RESIDENTIAL ETHNIC SEGREGATION AND STROKE RISK IN MEXICAN AMERICANS: THE BRAIN ATTACK SURVEILLANCE IN CORPUS CHRISTI PROJECT

Objective: Residential ethnic segregation may operate through multiple mechanisms to increase stroke risk. The current study evaluated if residential ethnic segregation was associated with stroke risk in a bi-ethnic population.

Design: Incident strokes were identified in Nueces County, Texas from 2000 to 2010. Residential ethnic segregation (range: 0-1) was derived for each census tract in the county (n=64) using 2000 US Census data, and categorized into: predominantly non-Hispanic White (NHW, <.3); ethnically mixed (.3–.7); predominantly Mexican American (MA, >.7). Multilevel Poisson regression models were fitted separately for NHWs and MAs to assess the association between residential ethnic segregation (predominantly NHW referent) and relative risk for stroke, adjusted for age category, sex and census tract-level median per capita income. Effect modification by age was also examined.

Results: In adjusted models, residential ethnic segregation was not associated with stroke risk in either ethnic group. Effect modification by age was significant in both groups. Young MAs and NHWs living in predominantly MA census tracts were at greater relative risk for stroke than those living in predominantly NHW census tracts, but this association was only significant for MAs (MAs: RR = 2.38 [95% CI: 1.31-4.31]; NHWs: RR = 1.53 [95% CI: .92-2.52]).

Conclusion: Our findings demonstrate that residential ethnic segregation may influence downstream stroke risk in young MAs. Pathways between residential ethnic segregation and stroke in young MAs should be explored. (*Ethn Dis.* 2015;25[1]:11–18)

Key Words: Stroke, Epidemiology, Risk Factors, Segregation

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INTRODUCTION

Stroke is the fourth leading cause of mortality in the United States and a leading cause of long-term disability.¹ In 2013, it was estimated that every year 610,000 individuals experience their first stroke, which costs the United States \$36.5 billion in direct and indirect costs.¹ Stroke incidence is higher among minority populations in the United States, including Mexican Americans (MAs).²

Previous research has shown that known behavioral and biological risk factors have a similar effect on the risk of stroke in MAs as compared to non-Hispanic whites (NHWs).³ However, several stroke risk factors are more prevalent among MAs. Specifically, MAs have a higher prevalence of diabetes, lower socioeconomic status, and limited access to health care services compared to NHWs.⁴⁻⁸ In contrast, hypertension prevalence is similar between MAs and NHWs, and atrial fibrillation is more prevalent in NHWs.¹ Currently identified biological and behavioral risk factors likely only partially explain disparities in stroke risk between NHWs and MAs.³

Recently, researchers have focused on how neighborhoods shape health because of a growing sense that individual-based explanations of poor health fail to capture underlying disease mechanisms that may contribute to health disparities.^{9,10} Studies have shown that even after controlling for individuallevel socioeconomic status, neighborhood characteristics are associated with exposure to risk factors and a range of health outcomes.^{10,11} Specifically, residing in disadvantaged neighborhoods has been associated with increased coronary heart disease incidence, cardiovascular disease mortality, and ischemic stroke risk.^{12–14} These findings raise the hypothesis that characteristics of neighborhoods influence health, including stroke, beyond the characteristics of the individual. Several mechanisms by which neighborhoods may affect health have been proposed.

Residential racial-ethnic segregation is the degree to which two or more groups live separately from one another in the urban environment.¹⁵ Housing discrimination, limited social and economic capital, and preference to live in neighborhoods with a similar ethnic group, or avoid another ethnic group all play a role in neighborhood selection.^{9,16} These sorting mechanisms have produced segregated neighborhoods composed of predominantly racial-ethnic minorities across the United States that are often disadvantaged; with a detrimental physical and social environment that may negatively impact health.9

Measures of residential racial-ethnic segregation, usually measured at the census tract or metropolitan level, may provide information about living conditions associated with stroke risk not captured by individual-level variables.¹⁷ For example, higher rates of violence and crime, lack of opportunity or upward mobility, social disorganization, low neighborhood trust and habitual illness are forms of chronic stress associated with segregated neighborhoods.^{9,18-23} These biological and psychosocial chronic neighborhood stressors likely propagate the atherosclerosis process by inducing hypertension or other inflammatory processes, leading to a higher risk of stroke.^{24–26} Residential racial-ethnic segregation may also affect

stroke risk by encouraging uptake of unhealthy dietary habits that increase risk of hypertension and other stroke risk factors, or by inhibiting physical activity.^{22,27–31}

On the other hand, areas of high residential racial-ethnic segregation might foster social cohesion.³² Neighborhood cohesion has been associated with reduced stroke risk.³³ Social cohesion in minority neighborhoods can provide some mitigation of stroke risk factors though social support, known as the barrio effect.³⁴ Social cohesion also can create an aura of trust and safety within the neighborhood, which can impact stroke risk factors, such as engagement in physical activity.³⁵

Previous studies on residential racial-ethnic segregation and stroke have used cross-sectional data on stroke mortality, and have focused on African-Americans in urban settings.36-38 However, studying the effects of residential ethnic segregation on stroke risk in MAs has not been considered. Existing literature on the effects of residential segregation on health outcomes for Hispanics more broadly is mixed. For example, previous research has found that the deleterious effects of residential segregation on self-reported indices of physical health and disability are greater for Puerto Ricans as compared to MAs, possibly to due higher levels of segregation among Puerto Ricans.^{39,40} Other studies have shown consistent protective effects of residential segregation among older MAs across a range of health outcomes, but both protective and harmful effects of residential segregation when examining MAs by generational status.^{34,41–43} The subpopulation studied, geographic location, generational status, and study methodology may contribute to the heterogeneous findings across previous studies.^{34,40,43–45} Investigation of the relationship between residential ethnic segregation and stroke risk in MAs may be particularly relevant given the persistent stroke disparities in this subpopulation, as well as the large number of MAs in the United States at risk for stroke. Such investigation could inform novel interventions to decrease ethnic stroke disparities.

The objective of our study was to determine if greater residential ethnic segregation, measured at the census tract-level, was associated with higher stroke risk in a bi-ethnic populationbased stroke study. Given the high incidence of stroke among MAs, we hypothesized that greater residential ethnic segregation would be associated with higher stroke risk for MAs, but not for NHWs. Secondarily, we sought to determine whether age modified the association between residential ethnic segregation and risk of stroke within each ethnic group.

METHODS

Study Population

Stroke cases obtained from surveillance data consisted of MA and NHW patients, aged \geq 45 years, who were residents of Nueces County, Texas. The population at risk for stroke was determined by census tract population counts of MAs and NHWs, aged \geq 45 years who were residents of Nueces County, Texas in 2000. In 2012, Nueces County had a total population

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Data Sources

The Brain Attack Surveillance in Corpus Christi (BASIC) project is an ongoing stroke surveillance project in Nueces County, Texas that began in January 2000. The project uses active and passive surveillance to capture all cerebrovascular events of Nueces county residents aged >44 years.^{47,48} During active surveillance, abstractors examine admissions logs for a set of validated screening terms in emergency and inpatient departments. In addition, abstractors search for in-house strokes or those not ascertained through screening logs in intensive care units and hospital floors. For passive surveillance, abstractors review hospital ICD-9 stroke discharge codes. Potential stroke cases are reviewed for study eligibility and then validated by trained stroke physicians blinded to age and race-ethnicity. The criteria for stroke validation is based on previously published international criteria.⁴⁹ The data utilized for this study span from 2000 to 2010.

Race-ethnicity, sex, age, health insurance status, and stroke risk factors were obtained from patient medical records. Race-ethnicity was coded as NHW or MA, sex was coded as male or female, and age was recorded as a continuous variable. A previous study reported high agreement between raceethnicity recorded in the medical record and self-report in this community (94%).⁴⁷ Patients' home addresses were also obtained from medical records and sent to an independent company for geocoding. The geocodes were used to determine census tract residence, which served as a proxy for neighborhoods.

A residential ethnic segregation value, the primary exposure variable, census tract population counts of MAs and NHWs, and ethnicity specific median per capita income were derived from the 2000 US Census for each census tract in the county. Median per capita income for each ethnicity was calculated such that income contributions of people aged ≤ 15 years are not included in the numerator but are included in the denominator.⁵⁰

Residential Ethnic Segregation

The residential ethnic segregation index was calculated based on the isolation index.¹⁵ The index ranges from 0 to 1, and reflects the probability that two members randomly drawn from the same unit area (ie, census tract) are both MA. Higher values represent greater segregation. The residential ethnic segregation index was calculated as follows:

$$P_X = \sum_{i=1}^n \left[\frac{x_i}{X}\right] \left[\frac{x_i}{t_i}\right]$$

Where x_i is the number of MAs in each block-group *i* within each census tract, t_i is the total population in blockgroup *i*, and X is the number of MAs in the census tract. The value is then summed across all *n* block groups in the census tract to create a residential ethnic segregation index for each census tract. Using the ethnic segregation index, the census tracts were categorized as predominantly NHW (<.3), ethnically mixed (.3–.7), or predominantly MA (>.7) to be consistent with previous studies of residential racial-ethnic segregation that have used this index.^{36–38}

Statistical Analysis

Overall stroke counts, demographic characteristics, and risk factor prevalences were reported by residential ethnic segregation category (predominantly NHW, <0.3; ethnically mixed, .3–.7; predominantly MA, >.7) for NHW and MA stroke cases. Because individual-level data were not available for stroke-free controls, group-level data were analyzed using Poisson regression models with population counts from the 2000 US Census used to represent the population at-risk. Multilevel Poisson regression models were fitted separately for NHWs and MAs to assess the association between residential ethnic segregation and stroke risk within each ethnic group.14 Residential ethnic segregation was modeled as two indicator variables representing ethnically mixed and predominantly MA census tracts, with predominantly NHW as the referent. First, an unadjusted Poisson regression model was run to assess the crude association between residential ethnic segregation and stroke risk within each ethnic group. Next, to adjust for sociodemographics, a multilevel Poisson model was fitted to cross-classified cells of age category (45-59, 60-74, \geq 75 years) and sex, with additional adjustment for census tract-level ethnic specific median per capita income. Adjusted models included a random intercept for each census tract. Additionally, the adjusted models were used to examine possible effect modification of the residential ethnic segregationstroke risk association by age category given previous findings that the health impacts of segregation differ by age.^{36,37} Significance of effect modification was determined by the Wald test. Risk ratios and 95% confidence intervals were calculated from each model.

RESULTS

Table 1 presents stroke counts, demographic characteristics, and risk factor prevalences for NHWs and MAs by categories of residential ethnic segregation. The median residential ethnic segregation value across the 64 census tracts in the county was .52 (IQR: .38– .77). There were 2,855 strokes cases used in our analysis, 1585 among MAs (predominantly NHW: 47; ethnically mixed: 511; predominantly MA: 1027) and 1270 among NHWs (predominantly NHW: 289; ethnically mixed: 796; predominantly MA: 185).

Among MAs, median age of stroke did not vary considerably across residential segregation categories. Additionally, among MAs, predominantly MA census tracts had the lowest median per capita income (\$9,521; IQR: \$7,816-\$9,935) and percentage with health insurance (83.9%), along with the greatest prevalence of hypertension (75.2%), diabetes (50.3%), and excessive alcohol use (8.4%). Similarly, among NHWs, predominantly MA census tracts had the lowest median per capita income (\$17,701 [IQR: \$12,212-\$20,755]) and percentage with health insurance (86.5%), along with the greatest prevalence of diabetes (29.7%) and excessive alcohol use (8.1%).

Table 2 displays the unadjusted and adjusted risk ratios for the association between residential ethnic segregation and stroke risk for MAs and NHWs. After adjustment for age, sex and ethnicspecific median per capita income, both ethnically mixed and predominantly MA census tracts were not significantly associated with greater stroke risk among MAs or NHWs. Age did modify the association between residential ethnic segregation and stroke risk among MAs (Table 3, P<.0000) and NHWs (Table 3, P=.0010).

For MAs, age modified the association between residential ethnic segregation and stroke risk such that association between residential ethnic segregation and stroke risk was greatest in the youngest age-category (aged 45-59 years: ethnically mixed: RR = 1.49[95% CI: .82-2.70]; predominantly MA: RR = 2.38 [95% CI: 1.31-4.31]), decreased but was not significant in the 60-74 age category (aged 60-74 years: ethnically mixed: RR = 1.13[95% CI: .84-1.51]; predominantly MA: RR = 1.24 [95% CI: .95-1.64]), and was protective but not significant in the oldest age-category (aged \geq 75 years:

Table 1. Demographics and risk factor prevalence by segregation category and ethnicity. BASIC population in Nueces County, Texas from 2000–2010 (*N*=2855).^{a,b}

	Non-Hispanic White			
-	Predominantly NHW ^c	Ethnically Mixed ^d	Predominantly MA ^e	
Total stroke count	289	796	185	
Age				
45–59	66 (22.8)	114 (14.3)	38 (20.5)	
60-74	106 (36.7)	235 (29.5)	42 (22.7)	
≥75	117 (40.5)	447 (56.2)	105 (56.8)	
Demographics				
Median age (IQR)	71 (60–79)	77 (66–84)	77 (64–84)	
Female	137 (47.4)	428 (53.8)	99 (53.5)	
Health insurance	269 (93.1)	747 (93.8)	160 (86.5)	
Median per capita income (IQR)	23,679 (19,987–36,619)	26,030 (20,511–28,892)	17,701 (12,212–20,755)	
Risk factor prevalence				
Hypertension	195 (67.5)	579 (72.7)	127 (68.7)	
Diabetes	67 (23.2)	175 (22.0)	55 (29.7)	
Atrial fibrillation	36 (12.5)	145 (18.2)	33 (17.9)	
Coronary artery disease	98 (33.9)	254 (31.9)	51 (27.6)	
Smoker	76 (26.3)	125 (15.7)	32 (17.3)	
Excessive alcohol use	15 (5.2)	46 (5.8)	15 (8.1)	
		Mexican American		
-	Predominantly NHW ^c	Ethnically Mixed ^d	Predominantly MA ^e	
Total stroke count	47	511	1027	
Age				
45-59	13 (27.7)	173 (33.9)	313 (30.5)	
60–74	17 (36.2)	177 (34.6)	378 (36.8)	
≥75	17 (36.2)	161 (31.5)	336 (32.7)	
Demographics				
Median age (IQR)	67 (59–80)	66 (56–78)	69 (57–78)	
Female	26 (55.3)	249 (48.7)	515 (50.2)	
Health insurance	42 (89.4)	438 (85.7)	862 (83.9)	
Median per capita income (IQR)	15,083 (15,083–16,644)	14,251 (11,734–15,856)	9,521 (7,816–9,935)	
Risk factor prevalence				
Hypertension	34 (72.3)	382 (74.8)	772 (75.2)	
Diabetes	19 (40.4)	239 (46.8)	517 (50.3)	
Atrial fibrillation	6 (12.8)	33 (6.5)	89 (8.7)	
Coronary artery disease	11 (23.4)	153 (29.9)	296 (28.8)	
Smoker	8 (17.0)	107 (20.9)	185 (18.0)	
SHIOKEI	0 (17.0)	10/ (20.3)	105 (10.0)	

NHW, non-Hispanic White; MA, Mexican American.

^a Data are *n* (%) unless indicated otherwise.

^b Median residential ethnic segregation across the 64 census tracts in the county = .52; IQR: .38–.77.

^c n predominantly non-Hispanic census tracts =10.

^d n ethnically mixed census tracts =33.

 e n predominantly Mexican American census tracts =21.

ethnically mixed: RR = .89 [95% CI: .48-1.66]; predominantly MA: RR = .71 [95% CI: .38-1.34]).

For NHWs, the association between residential ethnic segregation and stroke risk was protective for NHWs aged 45– 59 years living in ethnically mixed census tracts (aged 45–59 years: ethnically mixed: RR = .77 [95% CI: .53-1.12]). Similar to MAs, young NHWs living in predominantly MA census tracts had the greatest relative risk for stroke as compared to young NHWs living in predominantly NHW census tracts (aged 45–59 years: predominantly MA: RR = 1.53 [95% CI: .92-2.52]). In the older age categories, the association between residential ethnic segregation and stroke risk in ethnically mixed census tracts remained protective, while the association in predominantly MA census tracts became more protective (aged 60-74 years: ethnically mixed: RR = .94 [95% CI: .72-1.22];

	Non-Hispanic White		Mexican American	
	Unadjusted RR (95% Cl)	Adjusted RR (95% Cl)	Unadjusted RR (95% Cl)	Adjusted RR (95% CI)
Residential ethnic segregation				
Predominantly NHW (<.3)	1	1	1	1
Ethnically mixed (.3–.7)	1.06 (.82-1.38)	.88 (.70–1.11)	1.20 (.84–1.71)	1.17 (.84–1.64)
Predominantly MA (>.7)	1.24 (.95–1.61)	.82 (.60–1.12)	1.53 (1.08-2.19)	1.31 (.92–1.86)
Age				
Category 1 (45–59)		1		1
Category 2 (60–74)		2.92 (2.42-3.53)		2.28 (2.00-2.61)
Category 3 (≥75)		9.12 (7.35–11.33)		4.80 (4.02-5.73)
Female		.70 (.5984)		.75 (.66–.85)
Median Per Capita Income		.89 (.80-1.00)		1.04 (.84-1.30)

Table 2. Multilevel Poisson models of the association between census-tract level residential ethnic segregation and stroke risk by ethnicity. BASIC study population in Nueces County, Texas from 2000–2010.

predominantly MA: RR = .76 [95% CI: .52-1.14]; aged \geq 75 years: ethnically mixed: RR = .88 [95% CI: .67-1.15]; predominantly MA: RR = .71 [95% CI: .49-1.02];). However, none of the associations in NHWs reached statistical significance.

DISCUSSION

We hypothesized that greater residential ethnic segregation would be associated with higher stroke risk among MAs, which was supported by our findings in younger MAs. Mexican Americans aged 45-59 years living in predominantly MA neighborhoods had significantly greater stroke risk compared to MAs aged 45-59 living in predominantly NHW neighborhoods. The association between residential ethnic segregation and stroke risk was attenuated for older MAs, and became protective in the oldest age-category, but did not reach statistical significance. Non-Hispanic Whites aged 45-59 residing in predominantly MA neighborhoods also had increased stroke risk compared with those residing in predominantly NHW neighborhoods but the magnitude of this association was

Mexican Americans (MA) aged 45–59 years living in predominantly MA neighborhoods had significantly greater stroke risk compared to MAs aged 45–59 living in predominantly non-Hispanic White neighborhoods.

Table 3. Age-effect modification of the association between census-tract level residential ethnic segregation and stroke risk by ethnicity. BASIC study population in Nueces County, Texas from 2000–2010.^a

	Non-Hispanic W	hite	
Residential ethnic segregation	Age 45–59 RR (95% Cl)	Age 60–74 RR (95% Cl)	Age ≥75 RR (95% CI)
Predominantly NHW (<.3)	1	1	1
Ethnically mixed (.3–.7)	.77 (.53–1.12)	.94 (.72–1.22)	.88 (.67-1.15
Predominantly MA (>.70)	1.53 (.92–2.52)	.76 (.52–1.14)	.71 (.49–1.02
	Mexican Americ	can	
Residential ethnic segregation	Age 45–59 RR (95% Cl)	Age 60–74 RR (95% Cl)	Age ≥75 RR (95% CI)
Predominantly NHW (<.3)	1	1	1
Ethnically mixed (.37)	1.49 (.82-2.70)	1.13 (.84–1.51)	.89 (.48-1.66
Predominantly MA $(>.70)$	2.38 (1.31-4.31)	1.24 (.95-1.64)	.71 (.38–1.34

not as strong as that observed in MAs and did not reach significance likely due to the small number of stroke events observed in this exposure group.

There are multiple pathways by which greater residential ethnic segregation could increase stroke risk. Segregated neighborhoods often have limited access to healthy food, more exposure to unhealthy food, and inadequate spaces for physical activity, which may influence health behaviors and downstream stroke risk factors including hypertension.^{27–31} Lower levels of education and reduced health care utilization in more highly segregated neighborhoods may also contribute to increased stroke risk.^{9,51} In our study, the prevalence of diabetes, hypertension, and excessive alcohol use was greatest, while censustract level median per capita income and proportion of individuals with health insurance was lowest among MAs in predominantly MA census tracts as compared to MAs in ethnically mixed and predominantly NHW census tracts. However, absolute differences in the prevalence of risk factors across segregation categories among MAs were not large. These data suggest that residential ethnic segregation may influence resources for maintaining health; however, a different study design would be required to test this hypothesis directly. Chronic stress, induced through generalized distrust, constrained opportunities for upward mobility and social disorganization, is associated with segregation and may represent another pathway by which segregation impacts stroke risk.^{9,21,36} We did not have information on chronic stress in our study to consider how stress may vary by residential ethnic segregation.

Our study suggests that the pathways between residential ethnic segregation and stroke risk likely affect both young MAs and NHWs, but these mechanisms may be selective and/or operate to varying degrees in the two ethnic groups. Though all members living in segregated neighborhoods

may have limited access to healthy food, minorities have specifically been targeted by tobacco and alcohol advertising.^{52,53} Greater segregation is also associated with lower education and socioeconomic status, but NHWs living in predominantly MA census tracts may have better access to neighborhood social and economic resources, improving control over their life circumstances and accumulating less stress.^{54,55} Finally, social disorganization and greater generalized distrust among MAs living in segregated areas may also induce more stress among MAs.^{9,21,56} More research is needed to understand the specific pathways by which residential segregation may influence stroke burden in young MAs and NHWs and how these pathways may differ between ethnic groups.

Although not significant, the observed protective associations between residential ethnic segregation and stroke among older MA might be explained by a survival bias or a 'barrio' effect.³⁴ Older residents who survived exposure to stroke risk factors in early life may be less susceptible to the effects of residential ethnic segregation than younger residents. An alternative hypothesis is that greater social support in older cohorts, especially older MAs, may have provided social capital to buffer the effects of residential ethnic segregation.^{34,57}

Our study is different from previous studies in that it is the first study to examine the effects of residential ethnic segregation on incident stroke risk in MAs. In two different studies, Greer et al documented similar effect modification of the association between residential ethnic segregation and stroke mortality by age among African Americans.^{36,37} In these studies, greater African-American racial residential segregation (predominantly White as referent) was significantly associated with higher stroke mortality in younger age categories (35-64) for African-Americans, but non-significant for older (>65) African Americans. Fang et al also found a positive association between residential ethnic segregation (predominantly White as referent) and stroke mortality among Whites, but no difference among African-Americans living in neighborhoods with different levels of segregation without adjusting for neighborhood poverty.³⁸ Our results, in combination with these previous studies, support the hypothesis that residential ethnic segregation may be influencing downstream stroke risk and therefore could be a contributor to racialethnic stroke disparities.

Elucidating the pathways by which residential ethnic segregation influences stroke risk is important to contextualize more proximal stroke risk factors, and to determine if residential ethnic segregation is a fundamental cause of stroke. Fundamental causes, such as socioeconomic status, are hypothesized to be dynamically tied to access to important resources such as social networks, economic capital, power, knowledge, and prestige. Access to these multiple resources, in turn, helps to prevent risk factors, numerous disease outcomes, and consequences of disease.⁵⁸ Addressing downstream risk factors in an attempt to reduce disparities may have limited effectiveness if residential ethnic segregation is a fundamental cause of stroke.9 Instead, researchers should be focused on policies and interventions aimed at improving neighborhood conditions and resources in segregated neighborhoods.

One major strength of our study is the incident stroke data with comprehensive disease surveillance and stroke case capture provided by the BASIC project. There are a few limitations that warrant discussion. First, the results are derived from a single, bi-ethnic nonimmigrant community and may not extend to areas where the MA population is the minority relative to NHWs or to immigrant communities.¹⁵ Our study was unable to account for length of residence in each neighborhood, but census data suggest that residence in this

community has been stable.⁵⁹ In addition, our study did not control for individual level factors other than sociodemographics given the study design (eg, only individual-level data for stroke cases was available); however, stroke risk factors may be on the causal pathway as described above and including them in our model may be an overadjustment.

The exposure, residential ethnic segregation, was also limited. Residential ethnic segregation was measured as a cross-sectional exposure based on 2000 census data. If residential ethnic segregation in Nueces County was higher prior to 2000, the cross-sectional exposure measured in 2000 may underestimate the effect of earlier life exposure to residential ethnic segregation on stroke risk.⁶⁰ Residential ethnic segregation may also capture effects of neighborhood poverty, which may present an over-adjustment in our model as we controlled for neighborhood income. Additionally, calculating the residential ethnic segregation measure at the census-tract level may dilute the effect for smaller block groups with stronger associations between residential ethnic segregation and stroke risk.⁶¹ Lastly, there are numerous measures of residential ethnic segregation, and choice of segregation measure can impact if the association between exposure and outcome is significant or not.⁶²

To our knowledge, our study is the first to examine residential ethnic segregation and stroke risk in a predominantly MA population. Our study identified younger MAs living in areas of greater segregation are at higher stroke risk than those residing in predominantly NHW neighborhoods. Future studies should explore specific pathways associated with greater residential ethnic segregation that put young MAs and NHWs at greater stroke risk, and elucidate how these pathways may operate differently for specific ethnic groups. A shift in focus to understanding the upstream factors influencing stroke risk will provide a better understanding of the fundamental drivers of stroke disparities, and could help to inform more effective interventions or policies to reduce stroke risk.

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