glomerular filtration rate; HR = hazard ratio; LDL-C = low-density lipoprotein cholesterol; MI = myocardial infarction; SES = socioeconomic status; sHR = subdistribution hazard ratio.

patients without high-risk CVD, such as HF or previous MI, may have less exposure to healthcare and less robust social support—these factors would result in a unique vulnerability to adverse outcomes.

This study has several limitations that merit discussion for appropriate context. Assessment of exposures occurred at a single time point and therefore precludes any inference of causality between nSES and outcomes. Furthermore, we do not have residential mobility information on our cohort; however, previous studies have shown that even when patients move, they generally move laterally within nSES strata.[30] All participants were limited to the state of Georgia, which affects the generalizability of our findings and may mask regional influences specific to the Southeast United States regarding the associations between nSES and adverse cardiovascular outcomes. Furthermore, because not all subjects enrolled in the Emory Cardiovascular Biobank were able to be geocoded, there is potential selection bias. Patients who have survived an initial MI or diagnosis of HF are more likely to survive in the long term, and survival bias cannot be excluded, however, it is less likely given that those with high-risk CVD experienced higher crude rates of adverse events. Lastly, given the observational nature of this analysis, residual confounding at both the neighborhood and individual level are possible contributors to bias in our study.

In conclusion, our study showed that neighborhood SES was independently associated with adverse outcomes in a high-risk cohort of patients with CAD after adjustment for individual-level traditional risk factors. Furthermore, the association between low nSES on incident all-cause death and cardiovascular death or MI was greater in patients without a history of HF or MI. Identifying mechanisms to improve care delivery to high-risk, low nSES patients without prevalent CVD may help close nSES-related disparities in cardiovascular disease outcomes.

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Disclosures

The authors have no conflicts of interest to disclose.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.amjcard.2018.10.011.

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