Neighborhood Psychosocial Hazards and Cardiovascular Disease: The Baltimore Memory Study

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Objectives. We examined associations between cardiovascular disease and neighborhood psychosocial hazards, such as violent crime, abandoned buildings, and signs of incivility, to evaluate whether features of place are associated with older adult health.

Methods. We analyzed first-visit data from the Baltimore Memory Study of randomly selected residents aged 50 to 70 years (n = 1140) of 65 contiguous neighborhoods in Baltimore, Maryland. We looked for associations between self-reports of history of selected cardiovascular diseases and scores on the 12-item neighborhood psychosocial hazards scale.

Results. After adjustment for established individual risk factors for cardiovascular disease, residents in neighborhoods with scores in the highest quartile of the psychosocial hazards scale had more than 4 times higher odds of a history of myocardial infarction and more than 3 times higher odds of myocardial infarction, stroke, transient ischemic attack, or intermittent claudication compared with residents living in neighborhoods scoring in the lowest quartile.

Conclusions. Neighborhood psychosocial hazards were significantly associated with self-reported cardiovascular disease after adjustment for individual-level risk factors. This is consistent with the hypothesis that environmental stress plays a role in the etiology of cardiovascular disease. (*Am J Public Health.* 2008; 98:1664–1670. doi:10.2105/AJPH.2007.125138)

The effect on health of the places in which people live—apart from individual, genetic, or lifestyle characteristics—is of increasing interest to researchers.[1] A new wave of research is examining the health consequences of various aspects of residential neighborhoods. Moving beyond the study of individual risk factors to the study of neighborhoods may be a key to understanding widening health disparities across racial/ethnic and sociodemographic groups.[2,3]

Many aspects of neighborhoods are hypothesized to influence cardiovascular disease (CVD) risk through several different and potentially interrelated mechanisms.[4–6] Health behavior has received significant attention. Some data suggest that residents of socioeconomically deprived neighborhoods are more likely to engage in high-risk health behaviors, including inactivity,[7,8] poor diet,[9] illicit drug use,[10,11] and smoking.[12] However, studies that found an association between neighborhood of residence and CVD after adjustment for many of these health behaviors raised doubts that behavior was the sole mechanism.[13,14]

Several studies have examined the relationship between neighborhood socioeconomic characteristics (such as percentage of residents living in poverty) and CVD. Living in disadvantaged neighborhoods was found to be independently associated with increased risk of subclinical CVD,[15] coronary heart disease,[13,16] and stroke,[17] as well as poorer prognosis and decreased survival after myocardial infarction.[18] Several explanations have been suggested for this association.[4]

Psychosocial hazards in the neighborhood may be an important link between neighborhood socioeconomic disadvantage and adverse health outcomes.[5,19] Psychosocial hazards are visible characteristics of neighborhoods—such as violent crime, abandoned buildings, and signs of incivility—that give rise to a heightened state of vigilance, alarm, or threat.[6,20] Daily exposure to psychosocial hazards in the neighborhood is known to activate a physiological stress response.[21,22] Chronic stress may in turn lead to dysregulation of either the autonomic nervous system[23] or the hypothalamic–pituitary–adrenal axis, or both. Dysregulation of the latter has been linked to key CVD risk factors, including the deposition of abdominal fat,[24,25] acute and chronic elevations in blood pressure,[26] and various inflammatory processes.[27]

We tested the hypothesis that a higher level of neighborhood psychosocial hazards is associated with increased odds of self-reported myocardial infarction, stroke, transient ischemic attack, and intermittent claudication, independent of individual-level risk factors.

METHODS

The Baltimore Memory Study is a population-based cohort study of risk factors for cognitive decline in residents sampled at random from 65 contiguous urban neighborhoods. The methods are described elsewhere in detail.[28] In short, households in the predetermined study area were linked to telephone numbers, and households with telephones were randomly selected for recruitment. The sampling frame was taken from the Department of Assessments and Taxation listing of all residential properties in Baltimore City and accounted for multiunit dwellings such as apartment buildings and condominiums. Eligibility was determined for 2351 respondents (aged 50–70 years, resident in selected household, and resident of Baltimore for at least 5 years), and of these respondents, 60.8% were scheduled for an enrollment visit. Of the 1403 scheduled for an appointment, 1140 (81.3%) were enrolled and subsequently tested.

Individual Measures

The baseline examinations took place between 2001 and 2002. Data on individual study participants was collected at the study clinic by trained research assistants. During the first study visit, a structured interview obtained information on age in years, gender,

housing, residential history, and smoking history. Participants were categorized as Black, White, or other race/ethnicity (including Hispanic Whites, Hispanic Blacks, other Blacks, and other Whites) by self-report. Educational attainment was assessed by self-reported years of education completed as well as credentials acquired (e.g., degrees, certificates, trade school).[28] For analysis, participants were classified as having less than a high school education or more than a high school education.

The CVD outcomes were self-reported history of physician diagnosis of myocardial infarction (MI), stroke, transient ischemic attack (TIA), or intermittent claudication (IC). For the first 3, participants were asked whether a doctor had ever told them that they had these conditions. Only yes responses were counted; participants who answered "possible" were treated as negative for all outcomes to increase the specificity of outcome classification (in this case, higher specificity was preferable to greater sensitivity). Participants were considered to have IC if they answered yes to having all of the following symptoms from the World Health Organization Rose Questionnaire[29]: (1) pain in either leg on walking, (2) pain does not begin when standing still, (3) pain involves the calves, (4) pain is present when walking uphill or hurrying, and (5) pain is relieved in 10 minutes or less after rest.

Blood pressure measurements were taken 3 times in the sitting position with a sphygmomanometer. Presence of hypertension was defined as mean systolic blood pressure of 140 mm Hg or more, mean diastolic blood pressure of 90 mm Hg or more, or use of antihypertensive medications. Low-density lipoprotein cholesterol was calculated with the Friedewald formula from measurements of total cholesterol, high-density lipoprotein, and triglycerides.[30] In 11% of participants, the formula could not be applied because of high triglyceride values; in this group, missing values were imputed from model-based multiple-imputation procedures.[31] Serum low-density lipoprotein cholesterol levels of 160 mg/dL or more were classified as high.[32] Functional status was measured with a standard 7-item index of ability to perform instrumental activities of daily living (IADL).[33] For analysis, participants were classified as no IADL disability (0) versus 1 or more domains of disability.[1] Self-reports of physician-diagnosed diabetes were obtained during the baseline interview.

Neighborhood Measures

Neighborhood boundaries were derived by the Baltimore City Department of Planning from community definitions of existing neighborhoods. City planners held focus groups, identified neighborhood groups and committees, and conducted surveys to determine which neighborhoods were recognized by members of the community and which were historically real and geographically definable. Thus, we defined our spatial units as city neighborhoods, not census units or zip codes, which are used as proxies for neighborhoods in most studies.

Data on neighborhood characteristics came from the 2000 US Census and the Baltimore City departments of planning, public works, and police. Block-level census data were recombined into neighborhoods by the US Census Bureau. Block-level census data are normally not made available to researchers because of confidentiality concerns; they represent the smallest level of aggregation collected. For this study, the Census Bureau tabulated these data to the precise boundaries of Baltimore neighborhoods by special request. Number and location of violent crimes, off-site liquor licenses, and 911 emergency telephone calls were individually mapped and aggregated at the neighborhood level with a geographic information system. Participants were linked to their neighborhood of residence by their home address at baseline.

Development of the neighborhood psychosocial hazards scale (NPH) was previously reported.[20] In brief, this scale was constructed by selecting an array of neighborhood indicators drawn from sources other than study participants, hypothesized to measure the presence of psychosocial hazards in the neighborhood. Factor analysis was used to identify a final set of 12 indicators (e.g., violent crime rate, calls to city agencies about street problems, and number of off-site liquor licenses; the complete list of indicators is available as a supplement to the online version of this article at http://www.ajph.org) that met standard criteria indicating this to be a reliable and valid measure of the construct of neighborhood psychosocial hazard.[6,21] The NPH scale is normally distributed (range=19.0–19.2; mean=0.1; SD=9.6); higher values indicate more neighborhood psychosocial hazards. For modeling, we divided the NPH values into quartiles at the neighborhood level; the lowest quartile was the reference category. We based the quartile cutpoints on the distribution of neighborhoods, not the distribution of participants. In sensitivity analyses (not shown), we verified that these results were not dependent on the use of quartiles or another set of cutpoints: similar inferences resulted from alternative choices.

To evaluate the strength of association of neighborhood psychosocial hazards compared with traditional measures of socioeconomic status, we examined 2 other measures. The Townsend index of socioeconomic deprivation is a standard summary measure developed specifically for use in public health research.[34] The index of neighborhood affluence combines 5 indicators of neighborhood wealth (per capita income of $20 000 or higher, median family income of $50 000 or higher, 30% of adults with undergraduate degrees or higher educational level, median house value of $75 000 or more, and 35% or more children enrolled in private school).[35] Both of these indexes are intended to measure neighborhood socioeconomic status rather than specific psychosocial hazards, although these concepts are known to overlap.

Statistical Analysis

We evaluated bivariate associations between the 3 neighborhood measures and self-reported history of CVD with the χ^2 test for pairs of ordinal variables and 1-way analysis of variance for unbalanced designs for pairs of continuous and discrete variables. Our data had a 2-level structure: individuals nested within neighborhoods. We used multilevel analysis to account for the correlation that arose from this nesting.[36] Stata version 9 (StataCorp LP, College Station, TX) was used to estimate random-effects logistic regression. In this model, a random intercept for each neighborhood corrected for autocorrelation arising from the 2-level data structure. The interpretation of results from this type of model is similar to that of traditional fixed-effects logistic regression models.

TABLE 1—Self-Reported History of Cardiovascular Disease and Characteristics of Study Participants at Enrollment: Baltimore Memory Study, 2001–2002

		MI Only		MI, Stroke, or TIA		MI, stroke, TIA, or IC	
	Entire Cohort, No. (%)	No. (%)	*P* for trend[a]	No. (%)	*P* for trend[a]	No. (%)	*P* for trend[a]
Participants	1140 (100)	59 (5)		102 (9)		137 (12)	
Age, y			.124		.006		.001
50–55	362 (32)	17 (5)		28 (8)		37 (10)	
55–60	295 (26)	9 (3)		17 (6)		27 (9)	
60–65	234 (21)	15 (6)		22 (9)		25 (11)	
65–70	249 (22)	18 (7)		35 (14)		48 (19)	
Gender			.030		.196		.831
Women	749 (66)	31 (4)		61 (8)		91 (12)	
Men	391 (34)	28 (7)		41 (10)		46 (12)	
Race/ethnicity			.008		<.001		<.001
Black	474 (42)	29 (6)		57 (12)		76 (16)	
White	597 (52)	22 (4)		35 (6)		49 (8)	
Other	69 (6)	8 (12)		10 (15)		12 (18)	
Education			.697		.023		.002
Less than high school	154 (14)	7 (5)		21 (14)		30 (19)	
High school or more	985 (86)	52 (5)		81 (8)		107 (11)	
Current smoker			.625		.061		.377
Yes	242 (21)	14 (6)		29 (12)		104 (12)	
No	898 (79)	45 (5)		73 (8)		33 (14)	
Hypertension[b]			<.001		<.001		<.001
Yes	710 (38)	50 (7)		84 (12)		25 (6)	
No	430 (62)	9 (2)		18 (4)		112 (16)	
Diabetes			<.001		<.001		<.001
Yes	200 (18)	22 (11)		36 (18)		90 (10)	
No	940 (82)	37 (4)		66 (7)		47 (24)	
IADL status			<.001		<.001		<.001
Disabled	123 (11)	16 (13)		32 (26)		40 (33)	
Not disabled	1010 (89)	43 (4)		70 (7)		97 (10)	
LDL cholesterol, mg/dL			.686		.568		.499
≥160	123 (12)	7 (6)		12 (10)		105 (12)	
<160	887 (88)	43 (5)		73 (8)		12 (10)	
NPH scale[c]			<.001		<.001		<.001
Quartile 1	233 (21)	5 (2)		6 (3)		8 (3)	
Quartile 2	432 (38)	18 (4)		35 (8)		46 (11)	
Quartile 3	225 (20)	11 (5)		24 (11)		34 (15)	
Quartile 4	245 (22)	25 (10)		37 (15)		49 (20)	

Note. MI = myocardial infarction; TIA = transient ischemic attack; IC = intermittent claudication; IADL = instrumental activities of daily living; LDL = low-density lipoprotein; NPH = neighborhood psychosocial hazards.
[a]For variables with more than 2 categories, *P* is for the χ^2 test of differences across row in the percentage reporting the outcome. Otherwise, *P* is for a 2-tailed test of difference between 2 groups in the proportion reporting each outcome.
[b]Defined as systolic blood pressure of 140 mm Hg or more, diastolic blood pressure of 90 mm Hg or more, or use of antihypertensive medications.
[c]The NPH scale comprises 12 indicators of psychosocial hazards in the neighborhood that lead to a heightened sense of threat in residents.[20] The observed range on the original scale for quartile 1 (reference) was −19.2 to −7.3; for quartile 2, −7.2 to −1.2; for quartile 3, −1.1 to 7.5; and for quartile 4, 7.6 to 19.0. The frequencies were not equal because the quartile cut points were determined by the observed distribution of NPH scores across neighborhoods, not individuals.

We constructed 2 sets of models. Partially adjusted models included the NPH score (in quartiles) and only sociodemographic risk factors not believed to be on the causal pathway (including age, gender, race/ethnicity, and education). In the fully adjusted models, health behaviors (smoking), medical history (hypertension, diabetes, high levels of low-density lipoprotein cholesterol), and presence of IADL disability were added. The interpretation of the fully adjusted models was more complex because several of the variables may be intermediate end points (or mechanisms) that lie between neighborhood exposure and outcomes. Because differences in the health status of persons living in different neighborhoods could be attributable to selection rather than to the effects of the neighborhood itself, the fully adjusted models should be interpreted with caution.

Because some of our CVD outcomes were rare, and to check the robustness of our findings, we evaluated separate models in which 3 outcome sets were examined with increasing prevalence. Outcome group 1 was limited to persons with a self-reported history of MI. Outcome group 2 included participants who reported a history of MI, stroke, or TIA. Outcome group 3 included participants who reported a history of MI, stroke, TIA, or IC. All outcomes were coded 1 for yes, 0 for no, or possible. All of these diseases involve peripheral vascular lesions, generally atherosclerotic, and are likely to share a common pathologic process. This common pathologic process could plausibly involve the stress pathways that we hypothesized are involved in how neighborhoods affect health, and combining outcomes had the advantage of increasing the statistical power of our analyses.

RESULTS

At enrollment, participants in the Baltimore Memory Study were 66% female and 42% Black; their mean age was 59 years (Table 1). Overall, 59 (5.2%) reported a previous history of MI, 51 (4.5%) reported a history of stroke or TIA, and 41 (3.6%) reported a history of IC. Among participants reporting MI, 13% also reported a history of stroke or TIA (not shown). The prevalence of any CVD (including IC) was 12.1% (n=137). We found

TABLE 2—Association of Neighborhood Psychosocial Hazards Scale Scores With Self-Reported History of Cardiovascular Disease: Baltimore Memory Study, 2001–2002

	MI Only		MI, Stroke, or TIA		MI, stroke, TIA, or IC	
	OR (95% CI)	*P* for trend	OR (95% CI)	*P* for trend	OR (95% CI)	*P* for trend
Partially adjusted models (n = 1133)[a]						
NPH scale[b]		.001		.003		<.001
Quartile 1 (Ref)	1.00		1.00		1.00	
Quartile 2	2.15 (0.76, 6.13)		2.75 (1.10, 6.89)		2.69 (1.21, 5.99)	
Quartile 3	2.34 (0.76, 7.20)		3.51 (1.35, 9.13)		3.79 (1.65, 8.69)	
Quartile 4	5.56 (1.82, 17.04)		4.51 (1.68, 12.1)		4.62 (1.94, 11.0)	
Fully adjusted models (n = 1130)[c]						
NPH scale[b]		.003		.003		
Quartile 1 (Ref)	1.00		1.00		1.00	
Quartile 2	1.89 (0.65, 5.46)		2.47 (0.98, 6.27)		2.46 (1.09, 5.54)	
Quartile 3	2.15 (0.69, 6.64)		3.07 (1.17, 8.07)		3.37 (1.46, 7.81)	
Quartile 4	4.68 (1.50, 14.6)		3.57 (1.30, 9.75)		3.81 (1.58, 9.23)	

Note. MI = myocardial infarction; TIA = transient ischemic attack; IC = intermittent claudication; OR = odds ratio; CI = confidence interval; NPH = neighborhood psychosocial hazards. ORs and 95% CIs were obtained from random-effects logistic regression models.

[a]Included age, gender, race/ethnicity, education, and quartiles of the NPH scale.

[b]The NPH scale comprises 12 indicators of psychosocial hazards in the neighborhood that lead to a heightened sense of threat in residents.[20] The observed range on the original scale for quartile 1 (reference) was –19.2 to –7.3; for quartile 2, –7.2 to –1.2; for quartile 3, –1.1 to 7.5; and for quartile 4, 7.6 to 19.0.

[c]Added current smoking, hypertension, diabetes, low-density lipoprotein cholesterol, and instrumental activities of daily living disability to the variables in the partially adjusted models.

higher prevalence among older participants, males (MI only), Blacks, and those with less than a high school education (outcome groups 2 and 3 only). Hypertension, diabetes, and disability were also associated with higher CVD prevalence for all outcome groups. In crude analysis, prevalence of all 3 outcome groups increased in higher quartiles of the NPH scale. Participants residing in neighborhoods with NPH scores in the highest quartile had nearly 5 times the odds of reporting a history of MI and nearly 6 times the odds of reporting a group-2 or group-3 outcome as residents of the lowest-risk neighborhoods.

We used a multilevel (random effects) logistic regression to examine associations of NPH with CVD after adjustment for sociodemographic variables (partially adjusted models) and the full set of covariates (fully adjusted models; Table 2). In the partially adjusted models, residing in a neighborhood in the highest quartile of the NPH scale was associated with significantly increased odds of self-reported history of MI compared with living in a neighborhood with the lowest NPH score (odds ratio [OR]=5.6; 95% confidence interval [CI]=1.8, 17; P=.001, for trend). For outcome group 2 (MI, stroke, or TIA), we found a significant association between quartiles 2 (OR=2.8), 3 (OR=3.5), and 4 (OR=4.5) of the NPH scale and the reference quartile (P=.003, for trend). For outcome group 3, the pattern was similar to that of outcome group 2; we noted a dose–response relationship with increasing ORs across quartiles of the NPH scale (OR=2.7, 3.8, and 4.6, respectively; P<.001, for trend).

After we adjusted for current smoking, hypertension, diabetes, low-density lipoprotein cholesterol, and IADL disability (fully adjusted model), the associations remained consistent and CIs narrowed. As indicated by tests for trend, the odds of CVD increased across increasing levels of NPH scores. In outcome groups 2 and 3, the 2 highest levels of NPH scores were associated with significantly increased odds of CVD (group 2, OR=3.1 and 3.6 for the highest and next quartile, respectively; P=.003, for trend) and for group 3 (OR=3.4 and 3.8 for the highest and next quartile, respectively; P=.003, for trend). These results were attributable in part to the additional power from the inclusion of more events in outcome groups 2 and 3. Adjustment for smoking, hypertension, diabetes, low-density lipoprotein cholesterol, and IADL disability attenuated the association between NPH scores and CVD between 8% (quartile 2, outcome group 3) and 21% (quartile 4, outcome group 2).

We compared the results for the NPH scale with the results of similar models that included the Townsend index of socioeconomic deprivation and the index of neighborhood affluence. After adjustment for the full set of covariates (Table 3), we found a pattern of associations of CVD with the Townsend index that was similar to our findings with the NPH scale; however, for all significant associations, the Townsend index showed weaker associations with CVD. We found significant trends indicating an association between odds of CVD and higher Townsend index score for outcome group 1 (OR=1.85, 1.92, and 3.33 for the second, third, and fourth quartiles, respectively, compared with the first; P=.015, for trend) and outcome group 2 (OR=1.39, 1.72, 2.04 for the 3 highest quartiles, respectively; P=.015, for trend), but not for outcome group 3 (OR=1.90, 2.14, 2.09 for the 3 highest quartiles, respectively; P=.057, for trend).

Higher quartiles of the index of neighborhood affluence represent greater affluence, and thus the associations were reversed. Neighborhood affluence was generally more weakly associated with all outcome groups than were NPH scale or Townsend index scores, although we noted a significant trend for all outcome groups. Residents of the most affluent neighborhoods were less likely to belong to outcome group 2 (OR=1.01, 0.76, and 0.37 for the second, third, and fourth quartiles, respectively, compared with the first; P=.049, for trend) or outcome group 3 (OR=1.02, 0.69, 0.32; P=.005, for trend).

DISCUSSION

In a large community-based population of adults aged 50 to 70 years who were randomly selected from contiguous urban Baltimore neighborhoods, residence in

TABLE 3—Associations of 3 Neighborhood Measures With Self-Reported History of Cardiovascular Disease in Fully Adjusted Models (n = 1130): Baltimore Memory Study, 2001–2002

	NPH Scale[a]		Townsend Index[b]		Index of Neighborhood Affluence[c]	
	OR (95% CI)	*P* for trend[d]	OR (95% CI)	*P* for trend[d]	OR (95% CI)	*P* for trend[d]
MI Only		.003		.015		.049
Quartile 1 (Ref)	1.00		1.00		1.00	
Quartile 2	1.89 (0.65, 5.46)		1.85 (0.72, 4.78)[d]		1.10 (0.52, 2.34)[d]	
Quartile 3	2.15 (0.69, 6.64)		1.92 (0.84, 4.38)		0.63 (0.29, 1.39)	
Quartile 4 (highest)	4.68 (1.50, 14.6)		3.33 (1.38, 8.05)		0.39 (0.13, 1.15)	
MI, stroke, or TIA		.003		.015		.049
Quartile 1 (Ref)	1.00		1.00		1.00	
Quartile 2	2.47 (0.98, 6.27)		1.39 (0.66, 2.91)		1.01 (0.55, 1.86)	
Quartile 3	3.07 (1.17, 8.07)		1.72 (0.93, 3.17)		0.76 (0.42, 1.38)	
Quartile 4 (highest)	3.57 (1.30, 9.75)		2.04 (1.02, 4.10)		0.37 (0.15, 0.93)	
MI, stroke, TIA, or IC		.003		.057		.005
Quartile 1 (Ref)	1.00		1.00		1.00	
Quartile 2	2.46 (1.09, 5.54)		1.90 (1.00, 3.63)		1.02 (0.59, 1.75)	
Quartile 3	3.37 (1.46, 7.81)		2.14 (1.23, 3.74)		0.69 (0.40, 1.18)	
Quartile 4 (highest)	3.81 (1.58, 9.23)		2.09 (1.09, 3.98)		0.32 (0.14, 0.73)	

Note. NPH = neighborhood psychosocial hazards; OR = odds ratio; CI = confidence interval; MI = myocardial infarction; TIA = transient ischemic attack; IC = intermittent claudication. ORs and 95% CIs were obtained from random-effects logistic regression models. Fully adjusted models included age, gender, race/ethnicity, education, current smoking, hypertension, diabetes, low-density lipoprotein cholesterol, instrumental activities of daily living disability, and quartiles of the NPH scale.
[a]The NPH scale comprises 12 indicators of psychosocial hazards in the neighborhood that lead to a heightened sense of threat in residents.[20]
[b]The Townsend index of socioeconomic deprivation is a standard summary measure of neighborhood socioeconomic status that was developed specifically for use in public health research.[34]
[c]The index of neighborhood affluence combines 5 indicators of neighborhood wealth (per capita income ≥ $20 000, median family income ≥ $50 000, 30% of adults with undergraduate degrees or higher educational level, median house value of ≥ $75 000 or more, and ≥ 35% children enrolled in private school).[35]
[d]2-tailed χ^2 test.

neighborhoods with more psychosocial hazards was associated with significantly higher odds of self-reported history of physician-diagnosed CVD. All 3 outcome groups examined had increased odds of CVD if they lived in neighborhoods with higher scores on a measure designed to assess the presence of psychosocial hazards through information about neighborhoods gathered independently of study participants.

Previous studies investigated the association of neighborhood material deprivation with cardiovascular disease and its risk factors.[4,12–16,37] However, most of these studies used global measures of area socioeconomic status that did not allow for testing specific hypotheses about the mechanisms through which material deprivation operates. We sought to address this limitation by comparing the strength of associations between global measures of socioeconomic status (the Townsend index of socioeconomic deprivation and the index of neighborhood affluence) and a more specific measure of neighborhood psychosocial hazards.

Ours was among the first studies to find support for the hypothesis that environmental stress is involved in the relationship of neighborhood of residence and CVD. Growing evidence links environmental stress to CVD in both animals[38,39] and humans.[40–44] Fewer studies have looked for neighborhood factors that elicit dysregulation of the stress response system.[45,46] Because our study was cross-sectional, we could not infer causality. Also, because we did not include biomarkers of stress response, we could not confirm that this is a physiologic mechanism linking neighborhood environment to CVD. However, we consider our measure of psychosocial hazards to be a novel response to criticisms by Sampson,[47] among others, that called for detailed measures of particular mechanisms that might underlie the well-established association between neighborhood deprivation and health. Animal and mechanistic studies strongly indicate that stress is involved in atherosclerotic disease. However, almost no studies have demonstrated that adverse environments are stressful, even though there is consensus that stress comes from something in the environment. We attempted to carefully measure what that might be.

A criticism of neighborhood studies is that observed associations may be attributable to confounding from individual-level risk factors that are clustered by neighborhood, such as health behavior, medical history, or individual socioeconomic status. If, for example, persons who smoke, have low educational attainment, or suffer from chronic diseases self-select into neighborhoods that are high in psychosocial hazards, an association could be detected that is not causal. Because our study was cross-sectional, this possibility could not be ruled out. To address this concern, we adjusted for multiple individual CVD risk factors. The association of NPH with CVD was only slightly attenuated with the addition of these variables, suggesting that the association between neighborhood psychosocial hazards is not explained solely by differences in these characteristics. To address residual confounding by individual socioeconomic status, we adjusted for individual household income; however, our inferences were unchanged.

Participants were included in the Baltimore Memory Study only if they had lived in their current residence for 5 or more years, decreasing the likelihood that recent relocation necessitated by chronic conditions could lead to bias. Most (74%) study participants lived in their current address for 10 years or longer. We conducted a sensitivity analysis by excluding persons who reported residing in their current neighborhood for less than 10 years and found no change in our results. Therefore, it is unlikely that self-selection by persons with adverse risk factors moving into

neighborhoods with more psychosocial hazards account for our results.

This study relied on self-reported history of CVD. However, participants were asked whether a doctor ever told them they had a cardiovascular event or disease. Previous studies found that self-reports of MI, stroke, and TIA were generally in good agreement with more-objective methods of case determination.[48,49] Self-reported history of stroke and MI have been found to have a high sensitivity (80%–90%) and specificity (99%).[50–52] The World Health Organization Rose Questionnaire for IC was developed for use in epidemiological surveys and has a high specificity for peripheral vascular disease.[53] Given the high specificity of self-reports, misclassification is more likely to lead to a bias toward the null, rather than exaggeration of the true association.

In the Baltimore Memory Study, all medications were presented to the interviewer during the baseline clinic visit and carefully recorded and catalogued by type. We found that those who reported a history of any CVD had 4.4 times the odds of taking 1 or more CVD-related medications (antihypertensives, beta blockers, calcium channel blockers, statins, or other lipid-lowering drugs). This added to our confidence that outcome misclassification was unlikely to be severe. Some unknown proportion of each condition may not have been related to atherosclerosis. For MI and IC, this proportion was likely very low. For strokes, almost 15% are known to be hemorrhagic; the rest are ischemic.[54] However, this would be likely to decrease the strength of associations observed.

Our findings agree with previous studies showing that neighborhood of residence is independently associated with CVD. Our results suggest that chronic exposure to psychosocial hazards in the residential neighborhood influences CVD risk and may explain some of the substantial socioeconomic disparities seen in the distribution of CVD. Future research should examine the biological mechanisms by which neighborhood psychosocial hazards become internalized and the risk of CVD is increased. Previous literature suggests that neighborhood psychosocial hazards may have negative effects on the autonomic nervous system, the hypothalamic–pituitary–adrenal axis, and cytokines and other inflammatory mediators.[20,55,56] Our study illustrates the utility of considering place-level characteristics in the array of factors that may contribute to the etiology of CVD. These findings also suggest new targets for intervention and policy change. ■

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Contributors

T. Augustin envisioned the project, conducted analyses, and contributed to the writing. T.A. Glass supervised the analysis and edited the article. B.D. James edited the article and helped refine the analysis. B.S. Schwartz oversaw the data collection and edited the article.

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Human Participant Protection

This study was approved by the internal review board at the Johns Hopkins Bloomberg School of Public Health.

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