# RACIAL RESIDENTIAL SEGREGATION AND STROKE MORTALITY IN ATLANTA

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**Objective:** To assess the association between neighborhood-level racial residential segregation and stroke mortality using a spatially derived segregation index.

**Design:** Cross-sectional study

**Setting:** Atlanta Metropolitan Statistical Area

**Methods:** The study population consisted of non-Hispanic Black and White residents of the Atlanta Metropolitan Statistical Area during the time period Jan 1, 2000 to December 31, 2006. Census tract-level stroke death rates for Blacks and Whites were modeled as a function of the segregation index while controlling for two neighborhood-level chronic stressors (poverty, low education).

**Results:** Racial segregation was positively associated with stroke mortality for both Blacks and Whites aged 35–64 years. Among Blacks and Whites aged 65 or older, segregation was negatively associated with stroke mortality after controlling for the two stressors, suggesting that they were pathways between segregation and stroke death rates.

**Conclusion:** Future studies are needed to identify additional pathways between residential segregation and other health outcomes, and to collect data that support a life course approach to understanding the impact of residential segregation on health. (*Ethn Dis*. 2011;21(4):437-443)

**Key Words:** Residential Segregation, Stroke, Mortality, Neighborhood Environment

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## INTRODUCTION

Racial residential segregation is increasingly recognized as a fundamental risk factor for many undesirable health outcomes.[1,2] While the majority of studies on residential segregation have focused on associations with infant mortality[3–6] or all-cause mortality,[1,7,8] only one has examined the association with stroke.[9] And yet, stroke mortality is an important health outcome to study in this context: many of the chronic stressors associated with racial residential segregation are also associated with risk factors for stroke, and stroke ranks fourth among the leading causes of mortality in the United States.[10]

Racial residential segregation has been defined as the spatial separation of two or more racial/ethnic groups that stems from historical laws, residential mortgage lending practices, and the unintended consequences of federal housing policies.[2] Such segregation often results in Blacks living in less desirable areas that have limited social, economic, and health care opportunities for residents of color as well as exposure to high levels of chronic stressors. The chronic stress of living in socially and economically deprived neighborhoods has been associated with many risk factors for stroke, including a higher prevalence of hypertension, cigarette smoking, and depression.[11–13] Therefore, it is important to examine such chronic stressors as potential pathways between racial residential segregation and stroke mortality.

Because racial residential segregation is an inherently spatial phenomenon, it is important to have a measure of segregation that accounts for the characteristics of neighboring communities as well as the neighborhood of interest. Fortunately, the field of spatial statistics has advanced significantly in recent years, such that enhanced spatial measures of racial segregation that take into account the degree of segregation in these adjacent communities are now available.[14,15] These new measures substantially increase the accuracy of capturing neighborhood-level residential segregation by race.[16]

In this study, we use a spatially indexed measure of racial isolation to examine the association between racial residential segregation and stroke death rates for Blacks and Whites, aged 35 years or older, living in the metropolitan statistical area (MSA) of Atlanta, Georgia, which has been identified as having substantial neighborhood-level variation in the distribution of both race and mortality.[17] We examine the role of two chronic stressors (poverty and low educational profile) as potential pathways between residential segregation and stroke death rates.

## METHODS

### Mortality data and population

The Georgia Department of Community Health, Office of Vital Records,

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provided death records for the Atlanta MSA for January 1, 2000–December 31, 2006; these records were recorded by census tract of the decedent's residence at death, primary cause of death, age, and race. We abstracted death records for non-Hispanic Blacks and non-Hispanic Whites aged ≥35 years whose primary cause of death was reported as stroke. Stroke was classified according to the *International Classification of Diseases 10th Revision* codes for cerebrovascular disease (I60–I69).

We obtained population counts by race and age from the 2000 U.S. Decennial Census for the census tracts in Atlanta. In addition, by census tract, we obtained the percentage of people living below poverty and the percentage aged 25 years or older who had not completed high school and divided these distributions into tertiles.

## Isolation index

Racial residential segregation was measured using a spatial derivation of the isolation index originally developed by Massey and Denton.[14,18] This derivation, which was developed by Reardon and O'Sullivan,[14] can be interpreted as the probability that any two individuals randomly drawn from a census tract would be Black. The index ranges from 0 to 1, with 0 indicating no chance for two Black individuals to be randomly drawn from the same neighborhood and 1 indicating a 100% chance that two individuals drawn randomly from the same neighborhood would both be Black.

The isolation index is traditionally calculated using census tracts as the subunit of MSAs and typically ignores the racial composition of neighboring census tracts, but the variation of the index introduced by Reardon and O'Sullivan[14] accounts for the racial composition both within the tract (eg, between census blocks within tracts) and in the areas surrounding each tract. The calculation of the index was implemented via a freely available script[19] in ArcGIS software (version 9.1; Environmental Systems Research Institute Inc., Redlands, CA). Estimates of the total population and Black-only population were calculated by estimating a smoothed density surface[20] on a 50m × 50m grid of cells over the census blocks making up the Atlanta MSA. The Black population density for grid cell $r$ ($\tau_r$) and the proportion of the neighborhood population around $r$ that was Black ($\pi_r$) were calculated for each grid cell. The neighborhood of $r$ was defined as all grid cells within a 500-meter radius, and a truncated Gaussian decay function centered on $r$ was used to spatially weight the contributions of neighboring cells to $\pi_r$. Because stroke death rates were reported at the tract level, the point-level population densities were aggregated across the tract, giving a single value representing the isolation index for census tract $i$, which was calculated as in Equation 1.

$$P_i^* = \sum_{r \in i} \frac{\tau_r}{T_i} \pi_r \qquad (1)$$

Here it is seen that $P_i^*$ is the weighted average using the total Black population in the census tract to weight the sum of the product of $\tau_r$ and $\pi_r$ for all grid cells that fall within the census tract.

The census tracts were categorized as predominantly White (<0.3), racially mixed (0.3–0.7), or predominantly Black (>0.7). The cutpoints were chosen to be consistent with categories of racial residential segregation used in previously published studies.[7,8] For the remainder of this article, we will refer to the isolation index as the segregation index.

## Statistical analysis

Using Poisson regression, we fit a series of models to investigate the associations between residential segregation and stroke death rates for the time period 2000–2006. Poisson generalized estimating equations (GEE) with an unstructured covariance matrix were used to account for the clustering of stroke deaths within the census tracts. Model A assessed the association between the segregation index and stroke death rates while controlling for age; Model B included the two chronic stressor variables (poverty [percent of people in each tract living below poverty level] and low educational profile [percent of people without a high school diploma]). To assess the heterogeneity of the effect of segregation by age, both models included effects by age and segregation. Models were run for Blacks and Whites combined as well as stratified by race. In addition, using the parameters from Model B, rate ratios (RRs) comparing death rates for Blacks to death rates among Whites were calculated for each category of racial residential segregation. In the race-stratified models, census tracts with a population of 0 for either Blacks or Whites were excluded in order to stabilize the model (census tracts excluded: $n$=127 for model including Blacks, $n$=41 for model including Whites). All analyses for race stratified models were conducted using SAS (version 9.2; SAS institute; Cary, NC).

# RESULTS

All 660 census tracts within the Atlanta MSA were included in this analysis (except in the race-stratified models). Forty-two percent of the census tracts were categorized as predominantly White, 31% were racially mixed, and 27% were predominantly Black. The overall segregation index for the Atlanta MSA was 0.73. Predominantly Black neighborhoods were most often in the central portion of the MSA, while predominantly White neighborhoods were almost always outside the central city. Most of the racially mixed neighborhoods were in the southern half of the MSA.

Among Blacks, people in the older age group (≥65 years) were more likely to reside in predominantly Black neigh-

borhoods (72%) than were those in the younger group (60%) (Table 1). Among Whites, the opposite pattern was observed in predominantly White neighborhoods: older Whites were less likely to live there (57%) than were their younger counterparts (67%). During the study period, 10,451 stroke deaths were reported among non-Hispanic Blacks and non-Hispanic Whites aged 35 or older in the Atlanta MSA (Blacks: $n$=2,994; Whites: $n$=7,457). In both age groups among Blacks, the largest proportion of stroke deaths occurred in the predominantly Black census tracts; among Whites (for both age groups), the largest proportion of stroke deaths occurred in predominantly White neighborhoods. In the racially mixed neighborhoods the percentage of all stroke deaths (calculated by race) was higher for Whites than for Blacks.

Examination of the two chronic stressors across categories of residential segregation revealed that the mean percentages of the population living below poverty and of persons without a high school education worsened as segregation categories went from predominantly White to predominantly Black (Table 1). The predominantly Black census tracts had a mean percentage of the population living below poverty that was more than four times as large as the percentage in poverty for the predominantly White census tracts. In addition, predominantly Black census tracts had the highest percentage of people without high school diplomas, with those census tracts having more than twice the percentage of such people as the predominantly White census tracts.

For both Blacks and Whites, results for Model A (included age and segregation index) indicated a positive association between residential segregation and stroke mortality for the younger group (35–64) (Table 2). In predominantly Black neighborhoods, the RR was 1.60 for Blacks and 1.68 for Whites (predominantly White neighborhoods as the referent). For ages 35–64 in racially mixed neighborhoods, again with predominantly White neighborhoods as the referent, stroke death rates were significantly higher for Whites (RR = 1.37) but not for Blacks. When poverty and education were included in the model (Table 2, Model B), the magnitude of the three significant associations seen in Model A was attenuated somewhat but remained significant.

In contrast, for residents aged 65 or older, in Model A (Table 2) stroke death rates were slightly lower (but not significantly so) in predominantly Black neighborhoods when predominantly White neighborhoods were the referent (Blacks: RR = 0.87, Whites: RR = 0.90). The effect of residential segregation became protective (and significant) when poverty and education were included in the model (Table 2, Model B).

**Table 1. Distribution of population and deaths in the Atlanta Metropolitan Statistical Area (MSA) by racial residential segregation, race/ethnicity, and age group**

| | | Racial residential segregation | | |
|---|---|---|---|---|
| | Total | Predominantly White | Racially mixed | Predominantly Black |
| **Segregation index** | | ≤ 0.3 | 0.3–0.7 | ≥ 0.7 |
| **Number of census tracts** | 660 | 276 | 206 | 178 |
| **Population (% distribution by age in years)** | | | | |
| **Blacks** | 468,624 | 12.1 | 26.3 | 61.6 |
| 35–64 | 412,108 | 12.7 | 27.1 | 60.3 |
| ≥ 65 | 56,516 | 7.7 | 20.7 | 71.7 |
| **Whites** | 1,329,143 | 64.7 | 30.0 | 5.3 |
| 35–64 | 1,082,739 | 66.7 | 28.9 | 4.5 |
| ≥ 65 | 246,404 | 56.5 | 34.7 | 8.8 |
| **Stroke deaths (% distribution by age in years)** | | | | |
| **Blacks** | 2,994 | 9.8 | 22.1 | 68.1 |
| 35–64 | 1,121 | 9.2 | 20.9 | 69.9 |
| ≥ 65 | 1,873 | 10.1 | 22.9 | 67.0 |
| **Whites** | 7,457 | 56.1 | 36.1 | 7.8 |
| 35–64 | 994 | 58.7 | 34.6 | 6.7 |
| ≥ 65 | 6,463 | 55.7 | 36.4 | 7.9 |
| **Percentage below poverty level** | | | | |
| Mean (SD) | | 5.3 (4.8) | 11.4 (6.8) | 22.5 (16.3) |
| **Percentage ≥ 25 without high school diploma** | | | | |
| Mean (SD) | | 10.8 (8.9) | 22.4 (11.4) | 26.7 (14.6) |

Within each category of residential segregation, stroke death rates were higher for Blacks than for Whites in both age groups (Table 3). In each category, the larger Black:White RR was in the younger age group.

## DISCUSSION

In this study based in the Atlanta MSA, we found that, in the younger age group (aged 35–64), the stroke death rates were significantly higher in predominantly Black neighborhoods than in predominantly White neighborhoods for both Blacks and Whites. This finding held true among both Blacks and Whites. Our finding of an opposite effect (after entering two chronic stressors into the model) for residents of both races aged 65 and older, that racial segregation was protective, seems counterintuitive but was not entirely unex-

**Table 2. Rate Ratios of Stroke Mortality by Category of Racial Residential Segregation, Age Group, and Race, Atlanta Metropolitan Statistical Area (MSA), 2000–2006**

| | Blacks | | | | | | Whites | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Model A* | | | Model B† | | | Model A* | | | Model B† | | |
| | RR | 95% CI | p | RR | 95% CI | p | RR | 95% CI | p | RR | 95% CI | p |
| Segregation by age in years | | | | | | | | | | | | |
| 35–64 | | | | | | | | | | | | |
| Group 1 (ref) (predominantly White) | (<0.3) | — | — | — | — | — | — | — | — | — | — | — |
| Group 2 (racially mixed) | (0.3–0.7) | 1.07 | (0.82, 1.40) | 0.63 | 0.97 | (0.73, 1.28) | 0.81 | 1.37 | (1.18, 1.59) | <.0001 | 1.22 | (1.05, 1.41) | 0.009 |
| Group 3 (predominantly Black) | (≥0.7) | 1.60 | (1.28, 2.01) | <.0001 | 1.49 | (1.17, 1.90) | 0.001 | 1.68 | (1.27, 2.22) | 0.0002 | 1.51 | (1.14, 2.00) | 0.004 |
| ≥ 65 | | | | | | | | | | | | | |
| Group 1 (ref) (predominantly White) | (<0.3) | — | — | — | — | — | — | | | | — | — | — |
| Group 2 (racially mixed) | (0.3–0.7) | 1.02 | (0.82, 1.28) | 0.86 | 0.90 | (0.71, 1.14) | 0.40 | 1.06 | (0.98, 1.15) | 0.12 | 0.95 | (0.87, 1.03) | 0.21 |
| Group 3 (predominantly Black) | (≥0.7) | 0.87 | (0.71, 1.07) | 0.19 | 0.78 | (0.63, 0.97) | 0.03 | 0.90 | (0.77, 1.05) | 0.19 | 0.81 | (0.69, 0.96) | 0.02 |
| Percentage of people below poverty level | | | | | | | | | | | | | |
| Low (ref) | (<5.25%) | | | | — | — | — | | | | — | — | — |
| Medium | (5.25–11.75%) | | | | 0.92 | (0.79, 1.06) | 0.25 | | | | 1.07 | (0.86, 1.01) | 0.07 |
| High | (>11.75%) | | | | 0.88 | (0.75, 1.04) | 0.13 | | | | 0.97 | (0.83, 1.14) | 0.73 |
| Percentage of people without high school diploma | | | | | | | | | | | | | |
| Low (ref) | (≤10%) | | | | — | — | — | | | | — | — | — |
| Medium | (10–20%) | | | | 1.36 | (1.18, 1.58) | <.0001 | | | | 1.19 | (1.09, 1.29) | <.0001 |
| High | (≥20%) | | | | 1.45 | (1.22, 1.73) | <.0001 | | | | 1.37 | (1.26, 1.50) | <.0001 |

* Model A: segregation, age, age × segregation.
† Model B: segregation, age, % below poverty, % without high school diploma, age × segregation.

pected. That the two chronic stressors (poverty and low educational profile) attenuated our findings for younger residents and brought the findings for older residents into statistical significance suggests that they may be important pathways between racial segregation and stroke mortality.

Our finding of effect modification by age group in the association between a socio-environmental condition (in this case racial residential segregation) and a health outcome is not unique. Furthermore, previous research has shown that socioenvironmental conditions have the strongest health effects on people aged 35–64.[21–24] Possible hypotheses for explaining the observed effect modification we found among Blacks by age include: a) an ethnic density effect in the older age group, and b) survival bias. The former mechanism refers to a protective health effect among minority populations living in areas in which they are the predominant racial/ethnic group.[7] It is hypothesized that this phenomenon operates via social support, which shields members of the community from the ill effects of prejudice and marginalization while also building intra-group trust and increasing Black political power. This effect may not have been present in our

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**Table 3. Black:White Rate Ratios for Stroke Mortality by Age Group and Category of Racial Residential Segregation, Atlanta MSA, 2000–2006***

| | Predominantly White | | Racially Mixed | | Predominantly Black | |
|---|---|---|---|---|---|---|
| Age group (years) | RR | 95% CI | RR | 95% CI | RR | 95% CI |
| 35–64 | 2.33 | (1.83, 2.98) | 1.85 | (1.52, 2.25) | 2.32 | (1.73, 3.11) |
| ≥ 65 | 1.37 | (1.12, 1.69) | 1.31 | (1.15, 1.50) | 1.31 | (1.15, 1.50) |

* Model controls for percentage below poverty and percentage without high school diploma.

younger group, as the effects of the spatial concentration of poverty, high unemployment rates, and low social capital that are associated with residential segregation may have outweighed any benefits of ethnic density in this working-age population. The second hypothesis, survival bias, as applied to our study, would assume that the residents who survived the adverse health effects of poverty and discrimination during their working years were less susceptible as a group to the health effects of racial residential segregation than were their younger counterparts.

For the younger age group, our findings mirror components of an earlier study that investigated the association between residential segregation and stroke mortality at the zip code level in New York City[9]; in that study, Fang and coworkers found a positive association of segregation for Whites but no associations (positive or negative) for Blacks. Methodological factors that could contribute to the difference in results between our study and that by Fang et al include the use of different measures of racial residential segregation and different geographic units of analysis. Fang and associates calculated racial composition for each neighborhood, which measures the percentage of each neighborhood that is Black. In contrast, our study used the isolation index, which measures the spatial distribution of people of different racial/ethnic groups and identifies areas with spatial concentrations of minority residents. There were also differences in unit of analysis used; the Fang study used zip code while our study used census tracts. The phenomenon of different associations resulting from the use of different geographic scales is referred to as the modifiable areal unit problem (MAUP)[25] and could occur if the measure of segregation is sensitive to the neighborhood measurement scale even in the setting of identical underlying population distributions.

Our finding that the association between racial residential segregation and stroke mortality is partially explained via chronic stressors (ie, neighborhood poverty and low educational profile) is consistent with studies that have documented a myriad of chronic stressors associated with racial residential segregation and is supported by studies that documented associations between chronic stressors and many risk factors for stroke. For instance, in addition to higher poverty rates and lower educational profiles, racially segregated residential areas have been found to have fewer educational opportunities, fewer employment opportunities, increased crime, increased environmental risks, and poorer quality of the built environment.[1,26–29] These types of chronic stressors have also been found to be associated with three of the major risk factors for stroke – hypertension, smoking, and diet.[11,12,30] Chronic stressors reported to be associated with hypertension include residing in a neighborhood with a large proportion of residents without tertiary education[11] and living in an economically deprived neighborhood.[31–34]

Regarding the chronic stressor of cigarette smoking, studies have found that poor neighborhood social cohesion is associated with the prevalence of this behavior,[12] and deprived neighborhood conditions have been associated with the risk of cigarette smoking.[35,36] In addition, studies have repeatedly shown that socially disadvantaged neighborhoods have less access to healthy foods, a disturbing phenomenon in that access to healthy foods is critical for a healthy diet. Areas of concentrated poverty have only a limited availability of fresh foods[37,38]; predominantly Black neighborhoods often have fewer supermarkets and more fast-food restaurants[37,39]; and, a high density of fast-food restaurants has been associated with an increased neighborhood-level risk of stroke.[40] In addition to increasing the likelihood of behavioral risk factors for stroke such as poor diet and smoking, neighborhood-level chronic stressors may directly affect physiologic processes related to stroke through chronic or persistent activation of the hypothalamic-pituitary-adrenal (HPA) axis. Such physiologic dysfunction may result from wear and tear on the HPA axis, often termed allostatic load. The result of this response can be an increased risk of stroke.[41]

One strength of our study is use of the spatial approach developed by Reardon and O'Sullivan for calculating the segregation index. This spatial approach is particularly appropriate for studying segregation at the neighborhood/micro level because, for census tracts that are predominantly Black, it distinguishes between those surrounded by predominantly Black census tracts and those surrounded by predominantly White or racially mixed census tracts. Although

the Reardon derivation of the segregation index can be used to represent the segregation of a point location, we aggregated the index to the census-tract level so that it would be compatible with the smallest geographic unit for which the stroke mortality data were available. For this study, we chose to focus on racial residential segregation at the micro level, given our interest in exploring the role of chronic stressors. The diversity of neighborhoods in the study area, which included both central-city and suburban neighborhoods, supported the neighborhood-level focus of this study.[42]

We should note, however, that several aspects of our study design limit the inferences that can be drawn about the causal relationship between segregation and stroke mortality. While the proposed pathways by which segregation might increase the prevalence of risk factors for stroke are cumulative and chronic in nature, our measure of residential neighborhoods is cross-sectional, taken at the time of death. Differences in migration over the life course and mobility patterns by age group and race/ethnicity could bias the results. There may also be unmeasured confounding of the segregation-stroke association by individual poverty, such that poor individuals are more likely to live in highly segregated neighborhoods. Alternatively, segregation can be viewed as a structural social phenomenon that constrains housing choice by race and income, and thus it would be a determinant and not a consequence of the residential location of poor families. In this view, individual poverty is not a confounder but rather a mediator. Another limitation is that the poverty distributions for predominantly Black and White neighborhoods did not overlap substantially, which could bias the perceived effects of the two chronic stressors. Our stratification of the results by race minimizes the impact of this potential bias, however. Finally, because we focused on the neighborhood-level pathways through which racial residential segregation could affect stroke mortality, conclusions cannot be drawn regarding the larger context of city, state, or national effects of segregation on mortality.[42]

In conclusion, racial residential segregation has been established as a key contextual factor that influences neighborhood health, but more work is needed to examine health outcomes other than stroke mortality (especially those that are sensitive to chronic stressors). Such work could help to identify the various pathways through which racial residential segregation affects the health status of populations at the neighborhood level as well as at the city, county, or state level, and to explore the extent to which effect modification by age group exists in other communities. From a data perspective, it is also important that epidemiologic surveys and health surveillance systems collect information from participants on the location of their previous residences in order to accommodate a life-course approach to the study of racial residential segregation and health.

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