Neighborhood Disadvantage, Poor Social Conditions, and Cardiovascular Disease Incidence Among African American Adults in the Jackson Heart Study

Sharrelle Barber, ScD, MPH, DeMarc A. Hickson, PhD, MPH, Xu Wang, MS, Mario Sims, PhD, Cheryl Nelson, MSPH, and Ana V. Diez-Roux, MD, PhD

Objectives. To examine the impact of neighborhood conditions resulting from racial residential segregation on cardiovascular disease (CVD) risk in a socioeconomically diverse African American sample.

Methods. The study included 4096 African American women (n = 2652) and men (n = 1444) aged 21 to 93 years from the Jackson Heart Study (Jackson, Mississippi; 2000–2011). We assessed neighborhood disadvantage with a composite measure of 8 indicators from the 2000 US Census. We assessed neighborhood-level social conditions, including social cohesion, violence, and disorder, with self-reported, validated scales.

Results. Among African American women, each standard deviation increase in neighborhood disadvantage was associated with a 25% increased risk of CVD after covariate adjustment (hazard ratio = 1.25; 95% confidence interval = 1.05, 1.49). Risk also increased as levels of neighborhood violence and physical disorder increased after covariate adjustment. We observed no statistically significant associations among African American men in adjusted models.

Conclusions. Worse neighborhood economic and social conditions may contribute to increased risk of CVD among African American women. Policies directly addressing these issues may alleviate the burden of CVD in this group. (*Am J Public Health.* 2016; 106:2219–2226. doi:10.2105/AJPH.2016.303471)

See also Galea and Vaughan, p. 2091.

Cardiovascular disease (CVD) remains the leading cause of mortality among most racial groups in the United States, accounting for 1 in 4 deaths annually.[1] A substantial amount of research has examined the role neighborhood environments play in influencing CVD risk factors and subsequent disease onset[2,3] with strong empirical evidence linking socioeconomically disadvantaged residential environments to greater disease risk[3] and higher rates of CVD morbidity and mortality.[2,4–6] More recently, attention has focused on specific built and social environment characteristics that may link these settings to higher disease risk. For example, limited access to affordable, healthy foods and deficiencies in the built environment have been found to be associated with behavioral risk factors such as diet and physical activity.[7,8] Moreover, neighborhoods with low levels of social cohesion and high rates of crime, violence, and disorder have been found to be particularly detrimental to health as they create environments that induce stress,[9] elevate blood pressure,[10] and may not be conducive to healthy behaviors such as physical activity.[11] Combined, these behavioral, psychosocial, and biological risk factors have the potential to lead to subsequent CVD onset.[5]

Structural factors such as residential segregation by both race and class have contributed to the disproportionate exposure of African Americans to socioeconomically deprived residential settings warranting investigations into how these contexts shape disease risk among this group.[12] Although a number of studies have examined how neighborhood economic[2,4,6] and, to a lesser extent, social conditions[13,14] influence CVD risk and onset longitudinally, prospective analyses among African Americans remain sparse and findings have been mixed.[2,4] Studies that have examined associations among African Americans have often included relatively small sample sizes with few incident CVD cases limiting statistical power to detect associations in this racial group.[4] In addition, existing studies have not included African American samples that are heterogeneous with regard to socioeconomic status, limiting the ability to investigate these factors. Moreover, investigations in urban residential areas in the Deep South, which often have high proportions of African Americans and residents that have been historically exposed to the confluence of discrimination and poverty, have been scant.

ABOUT THE AUTHORS

Sharrelle Barber, Xu Wang, and Ana V. Diez-Roux are with Drexel University School of Public Health, Department of Epidemiology and Biostatistics, and Center for Integrative Approaches to Health Disparities, Philadelphia, PA. DeMarc A. Hickson and Mario Sims are with University of Mississippi Medical Center, Jackson. Cheryl Nelson is with National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, MD.

Correspondence should be sent to Sharrelle Barber, ScD, MPH, Drexel University School of Public Health, Department of Epidemiology and Biostatistics, Nesbit Hall, 5th Floor, 3215 Market St, Philadelphia, PA 19104 (e-mail: smb483@drexel.edu). Reprints can be ordered at http://www.ajph.org by clicking the "Reprints" link.

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We used data from the Jackson Heart Study (JHS)—the largest single-site cohort study of CVD among African Americans conducted in the United States—to examine the prospective association between features of the neighborhood economic and social environment and CVD incidence. Specifically, we examined the associations of neighborhood disadvantage and neighborhood social cohesion, violence, and physical disorder with CVD events. We hypothesized that higher levels of neighborhood disadvantage, violence, and disorder and lower levels of social cohesion would be associated with an increased risk of CVD.

METHODS

The JHS is a large cohort study designed to examine the etiology of CVD among African Americans.[15] The study population included adults aged 21 to 93 years residing in the Jackson, Mississippi, metropolitan area (i.e., Hinds, Rankin, and Madison counties). Trained study staff recruited a total of 5301 participants into the JHS at baseline,[15] most of whom (n = 5297) were retrospectively geocoded to census tracts in the Jackson metropolitan area by using information abstracted from the residential mailing address provided during the baseline home interview.[16] The sample has been shown to be approximately representative of the African American population in the geographic area of interest.[17]

Data collection methods have been described elsewhere.[15] Briefly, baseline data collection took place between September 2000 and March 2004 and involved a home interview and an on-site examination in which study staff obtained extensive clinical, demographic, social, cultural, and behavioral data. Two follow-up clinical visits took place between 2005–2008 and 2009–2012, and study staff contacted participants each year via telephone for an annual follow-up survey. All participants provided informed consent.

The analytic sample included all participants with geocoded information who resided in the Jackson metropolitan area and were free of CVD at baseline (n = 4698). We excluded individuals with missing information on neighborhood variables and key biomedical and behavioral covariates

(n = 602). To retain sample size and statistical power, we included an indicator variable denoting missing information for family income and educational attainment (n = 840). The resulting analytic sample was composed of 4096 participants residing in 111 of the 115 census tracts in the Jackson metropolitan area.

Measurement

Neighborhood disadvantage. Development of the neighborhood disadvantage score for the JHS has been described elsewhere.[18] Briefly, we used exploratory factor analysis based on census tracts in the Jackson metropolitan area to develop a composite score of sociodemographic indicators from the 2000 US Census (see the box on the next page). We developed the final score by summing the standardized z-scores for each indicator with higher scores denoting higher levels of disadvantage.

Neighborhood social environment. We considered 3 dimensions of the neighborhood social environment: social cohesion, violence, and physical disorder. These dimensions capture related, yet distinct, neighborhood-level social processes that tend to arise as neighborhood socioeconomic conditions decline and have been found to be associated with CVD risk factors[10,19,20] and disease onset.[13,14] We obtained information on the neighborhood social environment by using validated scales[21] during the third annual follow-up telephone survey with participants (see the box on the next page). Response options for each were on a 4-point Likert scale.

Consistent with previous work,[10] we used 3-level hierarchical models to derive empirical Bayes estimates (conditional on age and gender) for each census tract of residence at baseline by pooling responses of all respondents within a tract. This approach creates a more valid and reliable measure by aggregating information from multiple informants and by borrowing information across tracts to improve estimates for tracts with few observations. It also allows estimates to be adjusted for age and gender, thereby eliminating any systematic differences across tracts because of the age and gender of the respondents. Lower scores for social cohesion represented less cohesion and higher scores for neighborhood violence and disorder represented more violence and more physical

disorder. All neighborhood variables had satisfactory reliability (social cohesion, 0.72; violence, 0.75; disorder, 0.68) and intra–class correlations consistent with the literature (social cohesion, 0.09; violence, 0.12; disorder, 0.20).[21]

Cardiovascular Disease Events and Covariates

The primary outcome was incident CVD events, which included incident coronary heart disease (CHD) and incident stroke events. We defined a CHD event as a definite or probable myocardial infarction (MI), a definite fatal CHD, or cardiac procedure. We defined a stroke event as a definite or probable stroke on the basis of neuroimaging studies or autopsy according to criteria adapted from the National Survey of Stroke.[22] We followed participants with geocoded information free of CVD at baseline (n = 4968) from the time of their baseline examination in 2000 to 2004 to the date of their first CVD event, death, and loss to follow-up, or otherwise through December 31, 2011. Ascertainment of CVD events in the JHS has been described elsewhere.[23]

We controlled for age (in years), gender, education (≤ high school, some college, and ≥ college), and family income in the analysis. Family income categories (low, lower-middle, upper-middle, and high) accounted for family size and poverty level.

We also considered health behaviors and traditional biomedical risk factors in the analysis, as they may confound or partially mediate the association between neighborhood characteristics and CVD events. We assessed physical activity with the Active Living Index, a summary score of the frequency and duration of leisure-time physical activities (range: 1 = low to 5 = high).[24] We assessed consumption of dietary fat by using items from a 158-item validated self-report food-frequency questionnaire (FFQ) adapted for the study population[25] and calculated it as the percentage of calories from fat consumed per day. We assessed cigarette smoking status via self-report (current, former, or never) and we estimated alcohol consumption from the frequency and portion sizes of beer, wine, and liquor reported in the FFQ and included as a continuous variable (grams per day). We defined obesity (yes or

NEIGHBORHOOD VARIABLES USED TO ASSESS NEIGHBORHOOD DISADVANTAGE AND THE SOCIAL ENVIRONMENT IN THE JACKSON, MS, METROPOLITAN AREA

Neighborhood disadvantage indicators
    % individuals living below federal poverty threshold
    % households receiving public assistance
    % occupied housing units with no vehicle
    % adults aged 25 years and older with less than a high-school education
    % unemployed individuals aged 16 years and older in the civilian labor force
    % unoccupied housing units
    % occupied housing units with more than 1 person per room (crowding)
    % female-headed households

Social cohesion scale items
    This is a close-knit neighborhood.
    People around here are willing to help their neighbors.
    People in this neighborhood generally don't get along.
    People in this neighborhood can be trusted.
    People in this neighborhood don't share the same values.

Neighborhood violence scale items
    How often was there a fight in this neighborhood in which a weapon was used?
    How often was there a violent argument between neighbors?
    How often were there gang fights?
    How often was there a sexual assault or rape?
    How often was there a robbery or mugging?

Neighborhood disorder scale items
    Excessive noise
    Heavy traffic or speeding cars
    Trash and litter

Note. Neighborhood disadvantage indicators were derived from the 2000 US Census. Social environment scale items were administered in a follow-up phone interview approximately 3 years after the baseline examination. Response options for social cohesion were on a 4-point Likert scale ranging from strongly disagree (1) to strongly agree (4). Items 2 and 5 were reverse-coded. Response options for neighborhood violence were on a 4-point Likert scale ranging from never (1) to often (4). Response options for neighborhood disorder were on a 4-point Likert scale ranging from not really a problem (1) to very serious problem (4).

no) as having a body mass index (BMI; defined as weight in kilograms divided by the square of height in meters) greater than or equal to 30. We based type 2 diabetes (yes or no) on the American Diabetes Association 2010 criteria or use of antidiabetic medication.[26] We defined hypertension (yes or no) in accordance with the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure criteria as blood pressure greater than 140/90 millimeters of mercury or use of antihypertensive medication.[27] Finally, JHS study staff collected fasting high–density and low–density lipoprotein cholesterol and triglycerides via standard procedures during the baseline clinic examination.

Statistical Analysis

We examined the distribution of sociodemographic characteristics and CVD risk factors according to CVD status and tertiles of neighborhood disadvantage. We used Poisson regression to calculate gender-specific, age-adjusted incidence rates based on tertiles of each neighborhood characteristic and tested for trend by including neighborhood factors in models as ordinal variables.

To examine the association between neighborhood characteristics and CVD incidence, we fit Cox proportional hazards regression models to estimate adjusted hazard ratios (HRs) and 95% confidence intervals (CIs). On the basis of the presence of approximately linear relationships observed in descriptive analyses, we included neighborhood characteristics as continuous standardized scores. We reverse-scored standardized scores for social cohesion (i.e., multiplied by −1) so interpretation of the HRs would be consistent with the other neighborhood variables (i.e., correspond to worse social conditions). We fit 3 sequential models separately for each neighborhood variable. Model 1 adjusted for age; model 2 further adjusted for socioeconomic status; and model 3 further adjusted for behavioral and biomedical risk factors. We also examined associations separately for incident CHD and stroke events with the same modeling procedure.

In sensitivity analysis, we further adjusted for self-reported perceptions of the neighborhood built environment (e.g., access to adequate food or shopping, lack of playgrounds or parks, poorly maintained sidewalks) in models for the social environment as these factors may confound the association between the social environment and CVD risk. In addition, because neighborhood disadvantage and features of the social environment tend to be highly correlated— a phenomenon confirmed in our sample (social cohesion, r = −0.59; violence, r = 0.83; disorder, r = 0.87)—we sought to disentangle the effects by examining HRs for cross-classified categories of neighborhood disadvantage and each social environment variable in gender-specific models. We created categories (high vs low) for each neighborhood variable by dichotomizing the scores at the median.

We used the PROC PHREG procedure in SAS to fit all models,[28] and we adjusted for clustering within neighborhoods by using a robust sandwich covariance matrix estimate.[29] Given previous evidence that the social patterning of CVD risk may differ by age and gender in African Americans,[23,30] we tested for effect modification by these variables. We observed statistically significant interactions between gender and neighborhood disorder (P = .05); therefore, gender-specific models are presented for all neighborhood variables for consistency. We found no interactions for age. We carried out all analyses with SAS software, version 9.3 (SAS Institute Inc, Cary, NC).

	Total CVD Events[a]			Neighborhood Disadvantage[b]			
Characteristic	No	Yes	P	High	Medium	Low	P
No. of participants	3864	232		1352	1432	1312	
Age, y, mean	53.4	64.2	< .001	58.0	53.9	50.1	< .001
Gender, %			.38				< .001
Female	64.9	62.1		68.5	65.1	60.5	
Male	35.1	37.9		31.5	34.9	39.5	
Education, %			< .001				< .001
≤ high school or GED	34.6	50.9		50.0	35.8	20.4	
Some college	30.2	22.8		26.2	31.6	31.6	
≥ college	35.1	24.5		23.5	32.5	48.0	
Missing	0.1	1.7		0.4	0.1	0.1	
Family income level,[c] %			< .001				< .001
Low	11.7	16.8		18.1	11.3	6.3	
Lower-middle	19.5	28.0		28.3	21.0	10.4	
Upper-middle	26.3	24.6		24.7	26.3	27.6	
High	27.8	19.8		15.2	26.4	40.9	
Missing	14.7	10.8		13.7	15.0	14.8	
Smoking status, %			< .001				< .001
Current	11.7	18.0		14.0	13.6	8.4	
Former	16.9	25.4		20.4	18.0	13.7	
Never	71.4	56.5		65.6	68.5	77.9	
Alcohol consumption, grams, mean (SD)	4.4 (18.2)	2.8 (8.0)	.11	4.0 (16.4)	5.1 (12.4)	4.0 (10.6)	.06
Active living score,[d] mean (SD)	2.1 (0.8)	2.0 (0.8)	.005	1.9 (0.8)	2.1 (0.8)	2.2 (0.8)	< .001
% of calories from fat, mean (SD)	35.2 (6.9)	34.2 (7.0)	.005	34.8 (6.8)	34.9 (6.9)	35.9 (6.9)	< .001
Obesity status,[e] %	53.0	50.0	.38	55.4	52.5	50.4	.034
HDL[f] cholesterol, mean (SD)	52.1 (14.5)	52.2 (15.4)	.048	53.0 (15.0)	52.2 (14.8)	51.1 (13.6)	< .001
LDL[f] cholesterol, mean (SD)	126.8 (36.2)	130.3 (38.7)	.75	126.4 (36.0)	126.9 (35.8)	127.7 (37.3)	.78
Triglycerides, mean (SD)	100.2 (53.9)	114.1 (61.0)	< .001	101.7 (53.6)	103.5 (57.5)	97.5 (51.6)	.014
Diabetes status,[g] %	15.8	35.3	< .001	22.8	15.5	12.5	< .001
Hypertension status,[h] %	54.1	82.8	< .001	63.6	55.6	47.7	< .001
Neighborhood disadvantage, mean (SD)	2.49 (6.4)	5.22 (5.8)	< .001	9.7 (3.0)	3.1 (2.2)	−5.0 (2.2)	.014
Social cohesion,[i] mean (SD)	3.01 (0.1)	3.00 (0.1)	.09	2.9 (0.1)	3.0 (0.1)	3.1 (0.1)	< .001
Violence,[i] mean (SD)	1.26 (0.1)	1.29 (0.1)	< .001	1.4 (0.1)	1.3 (0.1)	1.1 (0.1)	< .001
Disorder,[i] mean (SD)	1.67 (0.2)	1.75 (0.2)	< .001	1.9 (0.1)	1.7 (0.1)	1.4 (0.1)	< .001

Note. CHD = coronary heart disease; CVD = cardiovascular disease; GED = general equivalency diploma; HDL = high-density lipoprotein; LDL = low-density lipoprotein. *P* values determined by χ^2 test for categorical variables and Kruskal-Wallis test for continuous variables.

[a]CVD events included definite or probable fatal or nonfatal CHD and stroke events.

[b]The neighborhood disadvantage score was constructed by summing *z*-scores of sociodemographic indicators from the 2000 US Census.

[c]Family income levels were based on the midpoint of self-reported family income categories and adjusted for family size and poverty.

[d]The active living score is a summary score of the frequency and duration of leisure-time physical activity. Higher scores represent higher physical activity.

[e]Obesity was defined as body mass index ≥ 30 kg/m².

[f]Fasting HDL and LDL cholesterol units were mg/dL.

[g]Diabetes status was based on the American Diabetes Association 2010 criteria or use of antidiabetic medication.

[h]Hypertension status was defined as blood pressure > 140/90 mm Hg or use of antihypertensive medication.

[i]Neighborhood social cohesion, violence, and disorder were assessed via validated scales measured during a follow-up phone interview.

RESULTS

A total of 232 CVD events occurred during a median follow-up time of 8.38 years. Individuals who developed CVD were older, had less family income and education, and generally had worse risk factor profiles (Table 1). They also resided in more disadvantaged neighborhood settings and in neighborhoods with slightly higher levels of violence and disorder (Table 1). Age-adjusted CVD incidence rates were slightly lower than recently reported estimates for African Americans[31] and generally increased as neighborhood conditions worsened for both women and men (Table 2).

Hazard ratios for women revealed a 25% increased risk of CVD for each standard deviation increase in neighborhood disadvantage (HR = 1.25; 95% CI = 1.05, 1.49) after adjustment for sociodemographic characteristics (Table 3). Risk was attenuated with the addition of traditional CVD risk factors, but only slightly, and the association remained statistically significant (HR = 1.23; 95% CI = 1.04, 1.45). Hazard ratios for men showed a similar pattern, but associations were weaker and were not statistically significant (HR = 1.08; 95% CI = 0.82, 1.41; model 3: HR = 1.03; 95% CI = 0.79, 1.36; Table 3).

For the neighborhood social environment (Table 3), each standard deviation increase in neighborhood violence and disorder was associated with an increased risk of CVD among women after adjustment for sociodemographic characteristics (HR = 1.13; 95% CI = 1.00, 1.28 and HR = 1.20; 95% CI = 1.02, 1.40, respectively). Findings for social cohesion were not statistically significant. For men, living in neighborhoods with higher levels of violence and disorder and lower levels of social cohesion was associated with a decreased risk of CVD; however, these associations were relatively weak and not statistically significant. Sensitivity analysis further adjusting for the resource environment did not substantially modify our results.

In separate analyses for stroke and CHD (available as a supplement to the online version of this article at http://www.ajph.org), patterns among women were similar for neighborhood disadvantage and the neighborhood social environment, although associations appeared stronger for stroke (neighborhood disadvantage: HR = 1.23; 95% CI = 1.00, 1.50; social cohesion: HR = 1.17; 95% CI = 1.00, 1.37; violence: HR = 1.21; 95% CI = 1.03, 1.41; disorder: HR = 1.31; 95% CI = 1.08, 1.60) than for CHD (neighborhood disadvantage: HR = 1.15; 95% CI = 0.94, 1.41; social cohesion: HR = 0.96; 95% CI = 0.81, 1.15; violence: HR = 1.02; 95% CI = 0.85, 1.21; disorder: HR = 1.04; 95% CI = 0.86, 1.26).

Finally, in sensitivity analysis examining cross-classified categories of neighborhood disadvantage and the social environment, adjusted HRs for women living in the worst neighborhood environments (i.e., high disadvantage and poor social conditions) were consistent with an increased risk of CVD compared with neighborhoods with the best economic and social conditions (social cohesion: HR = 1.37; 95% CI = 0.90, 2.09; disorder: HR = 1.49; 95% CI = 1.00, 2.21; violence: HR = 1.49; 95% CI = 1.01, 2.19).

TABLE 2—Age-Adjusted Cardiovascular Disease Incidence Rates According to Levels of Neighborhood Characteristics for Women and Men: Jackson Heart Study; Jackson, MS; 2000–2011

Variable	Women (n = 2652)			Men (n = 1444)		
	No. of Events	Incidence/1000 PY (95% CI)	P_{trend}	No. of Events	Incidence/1000 PY (95% CI)	P_{trend}
Overall	144	4.4 (3.6, 5.4)		88	5.6 (4.4, 7.1)	
Neighborhood disadvantage			.006			.27
High	78	5.5 (4.1, 7.4)		38	7.1 (4.9, 10.2)	
Medium	47	4.0 (3.1, 5.1)		28	6.0 (4.7, 7.8)	
Low	19	2.9 (2.0, 4.2)		22	5.2 (3.5, 7.6)	
Social cohesion			.20			.96
Low	55	4.7 (3.5, 6.4)		30	6.1 (4.2, 8.8)	
Medium	57	4.1 (3.3, 5.2)		25	6.0 (4.7, 7.8)	
High	32	3.6 (2.6, 5.0)		33	6.0 (4.2, 8.5)	
Violence			.026			.35
High	59	5.2 (3.9, 7.0)		32	6.9 (4.7, 9.9)	
Medium	67	4.1 (3.2, 5.2)		33	6.1 (4.7, 7.8)	
Low	18	3.2 (2.2, 4.5)		23	5.3 (3.7, 7.7)	
Disorder			.002			.75
High	76	5.7 (4.2, 7.5)		32	6.3 (4.3, 9.1)	
Medium	52	3.9 (3.1, 5.0)		33	6.0 (4.7, 7.8)	
Low	16	2.7 (1.9, 4.0)		23	5.8 (4.0, 8.3)	

Note. CI = confidence interval; PY = person-years. Poisson regression was used to estimate gender-specific, age-adjusted incidence rates (sum of events/PY) overall and according to levels of neighborhood characteristics. Cardiovascular disease events included definite or probable fatal or nonfatal coronary heart disease and stroke events. PY were calculated as time to first cardiovascular disease event. The neighborhood disadvantage score was constructed by summing z-scores for eight 2000 US Census indicators. Neighborhood social cohesion, violence, and disorder were assessed via validated scales during a follow-up phone interview approximately 3 years after the baseline examination.

TABLE 3—Adjusted Hazard Ratios for Cardiovascular Disease Incidence Corresponding to a 1-Standard Deviation Increase in Neighborhood Disadvantage, Social Cohesion, Neighborhood Violence, and Neighborhood Disorder: Jackson Heart Study; Jackson, MS; 2000–2011

Variable	Women (n = 2652)			Men (n = 1444)		
	Model 1, HR (95% CI)	Model 2, HR (95% CI)	Model 3, HR (95% CI)	Model 1, HR (95% CI)	Model 2, HR (95% CI)	Model 3, HR (95% CI)
Disadvantage	1.33 (1.12, 1.58)	1.25 (1.05, 1.49)	1.23 (1.04, 1.45)	1.10 (0.86, 1.40)	1.08 (0.82, 1.41)	1.03 (0.79, 1.36)
Social cohesion	1.11 (0.97, 1.26)	1.04 (0.90, 1.20)	1.04 (0.91, 1.19)	0.92 (0.76, 1.12)	0.88 (0.70, 1.10)	0.87 (0.69, 1.09)
Violence	1.18 (1.04, 1.34)	1.13 (1.00, 1.28)	1.14 (1.01, 1.29)	1.02 (0.82, 1.26)	0.98 (0.77, 1.25)	0.99 (0.77, 1.27)
Disorder	1.28 (1.10, 1.49)	1.20 (1.02, 1.40)	1.18 (1.01, 1.40)	0.99 (0.80, 1.23)	0.95 (0.74, 1.21)	0.93 (0.72, 1.20)

Note. CI = confidence interval; HR = hazard ratio. Model 1 adjusts for age; model 2 further adjusts for baseline family income and educational attainment; model 3 further adjusts for baseline cardiovascular risk factors (physical activity, dietary fat consumption, smoking status, alcohol consumption, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, body mass index, hypertension status, and diabetes status).

In addition, women living in neighborhoods with low levels of disadvantage but poor social conditions (i.e., high levels of violence and disorder and low levels of social cohesion) were at increased risk for CVD (social cohesion: HR = 1.38; 95% CI = 0.80, 2.38; disorder: HR = 1.49; 95% CI = 0.90, 2.47; violence: HR = 1.40; 95% CI = 0.83, 2.36). These estimates, however, were not statistically significant likely because of small sample sizes within discordant categories.

DISCUSSION

In the largest prospective study of neighborhood conditions and CVD incidence among African American adults, we found that women living in neighborhoods characterized by higher levels of disadvantage, violence, and disorder were at greater risk for CVD, independent of individual-level sociodemographic characteristics. Adjustment for traditional biological and behavioral risk factors attenuated associations, but they remained statistically significant, and associations with violence and disorder were robust to adjustment for self-reported measures of the built environment. We found no evidence that lower levels of social cohesion increased risk of CVD onset among women. Among men, estimates for neighborhood economic and social conditions were often weaker, and in some cases in the opposite direction compared with findings for women, but were not statistically significant. We also observed stronger associations for stroke compared with CHD in our sample, which, to our knowledge, has not been reported in other studies. However, because of limited statistical

power, this finding should be interpreted with caution, and future studies with more incident cases and longer follow-up time should examine this more closely.

Our findings among African American women are consistent with past prospective studies that examined neighborhood socioeconomic conditions and CVD onset,[2,6] although 1 longitudinal study of older adults based on data from the Cardiovascular Health Study found no association between neighborhood socioeconomic status and incident ischemic stroke for African Americans.[4] This null finding may have been because of the modest sample size (n = 785) and selection issues related to the age of the cohort.[4] Relatively few longitudinal studies have examined the extent to which social factors influence CVD incidence. In a large prospective population-based study in Sweden,[13] investigators found that residents residing in neighborhoods with higher rates of violent crime had higher rates of incident CHD among both women and men (women: 1.75; 95% CI = 1.37, 2.22; men: 1.39; 95% CI = 1.19, 1.63) similar to the findings we observed for overall CVD and for stroke. Our findings are also consistent with cross-sectional and longitudinal studies that have examined CVD risk factors such as hypertension[10] and diabetes.[32]

Our findings are consistent with a number of potential pathways linking neighborhood economic and social conditions to CVD risk. Neighborhoods characterized by socioeconomic disadvantage often lack material resources that are conducive to cardiovascular health such as access to affordable healthy foods[8] and access to outlets to engage in physical activity.[7] In addition, poor social

conditions such as violence and physical forms of disorder may be viewed as neighborhood-level stressors that may lead to unhealthy coping behaviors such as smoking[33] and higher alcohol use.[19] These stressors may also activate the body's natural "fight or flight" mechanisms resulting in increased heart rate and blood pressure as well as release of glucose into the bloodstream. Chronic exposure to these stressors may cause dysregulation of these physiological responses and subsequent disease onset.[34] In addition to these stress-mediated pathways, social conditions may also directly influence health behaviors related to CVD. There is evidence that suggests that individuals living in neighborhoods with higher rates of crime may feel unsafe and be less likely to engage in healthy behaviors such as physical activity.[11] We found some evidence that these traditional risk factors and health behaviors explain a portion of the association as estimates were attenuated when these factors were included in our models. However, attenuation was modest and future studies should explore these and additional pathways (e.g., inflammatory, psychosocial) with more rigorous mediation methods.

Findings among African American men have often been mixed with some studies reporting null findings or findings that suggest that African American men living in neighborhoods with the worst economic and social conditions are at lower risk for CVD.[2] These contradictory findings may be attributable to the smaller sample size among African American men, which reduces the statistical power to detect associations or selection issues. For example, in neighborhoods with the worst social conditions, overall

mortality rates are generally higher because of a number of "competing risks." As a consequence, African American men may experience other causes of death before developing CVD. Although we found some limited evidence of this in separate analyses examining neighborhood conditions and non–CVD-related mortality (data not shown), the relationship was similar for women and thus may not explain the gender differences we observed in our study. Relatedly, African American men who are selected from these settings may represent a "healthier" subset of men, which may also influence observed associations. It is difficult to determine whether this is the case in our study, and future studies should examine this more closely.

Limitations and Strengths

Our study is not without limitations. First, our measures of the social environment were derived from self-reported scales. Although these scales had satisfactory reliability in our population, some measurement error or misclassification is possible and more objective measures of the neighborhood social environment (e.g., crime data or systematic social observations) may provide a more accurate assessment of these constructs. We also had single, time-invariant measures of the neighborhood environment, which may not accurately reflect exposures relevant to the development of CVD. Although this is the case, approximately 80% remained in the same neighborhood during the study period. Also, although we adjusted for an extensive set of potential confounders in our analyses, we cannot rule out residual confounding. Finally, our sample was restricted to African American adults in a southern metropolitan area and may not be generalizable to African Americans in other settings.

Despite these limitations, our study has several strengths. Our study is the largest longitudinal study to examine the association between neighborhood economic and social conditions and incidence of CVD among African American adults, providing stronger causal evidence for a link between neighborhood conditions and CVD onset in this population. Also, relatively few studies have examined neighborhood social conditions in relation to incident CVD and we provide

compelling evidence for an association. This includes suggestive, albeit limited, evidence from our cross-classified analyses that even when neighborhood disadvantage is relatively low, poor social conditions may have an effect over and above their association with neighborhood disadvantage. Finally, our study examines these factors within the context of a Southern metropolitan area, a region of the country that has been underexplored in this area of research.

Conclusions

Poor neighborhood social conditions such as violence and disorder do not occur in a vacuum and are a byproduct of the larger context of racial and economic stratification by place in the United States.[12] Neighborhoods characterized by high rates of poverty and unemployment also experience higher rates of crime and physical decay, a phenomenon fueled by decades of disinvestment in urban residential areas across the country.[35] Furthermore, racial residential segregation places African Americans at increased risk for exposure to these health-damaging neighborhood environments, implicating these contexts as important shapers of disease risk in this group and as major drivers of observed racial inequities in CVD outcomes.[12]

Our findings also augment the existing literature on neighborhood contextual factors and CVD risk, which has tended to focus on physical features of the neighborhood environment (e.g., access to healthy foods). Although access to health-promoting resources may be important, it is only 1 part of the equation and prevention efforts that only focus on these features may be undermined without careful consideration of the broader economic and social conditions that also contribute to CVD risk. Thus, neighborhood-level policy efforts directly addressing economic and social conditions should be considered as viable strategies to reduce the burden of CVD among African Americans and ultimately mitigate observed racial inequities in this group. *AJPH*

CONTRIBUTORS
S. Barber, D. A. Hickson, and A. V. Diez-Roux originated the study concept and study design. S. Barber and X. Wang acquired the data and were responsible for data analysis. All authors were involved in data interpretation, article development, and providing substantive feedback on all drafts of the article.

HUMAN PARTICIPANT PROTECTION
All Jackson Heart Study participants provided informed consent and the study procedures were approved by the institutional review boards of Jackson State University, Tougaloo College, and the University of Mississippi Medical Center.

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