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Neighborhood Socioeconomic Status and Adverse Outcomes in Patients with Cardiovascular Disease

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Abstract

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 prior myocardial infarction (MI) compared to those without. We followed 3,635 individuals (mean age 63.2 years, 42% with HF, 25% with prior MI) with known or suspected CAD over a median of 3.3 years for all-cause death and cardiovascular death or non-fatal MI. Individuals 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 prior MI x nSES and HF x nSES were analyzed. Compared to high nSES individuals, low nSES individuals had increased risk of all-cause death (hazard ratio [HR] = 1.61; 95% CI = 1.20, 2.15) and cardiovascular death or MI (subdistrubution hazard ratio [sHR] = 1.82; 95% CI = 1.30, 2.54). Associations were more pronounced among patients without HF or prior MI. Low nSES individuals without HF had a higher risk of all-cause death (HR = 2.27; 95% CI = 1.41, 3.65) compared to those with HF (HR = 1.21; 95% CI = 0.82, 1.77, P-interaction = 0.04). Similarly, low nSES individuals without prior MI had a higher risk of cardiovascular death or MI (sHR = 2.72; 95% CI = 1.73, 4.28) compared to those with prior MI (sHR = 1.02; 95% CI = 0.58,

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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 prior MI compared to those with HF or MI.

Keywords

Social determinants; health status disparities; coronary artery disease

Introduction

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,[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 between 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 individuals living in low nSES areas would have worse outcomes, and that the association of low nSES would be greater among individuals with heart failure or prior MI.

Methods

We studied 3,635 adults, aged 21 years and older enrolled in the Emory Cardiovascular Biobank, a prospective cohort of patients undergoing left heart catheterization for suspected or confirmed CAD in Atlanta, GA, between 2004 and 2014. Participants were interviewed to collect demographic characteristics, medical history, medication use, and behavioral habits. Individuals 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, GA).

Participants’ residential addresses were geocoded using latitude and longitude coordinates with ArcMap 10.2 (Environmental Systems Research Institute (ESRI), Inc., Redlands, California) and 2010 US Census TIGER/Line Shapefiles based on the North American Industry Classification. Nine percent of individuals were not located in the state of Georgia (n=463) and were excluded due to incomplete follow-up. Additionally, 19% of individuals were unmatched due to missing addresses, use of P.O. boxes, or were not located by the GIS-software (n=967). Compared to individuals outside of Georgia or those who were not geocoded, geocoded individuals 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 2006–2010 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 individuals per census tract was 2 [IQR 1–4], and the maximum number of individuals from any census tract was 22.

As previously described,[6, 15] a composite neighborhood socioeconomic score (nSES) was determined from six 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 three 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 individuals from neighborhoods with greatest socioeconomic advantage.

Individuals enrolled in the Emory Cardiovascular Biobank underwent a detailed baseline evaluation using standardized self-report questionnaires and medical records review. Age (years), sex (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, heart failure, prior MI, and prior revascularization (either percutaneous coronary intervention or coronary artery bypass grafting). Individuals 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 (SBP, in mm Hg) were measured by trained staff. Routine laboratory data included fasting values of low-density lipoprotein cholesterol (LDL-C) and serum creatinine (mg/dL), which was used to calculate the estimated glomerular filtration rate (eGFR, in ml/min/1.73 m^2). All participants underwent a detailed medication questionnaire to document use of the following: anti-hypertensives (angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, 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 non-fatal MI. Cardiovascular death was defined as death from MI, heart failure, 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 two 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 ± SD. 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 groups. 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 using Fine and Gray's method, treating non-cardiovascular 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 individuals 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 x prior MI and nSES x heart failure 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 prior revascularization, ACS on admission, age (<60 years vs. ≥60 years), race, sex, 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, NC) was used for all analyses.

Results

Overall, the study cohort consisted of 3,635 individuals. Low nSES individuals were younger, more likely to be female and black, and were less likely to be married or college graduates (Table 1). Additionally, the prevalence of traditional cardiovascular risk factors and heart failure was higher, while the use of statins and aspirin was lower, among lower nSES individuals (Table 1).

Over a median follow-up period of 3.3 years (interquartile range, 1.6–6.4 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 to high nSES individuals, 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 individuals but remained statistically significant for low nSES individuals (Table 2). Similar findings were found in a subset of individuals 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 heart failure or prior MI at all levels of nSES (Table 4). There was a significant interaction between low nSES and heart failure for the outcome of all-cause death; those without heart failure had a higher risk for death than those with heart failure (Figure 2). Similarly, there was a significant interaction between low nSES and prior MI for the outcome of cardiovascular death or MI; those without prior MI had a higher risk for cardiovascular death or MI than those without prior MI (Figure 2).

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

Discussion

In this study, we demonstrate that among high-risk individuals with CAD, living in census tracts with low compared to 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 heart failure or prior MI, the relative association between low nSES and adverse outcomes was greater in those without heart failure or prior 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 individuals 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] And 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 heart failure or MI is “protective” in low nSES patients. Previous studies have shown that low nSES individuals generally receive secondary preventive medications and/or procedural intervention less than high nSES individuals;[19] however, individuals 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 heart failure or prior 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 six variables, primarily relating to the income, wealth, occupation and education of the census tract at large; however, several other exposures with biomechanistic ties to heart failure and prior MI follow along nSES gradients and may help explain the excess burden of disease in low nSES individuals. 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 individuals without heart failure or prior 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 individuals without high-risk CVD, such as heart failure or prior 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 individuals 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. Individuals who have survived an initial MI or diagnosis of heart failure 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 individuals without a history of heart failure or MI. Identifying mechanisms to improve care delivery to high-risk, low nSES individuals without prevalent CVD may help close nSES-related disparities in cardiovascular disease outcomes.

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Abbreviations:

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

References

1. Daniel H, Bornstein SS, Kane GC, Health and Public Policy Committee of the American College of Physicians. Addressing Social Determinants to Improve Patient Care and Promote Health Equity: An American College of Physicians Position Paper. Ann Intern Med 2018;168:577–578. [PubMed: 29677265]
2. Diez Roux AV and Mair C. Neighborhoods and health. Ann N Y Acad Sci 2010;1186:125–145. [PubMed: 20201871]
3. Diez Roux AV, Merkin SS, Arnett D, Chambless L, Massing M, Nieto FJ, Sorlie P, Szklo M, Tyroler HA and Watson RL. Neighborhood of residence and incidence of coronary heart disease. N Engl J Med 2001;345:99–106. [PubMed: 11450679]
4. Borrell LN, Diez Roux AV, Rose K, Catellier D, Clark BL and Atherosclerosis Risk in Communities Study. Neighbourhood characteristics and mortality in the Atherosclerosis Risk in Communities Study. Int J Epidemiol 2004;33:398–407. [PubMed: 15082648]
5. Coady SA, Johnson NJ, Hakes JK and Sorlie PD. Individual education, area income, and mortality and recurrence of myocardial infarction in a Medicare cohort: the National Longitudinal Mortality Study. BMC Public Health 2014;14:705. [PubMed: 25011538]
6. Howard VJ, McClure LA, Kleindorfer DO, Cunningham SA, Thrift AG, Diez Roux AV and Howard G. Neighborhood socioeconomic index and stroke incidence in a national cohort of blacks and whites. Neurology 2016;87:2340–2347. [PubMed: 27742815]

7. Pollack CE, Slaughter ME, Griffin BA, Dubowitz T and Bird CE. Neighborhood socioeconomic status and coronary heart disease risk prediction in a nationally representative sample. Public Health 2012;126:827–835. [PubMed: 23083844]
8. Stirbu I, Looman C, Nijhof GJ, Reulings PG and Mackenbach JP. Income inequalities in case death of ischaemic heart disease in the Netherlands: a national record-linked study. J Epidemiol Community Health 2012;66:1159–1166. [PubMed: 22685304]
9. Sundquist K, Malmstrom M and Johansson SE. Neighbourhood deprivation and incidence of coronary heart disease: a multilevel study of 2.6 million women and men in Sweden. J Epidemiol Community Health 2004;58:71–77. [PubMed: 14684730]
10. Bikdeli B, Wayda B, Bao H, Ross JS, Xu X, Chaudhry SI, Spertus JA, Bernheim SM, Lindenauer PK and Krumholz HM. Place of residence and outcomes of patients with heart failure: analysis from the telemonitoring to improve heart failure outcomes trial. Circ Cardiovasc Qual Outcomes 2014;7:749–756. [PubMed: 25074375]
11. Brown AF, Liang LJ, Vassar SD, Merkin SS, Longstreth WT, Jr., Ovbiagele B, Yan T and Escarce JJ Neighborhood socioeconomic disadvantage and mortality after stroke. Neurology 2013;80:520–527. [PubMed: 23284071]
12. Gerber Y, Benyamini Y, Goldbourt U, Drory Y and Israel Study Group on First Acute Myocardial I. Neighborhood socioeconomic context and long -term survival after myocardial infarction. Circulation 2010;121:375–383. [PubMed: 20065165]
13. Agarwal S, Garg A, Parashar A, Jaber WA and Menon V. Outcomes and resource utilization in ST-elevation myocardial infarction in the United States: evidence for socioeconomic disparities. J Am Heart Assoc 2014;3:e001057. [PubMed: 25399775]
14. Rao SV, Kaul P, Newby LK, Lincoff AM, Hochman J, Harrington RA, Mark DB and Peterson ED. Poverty, process of care, and outcome in acute coronary syndromes. J Am Coll Cardiol 2003;41:1948–1954. [PubMed: 12798563]
15. Diez-Roux AV, Kiefe CI, Jacobs DR, Jr., Haan M, Jackson SA, Nieto FJ, Paton CC and Schulz R Area characteristics and individual-level socioeconomic position indicators in three population-based epidemiologic studies. Ann Epidemiol 2001;11:395–405. [PubMed: 11454499]
16. Wei LJ, Lin DY and Weissfeld L. Regression-Analysis of Multivariate Incomplete Failure Time Data by Modeling Marginal Distributions. J Am Stat Assoc 1989;84:1065–1073.
17. Osypuk TL, Ehntholt A, Moon JR, Gilsanz P and Glymour MM. Neighborhood Differences in Post-Stroke Mortality. Circ Cardiovasc Qual Outcomes 2017;10.
18. Rabi DM, Edwards AL, Svenson LW, Graham MM, Knudtson ML, Ghali WA and Alberta Provincial Project for Assessing Outcomes in Coronary Heart Disease I. Association of median household income with burden of coronary artery disease among individuals with diabetes. Circ Cardiovasc Qual Outcomes 2010;3:48–53. [PubMed: 20123671]
19. Subherwal S, Patel MR, Tang F, Smolderen KG, Jones WS, Tsai TT, Ting HH, Bhatt DL, Spertus JA and Chan PS. Socioeconomic disparities in the use of cardioprotective medications among patients with peripheral artery disease: an analysis of the American College of Cardiology's NCDR PINNACLE Registry. J Am Coll Cardiol 2013;62:51–57. [PubMed: 23643497]
20. Emdin CA, Hsiao AJ, Kiran A, Conrad N, Salimi-Khorshidi G, Woodward M, Anderson SG, Mohseni H, McMurray JJ, Cleland JG, Dargie H, Hardman S, McDonagh T and Rahimi K. Referral for Specialist Follow-up and Its Association With Post-discharge Mortality Among Patients With Systolic Heart Failure (from the National Heart Failure Audit for England and Wales). Am J Cardiol 2017;119:440–444. [PubMed: 27884420]
21. Maddox TM, Chan PS, Spertus JA, Tang F, Jones P, Ho PM, Bradley SM, Tsai TT, Bhatt DL and Peterson PN. Variations in coronary artery disease secondary prevention prescriptions among outpatient cardiology practices: insights from the NCDR (National Cardiovascular Data Registry). J Am Coll Cardiol 2014;63:539–546. [PubMed: 24184238]
22. Valtorta NK, Kanaan M, Gilbody S, Ronzi S and Hanratty B. Loneliness and social isolation as risk factors for coronary heart disease and stroke: systematic review and meta-analysis of longitudinal observational studies. Heart 2016;102:1009–1016. [PubMed: 27091846]

23. Sundquist K, Theobald H, Yang M, Li X, Johansson SE and Sundquist J. Neighborhood violent crime and unemployment increase the risk of coronary heart disease: a multilevel study in an urban setting. Soc Sci Med 2006;62:2061–2071. [PubMed: 16203075]

24. Brook RD, Rajagopalan S, Pope CA, 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, Peters A, Siscovick D, Smith SC, Jr., Whitsel L, Kaufman JD, American Heart Association Council on E, Prevention CotKiCD, Council on Nutrition PA and Metabolism. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation 2010;121:2331–2378. [PubMed: 20458016]

25. Gan WQ, Tamburic L, Davies HW, Demers PA, Koehoorn M and Brauer M. Changes in residential proximity to road traffic and the risk of death from coronary heart disease. Epidemiology 2010;21:642–649 [PubMed: 20585255]

26. Li S, Chiuve SE, Flint A, Pai JK, Forman JP, Hu FB, Willett WC, Mukamal KJ and Rimm EB. Better diet quality and decreased mortality among myocardial infarction survivors. JAMA Intern Med 2013;173:1808–1818. [PubMed: 23999993]

27. Havranek EP, Mujahid MS, Barr DA, Blair IV, Cohen MS, Cruz-Flores S, Davey-Smith G, Dennison-Himmelfarb CR, Lauer MS, Lockwood DW, Rosal M, Yancy CW, American Heart Association Council on Quality of C, Outcomes Research CoE, Prevention CoC, Stroke Nursing CoL, Cardiometabolic H and Stroke C. Social Determinants of Risk and Outcomes for Cardiovascular Disease: A Scientific Statement From the American Heart Association. Circulation 2015;132:873–898. [PubMed: 26240271]

28. Gallagher R, Luttik ML and Jaarsma T. Social support and self-care in heart failure. J Cardiovasc Nurs 2011;26:439–445. [PubMed: 21372734]

29. Marcus AF, Echeverria SE, Holland BK, Abraido-Lanza AF and Passannante MR. How Neighborhood Poverty Structures Types and Levels of Social Integration. Am J Community Psychol 2015;56:134–144. [PubMed: 26076667]

30. Sampson RJ and Sharkey P. Neighborhood selection and the social reproduction of concentrated racial inequality. Demography 2008;45:1–29. [PubMed: 18390289]

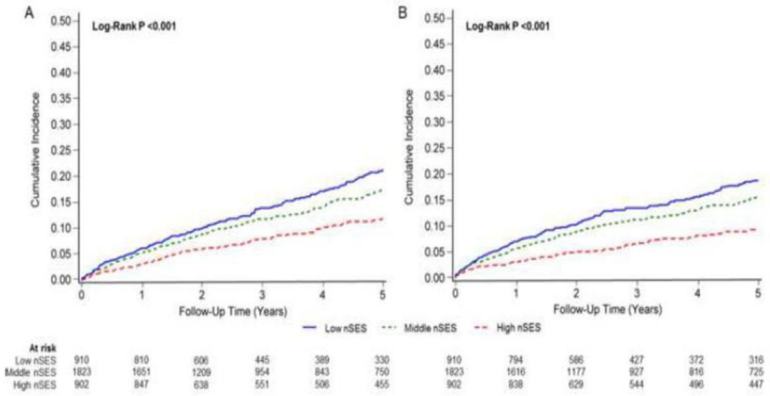

Figure 1. Cumulative incidence plots for adverse events.

Unadjusted cumulative incidence curves for (A) all-cause mortality and (B) cardiovascular death or non-fatal myocardial infarction across categories of neighborhood socioeconomic status (nSES). The lowest quartile of nSES (Q1) is denoted by the solid blue line.

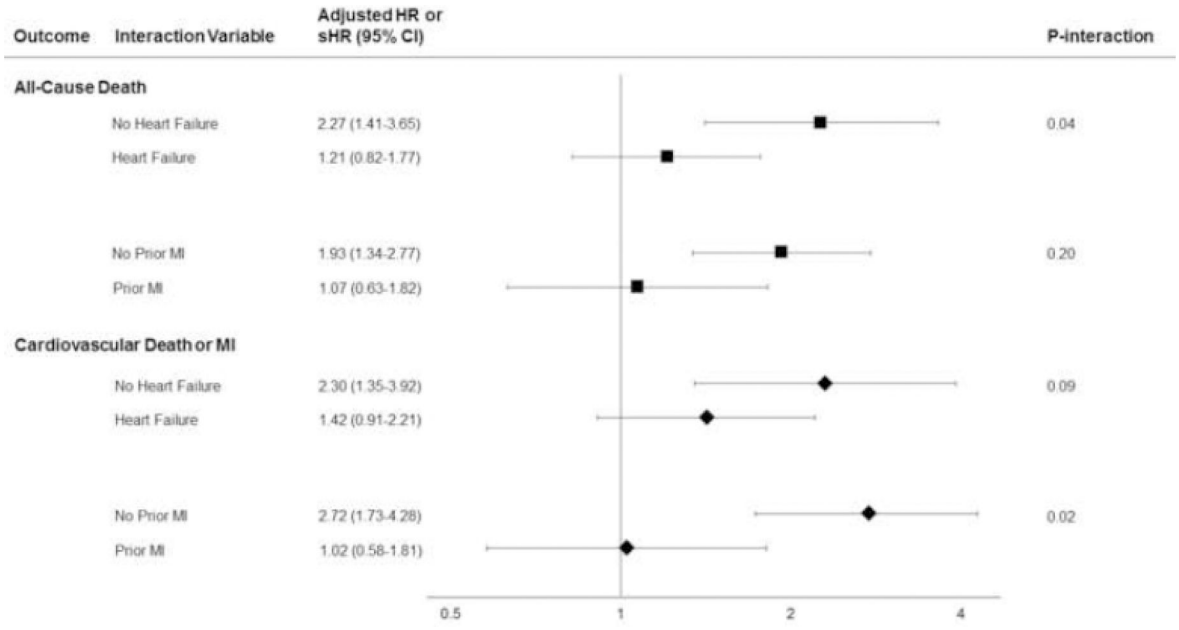

Figure 2. Interaction between low neighborhood SES and cardiovascular disease.

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 prior MI. Models are adjusted for age, sex, 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.

Table 1:

Baseline characteristics of the cohort

		Categories of Neighborhood Socioeconomic Status			
Variable	**Overall (n = 3,635)**	**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

		Categories of Neighborhood Socioeconomic Status			
Variable	**Overall (n = 3,635)**	**Low (n = 910)**	**Middle (n=1,823)**	**High (n=902)**	**p value**
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

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

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

*Model adjusted for age, sex, race, 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, prior MI, prior 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

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

*Model adjusted for age, sex, race, 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, prior MI, prior 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 prior 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)

nSES = neighborhood socioeconomic status; N = number of events; 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