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Neighborhood segregation and cognitive change: Multi-Ethnic Study of Atherosclerosis

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Abstract

INTRODUCTION: We investigated associations between neighborhood racial/ethnic segregation and cognitive change.

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METHODS: We used data (n=1,712) from the Multi-Ethnic Study of Atherosclerosis. Racial/ethnic segregation was assessed using Getis-Ord (Gi*) z-scores based on American Community Survey Census tract data (higher Gi*=greater spatial clustering of participant's race/ethnicity). Global cognition and processing speed were assessed twice, six years apart. Adjusted multilevel linear regression tested associations between Gi* z-scores and cognition. Effect modification by race/ethnicity, income, education, neighborhood socioeconomic status, and neighborhood social support was tested.

RESULTS: Participants were on average 67 years old; 43% were White, 11% Chinese, 29% African American/Black, 17% Hispanic; 40% had high neighborhood segregation (Gi*>1.96). African American/Black participants with greater neighborhood segregation had greater processing speed decline in stratified analyses, but no interactions were significant.

DISCUSSION: Segregation was associated with greater processing speed declines among African American/Black participants. Additional follow-ups and comprehensive cognitive batteries may further elucidate these findings.

Keywords

segregation; race; ethnicity; cognition; cognitive decline; processing speed; neighborhood; community; longitudinal; racial; social determinants of health; structural determinants

BACKGROUND

Cognitive impairment that characterizes Alzheimer's disease (AD) and related dementias (ADRD) has a significant cost for individuals, family caregivers, and society. However, the burden of ADRD is not distributed equally across groups, and research suggests that Hispanic and Black older adults are at greater risk for ADRD [1]. Moreover, only a handful of studies have examined ADRD risk in Asian American individuals, although they appear to have lower ADRD incidence [2–4]. Understanding the complex factors associated with cognitive decline in diverse older adults has important implications for public health prevention and intervention efforts. Recently, more attention has been paid to social determinants of health and structural factors that impact health broadly, and specifically, ADRD risk. Racial/ethnic segregation, or the spatial separation of racial/ethnic groups, is a form of structural racism posited to be a fundamental cause of health disparities in older adults [5, 6].

Black segregation in the Northeast and Midwest US started with the migration from the South around the turn of the 19th century. This period paralleled the pattern of other immigrant groups, including Hispanic and Asian American individuals, resulting largely from a desire to live with similar others, thus forming ethnic enclaves [7, 8].

Segregation is associated with separate and unequal access to health care, nutritious food sources, availability of greenspace, and clean unpolluted air, all of which impact risk for cognitive impairment and AD [9]. Racial/ethnic segregation is largely thought to be the product of individual and systemic discrimination and historical redlining practices, including housing discrimination; but it has also been part of the US racial/ethnic assimilation process [10–12].

Findings regarding associations between neighborhood segregation and health are mixed; however, living in ethnic enclaves offers opportunities for employment, connections to cultural and religious institutions, and social support. The advantage of such neighborhoods is demonstrated in findings related to the "Hispanic health paradox" wherein some first-generation Hispanic immigrants live longer than non-Hispanic Whites (NHWs) [12]. Additionally, residents of ethnic enclaves may be more likely to encounter culturally sensitive healthcare that could lead to better health outcomes [12]. However, Hispanic individuals disproportionately experience neighborhood disadvantage, which may have detrimental health consequences [13, 14]. Chinese residents in areas with a higher proportion of Chinese immigrants have been found to have lower socioeconomic status (SES) and lower health insurance coverage compared to those living in areas with fewer foreign-born Chinese individuals [15, 16]. These neighborhoods have been associated with worse walkability, lower neighborhood safety, worse social cohesion, fewer recreational exercise facilities, and lower neighborhood-based civic participation [15].

Few studies have examined the association of racial/ethnic segregation with longitudinal cognitive decline. In a nationally representative sample, greater neighborhood Hispanic composition (via percentage Hispanic) and segregation (via the isolation index) were positively correlated with cognitive function but negatively correlated with cognitive decline for NHW, non-Hispanic Black, and Hispanic older adults [17]. In another study of a diverse older cohort from Northern California, using the Getis-Ord (Gi*) statistic as a measure of racial/ethnic segregation, Black older adults living in areas with greater clustering of Black and Hispanic residents had lower baseline cognitive scores but exhibited no differences in their longitudinal cognitive change [18]. In contrast, Hispanic older adults living in neighborhoods with greater clustering of Hispanic or Black residents and White individuals living in neighborhoods with greater clustering of Hispanic residents demonstrated slower cognitive declines over time. A third study assessing older adults in the Washington Heights Inwood Columbia Aging Project (WHICAP) [19] examined associations between three segregation indices (dissimilarity, isolation, and interaction) [20] and memory, language, and visuospatial function. Black participants living in segregated neighborhoods (i.e., segregation measured via the dissimilarity index) scored worse on language and memory. In contrast, irrespective of the participant's race/ethnicity, living in neighborhoods that were desegregated was associated with higher scores on the three cognitive domains. Finally, in a cohort of Black adults (18- to 30-year-olds at enrollment) from four US regions, the Gi* statistic was used to examine accumulated exposure to segregation (mean value over follow-up) and cognition measured at a single time point using the Digit Symbol Substitution Test (DSST), Stroop color test, and Rey Auditory Verbal Learning Test [21]. Results showed that having more accumulated exposure to segregation in earlier adulthood was associated with worse processing speed in midlife. Given the paucity of longitudinal studies, as well as differing measures of residential segregation, samples, and cognitive outcomes assessed, it remains unclear how racial/ethnic segregation is associated with cognitive trajectories among diverse racial/ethnic groups. To our knowledge, no research studies have examined segregation and late-life cognitive change among Asian Americans.

This study aimed to investigate the association of racial/ethnic segregation on cognitive function cross-sectionally (at baseline) and longitudinally among NHW, Hispanic, African

American/Black, and Chinese older adults from the Multi-Ethnic Study of Atherosclerosis (MESA). We hypothesized that greater neighborhood segregation would be associated with baseline cognition and change in cognition over time and associations would vary by race/ethnicity. This study contributes substantially to the extant literature by including Chinese individuals and a population-based, geographically diverse cohort of older adults from six US cities/regions.

METHODS

Study Population

We obtained data from MESA, a longitudinal cohort study of subclinical cardiovascular disease (CVD) (clinical CVD excluded at baseline) [22]. MESA participants aged 45 to 84 years were enrolled from six US sites (Forsyth County, North Carolina; New York, New York; Baltimore, Maryland; St. Paul, Minnesota; Chicago, Illinois; Los Angeles, California) in 2000–2002 and have completed up to six in-person visits to date. Data collected at each exam included but were not limited to demographics, health history and health status, anthropometry, medications, and several medical assessments/procedures (e.g., imaging, blood pressure). The MESA study protocol was approved by each MESA site's Institutional Review Board and participants provided informed consent.

We restricted to participants with cognitive data at two time points (Exam 5, 2010–2012, and Exam 6, 2016–2018; no cognitive data collected before Exam 5), usable Cognitive Abilities Screening Instrument (CASI) data (excluded CASI missing ≥3 items and scores<20 that lack face validity), and Exam 5 data on neighborhood segregation (Figure 1).

Cognitive measures

Cognitive measures included the CASI (version 2) [23] and the Digit Symbol Coding (DSC) task [24]. CASI measures global cognition by assessing attention, concentration, judgment, orientation, abstraction, short-term and long-term memory, verbal fluency, language, and visual construction, and the DSC measures processing speed (range: 0–100 and 0–133, respectively; higher scores: better cognitive function). Unlike CASI and DSC, the Digit Span test (measuring working memory) was less consistently associated with neighborhood measures in prior cross-sectional studies [25, 26] and thus was excluded from our analyses to reduce multiple comparisons. CASI and DSC z-scores were calculated by subtracting from the participant's score the sample's mean score and dividing by the standard deviation (SD) (from Exam 5).

Neighborhood segregation

Racial/ethnic segregation based on addresses at Exam 5 was measured using the Getis-Ord (Gi*) statistic, which is calculated using a standard formula [27] based on percentage of non-Hispanic Black, Hispanic, non-Hispanic Asian, and non-Hispanic White residents in the US Census tract from 2007–2011 American Community Survey data. The Gi* z-score assesses spatial clustering of racial/ethnic groups for the participant's census tract compared to the racial/ethnic composition of ≥1 neighboring tracts and the broader region (i.e., counties). Larger negative Gi* z-scores indicate underrepresentation of the participant's

own race/ethnicity in their neighborhood and larger positive Gi* z-scores indicate an overrepresentation of their race/ethnicity.

Covariates

Participant characteristics included age (years), sex/gender, marital status (married/living as married versus other), self-reported race/ethnicity (White, Chinese, African American/Black, Hispanic), education (<high school, high school degree, some college, Bachelor's or higher), household family income (<$25,000/year, $25,000–49,999/year, $50,000–74,999/year, or $75,000+/year), and number of residential moves during MESA follow-up. Health-related variables included depressive symptoms (Center for Epidemiological Studies Depression Scale [CES-D] ≥16 versus <16) and the presence/absence of self-reported measures of diabetes, hypertension treated with medication, cardiovascular disease, and cerebrovascular disease (stroke or transient ischemic attack).

Neighborhood characteristics included socioeconomic status (SES) of the US Census tract, which was previously calculated using a principal components analysis of American Community Survey [28] variables (2007–2011) (neighborhood median household income, median home value, percentage households with rental income, and percentage of neighborhood residents with a managerial occupation, an annual household income >$50,000, high school degrees, and bachelor's degrees). Neighborhood population density (persons/km^2) was previously determined from 2010 Census block data for the ½-mile area (buffer) surrounding the residence by summing population densities of included blocks based on the percentage of the buffer in each block. Lastly, an index of the participant's self-reported appraisal of neighborhood social support was calculated by summing scores on four Likert scale questions (1=strongly agree to 5=strongly disagree) of whether neighbors are willing to help others, generally get along, are trustworthy, and share the same values.

Statistical analyses

Analyses.—We described demographic, health, and neighborhood characteristics (e.g., means, SDs) for all participants and stratified by participants' dichotomous Gi* z-scores (Gi*>1.96 versus ≤1.96, where >1.96 is an overrepresentation of participant's race/ethnicity in the neighborhood). Chi-square tests and ANOVA tested for differences in characteristics by Gi* z-score category. Scatterplots confirmed no violation of the linearity assumption. We tested associations between continuous Gi* z-scores and change (years since Exam 5/ initial assessment) in CASI and DSC using adjusted multilevel linear regression (participants clustered in census tracts) with random intercepts and slopes. Minimally adjusted models controlled for age, sex/gender, race/ethnicity, marital status, population density, site, and residential move during follow-up; moderately adjusted models additionally controlled for income, education, and neighborhood SES; and fully adjusted models additionally controlled for comorbidities (diabetes, hypertension, cardiovascular disease, cerebrovascular disease) and depressive symptoms. In addition, we stratified models to determine if associations varied by race/ethnicity because the interpretation of segregation-cognition associations is limited without considering the individual's race/ethnicity. We tested effect modification by race/ethnicity, education (categorical), income (categorical), neighborhood

social support index (discrete), and neighborhood SES (continuous) by including interaction terms in the regression models (e.g., Gi*×neighborhood SES×time).

Inverse probability weights (IPW) were used in all regression models to account for selection/attrition bias due to restricting to individuals still actively followed at Exam 5 with non-missing data. The probability of being in our sample was calculated from logistic regression models including age, sex, education, race/ethnicity, marital status, site, and health status at Exam 1 (body mass index, smoking status, diabetes, and hypertension). The stabilized weights were applied to the regression models based on the inverse of the calculated probabilities.

In sensitivity analyses, we examined whether associations from the main models (no stratification) remained similar using dichotomized Gi* z-scores, which compared associations for individuals with an overrepresentation (Gi*>1.96) of their race/ethnicity in the neighborhood to those with no neighborhood segregation or an underrepresentation of their race/ethnicity. The number of African American/Black, Chinese, and Hispanic participants living in neighborhoods with an underrepresentation of their race/ethnicity was too sparse (n=0, 5, and 10 participants, respectively) to further categorize Gi* z-scores.

RESULTS

The sample (n=1,712) was on average 67 years old (SD=8.3) (Table 1). Fifty-three percent were women, 74% had at least some college education, and 11% were Chinese, 29% African American/Black, 17% Hispanic, and 43% White. Mean CASI and DSC scores at Exam 5 were 89.7 (SD=6.9) and 55.0 (SD=17.0), respectively. Participants living in neighborhoods with an overrepresentation of their own race/ethnicity (compared to neighboring tracts and the county) had less education, were less often White, and were more often of Chinese, African American/Black, or Hispanic race/ethnicity. CASI and DSC scores were slightly lower on average for individuals living in segregated neighborhoods (i.e., their race/ethnicity was overrepresented). Individuals living in neighborhood with greater segregation had lower annual incomes. Mean time between cognitive assessments was 6.3 years (SD=0.5).

On average, participants' neighborhoods were composed of 42% White, 26% African American/Black, 9% Asian, and 21% Hispanic residents (Table 2). Participants living in neighborhoods with a high representation of their own race/ethnicity more often lived in neighborhoods with lower SES, higher population density, and lower self-reported social support. Those living in neighborhoods with an overrepresentation of their own race/ethnicity were more likely to have a greater neighborhood percentage of Asian, African American/Black, or Hispanic residents and lower neighborhood percentage of White residents.

In adjusted regression analyses, greater neighborhood representation of the participant's race/ethnicity was associated with faster annual declines in DSC in minimally, moderately, and fully adjusted models (Table 3). No associations were observed with baseline CASI or DSC scores or longitudinal change in CASI scores. When stratifying the fully adjusted models by race/ethnicity, greater Gi* z-scores were associated with greater declines in DSC

scores among African American/Black participants, but not among other races/ethnicities (Table 4). However, none of the examined interactions were significant (interaction term p-values were >0.05, data not shown).

In sensitivity analyses of the dichotomized Gi* z-scores, no associations were observed for baseline CASI and DSC scores or longitudinal change in scores (Supplemental Table 1). In posthoc analyses, we use similar models (i.e., same covariates) and found no association between neighborhood percentage of African American/Black residents and longitudinal change in DSC (estimate: 0.118, 95% CI: −0.122, 0.358). Thus, among the African American/Black participants, greater segregation of African American/Black individuals but not neighborhood percentage of African Americans/Black residents was associated with greater longitudinal decline in DSC score.

DISCUSSION

We found that African American/Black, Chinese, and Hispanic participants were more likely to live in neighborhoods with an overrepresentation of their own race/ethnicity, and individuals in more segregated neighborhoods had lower global cognition and processing speed scores on average. Overall, greater representation of the participant's race/ethnicity in their neighborhood was associated with a steeper decline in processing speed. Similarly, in race/ethnicity stratified analyses, residential clustering of African American/Black participants was associated with a steeper decline in processing speed (i.e., 0.23 SD difference over 6.3 years average follow-up), but not global cognition. While a 0.23 SD difference is not consistent with a clinical diagnosis of cognitive impairment, subtle differences in cognition are detectable among individuals with normal cognition and underlying Alzheimer's neuropathology [29].

Although some research suggests that residing within an ethnic enclave reduces exposure to social isolation, discrimination, and prejudice–and these factors may reduce the risk of cognitive impairment and decline–we found that segregation was not protective for Black residents and found no associations for the other racial/ethnic groups, even after controlling for important confounders such as SES. Our findings highlight that the impact of racial/ethnic segregation may be nuanced–depending on individual race/ethnicity (as in the current study) or educational level [17]. Differences in historical and recent trends that contribute to racial/ethnic patterning of neighborhoods may contribute to the discrepant results. While some ethnic enclaves may have emerged organically as community members sought to preserve cultural or linguistic practices, others result from ethno-cultural commodification [30] or racial/ethnic discrimination [31]. Differences in these experiences may shape the risk and protective factors for those living in racially/ethnically segregated communities. The historical patterns contributing to predominately Black neighborhoods produced areas of concentrated poverty and fewer resources, which have a myriad of potential health consequences including faster cognitive decline. However, this is the first known study to find this detrimental association between Black segregation and longitudinal change in cognition in older adults, with other studies demonstrating no such association [17, 18].

As mentioned in the introduction, only a handful of studies have examined longitudinal associations between segregation and cognitive decline. Our study mirrors that of Kovalchik et al. [17], which suggested that segregation may negatively impact cognition over time in later life. In contrast, Meyer et al. [18] showed no association between segregation and greater cognitive decline. Unlike our study, which used a single Gi* statistic that referred to neighborhood clustering of the participant's race/ethnicity, Meyer et al. used multiple Gi* statistics specific to the clustering of each race/ethnicity (e.g., how greater clustering of Hispanic residents and greater clustering of Black residents affected cognition by participant race/ethnicity). The difference in findings also may have resulted from differences in the cognitive measures, geographic regions, and populations studied. Caunca et al. found that having more accumulated exposure to segregation in earlier adulthood was associated with worse processing speed in midlife [21]. Although we did not observe associations between greater segregation and baseline processing speed, we did find associations with faster longitudinal decline in processing speed. Considered together, the Caunca et al. study and our study suggest that early and later-life exposure to segregated neighborhoods may be associated with worse processing speed among African American/Black individuals.

We acknowledge several limitations. First, selection bias is possible as cognitive health may drive self-selection into or out of various neighborhoods (e.g., to live closer to family/children for support). While we controlled for many factors thought to be associated with reasons for neighborhood selection (i.e., demographics), we cannot eliminate the possibility of self-selection bias. Relatedly, our study used residential information collected at the same time as baseline cognition. Although we adjusted our models for relocation, residential tenure in a racially/ethnically homogenous neighborhood and historical changes to neighborhood demography may differentially shape the risk and protective factors that contribute to late-life cognition and cognitive decline. For Chinese participants, racial/ethnic segregation was assessed using data for Asian residents combined, since Chinese only data were not available. We also did not account for acculturation or immigration processes, which may influence cognition. Future studies will need to examine potential mediation by neighborhood access to resources, crime, safety, or other factors that may explain the associations. In addition, future studies would benefit from more follow-ups and a more comprehensive battery of cognitive tests covering multiple domains. Supplemental Table 3 demonstrates that individuals excluded due to missing cognitive data at two time points were more likely to be Chinese or Hispanic, have lower educational attainment and lower income, and live in neighborhoods with lower SES and greater racial/ethnic segregation. While IPW helped account for selection bias from excluding those missing cognitive data, residual confounding remains a possibility. Furthermore, the derivation of cognitive z-scores based on the sample mean and SD at Exam 5 may have resulted in attenuated findings. Statistical power may have been reduced for the race-stratified findings and interaction testing. Genetic ancestry would lend a new perspective to these studies to these studies, although we conceptualize race as a social construct, a proxy that is linked to specific life experiences and social exposures and that is informative in the context of segregation. Lastly, while the diversity of MESA participants is a strength, because recruitment balanced certain races/ethnicities by site (e.g., Minnesota recruited only White and Hispanic individuals), this limited our ability to stratify analyses by both site and race/ethnicity. Site-stratified analyses

would be best conducted using multi-city cohorts with large samples of each race/ethnicity by site.

Our study advances prior work in this area by using a population-based, multi-site cohort of African American/Black, Hispanic, Chinese, and White older adults. We used a well-established, formal measure of local spatial association, the Gi* statistic, which considered spatial clustering of races/ethnicities across the regions under study. We controlled for multiple important potential confounders, including individual-level and neighborhood SES. Importantly, the MESA cohort allowed us to investigate whether segregation was associated with cognitive change among Chinese older adults. This is important as processes that affect racial/ethnic segregation may differ for Chinese and other Asian Americans compared to Hispanic and African American/Black older adults.

The mechanistic reasons for different associations with processing speed versus global cognition is unclear. Factors such as stress due to poverty and crime that are associated with neighborhood segregation [32] may differentially impact certain brain regions and cognitive domains including processing speed. For instance, one study found that childhood stressors were associated with poorer processing speed and working memory, but not episodic memory or executive function among 20- to 50-years olds [33]. In addition, segregated neighborhoods may have lower walkability (less walking infrastructure, fewer destinations, more physical disorder) and thus discourage neighborhood-based physical activity. More physical activity has been associated with better processing speed over time in later life [34], and thus serves as another possible mechanism relating segregation and cognition. Global cognition measured by the CASI may not be specific enough to detect brain changes that occur in response to exposure to neighborhood segregation, but this will need to be elucidated in future studies. Further, speed of processing and executive function appear more strongly associated with cardiometabolic disorders than other domains [35, 36]. Thus, future population-based studies should examine associations with longitudinal change in processing speed and executive function, which may provide evidence for cardiometabolic pathways linking segregation and cognitive change.

Future research should seek to disentangle the pathways by which racial/ethnic segregation contributes to cognitive aging outcomes among diverse racial/ethnic groups. Qualitative data assessing residents' perceptions of and experiences with segregation, discrimination, and social cohesion would add to the literature and further delineate the risks and protections of neighborhoods. Reducing structural racism and providing equitable access to resources and opportunities for future generations in Black neighborhoods may address root causes of health disparities [37]. Additionally, identifying the intermediary factors on the causal pathway may increase opportunities for interventions that help healthy brain development in younger age and slow cognitive decline in middle and later age.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

[1]. Tang M-X, Cross P, Andrews H, Jacobs D, Small S, Bell K, et al. Incidence of AD in African-Americans, Caribbean Hispanics, and Caucasians in Northern Manhattan. Neurology. 2001;56(1):49–56. [PubMed: 11148235]

[2]. Mayeda ER, Glymour MM, Quesenberry CP, Whitmer RA. Inequalities in dementia incidence between six racial and ethnic groups over 14 years. Alzheimers Dement. 2016;12(3):216–24. [PubMed: 26874595]

[3]. Mayeda ER, Glymour MM, Quesenberry CP Jr, Whitmer RA. Heterogeneity in 14-year dementia incidence between Asian American subgroups. Alzheimer Dis Assoc Disord. 2017;31(3):181–6. [PubMed: 28406845]

[4]. Borenstein AR, Wu Y, Bowen JD, McCormick WC, Uomoto J, McCurry SM, et al. Incidence rates of dementia, Alzheimer disease, and vascular dementia in the Japanese American population in Seattle, WA: the Kame Project. Alzheimer Dis Assoc Disord. 2014;28(1):23–9. [PubMed: 24045327]

[5]. Williams DR, Collins C. Racial residential segregation: a fundamental cause of racial disparities in health. Public Health Rep. 2001; 116(5):404–16. [PubMed: 12042604]

[6]. White K, Borrell LN. Racial/ethnic residential segregation: framing the context of health risk and health disparities. Health Place. 2011;17(2):438–48. [PubMed: 21236721]

[7]. Iceland J, Nelson KA. Hispanic segregation in metropolitan America: exploring the multiple forms of spatial assimilation. Am Sociol Rev. 2008;73:741–65.

[8]. Rumbaut R, Portes A. Introduction: ethnogenesis: coming of age in immigrant America. In: Rumbaut R, Portes A, editors. Ethnicities: children of immigrants in America. Berkeley: University of California Press; 2001. p. 1–20.

[9]. Landrine H, Corral I. Separate and unequal: residential segregation and black health disparities. Ethn Dis. 2009;19(2):179–84. [PubMed: 19537230]

[10]. Kramer MR, Hogue CR. Is segregation bad for your health? Epidemiol Rev. 2009;31:178–94. [PubMed: 19465747]

[11]. Logan JR. Separate and unequal: the neighborhood gap for Blacks, Hispanics and Asians in Metropolitan America. Project US2010 Report. 2011:1–22.

[12]. Gibbons J, Yang T-C, Brault E, Barton M. Evaluating residential segregation's relation to the clustering of poor health across American cities. Int J Environ Res Public Health. 2020;17(11):3910. [PubMed: 32492811]

[13]. Firebaugh G, Farrell CR. Still large, but narrowing: the sizable decline in racial neighborhood inequality in metropolitan America, 1980–2010. Demography. 2016;53(1):139–64. [PubMed: 26685905]

[14]. Do DP, Frank R, Zheng C, Iceland J. Hispanic segregation and poor health: it's not just Black and White. Am J Epidemiol. 2017;186(8):990–9. [PubMed: 28541384]

[15]. Osypuk TL, Diez Roux AV, Hadley C, Kandula NR. Are immigrant enclaves healthy places to live? The Multi-ethnic Study of Atherosclerosis. Soc Sci Med. 2009;69(1):110–20. [PubMed: 19427731]

[16]. Lim S, Yi SS, Lundy De La Cruz N, Trinh-Shevrin C. Defining ethnic enclave and its associations with self-reported health outcomes among Asian American adults in New York City. J Immigr Minor Health. 2017;19(1):138–46. [PubMed: 26699378]

[17]. Kovalchik SA, Slaughter ME, Miles J, Friedman EM, Shih RA. Neighbourhood racial/ethnic composition and segregation and trajectories of cognitive decline among US older adults. J Epidemiol Community Health. 2015;69(10):978–84. [PubMed: 26002968]

[18]. Meyer OL, Besser L, Mitsova D, Booker M, Luu E, Tobias M, et al. Neighborhood racial/ethnic segregation and cognitive decline in older adults. Soc Sci Med. 2021;284:114226. [PubMed: 34303293]

[19]. Pohl D, Seblova D, Avila J, Dorsman K, Kulick E, Casey J, et al. Relationship between residential segregation, later-life cognition, and incident dementia across race/ethnicity. Int J Environ Res Public Health. 2021;18(21):11233. [PubMed: 34769752]

[20]. Massey DS, Denton NA. The dimensions of residential segregation. Soc Forces. 1988;67:281–315.

[21]. Caunca MR, Odden MC, Glymour MM, Elfassy T, Kershaw KN, Sidney S, et al. Association of racial residential segregation throughout young adulthood and cognitive performance in middle-aged participants in the CARDIA Study. JAMA Neurol. 2020;77(8):1000–7. [PubMed: 32364578]

[22]. Bild DE, Bluemke DA, Burke GL, Detrano R, Diez Roux AV, Folsom AR, et al. Multi-Ethnic Study of Atherosclerosis: objectives and design. Am J Epidemiol. 2002;156(9):871–81. [PubMed: 12397006]

[23]. Teng EL, Hasegawa K, Homma A, Imai Y, Larson E, Graves A, et al. The Cognitive Abilities Screening Instrument (CASI): a practical test for cross-cultural epidemiological studies of dementia. Int Psychogeriatr. 1994;6(1):45–62. [PubMed: 8054493]

[24]. Wechsler D Wechsler Adult Intelligence Scale. Third edition ed. San Antonio TX: The Psychological Corporation; 1997.

[25]. Besser LM, Rodriguez DA, McDonald N, Kukull WA, Fitzpatrick AL, Rapp SR, et al. Neighborhood built environment and cognition in non-demented older adults: the Multi-Ethnic Study of Atherosclerosis. Soc Sci Med. 2018;200:27–35. [PubMed: 29355828]

[26]. Besser L, Galvin JE, Rodriguez D, Seeman T, Kukull W, Rapp SR, et al. Associations between neighborhood built environment and cognition vary by apolipoprotein E genotype: Multi-Ethnic Study of Atherosclerosis. Health Place. 2019;60:102188. [PubMed: 31797769]

[27]. Getis A, Ord JK. The analysis of spatial association by use of distance statistics. Geogr Anal. 1992;24(3):189–206.

[28]. US Census Bureau. American Community Survey (ACS). https://www.census.gov/programs-surveys/acs. Last accessed: 10/31/2021.

[29]. Hassenstab J, Monsell SE, Mock C, Roe CM, Cairns NJ, Morris JC, et al. Neuropsychological markers of cognitive decline in persons with Alzheimer Disease neuropathology. J Neuropathol Exp Neurol. 2015;74(11):1086–92. [PubMed: 26469250]

[30]. Pang C, Rath J. The force of regulation in the land of the free: the persistence of Chinatown, Washington DC as a symbolic ethnic enclave. Res Sociol Organ. 2007;25:191–216.

[31]. Rothstein R The color of law: a forgotten history of how our government segregated America. New York, NY: Liveright Publishing; 2007.

[32]. Williams DR, Collins C. Racial residential segregation: a fundamental cause of racial disparities in health. Public Health Rep. 2001;116(5):404–16. [PubMed: 12042604]

[33]. Saleh A, Potter GG, McQuoid DR, Boyd B, Turner R, MacFall JR, et al. Effects of early life stress on depression, cognitive performance and brain morphology. Psychol Med. 2017;47(1):171–81. [PubMed: 27682320]

[34]. Frederiksen KS, Verdelho A, Madureira S, Bazner H, O'Brien JT, Fazekas F, et al. Physical activity in the elderly is associated with improved executive function and processing speed: the LADIS Study. Int J Geriatr Psychiatry. 2015;30(7):744–50. [PubMed: 25363336]

[35]. Lyall DM, Celis-Morales CA, Anderson J, Gill JM, Mackay DF, McIntosh AM, et al. Associations between single and multiple cardiometabolic diseases and cognitive abilities in 474 129 UK Biobank participants. Eur Heart J. 2017;38(8):577–83. [PubMed: 28363219]

[36]. Gardener H, Caunca M, Dong C, Cheung YK, Rundek T, Elkind MSV, et al. Obesity measures in relation to cognition in the Northern Manhattan study. J Alzheimers Dis. 2020;78(4):1653–60. [PubMed: 33164939]

[37]. Phelan JC, Link BG. Is racism a fundamental cause of inequalities in health? Annu Rev Sociol. 2015;41:311–30.

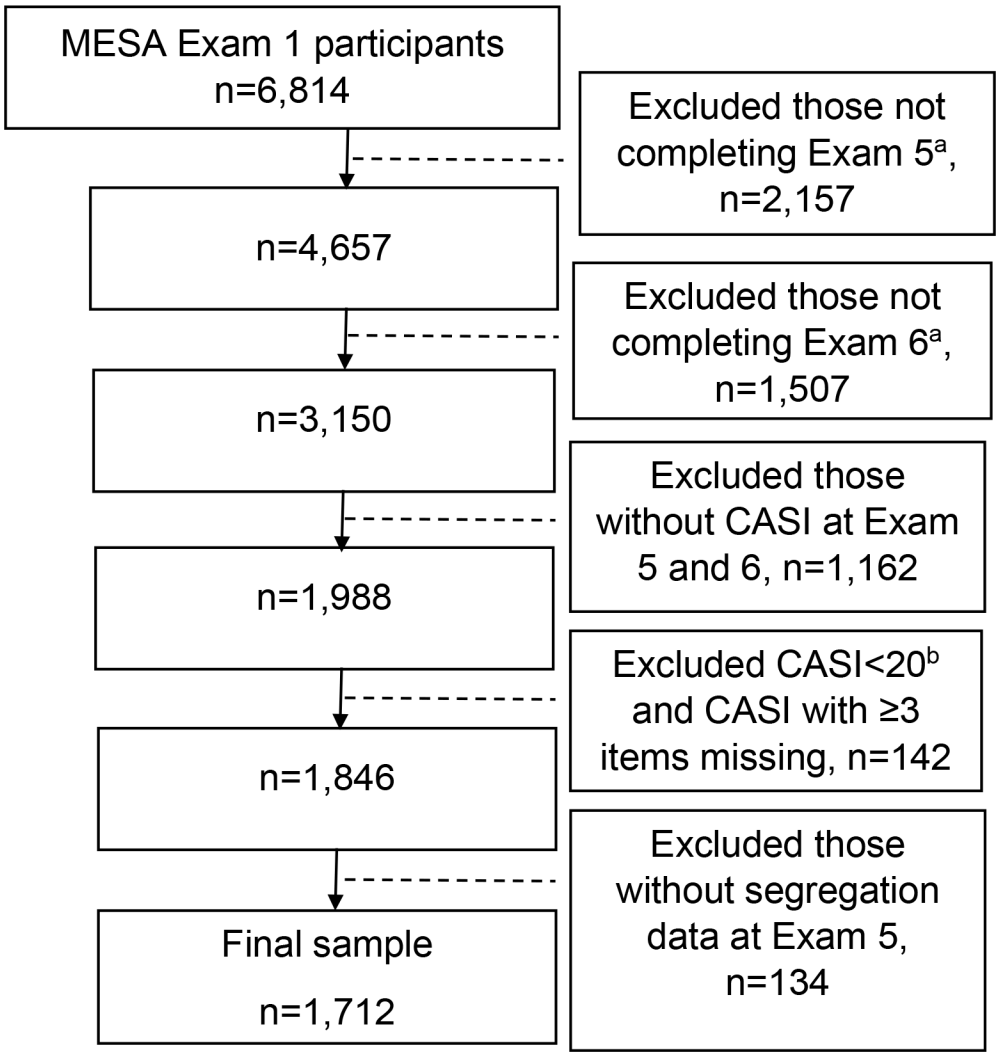

Figure 1.
Sample selection flow chart
[a] Cognitive data only available from Exams 5 and 6
[b] Lacks face validity

Table 1.

Participant characteristics by level of neighborhood racial/ethnic segregation

	Neighborhood segregation			
Characteristic at Exam 5	**Total Sample n=1,712**	**Gi z-score ≤1.96* n=1,031 (60.2%)**	**Gi z-score>1.96† n= 681 (39.8%)**	**p-value**
Age (years), mean (SD)	67.3 (8.3)	67.0 (8.0)	67.8 (8.7)	0.06
Female, n (%)	903 (52.8%)	555 (53.8%)	348 (51.1%)	0.27
Education, n (%)				
< High school degree	166 (9.7%)	64 (6.2%)	102 (15.0%)	<.0001
High school degree	267 (15.6%)	146 (14.2%)	121 (17.8%)	
Some college, no bachelor's degree	519 (30.4%)	327 (31.8%)	192 (28.3%)	
Bachelor's degree or higher	756 (44.3%)	492 (47.8%)	264 (38.9%)	
Race/ethnicity, n (%)				
White	731 (42.7%)	594 (57.6%)	137 (20.1%)	<.0001
Chinese	187 (10.9%)	46 (4.5%)	141 (20.7%)	
African American/Black	496 (29.0%)	260 (25.2%)	236 (34.7%)	
Hispanic	298 (17.4%)	131 (12.7%)	167 (24.5%)	
Married, n (%)	1100 (64.9%)	675 (66.1%)	425 (63.1%)	0.21
Income, n (%)				
<$25,000/year	385 (22.5%)	185 (17.9%)	200 (29.4%)	<.0001
$25,000–49,999/year	469 (27.4%)	270 (26.2%)	199 (29.2%)	
$50,000–74,999/year	352 (20.6%)	227 (22.0%)	125 (18.4%)	
$75,000+	506 (29.6%)	349 (33.9%)	157 (23.1%)	
CES-D≥16, n (%)	213 (12.6%)	124 (12.1%)	89 (13.2%)	0.52
Diabetes, n (%)	157 (9.2%)	92 (9.0%)	65 (9.6%)	0.84
Hypertension medication use, n (%)	855 (49.9%)	502 (48.7%)	353 (51.8%)	0.20
Cardiovascular disease n (%)	101 (5.9%)	58 (5.6%)	43 (6.3%)	0.56
Cerebrovascular disease n (%)	35 (2.1%)	17 (1.7%)	18 (2.6%)	0.16
Moved since Exam 1, n (%)	446 (26.1%)	286 (27.7%)	160 (23.5%)	0.44
CASI, mean (SD)	89.7 (6.9)	90.8 (6.4)	88.1 (7.3)	<.0001
Digit Symbol Coding, mean (SD)	55.0 (17.0)	56.9 (16.1)	52.2 (7.3)	<.0001

SD, standard deviation; CES-D, Center for Epidemiologic Studies Depression scale; CASI, Cognitive Abilities Screening Instrument

*no segregation or race/ethnicity underrepresented in neighborhood

†participant's race/ethnicity overrepresented in neighborhood

Table 2.

Neighborhood characteristics by level of neighborhood racial/ethnic segregation

	Neighborhood segregation			
	Total Sample	**Gi z-score ≤1.96[§]**	**Gi z-score >1.96[¶]**	
	n=1,712	**n=1,031 (60.2%)**	**n= 681 (39.8%)**	
Neighborhood characteristic at Exam 5	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**p-value**
Neighborhood socioeconomic status[*]	−0.53 (1.21)	−0.62 (1.15)	−0.40 (1.27)	0.0002
Population density (people/km^2)	6873 (9708)	5902 (8943)	8343 (10602)	<.0001
Neighborhood racial/ethnic composition[†]				
White	42.1% (0.0, 99.2)	50.1% (3.0, 97.1)	30.0% (0.1, 99.2)	<.0001
African American/Black	26.0% (0.0, 98.9)	22.2% (0.0, 98.2)	31.7% (0.0, 98.9)	<.0001
Asian[a]	9.2% (0.0, 87.2)	7.2% (0.0, 71.6)	12.2% (0.0, 87.2)	<.0001
Hispanic	20.5% (0.1, 98.7)	18.1% (0.2, 91.9)	24.1% (0.1, 98.7)	<.0001
Index measure of neighborhood appraisal of social support[‡]	14.8 (2.3)	15.0 (2.3)	14.5 (2.4)	0.0002

Abbreviations: SD = standard deviation; Gi = Getis-Ord statistic

[*] Derived from principal component analysis of 7 US Census demographic variables (see methods section); more positive=worse

[†] Variable for neighborhood % Asian includes all Asians (e.g., Chinese, Japanese) whereas the MESA cohort only includes Chinese participants

[‡] observed range: 6 to 20; derived from 4 self-reported neighborhood measures (described in methods section)

[§] no segregation or participant's race/ethnicity underrepresented in neighborhood

[¶] participant's race/ethnicity overrepresented in neighborhood

Table 3.

Adjusted association between neighborhood segregation (Gi z-score) and change in cognition in overall sample

		Minimally adjusted model[*,†]		Moderately adjusted model[†,‡]		Fully adjusted model[‡,§]	
Variable	**Outcome**	**Estimate (95% CI)**	**p-value**	**Estimate (95% CI)**	**p-value**	**Estimate (95% CI)**	**p-value**
Yrs[¶]	CASI	−0.07 (−0.14, −0.00)	0.04	−0.08 (−0.15, −0.01)	0.03	−0.06 (−0.13, 0.01)	0.09
Gi z-score[#]		−0.021 (−0.045, 0.004)	0.10	−0.012 (−0.033, 0.008)	0.24	−0.011 (−0.031, 0.009)	0.28
Gi z-score[#]×Yrs[¶]		0.033 (−0.019, 0.026)	0.77	0.000 (−0.022, 0.023)	0.97	−0.004 (−0.027, 0.019)	0.73
Yrs[¶]	DSC	−0.32 (−0.37, −0.27)	<.0001	−0.31 (−0.36, −0.26)	<.0001	−0.31 (−0.36, −0.26)	<.0001
Gi z-score[#]		−0.006 (−0.032, 0.019)	0.63	0.001 (−0.015, 0.016)	0.94	0.000 (−0.016, 0.016)	0.98
Gi z-score[#]×Yrs[¶]		−0.016 (−0.027, −0.004)	0.009	−0.017 (−0.028, −0.005)	0.006	−0.016 (−0.028, −0.003)	0.01

Abbreviation: CASI = Cognitive Abilities Screening Instrument; DSC = Digit Symbol Coding; CI = Confidence Interval; Yrs = Years since Exam 5 cognitive assessment

[*] Controlling for age, sex, race/ethnicity, married, population density, site, moved residences during MESA follow-up

[†] Inverse probability weighted

[‡] Additionally controlling for education, income, and neighborhood SES

[§] Additionally controlling for comorbidities (diabetes, hypertension, cardiovascular disease, cerebrovascular disease); and depressive symptoms

[¶] Years indicates years since the initial cognitive assessment (Exam 5)

[#] Higher Gi z-score: greater overrepresentation of participant's race/ethnicity in neighborhood

Table 4.

Adjusted association between neighborhood segregation (Gi z-score) and change in cognition by participant race/ethnicity

		CASI		DSC	
Variable	**Participant race/ethnicity**	**Estimate (95% CI)*,†,‡**	**p-value**	**Estimate (95% CI)*,†,‡**	**p-value**
Yrs§	White	−0.08 (−0.16, 0.00)	0.05	−0.28 (−0.34, −0.21)	<.0001
Gi z-score¶		−0.008 (−0.042, 0.027)	0.67	0.042 (−0.005, 0.088)	0.08
Gi z-score¶×Yrs§		0.021 (−0.019, 0.061)	0.30	−0.013 (−0.044, 0.018)	0.41
Yrs§	Chinese	0.18 (−0.10, 0.46)	0.20	−0.55 (−0.74, −0.36)	<.0001
Gi z-score¶		−0.009 (−0.037, 0.020)	0.55	−0.009 (−0.034, 0.017)	0.50
Gi z-score¶×Yrs§		−0.007 (−0.060, 0.046)	0.79	0.011 (−0.009, 0.030)	0.28
Yrs§	African American/Black	−0.07 (−0.20, 0.06)	0.27	−0.15 (−0.25, −0.06)	0.001
Gi z-score¶		0.012 (−0.036, 0.059)	0.63	0.009 (−0.034, 0.052)	0.69
Gi z-score¶×Yrs§		−0.002 (−0.037, 0.032)	0.89	−0.036 (−0.067, −0.006)	0.02
Yrs§	Hispanic	−0.03 (−0.23, 0.17)	0.77	−0.49 (−0.61, −0.37)	<0.0001
Gi z-score¶		0.056 (−0.028, 0.139)	0.19	−0.001 (−0.053, 0.051)	0.96
Gi z-score¶×Yrs§		−0.047 (−0.114, 0.019)	0.16	0.008 (−0.028, 0.043)	0.67

Abbreviation: CASI = Cognitive Abilities Screening Instrument; DSC = Digit Symbol Coding; CI = Confidence Interval

*Fully adjusted model controlling for age, sex, race/ethnicity, education, married, income, population density, site, moved residences during MESA follow-up, comorbidities (diabetes, hypertension, cardiovascular disease, cerebrovascular disease), neighborhood SES, depressive symptoms. Results from minimally and moderately adjusted models provided in Supplemental Table 2

†Inverse probability weighted

‡All Gi z-score×race/ethnicity×time interaction terms were insignificant (p>0.05) (data not shown)

§Years indicates years since the baseline cognitive assessment (Exam 5)

¶Higher Gi z-score: greater overrepresentation of participant's race/ethnicity in neighborhood