HHS Public Access

Author manuscript

Circ Cardiovasc Qual Outcomes. Author manuscript; available in PMC 2018 February 22.

Published in final edited form as:

Circ Cardiovasc Qual Outcomes. 2017 February ; 10(2): . doi:10.1161/CIRCOUTCOMES.116.002547.

Neighborhood Differences in Post-Stroke Mortality

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Abstract

Background—Post-stroke mortality is higher among residents of disadvantaged neighborhoods, but it is not known whether neighborhood inequalities are specific to stroke survival or similar to mortality patterns in the general population. We hypothesized that neighborhood disadvantage would predict higher post-stroke mortality and neighborhood effects would be relatively larger for stroke patients than for individuals with no history of stroke.

Methods and Results—Health and Retirement Study participants aged 50+ without stroke at baseline (n=15,560) were followed up to 12 years for incident stroke (1,715 events over 159,286 person-years) and mortality (5,325 deaths). Baseline neighborhood characteristics included objective measures based on census tracts (family income, poverty, deprivation, residential stability, and percent white, black or foreign-born) and self-reported neighborhood social ties. Using Cox proportional hazard models, we compared neighborhood mortality effects for people with versus without a history of stroke. Most neighborhood variables predicted mortality for both stroke patients and the general population in demographic-adjusted models. Neighborhood percent white predicted lower mortality for stroke survivors (HR=0.75 for neighborhoods in highest 25th percentile vs. below, 95 % CI: 0.62, 0.91) more strongly than for stroke-free adults (HR=0.92 (0.83, 1.02); p=0.04 for stroke-by-neighborhood interaction). No other neighborhood characteristic had different effects for people with versus without stroke. Neighborhood-mortality associations emerged within three months after stroke, when associations were often stronger than among stroke-free individuals.

Conclusions—Neighborhood characteristics predict post-stroke mortality, but most effects are similar for individuals without stroke. Eliminating disparities in stroke survival may require addressing pathways that are not specific to traditional post-stroke care.

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Conflict of Interest Disclosures: None.

Keywords

stroke; socioeconomic position; mortality; community; psychology and behavior; neighborhood; social ties; social support; social networks; stroke incidence

Journal Subject Terms

Cerebrovascular Disease/Stroke; Epidemiology; Lifestyle; Primary Prevention; Secondary Prevention

Introduction

Despite declines in overall stroke mortality and case-fatality rates[1], improving long-term survival of stroke patients and eliminating racial, socioeconomic, and geographic disparities in stroke outcomes remains a major public health priority[2]. A growing body of evidence suggests that neighborhood context is associated with stroke incidence and mortality after stroke;[3–15] for example, not only does a patient's own socioeconomic position (SEP) predict higher mortality following stroke,[16] but so does the average SEP of his or her neighbors.

The association between neighborhood SEP and post-stroke mortality is not surprising because neighborhood SEP predicts mortality in the general population[17]. It is not known, however, whether the association between neighborhood SEP and mortality among stroke survivors is stronger, weaker, or similar to neighborhood-mortality associations that prevail in the general population. If the magnitudes of the neighborhood-mortality associations are comparable for stroke survivors and stroke-free adults, this may suggest shared pathways and intervention points. Neighborhood effects on post-stroke survival may be even stronger than neighborhood effects in the general population, however, due to disparities in access and quality of acute care, rehabilitation services, and post-stroke care pathways.

We hypothesized that lower neighborhood SEP, lower residential stability, higher concentrations of minorities, and weaker social ties would predict worse post-stroke survival, and that this mortality disadvantage would be stronger (worse) in relative terms for individuals who had survived a stroke compared to individuals with no history of stroke.

Materials and Methods

We used prospective cohort data from Health and Retirement Study (HRS)[18, 19] participants born 1900 to 1947 included in the 1998 assessment. Biennial interviews by telephone or in person, were conducted through 2010 (retention rates >80%). HRS was approved by the University of Michigan Health Sciences Human Subjects Committee and these analyses were approved by Harvard School of Public Health Office of Human Research Administration.

From the age-eligible sample (n=19,991), we excluded those with history of stroke at baseline (n=1,211) and with missing or implausible stroke date information (n=64). An additional 2,889 participants were excluded due to missing covariates for neighborhood

social ties (n=1,666), friend information (n=8), race (n=1), marital status (n=22), education (n=54), physical activity (n=9), functional impairment (n=27), body mass index (BMI) (n=229), alcohol intake (n=3), smoking (n=120), blood pressure (BP) (n=293), diabetes (n=85), self-reported health (n=5), Census tract (n=12), and residential stability (n=355). Additionally, 267 individuals were excluded due to loss to follow-up prior to first exposure wave, leaving a final analytic sample of 15,560. In supplemental analyses, we used multiple imputation to compare models retaining individuals with partial covariate missingness, resulting in an analytic sample of 17,960.

Outcome: All-cause mortality

Mortality was obtained via linkage to the National Death Index (NDI) through 2008. If NDI information was missing we used exit interview information from proxies.

Effect Modifier: Incident Stroke

We evaluated stroke as a modifier of the association between neighborhood and mortality. First stroke was based on time-updated self- or proxy-report of a doctor's diagnosis ("Has a doctor ever told you that you had a stroke?"). No information on transient ischemic attacks, stroke subtypes, or stroke severity was available. Interviews were conducted with proxy informants (<15%), predominantly spouses, for participants not available for direct interviews (e.g., due to death). Proxy interviews were included by design in HRS to avoid bias due to excluding respondents with low cognitive function or declining health; previous evaluations of HRS indicate that inclusion of proxy reports reduces bias due to attrition, and raises response rates.[20–22] This outcome was validated using respondents with data that could be linked to records from the Centers for Medicare and Medicaid Services (CMS) (n=6,223 aged 65+ not enrolled in Medicare Health Maintenance Organizations); the self/proxy reported stroke outcome had a sensitivity of 74% and specificity of 93% to detect strokes as recorded in CMS (or 79% sensitivity and 91% specificity for identifying strokes recorded as the primary diagnosis on CMS records). Sensitivity and specificity was similar across sociodemographic and health factors (see Appendix Table 1). Respondents reported month and year of stroke diagnosis, used to calculate time since stroke in secondary analyses. We classified mortality of stroke patients based on time since stroke: less than 3 months, 3–12 months, and more than 12 months (compared to those who did not experience stroke).

Exposure: Neighborhood Environment

We considered three domains of neighborhood measures: social ties to neighbors, neighborhood SEP, and neighborhood demographic composition.

Social ties to neighbors were assessed based on presence of friends and (separately) relatives via this item "Do you have any close friends (relatives) in the neighborhood?" Neighborhood-based social interactions were assessed by two items: "Do you get together with any of your neighbors for social reasons?" and "How often do you get together with neighbors per month?" These two items were combined and dichotomized at one or more times/month (versus zero). We then created an index by averaging these three dichotomous

variables (each coded 0, 1), for an index ranging 0–1, with higher values denoting better social integration.

We geocoded participants' 1998 addresses and linked to 1990 Census tract data for the remainder of neighborhood variables described below. Using census tracts to proxy for neighborhood definitions is common and valid, since tracts correspond roughly to a spatial unit of a neighborhood.[23–25] We chose the functional form of the variables (i.e., quartiles vs. binary breaks) based on preliminary bivariate associations. Neighborhood SEP was measured as average tract family income (in quartiles, modeled ordinally), % of residents below the poverty line (dichotomized at the sample's 75th percentile, above 17.7% poor), and an index of deprivation (in quartiles, modeled ordinally). We derived a deprivation score from a principal components analysis of five census-based deprivation variables including % households in poverty, % unemployed civilians aged 16+, % households receiving public assistance, % female-headed households with children, and % persons aged 25+ with less than a high school education[26, 27].

Finally, we examined Census-tract measures of neighborhood demographics: % residents who identified as non-Hispanic (NH) black (dichotomized at sample's 75th percentile: 12.7%), % NH white (dichotomized at sample's 25th percentile: 61.3%), % foreign born (dichotomized at sample's 95th percentile, 23.0%). Neighborhood residential stability was defined as % of residents living at the same address 5 years ago (dichotomized at sample's 25th percentile: 44.7%). To avoid bias if stroke caused individuals to move to different types of neighborhoods, we did not time-update neighborhood characteristics; all are based on 1998 residence, when everyone was stroke-free.

Covariates

All covariates are measured at the individual level, and reported prospectively in the 1998 (our baseline) survey. *Demographic variables* included race/ethnicity (non-Hispanic white, non-Hispanic black, Latino/Hispanic, or non-Hispanic other), baseline age, gender, birth in a southern state, marital status, and nativity. *Individual-level SEP* was measured by self-reported own years of completed education, parental education, self-reported household income and (separately) household wealth in 1998. Income and wealth were equivalized for household size. *Behavioral risk factors* included smoking status; vigorous physical activity; and weekly alcohol use. *Health conditions* included BMI and self-rated health. *Comorbidities/chronic health problems* included self-reported diagnoses of diabetes and (separately) of hypertension; elevated depressive symptoms (measured by a modified 8-item Center for Epidemiological Studies Depression (CES-D) Scale, modeled as binary, <3 vs. ≥3)[28]; limitations in activities of daily living (ADLs: needing help to get across a room, dress, bathe, eat, get in and out of bed, or use the toilet) and, separately, instrumental activities of daily living (IADLs : needing help to prepare meals, make telephone calls, shop for groceries, or take medications), each recoded as any vs. none. See Table 1 for additional coding detail.

Analyses

We applied Cox proportional hazard survival regression models for the outcome of death, measured by continuous failure time as date of death, or right censoring as the last contact date before loss to follow-up, or the 2010 survey. We estimated several sets of models; each neighborhood variable was always modeled one at a time. The first set of models tested the main effects of neighborhood context on mortality, first adjusted for stroke and demographic covariates (Model 1); in Model 2 we then added CVD risk factors including health behaviors (physical activity, alcohol use, tobacco use), health conditions (obesity, self-rated health), and comorbidities/chronic health problems (depressive symptoms, hypertension, diabetes, functional impairment (ADL, IADL)), in addition to demographics and stroke, but not individual-level SEP. Model 3 built on Model 1 to add individual-level SEP, in addition to stroke and demographics, but not CVD risk factors. Model 4 included all covariates simultaneously (stroke, demographic, health behaviors, health conditions, and comorbidities/chronic health problems, individual SEP). Extensive evidence suggests that neighborhood disadvantage influences health behaviors and comorbid conditions, so we consider models adjusted for these covariates to underestimate the total effects of neighborhood on mortality.

A second set of models estimated a covariate-adjusted association between incident stroke and mortality excluding neighborhood variables, using the same model-building strategy above (Models 1 and 4) (reported in the text).

The third set of models tested our primary hypothesis of equivalent effects for stroke patients and stroke-free individuals by specifying a stroke-neighborhood interaction predicting mortality, adjusted for demographic covariates (Model 1) and for all covariates (Model 4). We present the p-value from those interaction tests and effect estimates (and 95 % CI) from pooled interaction models of neighborhood associations with mortality for people with and people without history of stroke.

To test whether associations between neighborhood environment and mortality depended on time since stroke, we interacted neighborhood with time since stroke indicator variables, and report associations of neighborhood on mortality within each time since stroke stratum.

We confirmed the proportional hazards assumptions held for the main effects models and directly evaluated heterogeneity in effects for time since stroke models. We used SAS 9.3 (Cary, NC) PROC PHREG and accounted for clustering of individuals in tracts using robust sandwich estimators[29]. We applied HRS sampling weights to render the sample representative of the 1998 US population aged 50+ years. We estimated a subset of our models using multiple imputation to retain individuals with partially missing data and found substantively identical results.

Results

In our sample (N=15,560), 1,715 participants (11.0%) experienced stroke, and 5,325 participants died (34.2%), from 1998–2010. Mean follow-up time was 10.2 years, and the cohort accrued 159,286 person years of follow-up (Table 1).

Main Effects of Stroke on Mortality

After adjustment for baseline demographic covariates, respondents who had ever experienced a stroke had 2-fold higher mortality risk, hazard ratio (HR)=2.17 (95 % CI: 2.00, 2.36). This association declined to 1.90 (95 % CI: 1.74, 2.08) after adjustment for all covariates. These associations were consistent regardless of the neighborhood variable modeled.

Main Effects of Neighborhood Context on Mortality

Figure 1 and Appendix Table 2 present the mortality HRs associated with neighborhood characteristics, adjusted for stroke and covariates, for the entire follow-up, pooled across stroke status. We found no statistically-significant evidence of proportional hazards violations in any model; p>0.15 for all tests. After demographic adjustment (Model 1), neighborhood social ties, higher neighborhood family income, and high neighborhood % white all predicted lower mortality. For example, those living in the highest (best) neighborhood family income quartile (Quartile 4) experienced 24% lower mortality (HR=0.76, 95 % CI: 0.70, 0.83), than those living in the lowest neighborhood family income quartile (Quartile 1)(p for trend <0.0001). As hypothesized, participants living in higher neighborhood deprivation and higher neighborhood poverty had significantly higher mortality risk.

Higher neighborhood family income predicted lower mortality even after adjusting for CVD risk factors (Model 2, HR for highest vs. lowest quartile neighborhood income=0.88, 95 %CI: 0.80, 0.96; p for linear trend=0.005). Other neighborhood SEP variables no longer significantly predicted mortality in Model 2, and mortality associations with all three neighborhood SEP variables were also attenuated after adjusting for individual-level SEP (Models 3, 4). However, neighborhood social ties (HR=0.86, 95 % CI 0.78, 0.96) and neighborhoods with high proportions of immigrants (HR=0.83, 95 % CI: 0.71, 0.97) significantly predicted lower mortality after adjustment for stroke, demographics, CVD risk factors, and individual SEP (Figure 1, Model 4).

Effect Modification of Neighborhood Context-Mortality Association by Stroke

Neighborhood social ties predicted significantly lower mortality for stroke patients (HR=0.76, 95 % CI: 0.59, 0.99) as well as for individuals with no history of stroke (HR=0.74, 95 % CI: 0.67, 0.83) after demographic adjustment; effect estimates were statistically comparable (stroke interaction with neighborhood social ties p=0.87) (Table 2, Model 1). Likewise, there were significant protective effects of neighborhood family income on mortality after demographic adjustment (Model 1, comparing 4^{th} to 1^{st} quartile), among both stroke-free (Model 1 HR=0.70, 95 % CI: 0.58, 0.85) and stroke populations (Model 1 HR=0.77, 95 % CI: 0.70, 0.85); effects were homogeneous by stroke (interaction p=0.36). Significantly harmful patterns were observed for other measures of neighborhood SEP (neighborhood poverty; deprivation) in Model 1, again with similar patterns by stroke. However, these mortality-neighborhood SEP associations were attenuated after adjustment for individual-level SEP (Table 2, Model 4).

We found few significant associations of neighborhood characteristics on mortality that were different by stroke status subgroups (our key hypothesis, Table 2); results were very similar when based on multiply imputed data (Appendix Table 3). Living in a predominantly white neighborhood, however, was associated with substantially better survival among stroke patients in demographically-adjusted models (HR mortality=0.75, 95 % CI: 0.62, 0.91), but not among stroke-free populations (HR=0.92, 95 % CI: 0.83, 1.02; interaction p=0.04; Table 2 Model 1). Associations changed little after comprehensive adjustment in Model 4 (HR mortality for stroke patients=0.80, 95 % CI: 0.66, 0.96; HR mortality among stroke-free populations =1.01, 95 % CI: 0.91, 1.11; interaction p=0.02).

Time Since Stroke

Estimated effects of neighborhoods on mortality were often different in the short term (stroke occurred <3 months from last contact) compared to those never experiencing stroke. Figure 2 demonstrates that neighborhood deprivation had adverse associations with mortality for recent stroke patients (Figure 2, Appendix Table 4: HR=1.35, 95 %CI: 1.03, 1.77; interaction vs. never stroke: p=0.02), with no effect for other stroke subgroups. Neighborhood % white was protective for recent stroke patients (HR=0.54, 95 %CI: 0.26, 1.11, interaction p=0.09), but the magnitude was less protective or null for other groups. Unexpectedly, recent stroke patients experienced adverse effects of more neighborhood social ties on mortality (HR=2.90, 95 %CI: 0.97, 8.61), while the association was significantly protective for the non-stroke population (HR=0.85, 95 %CI: 0.76–0.95; interaction p=.03). For follow-up periods 3 months or greater, there were few statistical differences for stroke, although estimates were imprecise. Results were similar when estimated in multiply imputed data sets (Appendix Table 5), although the unexpected adverse association between neighborhood social ties and mortality within 3 months after stroke was attenuated (HR=1.74, 95 % CI: 0.59, 5.16; interaction p=.31).

Discussion

In this nationally representative cohort, we found that several aspects of neighborhood context predicted better survival. Higher neighborhood SEP, living in a predominantly white neighborhood, and sharing social ties with neighbors predicted better survival for stroke patients, in partial support of our hypothesis. However, these associations were also present, and similar in relative magnitude, for those who never experienced a stroke, contrary to our hypothesis that associations would be larger in stroke survivors. Only the estimated effect of neighborhood racial composition (specifically, percent white) appeared specific to stroke survivorship. Although relative effects (e.g., ratios of mortality rates, such as hazard ratios) are similar, the absolute impact of neighborhood characteristics on mortality would be larger in stroke patients because they have higher underlying mortality.

Our findings complement a growing body of literature linking neighborhood disadvantage to shorter survival after stroke[3, 12–16] or after acute cardiovascular events[15, 27, 30–36]. Guidance on how to interpret and respond effectively to these disparities represents a major gap in previous literature. Our research attempts to address this gap by including individuals both with and without prior acute events, to evaluate whether inequalities were specific to post-

event care. Our findings have three important implications for stroke care. Several neighborhood factors are strong predictors of mortality among stroke patients; addressing these inequalities will probably require looking beyond conventional stroke care. For at least one domain (low neighborhood percent white), there may be mechanisms that are specifically detrimental to stroke survivors, and these mechanisms are relevant from the first months after stroke (particularly for neighborhoods that are deprived and low percent white). These neighborhood characteristics are presumably not causal, but proxies for other underlying neighborhood risk factors, such as access to high-quality acute or long-term stroke care (e.g., residential segregation of nursing homes)[37, 38]. Access to high-quality care is patterned by location, and may be driven by availability of specialized services in more affluent urban neighborhoods[13]. Therefore, interventions deriving from these findings might focus on ensuring access to high-quality care in the immediate aftermath of stroke, particularly for those living in racially segregated or deprived neighborhoods, with close follow-up soon after stroke. Our data on time of death were not sufficiently precise to evaluate whether early mortality was due to in-hospital deaths or mortality after discharge. Other data sources, such as the "Get With the Guidelines" stroke database[39] might support such analyses. However since many of the mortality associations were evident in both stroke patients and stroke-free populations, our results also point to the need to address social determinants of health in poorer-quality neighborhoods that may underlie vulnerability to mortality risk, for example community outreach to elders to prevent social isolation and provide both instrumental and emotional social support.

We documented that those with better neighborhood-based social ties exhibited lower risk of mortality, after comprehensive adjustment. This is consistent with prior findings that social isolation, social support, and social cohesion are associated with stroke outcomes.[10, 40–42] Our results extend these previous findings, suggesting the possibility of the specific relevance of ties to neighbors, by examining both stroke patients and stroke-free populations.

Lower neighborhood SEP predicted higher mortality rates in our study, although this association was substantially attenuated by careful control for individual-level SEP, which is generally in contrast with prior studies[3, 11–15]. This discrepancy with previous reports may be due to availability of unusually comprehensive measures of individual SEP available in our cohort. Prior reports of significant effects of neighborhood SEP on survival after stroke[12–14] may have attributed some individual-level SEP effects[16] to neighborhood characteristics[17, 43]. Our study better controls for individual SEP than any prior study on the topic. Prior studies have used medical records, which typically include *no* measures of individual SEP[12–14]. Notably, in models controlling only for demographic variables, all our indicators of neighborhood SEP were strongly associated with mortality, suggesting that studies not including individual SEP were likely picking up the strong association between individual SEP and mortality in the neighborhood SEP coefficients.

Neighborhood context may influence survival[17] via mechanisms related to both neighborhood SEP and neighborhood social context, such as receipt of social support; exposure to violence; physical environments that influence health behaviors like exercise; support for chronic disease management; and access to acute care and clinical services to manage comorbid conditions or to aid rehabilitation (see conceptualization of possible

mechanisms in Figure 3). Given the controversy about whether neighborhood effect models should be adjusted for individual SEP, with many arguing that individual SEP is a mediator of neighborhood effects on health,[17] we view the best estimates of effects of neighborhoods as falling somewhere between demographic-adjusted and SEP-adjusted models.

Although models examining time since stroke had less statistical power, we documented that recent stroke patients displayed elevated mortality in deprived neighborhoods, while nonstroke populations and other stroke patients had no such mortality association. These results suggest that harmful risks present in impoverished neighborhoods, whether health care-related (e.g., proximity to high-quality treatment facilities or response time by health care professionals), or not (e.g., stress from exposure to crime and violence in high-poverty areas) may interact with the physiological vulnerability of the recent stroke patient to exacerbate mortality risk. Unexpectedly, recent stroke patients also exhibited elevated risk of death in neighborhoods with higher neighborhood social ties, while nonstroke populations exhibited protective effects. Although we can speculate on potential explanations for this pattern including potential negative consequences of social ties,[44] we consider it likely to be a spurious association given that it did not hold up when evaluated in multiple imputed data sets.

Strengths and Limitations

Like many other studies,[10, 11, 13] HRS did not include measures of stroke severity. Since stroke severity is a strong predictor of mortality following a stroke, particularly soon after,[45] our associations of neighborhood context on mortality among recent stroke patients may reflect severity. However prior work suggests that stroke severity may not be influential in accounting for neighborhood associations with mortality.[12, 13] We adjusted for numerous measures of baseline health and frailty, far more than available in prior research in this area. HRS includes only self- or proxy-reported measures of stroke, which is a good but not perfect measure of clinical stroke and inevitably misses undiagnosed ischemic cerebrovascular injury[46]. However, we found that these reports had 74% sensitivity and 93% specificity for stroke diagnoses reported in Medicare billing records, demonstrating that HRS measures of self or proxy reports have good validity.

Although we did not model cause-specific mortality due to misclassification on death certificates[47], by calculating the attributable risk percent in the exposed, we find that the majority (54%) of the deaths among stroke patients were directly attributable to stroke.

As discussed above, causality remains uncertain in this observational study: we may have omitted important confounders or adjusted for factors on the causal chain. This causal inference challenge is unlikely to account for our finding that neighborhood-mortality associations are similar by stroke status. For example, we chose to model neighborhood context at baseline, to establish temporal order of neighborhood context prior to stroke or mortality. While neighborhood context after baseline, including after stroke, may be etiologically relevant, it is on the causal chain between baseline neighborhood and mortality and may be affected by the patient's level of impairment after stroke. In other words, the most impaired patients may be differentially moved to disadvantaged neighborhoods or neighborhoods where they have no social contacts, creating a spurious association between

neighborhood characteristics and post-stroke mortality. However, not accounting for such changes in neighborhood prior to stroke may also bias results if current neighborhood of residence is most relevant to mortality, although such bias may be minimal since residential mobility for elders is relatively low compared to younger populations.[48]

Since eligibility to enroll in HRS was restricted to those aged 50+, those who did not survive to age 50 or who had a stroke prior to 1998 were excluded. Strokes are rare below age 50 however,[49, 50] so such a selection is unlikely to introduce substantial bias. Nonetheless, there may be differential associations of neighborhood context with mortality among younger populations and this is an important topic for future research.

This study has several unique strengths and adds substantively to prior literature in this area, and to our conceptual understanding of the determinants of post-stroke mortality. This is one of few nationally representative cohorts with sufficient power to model effects of stroke on mortality. We avoid selection bias that may be present in hospital-based studies.[12] Moreover, studies based on administrative data sources would not include such detailed demographic, socioeconomic, and social variables available in HRS. These covariates, especially SEP, are important in order to evaluate whether neighborhoods *per se* have relevance beyond individual SEP for post-stroke outcomes. By including individuals with and without stroke, we were able to assess whether the neighborhood effects were most likely indicating mechanisms specific to stroke care.

Conclusions

Neighborhood disadvantage, racial composition, and social ties predict survival of stroke patients. Most characteristics of neighborhoods have similar estimated effects on stroke survivors and individuals never having a stroke. Many important pathways linking neighborhoods and post-stroke mortality are therefore likely not specific to conventional stroke care, but may include general mortality risk factors, including social determinants of health. These results represent an opportunity to improve long-term survival of stroke patients by identifying specific mechanisms accounting for geographic inequalities in mortality.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

We gratefully acknowledge assistance from Anna Kosheleva on validation of the self-reported stroke measures.

Funding Sources: Dr. Osypuk was supported by NIH grants R01MD006064, R03HD080848, R21HD066312. Dr. Glymour was supported by the American Heart Association 10SDG2640243 and National Institute on Aging R21AG34385. Dr. Gilsanz was supported by the National Heart, Lung, and Blood Institute F31HL112613, and the National Institute on Aging T32AG049663. Ms. Ehntholt was supported by the American Heart Association 10SDG2640243.

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What is known

- Post-stroke mortality is higher among residents of disadvantaged neighborhoods, but it is not known whether neighborhood inequalities are specific to stroke survival or similar to mortality patterns in the general population.
- Most studies examining how neighborhood characteristics influence mortality among stroke survivors do not include a comparison group of those who never experienced stroke.

What the study adds

- Neighborhood percent white predicted mortality for stroke survivors more strongly than for stroke-free adults, which may signal underlying risk factors, such as access to high-quality acute or long-term stroke care.
- Neighborhood-mortality associations emerged within three months after stroke, when associations were often stronger than among stroke-free individuals.
- This study found that characteristics of the neighborhoods where stroke patients reside predict post-stroke mortality over 12 years of follow up, but estimated effects of most neighborhood characteristics are similar for individuals without stroke**;** addressing these neighborhood-mortality inequalities will likely require looking beyond conventional stroke care.

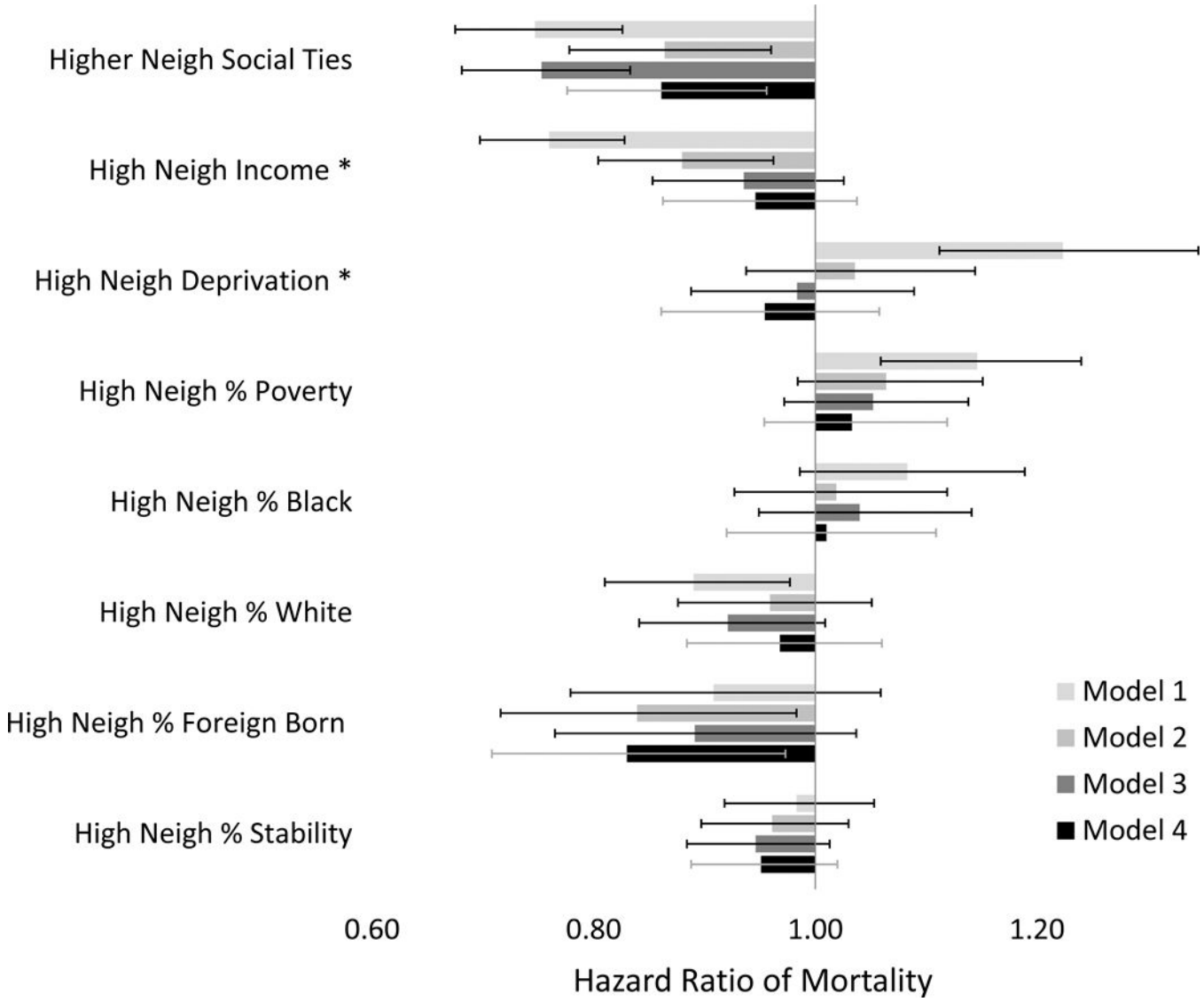

Figure 1. Main Effects of Neighborhood Context on Hazard Ratio of Mortality

Estimates, confidence intervals, and p-values reported in Appendix Table 2. All models adjusted for stroke status. Model 1 adjusted for demographic variables. Model 2 adjusted for demographics plus CVD risk factors. Model 3 adjusted for demographics plus SEP variables. Model 4 adjusted for demographic, CVD, and SEP variables. * Neighborhood family income and neighborhood deprivation are modeled in quartiles modeled ordinally; hazard ratio models a change from 4th vs. 1st quartiles.

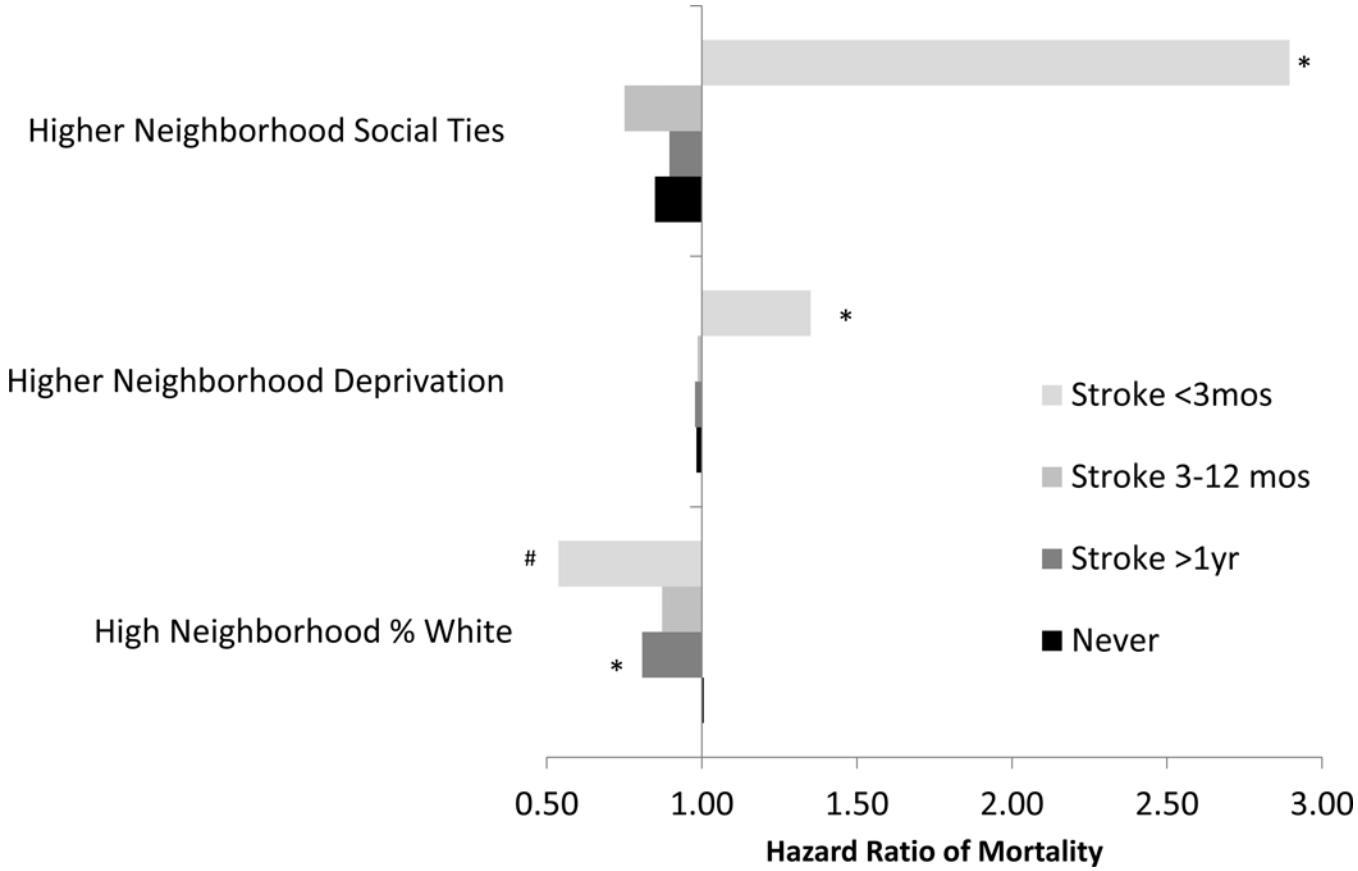

Figure 2. Stratum-Specific Estimates of Neighborhood Context on Hazard Ratios of Mortality within Strata of Time Since Stroke

* P<.05 # P<.10, where P denotes the P-value for the interaction (compared to never stroke). All Interaction P-values, and stratum specific estimates and confidence intervals for all neighborhood variables are reported in Appendix Table 4. Model 4 adjusted for demographic, CVD risk factors, and SEP variables, in addition to modeling the effect modification of neighborhood context with stroke status.

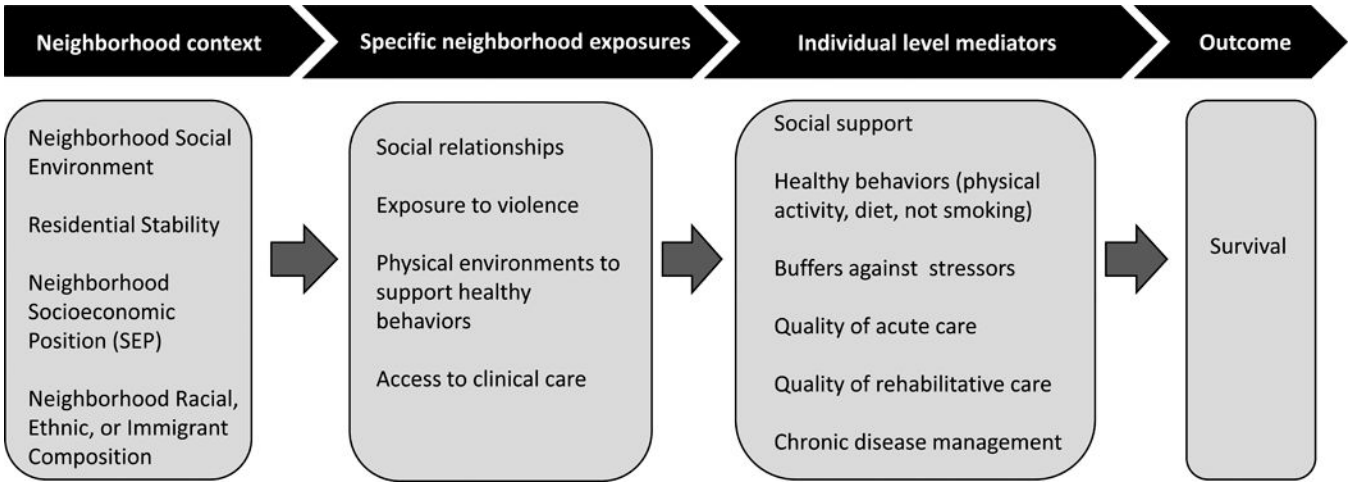

Figure 3.
Potential mechanisms linking neighborhood context to survival.

Table 1

Baseline Sample Characteristics and Incident Disease: HRS 1998 (Unweighted).

	n/mean/median	%/SD/Q
Total (n, %)	15,560	100%
Incident stroke by 2010 (n,%)	1,715	11.0%
Mortality by 2010 (n, %)	5,325	34.2%
Years of follow-up 1998–2010 (mean)	10.2	
Total person-years of follow-up 1998–2010 (sum)	159,286	
Demographic Variables		
Age in 1998 (mean, SD)	66.2	10.0
Male (n, %)	6,700	43.1%
Race/Ethnicity (n, %)		
Non-Hispanic White (n, %)	12,002	77.1%
Non-Hispanic Black (n, %)	2,160	13.9%
Hispanic (n, %)	1,097	7.1%
Non-Hispanic Other * (n, %)	301	1.9%
Married (vs. widowed/divorced/never married) (n, %)	10,584	68.0%
Foreign-born (n, %)	1,355	8.7%
Southern birth state (n, %)	5,534	35.6%
Socioeconomic Variables		
Parental education † ≥ 8 yrs (n, %)	10,905	70.1%
Years of education attained (mean, SD)	12.1	3.2
Equivalized household income ‡ (mean, SD)	$34,813	$54,429
Equivalized household wealth ‡ (mean, SD)	$232,184	$890,808
Behavioral Risk Factors		
Vigorous Physical Activity (≥3 times/wk) (n, %)	6,973	44.8%
Body Mass Index (BMI)		
Normal weight (BMI<25) (n, %)	5,897	37.9%
Overweight (25 <=BMI< 30) (n, %)	6,106	39.2%
Obese (BMI >= 30) (n, %)	3,557	22.9%
Alcohol use (past week)		
No alcohol use (n, %)	10,687	68.7%
Moderate alcohol use (1–2 days drinking) (n, %)	2,322	14.9%
Heavy alcohol use (≥3 days drinking) (n, %)	2,551	16.4%
Tobacco Use		
Never smoker (n, %)	6,373	41.0%
Current Smoker (n, %)	2,582	16.6%
Former Smoker (n, %)	6,605	42.5%
Activities of Daily Living (≥1 limitation) (n, %)	2,177	14.0%

	n/mean/median	%/SD/Q
Instrumental Activities of Daily Living (≥1 limitation) (n, %)	1,801	11.6%
Chronic Conditions		
CES-D Depressive Symptom Score (mean, SD)	1.5	1.8
Fair/Poor (v.s. excellent/very good/good) Self-Assessed Health (n, %)	4,453	28.6%
Hypertension (n, %)	7,231	46.5%
Diabetes (n, %)	2,135	13.7%
Neighborhood Variables		
1990 Census Variables		
neighborhood deprivation score (1990)	−0.013	0.91
neighborhood family income (1990) (mean, SD)	$42,476	$20,999
neighborhood % poverty (mean, SD)	13.4%	11.8
neighborhood % black (mean, SD)	13.9%	25.4
neighborhood % non-hispanic white (mean, SD)	74.1%	30.6
neighborhood % foreign-born (mean, SD)	6.3%	9.3
neighborhood % residential stability (5+ years) (mean, SD)	52.3%	12.1
75th percentile neighborhood % poverty (>17.7%) (n)	3897	
75th percentile neighborhood % black (> 12.7%) (n)	3883	
75th percentile neighborhood % white (> 61.3%) (n)	3890	
95th percentile neighborhood % foreign born (> 23.0%) (n)	778	
25th percentile neighborhood % residential stability (> 44.7%) (n)	11670	
Survey Based Variables		
Any Relatives in the neighborhood (n, %)	4,654	29.9%
Any Close friends in the neighborhood (n, %)	11,061	71.1%
Any Monthly contact with neighbors (n, %)	11,759	75.6%
3-Item Neighborhood Social Ties Index (mean, SD)	0.59	0.30

Notes: Baseline defined in 1998 for this analytic sample. All variables except stroke were defined in 1998. Sample members had never experienced stroke at baseline (1998). SD = standard deviation; Q1 = first quartile, Q3 = 3rd quartile, CES-D=Center for Epidemiologic Studies Depression Scale.

*Non-Hispanic other race/ethnicity was combined with the Non-Hispanic White group in regression models, due to small sample size.

†Parental education modeled as the highest education of the two parents.

‡Income and wealth were equivalized by dividing by the square root of the number of household members.

Table 2

Stratum-Specific Estimates of Neighborhood Context on Survival (Hazard Ratios of Mortality) within Strata of Ever-Stroke Status.

	Model 1						Model 4					
	Ever Stroke			Never Stroke		Inter-action	Ever Stroke			Never Stroke		Inter-action
	HR	95 % CI		HR	95 % CI	p	HR	95 % CI		HR	95 % CI	p
Higher Neighborhood Social Ties	0.76	(0.59	,0.99)	0.74	(0.67 ,0.83)	0.87	0.93	(0.71	,1.21)	0.85	(0.76 ,0.95)	0.53
Higher Neighborhood Family Income (ordinal, 4th vs. 1st Q)	0.70	(0.58	,0.85)	0.77	(0.70 ,0.85)	0.36	0.89	(0.72	,1.10)	0.96	(0.87 ,1.06)	0.54
Higher Neighborhood Deprivation (ordinal, 4th vs. 1st Q)	1.22	(0.99	,1.50)	1.22	(1.11 ,1.36)	0.96	0.98	(0.79	,1.23)	0.95	(0.85 ,1.06)	0.76
High Neighborhood % Poverty	1.19	(1.01	,1.41)	1.14	(1.04 ,1.24)	0.60	1.09	(0.92	,1.29)	1.02	(0.94 ,1.11)	0.50
High Neighborhood % Black	1.17	(0.97	,1.40)	1.07	(0.96 ,1.18)	0.38	1.10	(0.91	,1.32)	0.99	(0.90 ,1.10)	0.29
High Neighborhood % White	0.75	(0.62	,0.91)	0.92	(0.83 ,1.02)	0.04	0.80	(0.66	,0.96)	1.01	(0.91 ,1.11)	0.02
High Neighborhood % Foreign Born	1.13	(0.78	,1.65)	0.87	(0.74 ,1.03)	0.21	1.11	(0.76	,1.64)	0.79	(0.66 ,0.94)	0.12
High Neighborhood % Residentially Stable	0.94	(0.81	,1.10)	0.99	(0.92 ,1.07)	0.57	0.97	(0.82	,1.16)	0.95	(0.88 ,1.02)	0.79

Stratum-specific neighborhood-mortality estimates within strata of ever-stroke status derived from interaction models (interacting neighborhood context variable with stroke status). Model 1 adjusted for demographic variables (stroke status, age, gender, race, ethnicity, southern birth, nativity, and marital status). Model 4 additionally adjusted for SES variables (parental education, education, income, and wealth), and CVD risk factors (physical activity, ADL, IADL, obesity, alcohol use, smoking status, depressive symptoms, hypertension, diabetes, self-rated health). Q=Quartile. Neighborhood social ties modeled with a 3-item index; hazard ratio models a change from 0 to 3 social ties. Neighborhood family income and neighborhood deprivation are modeled in quartiles modeled ordinally; hazard ratio models a change from 4th vs. 1st quartiles.