

Research Article

Racial Segregation and Cognitive Function Among Older Adults in the United States: Findings From the REasons for Geographic and Racial Differences in Stroke Study

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Received: August 9, 2020; Editorial Decision Date: June 9, 2021

Decision Editor: Deborah S. Carr, PhD, FGSA

Abstract

Objectives: Residential segregation is one of the fundamental features of health disparities in the United States. Yet little research has examined how living in segregated metropolitan areas is related to cognitive function and cognitive decline with age. We examined the association between segregation at the metropolitan statistical area (MSA) level and trajectories of age-related cognitive function.

Method: Using data from Black and White older adults in the REasons for Geographic and Racial Differences in Stroke study ($n = 18,913$), we employed linear growth curve models to examine how living in racially segregated MSAs at baseline, measured by the degree of non-Hispanic Black (NHB) isolation and NHB dissimilarity, was associated with trajectories of age-related cognitive function and how the associations varied by race and education.

Results: Living in MSAs with greater levels of isolation was associated with lower cognitive function ($b = -0.093$, $p < .05$) but was not associated with rates of change in cognitive decline with age. No effects of living in isolated MSAs were found for those with at least a high school education, but older adults with less than a high school education had lower cognitive function in MSAs with greater isolation ($b = -0.274$, $p < .05$). The degree of dissimilarity was not associated with cognitive function. The association between segregation and cognitive function did not vary by race.

Discussion: Metropolitan segregation was associated with lower cognitive function among older adults, especially for those with lower education living in racially isolated MSAs. This suggests complex associations between individual socio-economic status, place, and cognitive health.

Keywords: Cognitive function, Multilevel linear model, Racial segregation, REGARDS study

Cognitive decline is a common symptom of dementia that has a wide range of consequences for independence, functional decline, institutionalization, and mortality (Langa et al., 2001; Plassman et al., 2007). In 2013/2014, it was estimated that approximately 11% of community-dwelling older Americans were living with dementia (Davydow et al., 2014; Hebert et al., 2013), and the number is projected to almost triple by 2060 (Matthews et al., 2019). There are large inequalities in cognitive function by race and education, with dementia risk estimated to be over twice as high in non-Hispanic Blacks (NHB) compared to non-Hispanic Whites (Barnes et al., 2005; Garcia et al., 2019; Zhang et al., 2016) and twice as high in those with less than a high school education compared to those with a college degree (Crimmins et al., 2018).

Previous research has highlighted the role of clinical (e.g., hypertension, depression) and social (e.g., social isolation, physical inactivity) factors as potentially modifiable risk factors for dementia and dementia disparities (e.g., Chin et al., 2011). Recently, a growing body of literature has highlighted the role of residential environments for cognitive function (Besser et al., 2017). Findings suggest that living in socioeconomically advantaged neighborhoods may promote cognitive function and/or buffer cognitive decline through their greater density of physical amenities (e.g., recreational centers, quality parks, walkable streets) as well as social and institutional resources (e.g., libraries, community centers) that promote cognitively beneficial health behaviors and facilitate mental stimulation (Clarke et al., 2012, 2015; Finlay et al., 2020, 2021; Ng et al., 2018). It is therefore increasingly recognized that cognitive health is the result of a complex interplay of factors across multiple levels, including biological/physiological factors, individual factors, and factors in the surrounding neighborhood environment (Livingston et al., 2020). Little attention, however, is given to how residential segregation is associated with cognitive function in older adults.

Racial Segregation and Cognitive Health

Racial residential segregation is one of the fundamental features of American society driving both the creation and perpetuation of inequality in the United States (Gee et al., 2012; Williams & Collins, 2001). Segregation at the metropolitan area level explains significant variation in educational attainment, infant mortality, obesity, diabetes, self-rated health, and all-cause mortality (Chang, 2006; Kramer et al., 2010; LaVeist, 1989, 2003). Among older adults, residence in more segregated neighborhoods is associated with a higher risk of functional decline and mortality (Sudano et al., 2013). Other research suggests no association between neighborhood segregation and older adult health, or even that racial segregation is positively associated with health for Black older adults after adjusting for neighborhood poverty (Robert & Ruel, 2006).

Only a handful of studies have examined the extent to which racial segregation is related to cognitive function. Using cross-sectional data from 4,525 aging Americans (age 54–65) in the 1996 wave of the U.S. Health and Retirement Study (HRS), Aneshensel and colleagues (2011) found that neighborhood segregation (measured as the proportion of African American residents in a census tract) was related to lower cognitive function, but only among those with lower levels of educational attainment (Aneshensel et al., 2011). Using data from the same cohort of HRS respondents 4 years later (age 58–69) and followed over a 10-year period (2000–2010), Kovalchik and colleagues (2015) found no relationship between segregation at the neighborhood or county level and trajectories of cognitive function; however, differences by individual educational level were not examined. A more recent study using data from only Black participants in the Coronary Artery Risk Development in Young Adults study found that greater cumulative exposure to segregated neighborhoods during 25 years of young adulthood was associated with worse cognitive function in midlife (age 43–55; Caunca et al., 2020).

Segregation and Spatial Scale

The previous studies that examined segregation and cognitive function used a measure of racial composition at the local neighborhood (i.e., census tract) level (Aneshensel et al., 2011; Caunca et al., 2020; Kovalchik et al., 2015). However, tract-level measures neglect the effects of residential segregation outside of the affected individual's neighborhood of residence (Quillian, 2014), even when adjusted by the racial composition of surrounding census tracts (Caunca et al., 2020). Many researchers argue that the processes related to segregation operate not only at the local neighborhood level, but also across the broader geographic space at the metropolitan area level (Kramer & Hogue, 2009; Quillian, 2014). Metropolitan areas include a city and the surrounding suburban and exurban areas that share the same administrative divisions and infrastructure. Rather than contrasting the cognitive function of older adults living in predominantly White or Black neighborhoods, measures of metropolitan segregation capture the effects of segregated space beyond the neighborhood where an individual lives. Metropolitan area segregation is not randomly created but has been imposed by economic systems, city plans, and housing policies over a long period of time (e.g., Rothwell & Massey, 2009). We argue that it is important to understand the consequences of segregation at this “supraneighborhood” level (Quillian, 2014) because it has consequences for the unequal distribution of resources relevant for cognitive health (e.g., availability of public transit, housing quality, social services) that are implemented across an entire metropolitan area (not just within individual neighborhoods). For example, decisions about urban planning at the metropolitan area level shape cognitively relevant features of the built environment

across municipal space, including sidewalks (and sidewalk maintenance), curb cuts, snow removal, and public transit systems. Decisions about the funding and location of cognitively stimulating social institutions (e.g., museums, public libraries) also apply across metropolitan areas, not just local neighborhoods. Thus, we argue that segregation at the metropolitan area is the most relevant spatial scale for studies of cognitive function.

Heterogeneity in the Effects of Segregation

We also expect that metropolitan segregation may not have the same consequences for cognitive function across all groups of older adults. The most disadvantaged older adults living within more segregated metropolitan areas are more at risk for cognitive impairment and decline due to differences in the ability to compensate for the lack of resources in their communities. More advantaged older adults (e.g., Whites and the higher educated) living in segregated metro areas can draw upon personal wealth, social capital, and social connections to offset the lack of resources in their environment (e.g., by accessing private gyms rather than relying on poor quality public recreation centers). Conversely, disadvantaged older adults (including Blacks and those with less than a high school education) have fewer personal resources to escape the lack of institutional resources in segregated areas (Aneshensel et al., 2011). Indeed, for Whites, segregation has been found to be either protective or not related to vascular risk factors for dementia (e.g., obesity, cardiovascular disease, hypertension), and some of the adverse health effects of living in segregated metropolitan areas are no longer statistically significant for Whites once neighborhood or individual characteristics are controlled (Chang, 2006; Greer et al., 2014; Kershaw et al., 2015; Usher et al., 2018). For example, Subramanian and colleagues (2005) found that segregation had almost no association with self-rated health in White adults (age 18+), but with a 10 percentage-point increase in metropolitan segregation (measured by the Black isolation index), Blacks were 5% more likely to report poor health compared to Whites. Only one study examined educational differences in the relationship between segregation and cognitive function (Aneshensel et al., 2011). Among older adults with less than a high school degree, segregation (measured by tract percent Black) was adversely related to cognitive function, but the racial composition of the neighborhood had no consequences for cognitive functioning among older adults with a high school degree (Aneshensel et al., 2011). We therefore expect that segregation is likely to be more deleterious for cognitive function among Black older adults and those with less than a high school education because of compound disadvantage (Wheaton & Clarke, 2003), where the combined effect of segregation and individual disadvantage is more than the additive effect of both.

Measuring Segregation

While residential segregation conceptually means the degree to which two (or more) groups live apart from each other, different measures capture distinct dimensions of segregation (Denton, 1994; Massey & Denton, 1988; Reardon & O'Sullivan, 2004). Empirical studies have shown few associations between health and a simple measure of the racial composition of a neighborhood (e.g., percent Black), but a measure of Black isolation (where the distribution of Black residents within a metropolitan area is most likely to be in census tracts with other Blacks rather than sharing tracts with Whites) has been shown to be significantly related to hypertension, obesity, and self-reported health (Chang, 2006; Kershaw et al., 2011; Subramanian et al., 2005). Following Reardon and O'Sullivan (2004), we focus on two dimensions of segregation: exposure/isolation and clustering/evenness. Exposure and isolation capture the degree to which different racial groups come into contact with each other (Massey & Denton, 1988). Clustering and evenness capture the degree to which different racial groups are evenly distributed spatially (Massey & Denton, 1988; Reardon & O'Sullivan, 2004). Both dimensions provide important information as Black and White residents may be evenly distributed in a metropolitan area but their interaction with each other may depend on the degree of exposure to other racial groups in their neighborhoods (Massey & Denton, 1988; Reardon & O'Sullivan, 2004).

In this study, we examine the association between segregation at the metropolitan statistical area (MSA) and trajectories of cognitive function over mid- to late-adulthood. We use more than 15 years of data (2003–2018) from the REasons for Geographic and Racial Differences in Stroke (REGARDS) study, a nation-wide longitudinal study of community-dwelling Black and White adults aged 45 years or older (Howard et al., 2005). We hypothesize that: (a) living in more segregated metropolitan areas will be associated with lower cognitive function and faster rates of cognitive decline, independent of neighborhood poverty, individual socioeconomic status, and vascular risk factors; and (b) that the most disadvantaged older adults (Blacks and those with lower levels of education) will be most vulnerable to the adverse effects of segregation on cognitive function.

Method

Data

REGARDS is an ongoing, nation-wide, prospective cohort study examining racial and regional disparities in stroke and cognitive function (Howard et al., 2005). Potential participants were randomly selected from commercially available lists of U.S. residents, stratified by age, race, sex, and geographic region. The cohort includes 30,239 Black or White individuals aged 45 years or older who did not have any medical conditions that prevented long-term participation

(e.g., cancer) and were not on a waiting list for a nursing home. Participants were enrolled between 2003 and 2007 and baseline computer-assisted telephone interviews gathered demographic information, medical history, and health status. An expanded cognitive battery including the word list learning/recall and the verbal fluency assessment was implemented in 2006 and conducted during follow-up telephone calls at 2-year intervals. Residential address was geocoded to latitude and longitude coordinates and we assigned participants to census tracts (and corresponding MSA) based on their geocoded address at baseline. The study procedures are reviewed and approved annually by the University of Alabama at Birmingham.

For the current study, we first selected 25,965 participants who had at least one cognitive assessment, and who did not report any stroke prior to the first cognitive assessment or at baseline (due to the known impact of stroke on cognitive function; Levine et al., 2015). Because we focused only on MSA-level segregation, we further excluded 6,237 participants who did not live in MSAs at baseline. Next, those who had missing data on covariates ($n = 815$) were excluded. Compared to those with missing data on covariates, the analytic sample was less likely to have a history of heart disease and diabetes, but more likely to ever drink, ever smoke, and be married; no differences were found in cognitive function scores and the degree of MSA segregation. The final analytic sample included 18,913 individuals living in 257 MSAs at baseline with an average of 343 participants ($SD = 283.1$, range 1–1,031) per MSA. On average, respondents participated in the study for 9.1 years ($SD = 4.0$, range 1–15 years) during 2006–2018. The analytic sample included 18,913 individuals with an average of 4.8 ($SD = 1.7$) observations per person (73,275 person-years).

Measures

Cognitive function

Global cognitive function was assessed by a composite index of five cognitive tests (Kobayashi et al., 2015; Weuve et al., 2015; Zhu et al., 2015): Word List Learning (WLL), Word List Delayed Recall (WLD), Animal Fluency Test (AFT), Letter Fluency Test (LF), and recall and orientation items from the Montreal Cognitive Assessment (MoCA). The WLL measures verbal learning (score range, 0–30) and the WLD measures verbal memory (score range, 0–10). The AFT and the LF assess language and executive function (i.e., complex cognitive processing used in problem solving or complex action sequences). Scores are based on the number of unique animals (AFT) and unique words beginning with “F” (LF) named in 1 min. Items from the MoCA include five-word delayed memory recall and six-item orientation (score range, 0–11). The composite index was a mean of standardized scores from the five cognitive tests at each follow-up; we used the mean scores only when there were at least two tests available. The composite z

score index was highly correlated with scores from each separate test (correlations ranged from 0.67 for LF to 0.84 for WLD) but had the advantage of extending beyond specific subdomains of cognitive function to capture global cognitive function.

MSA segregation

MSAs include at least one urbanized area of 50,000 population or more and consist of one or more whole counties as well as any adjacent counties having a high degree of social and economic integration with an urban core (U.S. Census Bureau, 2012). As of 2003, there were 362 MSAs in the United States (U.S. Census Bureau, 2016). In REGARDS, 8,742 Black participants lived in 155 MSAs and 11,231 White participants lived in 258 MSAs at baseline. We used two measures of MSA-level segregation at baseline: NHB isolation (the exposure/isolation dimension) and NHB dissimilarity (the evenness/clustering dimension; Massey & Denton, 1988; Reardon & O’Sullivan, 2004). The NHB isolation index is defined as the proportion of NHB residents in tracts, weighted by the proportion of NHB residents in MSAs (Massey & Denton, 1988):

$$P_{\text{NHB}}^* = \sum \left[\frac{\text{NHB}_{\text{tract}}}{\text{NHB}_{\text{MSA}}} \right] \left[\frac{\text{NHB}_{\text{tract}}}{\text{POP}_{\text{tract}}} \right],$$

where $\text{NHB}_{\text{tract}}$ and NHB_{MSA} represent the number of NHB residents in each tract (indexed within a MSA) and MSA, respectively, and $\text{POP}_{\text{tract}}$ represents the total population in each tract (indexed within a MSA). The isolation measure ranges from 0 to 1 and represents the probability that a randomly selected NHB resident of a MSA shares a tract with another NHB resident.

The NHB dissimilarity index is defined as the mean absolute deviation of each tract’s NHB population from the MSA’s NHB population, weighted by the population in the tract (Massey & Denton, 1988):

$$D = \frac{\sum \text{POP}_{\text{tract}} |\text{NHB}_{\text{tract}} - \text{NHB}_{\text{MSA}}|}{2\text{POP}_{\text{MSA}}\text{NHB}_{\text{MSA}} (1 - \text{NHB}_{\text{MSA}})},$$

where $\text{NHB}_{\text{tract}}$ (indexed within a MSA) and NHB_{MSA} represent the number of NHB residents in each tract and MSA, respectively, and $\text{POP}_{\text{tract}}$ (indexed within a MSA) and POP_{MSA} represent the total population of the tract and MSA, respectively. The measure represents the proportion of NHB residents that would have to change tracts to achieve an even distribution within their MSA.

Covariates

Age, race, and gender were self-reported by participants. Educational attainment was captured using three categories: less than high school (reference), high school diploma, and more than high school (some college or college degree). The year of baseline assessment (2003–2007) was included to account for any period differences.

Studies have shown that the association between segregation and health may be spurious due to chronic disadvantages that have long been embedded in individual lifestyle and underlying health conditions (e.g., Kershaw et al., 2011). We thus controlled for health behaviors and conditions at baseline, including ever drinking (1 = current or past drinker, 0 = never), ever smoking (1 = current or past smoker, 0 = never), self-reported body mass index (1 = overweight or obese, 0 = normal or underweight), hypertension (1 = self-reported hypertension, 0 = no), and diabetes (1 = self-reported diabetes, 0 = no). Social integration variables included marital status (1 = currently married, 0 = single/widowed/divorced) and the number of adults in the same household.

Because segregation measures vary by population size, the proportion of racial groups, and region (Massey & Denton, 1988), we adjusted for MSA population size (in millions), MSA % NHB, and census region (i.e., Northeast, Midwest, South, and West based on the geocoded address at baseline). While MSA % NHB is correlated with both isolation and dissimilarity ($\rho = 0.64\text{--}0.70$), we included it as a covariate to adjust for the effect of racial composition as distinct from the effect of segregation itself (Reardon & O'Sullivan, 2004). We also adjusted for neighborhood poverty (percentage of the population in a census tract with income below the 1999 federal poverty level) because it could confound the association between MSA segregation and cognitive function (Quillian, 2014).

Analyses

We first present sample statistics for baseline individual characteristics by the degree of isolation and dissimilarity (at each tertile of the segregation measure). We used growth curve models to examine the association between MSA segregation at baseline and trajectories of cognitive function over time. A three-level linear model was used to account for multiple observations per person, nested within MSAs. Age (in years) was used as the indicator of time (centered at age 45) and separate models were run for isolation and dissimilarity. The first model included only demographic characteristics to examine how trajectories of cognitive function vary by individual characteristics. Because previous studies have found racial differences in rates of age-related cognitive decline (e.g., Gross et al., 2015), we included an interaction term between race and age. Subsequent models added the segregation measures (testing effects on both the intercept and slope), and tested interaction terms between segregation and race, and segregation and education to examine differences in the cognitive effects of segregation by individual social position. The final models adjusted for MSA population size, MSA % NHB, census region, and neighborhood poverty, as well as individual health behaviors, health conditions, and social integration. The intraclass correlation coefficient (ICC) was

calculated for all models to compare the amount of variation in cognitive function (at both the individual and MSA levels) that was explained by model predictors. All analyses were performed in Stata MP 15.

Results

Sample Characteristics

The REGARDS study sample was 42% Black ($n = 8,336$) with a mean age of 64 years at baseline (Table 1). On average, global cognitive function z scores across the study period were -0.02 ($SD = 0.86$), and participants living in MSAs with a lower degree of either isolation or dissimilarity had higher cognitive scores than those exposed to a greater degree of MSA segregation. At baseline, participants lived in MSAs with mean isolation of 0.49 ($SD = 0.17$). This means that, on average, participants (regardless of their own race) lived in MSAs where there was a 49% probability that a NHB resident lives in a tract with another NHB resident; however, this probability ranged from 0.003 to 0.76. Participants lived in MSAs with mean dissimilarity of 0.58 ($SD = 0.12$), meaning that on average 58% of NHB residents within an MSA would need to move to a different tract to achieve an even distribution of NHB residents in the metropolitan area.

MSAs where REGARDS participants lived at baseline were as large as 21.2 million and as small as 0.07 million people, with an average 3.64 million people in each MSA. At baseline, approximately 63% of our analytic sample lived in the Southern region of the United States and 7% lived in the Northeast region. Participants lived in census tracts with a poverty rate of 15.2% on average (range 0.001%–82.2%). About 68% of participants had more than high school education (HS) and 9% had less than HS, but there were differences in educational attainment by levels of segregation: 5.6% of respondents in MSAs with lower isolation (T1) had less than HS compared to about 10% of those in MSAs with a greater degree of isolation (T2 and T3); 8.1% of participants in MSAs with a lower degree of dissimilarity (T1) had less than HS compared to 9.7% in MSAs with greater dissimilarity (T3).

Trajectories of Cognitive Function

Model 1 in Table 2 show that cognitive function declined linearly at a rate of 0.033 units for each year of age ($p < .001$). Black participants had significantly lower levels of cognitive function than Whites ($b = -0.442$, $p < .001$), but experienced slightly slower rates of cognitive decline with age ($b_{\text{interaction}} = 0.004$, $p < .001$). Participants with more than HS had higher levels of cognitive function compared to those with less than HS ($b_{\text{HS}} = 0.331$, $p < .001$; $b_{\text{HS}^+} = 0.649$, $p < .001$). After accounting for these demographic characteristics, about 46% of the variance (ICC) in

Table 1. Study Characteristics by Tertiles of MSA Segregation: REasons for Geographic and Racial Differences in Stroke (REGARDS) Study ($n = 18,913$)

	Overall			Isolation			Dissimilarity		
	Mean	Median	Min-max	T1 (lower segregation)	T2	T3 (greater segregation)	T1 (lower segregation)	T2	T3 (greater segregation)
Cognition z score	-0.02 ± 0.86	0.05	-5.21 to 4.33	0.05 ± 0.85	-0.05 ± 0.86	-0.06 ± 0.87	-0.00 ± 0.85	-0.01 ± 0.85	-0.05 ± 0.87
MSA population size (per 1,000,000)	3.63 ± 5.22	1.23	0.07-21.2	4.02 ± 5.70	3.49 ± 6.35	3.40 ± 3.00	0.83 ± 1.03	4.60 ± 5.70	5.30 ± 5.95
MSA % NHB	21.3%	20.2%	0.2%-51%	10.8%	23.7%	29.3%	21.6%	20.9%	21.4%
Census regions									
Northeast	7.3%			3.8%	18.4%	0.0%	0.7%	0.9%	18.7%
Midwest	18.0%			10.4%	11.8%	31.4%	4.1%	6.3%	40.3%
South	62.5%			49.2%	69.8%	68.6%	83.7%	67.2%	39.7%
West	12.2%			36.6%	0.0%	0.0%	11.5%	25.6%	1.3%
Neighborhood poverty	15.2%	11.6%	0%-82.2%	12.8%	19.4%	23.2%	17.1%	19.5%	16.0%
Age at baseline	64 ± 9	64	45-94	65 ± 9	64 ± 9	64 ± 9	64 ± 9	64 ± 9	64 ± 9
Black	41.6%			28.5%	41.8%	54.2%	27.1%	44.4%	52.0%
Year at baseline			2003-2007						
Female	56.3%			55.7%	56.3%	56.8%	55.3%	58.4%	55.4%
Education									
Less than HS	8.5%			5.6%	9.9%	9.9%	8.1%	7.4%	9.7%
HS	23.5%			20.9%	25.5%	24.1%	23.4%	21.4%	25.4%
More than HS	68.1%			73.5%	64.6%	66.0%	68.6%	71.3%	64.8%
Ever drink	72.7%			73.7%	71.5%	72.8%	70.3%	72.0%	75.4%
Ever smoke	52.9%			51.7%	53.3%	53.6%	51.4%	51.8%	55.0%
Overweight or obese (ref.: underweight or normal)	75.7%			74.1%	75.9%	77.2%	74.9%	75.6%	76.7%
Ever had heart disease	14.3%			14.4%	14.5%	14.1%	15.5%	12.7%	14.7%
Hypertension	55.3%			52.0%	55.8%	58.3%	52.9%	55.6%	57.3%
Diabetes	19.2%			17.2%	20.4%	20.1%	19.4%	18.8%	19.5%
Married	60.5%			63.9%	59.1%	58.5%	66.3%	59.4%	56.4%
Number of adults in household	1.0 ± 0.8	1	0-11	1.0 ± 0.8	0.9 ± 0.8	1.0 ± 0.8	1.0 ± 0.7	1.0 ± 0.8	1.0 ± 0.9

Notes: HS = high school; MSA = metropolitan statistical area; NHB = non-Hispanic Black; T = tertile. Mean ± SDs were included for continuous variables. $n = 18,913$ (73,275 person-years). NHB isolation T1 $n = 6,233$ (24,371 person-years), T2 $n = 6,265$ (24,198 person-years), and T3 $n = 6,415$ (24,706 person-years). NHB dissimilarity T1 $n = 6,165$ (23,758 person-years), T2 $n = 5,884$ (22,817 person-years), and T3 $n = 6,864$ (26,700 person-years).

Table 2. Coefficients From Three-Level Growth Curve Models for the Association Between MSA Isolation and Cognitive Function

	Model 1	Model 2	Model 3	Model 4	Model 5
Intercept (at age 45)	0.250 (0.026)***	0.290 (0.033)***	0.423 (0.058)***	0.525 (0.059)***	0.472 (0.061)***
Age	−0.033 (0.001)***	−0.033 (0.001)***	−0.033 (0.001)***	−0.033 (0.001)***	−0.031 (0.001)***
Black	−0.442 (0.023)***	−0.438 (0.023)***	−0.439 (0.023)***	−0.406 (0.023)***	−0.352 (0.023)***
Black × age	0.004 (0.001)***	0.004 (0.001)***	0.004 (0.001)***	0.004 (0.001)***	0.003 (0.001)***
Female (ref.: male)	0.207 (0.010)***	0.207 (0.010)***	0.207 (0.010)***	0.209 (0.010)***	0.233 (0.011)***
Baseline year	0.013 (0.004)***	0.013 (0.004)***	0.013 (0.004)***	0.014 (0.004)***	0.013 (0.004)***
Education (ref.: less than HS)					
HS	0.331 (0.016)***	0.331 (0.016)***	0.181 (0.055)***	0.169 (0.055)**	0.119 (0.054)*
More than HS	0.649 (0.017)***	0.649 (0.017)***	0.511 (0.052)***	0.497 (0.052)***	0.429 (0.050)***
Isolation		−0.093 (0.048)*	−0.339 (0.105)***	−0.254 (0.121)*	−0.274 (0.120)*
Isolation × less than HS					
Isolation × HS			0.278 (0.102)**	0.280 (0.103)**	0.333 (0.100)**
Isolation × more than HS			0.255 (0.098)**	0.246 (0.098)*	0.295 (0.094)**
MSA population size (per 1,000,000)				0.003 (0.003)	0.003 (0.003)
MSA % NHB				−0.072 (0.108)	−0.087 (0.106)
Census region (ref.: Northeast)					
Midwest				−0.005 (0.026)	−0.011 (0.026)
South				−0.098 (0.027)***	−0.091 (0.026)**
West				−0.056 (0.035)	−0.052 (0.035)
Neighborhood poverty				−0.357 (0.042)***	−0.284 (0.042)***
ICC					
MSA	0.011 (0.002)	0.010 (0.002)	0.010 (0.002)	0.004 (0.001)	0.004 (0.001)
Individual	0.460 (0.005)	0.460 (0.005)	0.460 (0.005)	0.454 (0.005)	0.446 (0.006)

Notes: HS = high school; ICC = intraclass correlation coefficient; MSA = metropolitan statistical area; NHB = non-Hispanic Black. $n = 18,913$ (73,275 person-years). Model 5 included health factors (ever drinking, ever smoking, body mass index, heart disease, hypertension, and diabetes) and social integration (marital status and number of household members).

* $p < .05$. ** $p < .01$. *** $p < .001$.

cognitive function remained between persons, while about 1.1% was between MSAs.

Association Between Isolation and Cognitive Function

Model 2 (Table 2) adds the measure of MSA isolation. Participants who lived in MSAs with greater levels of isolation had lower cognitive function compared to those in MSAs with a lower degree of isolation, net of age, race, gender, education, and baseline interview year ($b = -0.093$, $p < .05$). However, the effect estimate of isolation was almost completely attenuated among those with HS or more than HS (Model 3: $b_{\text{interaction}} = 0.278$ and 0.255 , respectively, $p < .01$), while isolation was adversely related to cognitive function among those with less than HS. The association between isolation and cognitive function did not vary by race, and rates of change in cognitive function did not vary by isolation or education level (see Supplementary Table 1).

Both neighborhood poverty and residing in Southern regions of the United States were associated with lower cognitive function (Model 4: $b_{\text{South}} = -0.098$, $p < .001$; $b_{\text{poverty}} = -0.357$, $p < .001$), but the associations among

isolation, education, and cognitive function were independent of geographic region, neighborhood poverty, and individual characteristics (Models 4 and 5). By the final Model 5, almost all of the variance in cognitive function between MSAs was explained (0.4% remaining), but significant between-person variation remained (ICC = 45%).

Figure 1 displays the predicted cognitive scores for three educational groups by the degree of isolation, based on Model 5 in Table 2. Participants with less than HS education who lived in MSAs with 90th percentile of isolation had lower levels of cognitive function than their counterparts in MSAs with 10th percentile of isolation. Among those with HS or more than HS, there were no differences in cognitive scores by the degree of isolation.

Association Between Dissimilarity and Cognitive Function

Table 3 presents the results from the growth curve models for MSA dissimilarity. Net of individual demographics, participants who lived in MSAs with greater dissimilarity had higher levels of cognitive function (Model 1: $b = 0.147$, $p < .05$). However, this was effectively explained by the

underlying propensity of more dissimilar MSAs to be located in regions outside the Southern United States and to have lower levels of neighborhood poverty, both of which were negatively associated with cognitive function (Model 3). There were no differences in the association between

dissimilarity and cognitive function by educational levels (Model 2) or race (Supplementary Table 2), and dissimilarity was not associated with rates of change in cognitive function (Supplementary Table 2).

Discussion

Despite the growing evidence showing links between socioenvironmental context and cognitive function (Besser et al., 2017; Clarke et al., 2012, 2015; Finlay et al., 2020, 2021; Ng et al., 2018), research on the relationship between racial segregation and cognition remains scarce. To date, only three studies have examined segregation and cognitive function (Aneshensel et al., 2011; Caunca et al., 2020; Kovalchik et al., 2015). Using data from adults in mid to later life, these studies found negative but also null associations between segregation and cognitive function and decline. Conflicting findings may be due to multiple methodological factors, including inconsistencies in considering heterogeneity in effects by race and education, and a focus almost exclusively on neighborhood segregation rather than segregation at the larger metropolitan area. This is the first study to examine the potential cognitive consequences of metropolitan area segregation, which

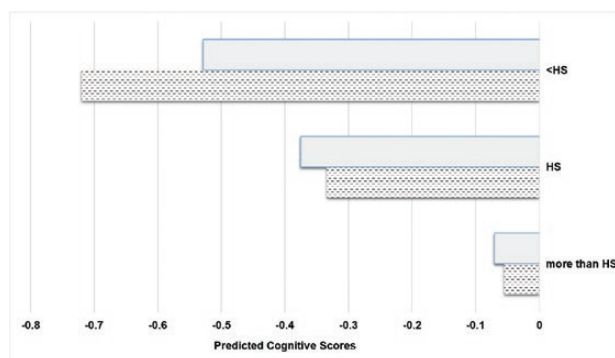


Figure 1. Predicted cognitive scores by REasons for Geographic and Racial Differences in Stroke study participants' educational levels and the degree of MSA NHB isolation. HS = high school; MSA = metropolitan statistical area; NHB = non-Hispanic Black. Solid line: lower isolation: 10th percentile of NHB isolation; dotted line: greater isolation: 90th percentile of NHB isolation. Predicted scores are based on Model 5 in Table 2. All other covariates, including age, were held constant at the mean.

Table 3. Coefficients From Three-Level Growth Curve Models for the Association Between MSA Dissimilarity and Cognitive Function

	Model 1	Model 2	Model 3	Model 4
Intercept (at age 45)	0.170 (0.044)***	0.289 (0.080)***	0.413 (0.055)***	0.335 (0.057)***
Age	-0.033 (0.001)***	-0.033 (0.001)***	-0.033 (0.001)***	-0.031 (0.001)***
Black	-0.444 (0.023)***	-0.445 (0.023)***	-0.406 (0.023)***	-0.350 (0.023)***
Black × age	0.004 (0.001)***	0.004 (0.001)***	0.004 (0.001)***	0.003 (0.001)***
Female (ref.: male)	0.207 (0.010)***	0.206 (0.010)***	0.209 (0.010)***	0.233 (0.011)***
Baseline year	0.014 (0.004)***	0.014 (0.004)***	0.014 (0.004)***	0.013 (0.004)***
Education (ref.: less than HS)				
HS	0.331 (0.016)***	0.183 (0.080)*	0.319 (0.016)***	0.298 (0.016)***
More than HS	0.650 (0.017)***	0.527 (0.072)***	0.630 (0.018)***	0.588 (0.019)***
Dissimilarity	0.147 (0.065)*	-0.053 (0.138)	-0.036 (0.078)	-0.024 (0.076)
Dissimilarity × education				
Dissimilarity × HS		0.251 (0.137)		
Dissimilarity × more than HS		0.208 (0.123)		
MSA population size (per 1,000,000)			0.003 (0.003)	0.003 (0.003)
MSA % NHB			-0.093 (0.066)	-0.079 (0.064)
Census region (ref: Northeast)				
Midwest			-0.005 (0.025)	-0.008 (0.025)
South			-0.098 (0.028)***	-0.091 (0.028)**
West			-0.062 (0.034)	-0.061 (0.034)
Neighborhood poverty			-0.359 (0.042)***	-0.286 (0.042)***
ICC				
MSA	0.010 (0.002)	0.010 (0.002)	0.004 (0.001)	0.004 (0.001)
Individual	0.460 (0.005)	0.460 (0.005)	0.455 (0.005)	0.446 (0.006)

Notes: HS = high school; ICC = intraclass correlation coefficient; MSA = metropolitan statistical area; NHB = non-Hispanic Black. $n = 18,913$ (73,275 person-years). Model 4 included health factors (ever drinking, ever smoking, body mass index, heart disease, hypertension, and diabetes) and social integration (marital status and number of household members).

* $p < .05$. ** $p < .01$. *** $p < .001$.

we argue is the most relevant spatial scale to capture the systematic organization and coordination of the social, transportation, and economic infrastructure relevant for cognitive health.

Using over 10 years of cognitive screening data from a nation-wide, racially diverse cohort of almost 20,000 community-dwelling aging Americans, we found that living in more segregated metropolitan areas (measured using the NHB Isolation Index) was associated with lower levels of cognitive function, but only among those with less than HS; cognitive function was not associated with any measure of segregation among those with HS or higher. These results hold after adjusting for multiple cognitive risk factors at the individual level, many metropolitan population characteristics, and for the poverty composition of the tract of residence. There was no association between segregation and the rate of change in cognitive function with age, nor did the effect estimates of segregation vary by race.

Our findings parallel those of Aneshensel and colleagues (2011) who used data from the U.S. HRS to show that neighborhood segregation was adversely associated with cognitive function among aging Americans with less than HS, but had little (or marginally positive) associations among those with higher levels of education. Our results add further evidence by showing that metropolitan segregation increases the cognitive risks of socioeconomically vulnerable older adults without harming the cognitive advantage of the socioeconomically advantaged. Higher educated older adults may be able to use alternative or compensatory resources to combat the lack of cognitively relevant community resources in more segregated metropolitan areas. For example, individual SES (i.e., education, income) has been shown to be protective against the risk of obesity or poor self-reported health in segregated MSAs (Chang, 2006; Subramanian et al., 2005). Because the isolation index is highly correlated with multiple indicators of concentrated disadvantage (Massey, 1990; Massey et al., 1987), highly isolated MSAs may lack resources that offer opportunities for maintaining cognitive health through health behaviors (Helzner et al., 2007; Weuve et al., 2004; Yaffe et al., 2001). However, higher educated adults living in segregated metropolitan areas may be able to pressure local organizations to provide services in their neighborhoods (Quillian, 2014), combatting the potential adverse cognitive effects that segregation would otherwise confer.

Our findings also show that different dimensions of segregation may have different implications for cognitive health (Massey & Denton, 1988). While the degree of NHB isolation was associated with cognitive function across all models adjusting for covariates, the degree of dissimilarity was not associated with cognitive function after adjusting for the region of the United States and neighborhood poverty. Studies have shown that dissimilarity is not as robustly associated with neighborhood quality (Denton, 1994) and health outcomes (Acevedo-Garcia et al., 2003) compared to other segregation measures. Scholars have argued that

Black isolation is the dimension of segregation most relevant for health because it is “spatially congruent” with concentrated disadvantage across multiple socioeconomic indicators (Acevedo-Garcia et al., 2003; Subramanian et al., 2005).

Contrary to our hypotheses, the association between segregation and cognitive function did not vary by race, nor was either measure of segregation associated with the rate of change in cognitive function with age. Previous research shows that racial inequalities in the association between MSA-level isolation and various health outcomes varies by age (Chang, 2006; Greer et al., 2014; Subramanian et al., 2005). For example, Greer and colleagues (2014) found that living in highly segregated MSAs was associated with increased mortality among Blacks age 35–64, but was not associated with mortality among older Blacks (aged 65 or older) and Whites. This selective survival could explain why we did not find racial differences in the association between segregation and cognition in REGARDS participants (where our sample was age 65 on average at baseline). Exposure to segregation during the period from young adulthood to midlife may be more critical for shaping trajectories of cognitive function, especially among Blacks (Caunca et al., 2020). Thus, rates of cognitive decline across metropolitan areas may already be established among older adults in our sample.

Strengths of this study include the use of multiple, well-established measures of cognitive function assessed repeatedly over 12 years in a large nation-wide biracial cohort of aging Americans. We used multiple measures of segregation at the metropolitan level, which we argue is the best spatial scale for capturing inequality in resources relevant for cognitive health. An additional advantage of using metropolitan segregation rather than neighborhood segregation is that it is less subject from bias due to self-selection into neighborhoods (Quillian, 2014). Despite these strengths, a few limitations should be noted. First, we used the degree of MSA-level segregation at baseline only and were unable to rule out selection bias due to the greater propensity of people at risk for cognitive decline to live in more segregated areas over the life course. Individuals may have experienced different residential contexts earlier in their life course (e.g., young or mid adulthood) or they may have moved to different segregated metropolitan areas after baseline. Residential history data for older adults are needed to better capture MSA exposures over time. Second, while we speculated about potential mechanisms by which segregation could be associated with cognitive health, we were unable to assess these pathways in our data. This is an avenue of research ripe for further investigation. Finally, by using a measure of global cognitive function we were unable to test whether specific domains of cognitive function were more sensitive (or not) to residential segregation.

Nonetheless, this is one of the first studies to investigate the role of metropolitan racial segregation for cognitive

function in a nation-wide study of Black and White aging Americans. Residential segregation has been formed, legislated, and reinforced for long periods of time in history via social and economic institutions such as housing policy and/or urban planning that may be directly relevant for cognitive function. Our findings suggest that living in segregated MSAs is adversely related to cognitive function among those with limited socioeconomic resources. With increasing urbanization, understanding the makeup of metropolitan areas and how they may support cognitive reserve with aging is a potential ecological strategy to address inequalities in cognitive impairment and dementia. Our findings support “upstream” (McKinlay, 1998) interventions that have the capacity to more effectively reduce disparities in cognitive function rather than modifying individual and clinical risk factors (Livingston et al., 2020). Directing attention to the structural, political, and organizational attributes of place may be more effective in ameliorating the burden of dementia in the years to come.

Supplementary Material

Supplementary data are available at *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences* online.

Funding

This research project is supported by cooperative agreement U01 NS041588 cofunded by the National Institute of Neurological Disorders and Stroke (NINDS) and the National Institute on Aging (NIA), National Institutes of Health (NIH), Department of Health and Human Service. Representatives of the NINDS were involved in the review of the manuscript but were not directly involved in the collection, management, analysis, or interpretation of the data. Additional funding was provided by RF1 AG057540 (P. J. Clarke, PI) from NIA. Representatives from NIA did not have any role in the design and conduct of the study, the collection, management, analysis, and interpretation of the data, or the preparation or approval of the manuscript. This work was also supported by the National Cancer Institute (NCI) institutional training grant T32-CA-236621 to Dr. Mullins. The content is solely the responsibility of the authors and does not necessarily represent the official views of NINDS, NIA, NIH, or NCI.

Conflict of Interest

None declared.

Acknowledgments

The authors thank the other investigators, the staff, and the participants of the REGARDS study for their valuable

contributions. A full list of participating REGARDS investigators and institutions can be found at <http://www.regardsstudy.org>.

Author Contributions

J. B. Jang planned the study, conducted statistical analysis, and drafted the manuscript. M. T. Hicken planned the study, supervised statistical analysis, drafted critical sections of the manuscript, and provided critical feedback on manuscript drafts. M. Mullins, M. Esposito, and K. Sol helped to plan the study, assisted with statistical analysis, and provided critical feedback on manuscript drafts. J. J. Manly, S. Judd, and V. Wadley supervised data collection and provided critical feedback on manuscript drafts. P. J. Clarke planned the study, supervised statistical analysis, drafted critical sections of the manuscript, and provided critical feedback on manuscript drafts.

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