# Neighborhood socioeconomic disadvantage and mortality after stroke

Arleen F. Brown, MD, PhD
Li-Jung Liang, PhD
Stefanie D. Vassar, MS
Sharon Stein Merkin, PhD
W.T. Longstreth, Jr., MD
Bruce Ovbiagele, MD
Tingjian Yan, PhD
José J. Escarce, MD, PhD

Correspondence to Dr. Brown: abrown@mednet.ucla.edu

## ABSTRACT

**Objective:** Residence in a socioeconomically disadvantaged community is associated with mortality, but the mechanisms are not well understood. We examined whether socioeconomic features of the residential neighborhood contribute to poststroke mortality and whether neighborhood influences are mediated by traditional behavioral and biologic risk factors.

**Methods:** We used data from the Cardiovascular Health Study, a multicenter, population-based, longitudinal study of adults ≥65 years. Residential neighborhood disadvantage was measured using neighborhood socioeconomic status (NSES), a composite of 6 census tract variables representing income, education, employment, and wealth. Multilevel Cox proportional hazard models were constructed to determine the association of NSES to mortality after an incident stroke, adjusted for sociodemographic characteristics, stroke type, and behavioral and biologic risk factors.

**Results:** Among the 3,834 participants with no prior stroke at baseline, 806 had a stroke over a mean 11.5 years of follow-up, with 168 (20%) deaths 30 days after stroke and 276 (34%) deaths at 1 year. In models adjusted for demographic characteristics, stroke type, and behavioral and biologic risk factors, mortality hazard 1 year after stroke was significantly higher among residents of neighborhoods with the lowest NSES than those in the highest NSES neighborhoods (hazard ratio 1.77, 95% confidence interval 1.17–2.68).

**Conclusion:** Living in a socioeconomically disadvantaged neighborhood is associated with higher mortality hazard at 1 year following an incident stroke. Further work is needed to understand the structural and social characteristics of neighborhoods that may contribute to mortality in the year after a stroke and the pathways through which these characteristics operate. *Neurology*® 2013;80:520–527

## GLOSSARY

**CHS** = Cardiovascular Health Study; **CI** = confidence interval; **CVD** = cardiovascular disease; **DBP** = diastolic blood pressure; **HDL** = high-density lipoprotein; **HR** = hazard ratio; **IRB** = institutional review board; **NSES** = neighborhood socioeconomic status; **SBP** = systolic blood pressure; **SES** = socioeconomic status; **TC** = total cholesterol.

Stroke is a leading cause of death in the United States. Among adults ages 65 years and older, mortality at 1 year after an initial stroke is over 30%.[1] An emerging literature suggests that place of residence may play an important role in stroke risk.[2–8] Recent evidence suggests that the association between neighborhood socioeconomic status (SES) and incident stroke is mediated by biologic risk factors, such as control of blood pressure, blood sugars, and lipids.[4] Fewer studies have explored whether neighborhood factors influence poststroke mortality,[2,9,10] and although socioeconomic features of neighborhoods, such as area-level deprivation[2,10] and neighborhood social cohesion,[9] have been implicated in poststroke mortality, the mechanisms remain poorly understood.

To examine the relationship between neighborhood SES (NSES) and mortality after stroke and whether these associations are mediated by traditional behavioral and biologic risk factors, we analyzed data from the Cardiovascular Health Study (CHS), a large population-based, longitudinal study of coronary heart disease and stroke in adults 65 years of age and older. We hypothesize that residence in a disadvantaged area is associated with higher mortality after incident stroke and that the

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From the Division of General Internal Medicine and Health Services Research (A.F.B., L.-J.L., J.J.E.), Department of Neurology (S.D.V., B.O., T.Y.), and Division of Geriatrics (S.S.M.), University of California, Los Angeles; Departments of Neurology and Epidemiology (W.T.L.), University of Washington, Seattle; Department of Neurosciences (B.O.), University of California, San Diego; SCAN Healthplan (T.Y.), Long Beach, CA; and RAND (J.J.E.), Santa Monica, CA.

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neighborhood effects are not mediated or moderated by individual socioeconomic, behavioral, and biologic stroke risk factors.

**METHODS Study population.** The CHS is a longitudinal, population-based study of cardiovascular disease (CVD), including coronary heart disease and stroke, and has been described previously.[11,12] Briefly, participants were randomly sampled from Medicare eligibility lists in 4 US communities: Forsyth County, North Carolina; Washington County, Maryland; Sacramento County, California; and Pittsburgh (Allegheny County), Pennsylvania. Eligible participants were 65 years or older at the time of the examination, not institutionalized, and did not require a proxy respondent at baseline. Approximately 57% of those eligible were enrolled in the study. The "initial cohort" (n = 5,201) was recruited between 1989 and 1990, and an additional African American "new cohort" (n = 687) was recruited from the North Carolina, California, and Pennsylvania counties in 1992. CHS collected survey and clinical data on study participants at regular intervals until 1999. These analyses use data from continued surveillance for cardiovascular events and mortality through June 30, 2006.

**Incident stroke, stroke type, and mortality after stroke.** The main outcome of these analyses is mortality hazard, or time to death after incident stroke, examined at 30 days and 1 year. Detailed descriptions of the ascertainment methods for incident and prevalent strokes, TIAs, and deaths are described elsewhere.[11,13–15] Participants with prevalent strokes (n = 205) at baseline were excluded from these analyses. Incident strokes were ascertained through interviews at annual visits, interim telephone contacts, notification of events by participants, and review of Medicare hospitalization and cause of death data. For suspected stroke events, inpatient and outpatient medical records, results of pertinent tests, and copies of brain images (CT and MRI) were obtained. All suspected strokes were adjudicated by a cerebrovascular disease endpoint committee that classified stroke subtype (ischemic, hemorrhagic, and unknown type) and determined whether death was caused by stroke.[11,13,14] Information on fatal events and classification of cause of death were obtained from death certificates, autopsy and coroner's reports, hospital records, and interview with attending physicians, next of kin, and witnesses.[11]

**Neighborhood socioeconomic status.** CHS participants' baseline home addresses were geocoded to identify the residential census tract defined in the 1990 US decennial Census. Census tracts were used as a proxy for neighborhood. Neighborhood definitions vary widely and these administrative units do not perfectly capture the social features of neighborhoods; however, census tract characteristics have been shown to be robust predictors of health.[16]

The NSES index used in this study has been previously described in studies of the CHS population.[17,18] It was constructed by summing the *z* scores of 6 census-derived SES indicators that represent the area's physical and social resources: 1) median household income; 2) median value of housing units; 3) percent households with interest, dividend, or rental income; 4) percent of residents ≥25 years of age with a high school degree; 5) percent of residents ≥25 years of age with a college degree; and 6) percent of residents in executive, managerial, or professional specialty occupations. Quartile 1 represented the highest residential NSES scores (least disadvantaged neighborhoods), and quartile 4, the lowest scores (the most disadvantaged neighborhoods).

**Covariates.** Sociodemographic characteristics of participants, including age, sex, race, income (median household income), and education, were all reported in the baseline survey.

Health-related behaviors reported in the interview included smoking history, physical activity, alcohol use, and diet. Participants were classified as nonsmokers if they had smoked fewer than 100 cigarettes (or 5 packs of cigarettes) in their lifetime. Former smokers were those who reported having smoked at least 100 cigarettes or 5 packs of cigarettes in their lifetime but had not smoked during the prior 30 days. Alcohol use was categorized as none (less than one drink per week), 1–7 drinks per week, and more than 7 drinks per week. Energy expenditure during leisure-time physical activity in the 2 weeks prior to the interview was categorized into quartiles. For participants in the original cohort, dietary intake in the 12 months before the baseline survey was estimated using a standardized food frequency inventory that was converted to nutrient intake per day.[19,20] Cereal fiber was reported in grams and sodium in milligrams.

Biologic characteristics of participants were obtained by combinations of participant report, medical record review, and CHS data collection. Body mass index was determined by height and weight measured during the examinations and reported in kg/m$^2$. Blood pressure measurements were made in a standardized manner during the clinical examination. Hypertension was defined as systolic blood pressure (SBP) ≥160 mm Hg, diastolic blood pressure (DBP) ≥95 mm Hg, or both a reported diagnosis of hypertension and use of an antihypertension medication. We defined diabetes as a fasting blood glucose ≥126 mg/dL on the clinical examination or both a reported diagnosis of diabetes and use of an oral antidiabetic medication or insulin, using the American Diabetes Association definition.[21] Fasting lipid panels, which included total cholesterol (TC), low-density lipoprotein cholesterol, and high-density lipoprotein (HDL) cholesterol, were measured in the study. A diagnosis of atrial fibrillation was based on self-report or EKG readings. Subclinical CVD was defined as evidence of any of the following on the survey, clinical examination, or both: ankle–arm index <0.9; carotid stenosis >25%; internal carotid thickness >80th percentile; common carotid thickness >80th percentile; major EKG abnormalities; abnormal ejection fraction or wall motion on echocardiogram; and claudication or angina on Rose Questionnaire.[18]

**Analyses.** Means and frequency distributions of demographic, behavioral, and biologic characteristics of participants who survived vs those who died after stroke were summarized. Based on prior work demonstrating differences in the factors that influence short term compared to longer term poststroke mortality,[22–25] we examined 2 points in time: 30 days after stroke and 1 year after stroke. Because the participants were clustered within census tracts, we constructed multilevel Cox proportional hazards models, or "frailty" models, which provide a mechanism for decomposing the variation in outcome variables of interest into separate components due to individual-level and neighborhood-level effects,[26,27] to examine whether NSES was associated with mortality after incident stroke. Participants who were alive 30 days after a stroke or 1 year after a stroke were censored at those time points, respectively. The base model was adjusted for participants' demographic characteristics (age, sex, income, and education categories) and stroke type. The full model was the base model plus the behavioral and biologic characteristics. To assess whether the association between NSES and death after a stroke was mediated by individual behavioral or biologic characteristics, we compared the results of the base model with the full model. Because the relationship between NSES and poststroke mortality did not differ appreciably between the full and partial models, we present here the results of the full model. We then tested the validity of the proportional hazards models for each of the final models (30-day and 1-year poststroke death) and found that the global tests of validity for both models were not significant, i.e., there was no indication of a violation of the proportionality assumption.[28]

To determine whether the relationship between NSES and mortality after stroke was moderated by individual sociodemographic

characteristics, interactions between NSES and selected individual-level variables that were significant in the full model were tested for statistical significance. We also conducted a sensitivity analysis that included CHS clinic site as a variable in the final model. This did not appreciably change the association between NSES and poststroke mortality, so we present here the results of the models without clinic site included.

Because nutrition assessments were only available for participants in the original cohort, we included measures of fiber, fish, and sodium intake in a series of models restricted to white participants. As another set of sensitivity analysis, we took 2 approaches to incorporating biologic risk factors that were measured longitudinally, such as SBP and DBP. In separate models, we first incorporated the baseline value of these covariates, then the last available measurement prior to death or study end date.

**Standard protocol approvals, registrations, and patient consents.** All participants gave written informed consent and all study protocols were approved by the institutional review boards (IRBs) of participating institutions. These analyses were reviewed and approved by the UCLA IRB.

**RESULTS** Baseline data were available for 5,888 participants. We excluded from the analyses 947 participants whose addresses were not geocoded or whose addresses matched to block groups with fewer than 100 persons, fewer than 30 housing units per block, or with more than 33% persons in group quarters, e.g., military bases. With these and other exclusions for prevalent stroke (n = 205) and "other" race/ethnicity (n = 35), 4,701 participants (80% of participants with available data) remained. Of these participants, 806 (19.8%) had an incident stroke during the mean 11.5 years of follow-up. These participants resided in 267 census tracts, for an average of 3 cases per census tract. This ratio did not differ appreciably by NSES quartile (table e-1 on the *Neurology*® Web site at www.neurology.org).

Among the 806 participants with an adjudicated incident stroke (82% ischemic, 11% hemorrhagic, and 7% unknown/other type) during the surveillance period, mortality was 20.0% at 30 days after stroke and 34.2% at 1 year. In bivariate analyses, for both the first 30 days and the first year after stroke, mortality was associated with hemorrhagic or other/unknown stroke type (relative to ischemic stroke), older age, and lower TC/HDL cholesterol ratio (table 1).

The Kaplan-Meier curves for survival after incident stroke at 30 days and 1 year show a separation of the higher SES neighborhoods from the lower SES neighborhoods (figure). The separation is significant at 1 year, with the highest mortality in the most disadvantaged neighborhoods (4th NSES quartile) and the lowest mortality in the least disadvantaged neighborhoods (1st and 2nd NSES quartiles).

At 30 days after the incident stroke, although the estimated hazard ratio (HR) for death was higher in the 4th NSES quartile (most disadvantaged neighborhoods) as compared to the 1st NSES quartile (the least disadvantaged neighborhoods), the association between NSES and mortality did not reach statistical significance in either the partial model (HR 1.43; 95% confidence interval [CI] 0.85–2.44) (data not shown) or the fully adjusted model (HR 1.53; 95% CI 0.89–2.62) (table 2). At 1 year after the incident stroke, however, residence in the most disadvantaged community was associated with a significantly higher poststroke mortality hazard, relative to the least disadvantaged community in the partial model (HR 1.70; 95% CI 1.14–2.54) and in the fully adjusted model (HR 1.77; 95% CI 1.17–2.68). Several other characteristics were associated with poststroke mortality in these models. Hemorrhagic stroke type and other/unknown stroke type, older age, and lower ratio of TC/HDL were associated with higher mortality at both 30 days and 1 year. Less physical activity was also associated with stroke mortality at 30 days, while a diagnosis of hypertension was also associated with mortality at 1 year. The only behavioral and biologic risk factors associated with higher stroke mortality was lower TC/HDL ratio (see table e-2). In the mediation analyses, we found no evidence of either full or partial mediation for the 30-day and 1-year models.

We also examined separate models that included interaction terms between NSES and participants' demographic characteristics (race, age, income, and education) and stroke type (ischemic, hemorrhagic, or unknown). None of the interaction terms was significant.

Our findings did not change appreciably in the sensitivity analyses. Incorporating dietary cereal fiber or salt intake into models restricted to white patients did not appreciably change the main findings. Use of baseline biologic measurements and the last observed biologic measurements produced comparable results; we present results using the last measurement available prior to the incident stroke.

**DISCUSSION** In this longitudinal study of the association between neighborhood context and mortality after incident stroke, we found higher mortality at 1 year among residents of the most socioeconomically disadvantaged census tracts relative to residents of the least disadvantaged census tracts. The association did not appear to be mediated by most traditional behavioral or biologic risk factors for stroke or poststroke mortality. Moreover, the associations did not differ by race, age, sex, income, or education.

Residence in a disadvantaged community may contribute to higher rates or greater severity of biologic stroke risk factors or higher frequency of deleterious behaviors, such as smoking or medication nonadherence, that, in turn, influence morality after stroke.[29] In this study, we did not find that behavioral and biological factors explained the association between living in a disadvantaged neighborhood and higher risk of mortality after stroke. However, considering that this risk persisted after adjusting for demographic factor and potential

**Table 1** Characteristics of Cardiovascular Health Study participants with incident stroke[a]

| | 30 Days after incident stroke | | 1 Year after incident stroke | |
|---|---|---|---|---|
| | Alive (n = 638) | Died (n = 168) | Alive (n = 530) | Died (n = 276) |
| Neighborhood SES | | | | |
| Q1 (Highest) | 151 (23.7) | 38 (22.6) | 130 (24.5) | 59 (21.4) |
| Q2 | 171 (26.8) | 38 (22.6) | 147 (27.7) | 62 (22.5) |
| Q3 | 158 (24.8) | 46 (27.4) | 129 (24.3) | 75 (27.2) |
| Q4 (Lowest) | 158 (24.8) | 46 (27.4) | 124 (23.4) | 80 (29.0) |
| Stroke type | | | | |
| Ischemic | 563 (88.2)[b] | 98 (58.3) | 471 (88.9)[b] | 190 (68.8) |
| Hemorrhagic | 41 (6.4)[b] | 50 (29.7) | 33 (6.2)[b] | 58 (21.1) |
| Unknown | 34 (5.3)[b] | 20 (11.9) | 26 (4.9)[b] | 28 (10.1) |
| Individual demographic characteristics | | | | |
| Age, y, mean (SD) | 73.3 (5.4)[b] | 74.7 (5.7) | 73.2 (5.4)[b] | 74.5 (5.6) |
| Female | 394 (61.8) | 103 (61.3) | 330 (62.3) | 167 (60.5) |
| White | 532 (83.4) | 142 (84.5) | 440 (83.0) | 234 (84.8) |
| Education | | | | |
| Less than high school | 198 (31.0) | 54 (32.1) | 163 (30.9) | 89 (32.3) |
| High school or GED | 195 (30.6) | 47 (28.0) | 158 (30.0) | 84 (30.4) |
| Some college | 139 (21.8) | 45 (26.8) | 120 (22.8) | 64 (23.2) |
| College graduate | 63 (9.9) | 9 (5.4) | 55 (10.4) | 17 (6.2) |
| Graduate/professional school | 40 (6.3) | 13 (7.7) | 31 (5.9) | 22 (8.0) |
| Income | | | | |
| Less than $12,000 | 177 (27.7) | 47 (28.0) | 147 (27.7) | 77 (27.9) |
| $12,000 to less than $25,000 | 230 (36.0) | 56 (33.3) | 191 (36.0) | 95 (34.4) |
| $25,000 to less than $35,000 | 80 (12.5) | 30 (17.9) | 69 (13.0) | 41 (14.9) |
| At least $35,000 | 114 (17.9) | 27 (16.1) | 91 (17.2) | 50 (18.1) |
| Missing income | 37 (5.8) | 8 (4.8) | 32 (6.0) | 13 (4.7) |
| Behaviors | | | | |
| Smoking status | | | | |
| Never smoked | 326 (51.1) | 79 (47.0) | 272 (51.3) | 133 (48.2) |
| Former smoker | 246 (38.6) | 69 (41.1) | 203 (38.3) | 112 (40.6) |
| Current smoker | 66 (10.3) | 20 (11.9) | 55 (10.4) | 31 (11.2) |
| Alcohol use | | | | |
| 0 drinks per week | 354 (55.5) | 84 (50.0) | 293 (55.4) | 145 (52.7) |
| 1–7 drinks per week | 211 (33.1) | 61 (36.3) | 177 (33.5) | 95 (34.6) |
| >7 drinks per week | 72 (11.3) | 22 (13.1) | 59 (11.2) | 35 (12.7) |
| Physical activity (kcal past 2 wk), mean (SD) | 1,691 (2,027) | 1,846 (1,976) | 1,698 (2,000) | 1,768 (2,050) |
| Biologic factors | | | | |
| Atrial fibrillation | 28 (4.4) | 7 (4.4) | 22 (4.2) | 13 (4.7) |
| TIA | 20 (3.1) | 5 (3.0) | 18 (3.4) | 7 (2.5) |
| Subclinical cardiovascular disease[c] | 135 (80.4) | 476 (74.6) | 395 (74.5) | 216 (78.3) |
| Hypertension[d] | 345 (54.1) | 98 (58.3) | 282 (53.2) | 161 (58.6) |
| Diabetes (ADA)[e] | 24 (14.9) | 123 (19.3) | 97 (18.6) | 51 (18.6) |
| Total/HDL ratio, mean (SD)[f] | 4.3 (1.3)[b] | 4.0 (1.2) | 4.3 (1.3)[b] | 4.1 (1.2) |

*Continued*

**Table 1** Continued

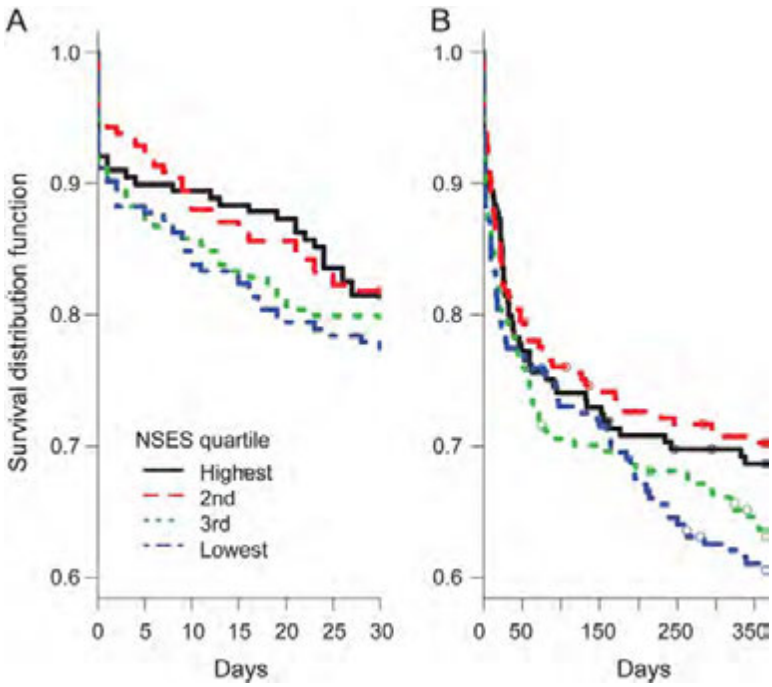
| | 30 Days after incident stroke | | 1 Year after incident stroke | |
|---|---|---|---|---|
| | Alive (n = 638) | Died (n = 168) | Alive (n = 530) | Died (n = 276) |
| Systolic BP, mean (SD)[f] | 142.3 (24.0) | 143.2 (22.9) | 143.0 (24.0) | 141.4 (23.4) |
| Diastolic BP, mean (SD)[f] | 71.2 (11.7) | 71.2 (13.9) | 71.6 (11.7) | 70.5 (13.0) |

Abbreviations: ADA = American Diabetes Association; BP = blood pressure; GED = General Educational Development; HDL = high-density lipoprotein; SES = socioeconomic status.
[a] Values represent number (%) unless indicated as mean (SD).
[b] $p < 0.05$ for difference comparing alive vs died.
[c] Ankle–arm index ≤0.9, carotid stenosis >25%, internal carotid thickness >80th percentile, major EKG abnormalities, abnormal ejection fraction or wall motion on echocardiogram, or claudication or angina on Rose Questionnaire.
[d] Hypertension = systolic blood pressure above 160 mm Hg, diastolic blood pressure above 95 mm Hg.
[e] Diabetes = fasting blood glucose above 126 mm/dL or diabetes diagnosis and diabetes medication.
[f] Last measurement available prior to stroke.

mediators, including individual-level SES, our findings suggest the factors unique to neighborhood characteristics may play a role in contributing to mortality risk within 1 year of stroke.

The difference in the association between NSES and stroke survival at 30 days and 1 year may point to mechanisms through which NSES influences stroke mortality. Stroke severity is the primary predictor of survival 1 month after stroke, and although predictive of longer term mortality, severity is of less importance in predicting 1-year survival.[23] These differences in risk factors for mortality after stroke suggest that NSES plays a less prominent role in the short term after stroke. Additional work, however, is needed to understand the relationship of NSES to other factors, such as the quality of prehospital and hospital care that may influence short-term stroke survival. Alternately, there are several mechanisms through which neighborhood factors may play an important role in longer term poststroke survival. Lower NSES communities may contribute to mortality if they have less supportive social environments with fewer formal and informal services for stroke survivors, or if they have fewer physical amenities (e.g., parks, other recreational facilities, or places to purchase healthy foods) that promote recommended poststroke behaviors. Additionally, health care system factors, including the availability and quality of primary care, specialty providers, rehabilitation facilities, and emergency services, may mediate the relationship between NSES and poststroke mortality. To date, the literature is scant on the contribution of the local and regional medical care infrastructure to neighborhood disparities in health. To address stroke disparities and improve population health, additional research is needed that integrates studies of neighborhood effects on health with work on the accessibility and quality of care to prevent and treat stroke.

**Figure** Kaplan-Meier curves of death after incident stroke in 806 Cardiovascular Health Study participants at (A) 30 days and (B) 1 year poststroke event




NSES = neighborhood socioeconomic status.

This study has several strengths. We have a large population-based cohort of older adults who were followed longitudinally for many years, and thus were able to assess a substantial number of incident strokes and subsequent poststroke events. We were also able to explore several behavioral and biologic mechanisms through which the neighborhood environment might be associated with poststroke mortality.

The study also has some potential limitations. Although participants were derived from a representative sample of Medicare enrollees in the counties studied, only 4 geographical areas in the United Sates were represented, and those who participated in the study were generally younger, were more educated, reported fewer chronic conditions, and had better health status than those who did not participate. Our analyses did not include a measure of stroke severity, which is an important predictor of mortality, particularly early after stroke[23,25]; however, we did include several CVD and

**Table 2** Significant multivariable risk factors for all-cause mortality at 30 days and 1 year after stroke[a]

| | 30-Day all-cause mortality | | 1-Year all-cause mortality | |
|---|---|---|---|---|
| | HR (95% CI) | *p* Value | HR (95% CI) | *p* Value |
| Neighborhood SES | | | | |
| Q1 (Highest) | 1.00 | — | 1.00 | — |
| Q2 | 1.05 (0.65–1.68) | 0.85 | 1.10 (0.76–1.60) | 0.61 |
| Q3 | 1.27 (0.79–2.05) | 0.33 | 1.43 (0.99–2.08) | 0.06 |
| Q4 (Lowest) | 1.53 (0.89–2.62) | 0.12 | 1.77 (1.17–2.68)[b] | 0.007[b] |
| Stroke type (ref: ischemic) | | | | |
| Ischemic | 1.00 | — | 1.00 | — |
| Hemorrhagic | 5.71 (3.94–8.27)[b] | <0.0001[b] | 4.11 (2.98–5.68)[b] | <0.0001[b] |
| Unknown | 2.96 (1.76–4.99)[b] | <0.0001[b] | 2.67 (1.77–4.03)[b] | <0.0001[b] |
| Individual demographic characteristics | | | | |
| Age (5-year intervals) | 1.39 (1.19–1.63)[b] | <0.0001[b] | 1.30 (1.15–1.46)[b] | <0.0001[b] |
| Behaviors | | | | |
| Physical activity, kcal | | | | |
| Q1 (lowest activity) (ref) | 1.00 | — | 1.00 | — |
| Q2 | 0.60 (0.38–0.94)[b] | 0.006[b] | 0.76 (0.53–1.10) | 0.14 |
| Q3 | 0.69 (0.44–1.10) | 0.12 | 0.92 (0.64–1.32) | 0.65 |
| Q4 (highest activity) | 0.53 (0.34–0.83)[b] | 0.03[b] | 0.73 (0.51–1.04) | 0.09 |
| Biologic factors | | | | |
| Hypertension[c] (ref: normal) | | | | |
| Borderline | 1.00 (0.59–1.70) | 0.99 | 1.03 (0.68–1.56) | 0.88 |
| Hypertensive | 1.30 (0.87–1.94) | 0.20 | 1.41 (1.03–1.92)[b] | 0.03[b] |
| Total/HDL ratio | 0.48 (0.28–0.84)[b] | 0.01[b] | 0.62 (0.41–0.96)[b] | 0.03[b] |

Abbreviations: CI = confidence interval; HDL = high-density lipoprotein; HR = hazard ratio; SES = socioeconomic status.
[a] Models are also adjusted for sex, race, education, income, smoking, alcohol use, diabetes, atrial fibrillation, TIA, and subclinical cardiovascular disease (ankle–arm index ≤0.9, carotid stenosis >25%, internal carotid thickness >80th percentile, major EKG abnormalities, abnormal ejection fraction or wall motion on echocardiogram, or claudication or angina on Rose Questionnaire).
[b] Significant.
[c] Hypertension categories: normal = normotensive; borderline = systolic blood pressure between 140 and 159 mm Hg or diastolic blood pressure between 90 and 94 mm Hg; hypertensive = systolic blood pressure above 160 mm Hg, diastolic blood pressure above 95 mm Hg.

non-CVD severity adjustors. We did not incorporate information on whether participants moved over the study period. However, residential mobility appears to be relatively low by age 35 and declines rapidly thereafter. Thus, using the characteristics of the neighborhood of residence at baseline for those who move is unlikely to bias the estimates that would be obtained if the characteristics of destination neighborhoods were available.[30–32] Another limitation of these analyses is that the CHS data included a relatively small number of African American subjects, and we excluded the few residents who were members of other racial and ethnic groups from these analyses. Nonetheless, among the white and African American subjects in this cohort, we found no interaction between race and NSES. Ideally, future studies of poststroke mortality should be conducted in racially and ethically varied communities. Finally, practice patterns for management of stroke and its risk factors have changed appreciably since the study's inception. Since diffusion of new therapies is often delayed to vulnerable individuals and communities, there is a strong possibility that the differences we observed may persist or be even more pronounced due to changes in clinical management.

There is increasing recognition of the role of community-level risk reduction in mitigating the risk and outcomes of CVD and stroke.[33] Additional research is needed to understand the structural and social exposures that may contribute to stroke in disadvantaged neighborhoods and to examine their relationships to the biologic, behavioral, and health care system influences that contribute to mortality in the year after a stroke. Additional work is also needed to understand how neighborhood exposures interact with individual characteristics, such as less education, low income, and low social support, to influence mortality and other outcomes after stroke. Ultimately it will be critical to incorporate this

knowledge into interventions to improve stroke outcomes at a population level.

## AUTHOR CONTRIBUTIONS

Dr. Brown: study supervision, study concept and design, analysis and interpretation, drafting/revising the manuscript. Dr. Liang: analysis and interpretation, drafting/revising the manuscript. S.D. Vassar: analysis and interpretation, drafting/revising the manuscript. Dr. Merkin: analysis and interpretation, drafting/revising the manuscript. Dr. Longstreth: analysis and interpretation, drafting/revising the manuscript. Dr. Ovbiagele: analysis and interpretation, drafting/revising the manuscript. Dr. Yan: analysis and interpretation, drafting/revising the manuscript. Dr. Escarce: study concept and design, analysis and interpretation, drafting/revising the manuscript.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## DISCLOSURE

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