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# Neighborhood Racial/Ethnic Residential Segregation and Cardiometabolic Risk: The Multi-Ethnic Study of Atherosclerosis

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## Abstract

**Background:** Racial residential segregation has been linked to adverse health outcomes, but associations may operate through multiple pathways. Prior studies have not examined associations of neighborhood-level racial segregation with an index of cardiometabolic risk (CMR), and whether associations differ by race/ethnicity.

**Methods:** We used data from the Multi-Ethnic Study of Atherosclerosis to estimate cross-sectional and longitudinal associations of baseline neighborhood-level racial residential segregation with a composite measure of CMR. Participants included 5,015 non-Hispanic black, non-Hispanic white, and Hispanic participants aged 45-84 years old over 12 years of follow-up (2000-2012). We used linear mixed effects models to estimate race-stratified associations of own-group segregation with CMR at baseline and with the rate of annual change in CMR. Models

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**Competing Interests**: None declared.

**Data sharing statement:** The Multi-Ethnic Study of Atherosclerosis (MESA) has hundreds of investigators, many active scientific working groups (on renal disease, eye disease and other topics) and dozens of ongoing analytic projects. The authors are always looking for outside investigators interested in using the data to answer their research questions. To help interested parties navigate the data and topics and find fruitful collaborations, they encourage you to contact the Coordinating Center or a MESA investigator. Here is a link to the website for more details: http://www.mesa-nhlbi.org/.

adjusted for socio-demographics, medication use, individual-level and neighborhood-level socioeconomic status (SES).

**Results:** In models adjusted for socio-demographics and medication use, high baseline segregation was associated with higher baseline CMR among blacks and Hispanics, but lower baseline CMR among whites. Individual and neighborhood-level SES fully explained observed associations between segregation and CMR for whites and Hispanics. However, associations of segregation with CMR among blacks remained (high versus low segregation: mean difference 0.17 SD units, 95% CI: 0.02, 0.32; medium versus low segregation: mean difference 0.18 SD units, 95% CI: 0.03, 0.33). Baseline segregation was not associated with change in CMR index scores over time.

**Conclusion:** Associations of own-group racial residential segregation with CMR varied by race/ethnicity. After accounting for SES, living in a more segregated neighborhood was associated with greater risk among black participants only.

### Keywords

Racial segregation; cardiometabolic risk; Neighborhood environment

## INTRODUCTION

The health inequalities between socially-constructed racial and ethnic groups are well-documented,[1] however the causes of these disparities are not completely understood. In the United States, racial residential segregation, disproportionately exposes black and Hispanic Americans to neighborhood disadvantage and poorer physical and social environments compared to white Americans[2-4] and may be an important determinant of racial disparities in disease risk. These effects on health may operate through multiple mechanisms, including differential access to economic and occupational opportunities and health-related resources, increased psychosocial stress, and greater exposure to environmental hazards.[2, 3, 5-8]

Studies of racial segregation and health have primarily used metropolitan statistical area (MSA) or county-level measures of segregation. However, this approach does not capture important heterogeneity in segregation experienced by different racial/ethnic groups throughout the MSA. Fewer studies have examined neighborhood-level segregation and health, often using neighborhood racial/ethnic composition as a proxy for segregation.[9, 10] However, these measures do not place each neighborhood's racial/ethnic composition in the context of the overall racial/ethnic composition of the larger MSA,[9, 10] and do not provide information about the racial/ethnic composition of contiguous neighborhoods. As segregation is theorized to result in the clustering of social, economic, and political constraints within a city,[3, 4, 6] a spatial measure of neighborhood-level segregation that captures the clustering of racial/ethnic groups within an MSA may be appropriate.

In addition, associations between segregation and health may vary for different racial/ethnic groups. Racial/ethnic clustering might limit exposure to interpersonal discrimination and allow for maintenance of strong social networks.[11-13] However, segregation of black

Americans is often a result of discrimination in housing and lending practices and accompanied by community disinvestment.[4] This limits socioeconomic opportunities and access to health-promoting resources for black residents of segregated neighborhoods and may increase exposure to social disorder.[14-18] Given these potential differences by race/ethnicity, it is important to investigate the effect of segregation on people of multiple racial/ethnic groups.

Because segregation may affect health through numerous mechanisms with varied physiological effects, it may be appropriate to examine a health outcome that reflects a cumulative, multi-system approach to conceptualizing health. We used data from the Multi-Ethnic Study of Atherosclerosis to examine associations between own-group racial/ethnic residential segregation and an index measure of cardiometabolic risk in non-Hispanic black, non-Hispanic white, and Hispanic adults. Our study extends the previous literature on segregation and health by examining an outcome that has been infrequently studied in the context of segregation, and by characterizing segregation using a neighborhood-level measure, the $G_i^*$ statistic, that contextualizes neighborhood segregation within a larger geographic area.

## METHODS

### Study Population

The Multi-Ethnic Study of Atherosclerosis (MESA) is a multi-site prospective cohort study of 6,814 U.S adults. Participants were aged 45-84 and free of cardiovascular disease at enrollment, and include self-identified White, African American, Hispanic, and Chinese-American adults enrolled from 6 U.S. sites (Chicago, Illinois; Los Angeles, California; New York City, New York; St. Paul, Minnesota; Baltimore, Maryland; and Forsyth County, North Carolina) using population-based sampling.[19] The baseline exams were conducted from July 2000 to August 2002, with four follow-up exams in 2002-2004 (exam 2), 2004-2005 (exam 3), 2005-2007 (exam 4), and 2010-2012 (exam 5). Retention rates were 94.2% at exam 2, 89.2% at exam 3, 86.8% at exam 4, and 75.5% at exam 5. The Institutional Review Board at each site approved the study. All participants provided written informed consent.

As only 28 Chinese participants lived in neighborhoods with low own-group segregation, we restricted our analysis to blacks, whites, and Hispanics. Our analyses included 5,015 participants with complete data on the outcome, exposure, and all covariates at baseline. Out of 6,010 black, white, and Hispanic MESA participants, 597 were excluded for missing baseline segregation, 18 for missing measures needed to calculate baseline cardiometabolic risk, and 380 for missing other baseline covariates.

### Cardiometabolic Risk Index

At each exam, we calculated a cardiometabolic risk (CMR) index composed of cardiovascular and metabolic measures, which included waist-hip ratio, triglycerides, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, glucose, systolic blood pressure, pulse pressure, and resting heart rate.[20] LDL and glucose were restricted to participants who fasted for at least 10 hours. Each measure was top and bottom

coded at the 1st and 99th percentile (calculated separately for each visit). For each indicator, we calculated standardized scores to indicate where individuals' values placed them relative to accepted clinical thresholds. The standardized scores were calculated by subtracting accepted clinical cutpoints from an individual's value for a given indicator and then dividing by the standard deviation (SD) of the indicator in the MESA population at the baseline exam. Clinical cutpoints included 0.90 waist-hip ratio for men and 0.85 for women,[21] 200 mg/dL for triglycerides,[22] 160 mg/dL for LDL cholesterol,[22] 40 mg/dL for HDL cholesterol,[22] 4.84 log of glucose (corresponding to 126 mg/dL),[21] 140 mm Hg for systolic blood pressure,[23] 60 mm Hg for pulse pressure,[24] and 90 beats per minute for heart rate.[25] HDL values were multiplied by −1 to match the direction of the other indicators, such that higher standardized scores for each indicator reflected higher biological risk. The standardized scores were summed to create a total CMR index score for each individual at each exam (range: −19.83 to 9.86). The CMR score was set to missing if participants were missing at least 5 of the 8 components in the score. For participants with fewer than 5 missing components, missing data were imputed using the participant's mean value of that component across all available exams.[20, 26] In total, 1,261 participants required imputation for at least 1 CMR score component. For multivariable models, CMR scores were transformed to z-scores by subtracting the mean CMR score across all participants and dividing by the SD, so model estimates could be interpreted as SD differences in CMR associated with baseline segregation category.

## Racial/ethnic Residential Segregation

Participants' residential addresses were geocoded to the census tract level at each exam and linked to tract-level data on racial/ethnic composition. Data from the 2000 U.S. census was used for years 2000-2004 (corresponding to exams 1, 2, and part of exam 3), the American Community Survey 2005-2009 was used for years 2005-2007 (corresponding to part of exam 3, and exam 4), and the American Community Survey 2007-2011 was used for exam 5.

Neighborhood racial/ethnic segregation was calculated separately for each racial/ethnic group using the local Getis-Ord $G_i^*$ statistic.[27] The $G_i^*$ statistic is a z-score for each census tract indicating the extent to which the racial composition of that tract and its neighbors deviates from the mean racial composition of a larger geographic area (in this case, counties surrounding each MESA site). Higher positive scores reflect greater clustering of a particular racial/ethnic group in that tract compared to the larger geographic area (overrepresentation), scores near 0 indicate racial integration, and lower negative scores reflect underrepresentation relative to the larger area. The contribution of neighboring tracts to the $G_i^*$ statistic were weighted such that tracts farther away from the focal tract exerted less influence compared to closer tracts. The $G_i^*$ statistic, by incorporating information on the racial composition of the larger geographic area in which the neighborhood is located, better reflects the sorting of groups across neighborhoods within a greater area compared to other measures such as racial/ethnic composition of a neighborhood alone. We examined segregation based on each participant's own race/ethnic group, and categorized segregation into three levels: (high: $G_i^*>1.96$, medium: $G_i^*$ 0-1.96, low: $G_i^*<0$). We focused on baseline

segregation because most participants (n=4,098, 82%) had no change in segregation category over follow-up.

## Covariates

Analyses were stratified by self-reported race/ethnicity, which was assessed at the baseline exam. Baseline socio-demographics included age, sex, study site, country of birth (U.S. versus foreign born), duration participants had lived in their current neighborhood (in years), and education (less than high school degree, high school graduate, some college, and college degree or higher). Individual-level time-varying factors included an income-wealth index and medication use. The income-wealth index is a composite measure that incorporates total family income and whether participants owned/were paying mortgage on a home, owned one or more cars, owned land, or owned investments (e.g. stocks, retirement investments) to create a score ranging from 0 (lowest income/wealth) to 8 (highest income/wealth). Details on the index have been published previously.[28, 29] Time-varying medication use included binary indicators of whether a participant was on antihypertensive medications, lipid-lowering medications, or insulin. Finally, a time-varying composite measure of neighborhood socioeconomic status (SES) was calculated based on factor analysis that incorporated the percentage of adult residences with a bachelor's degree, percentage of residents with management/professional occupations, median household income, percentage of households with interest, dividends, or rental income, and median housing value.[30]

## Statistical Analysis

To investigate whether baseline segregation was associated cross-sectionally with CMR at baseline, and whether the rate of change in CMR index scores over follow-up differed by baseline segregation, we used linear mixed effects models with subject-specific random intercepts and time slopes. All models, which were conducted separately for each race/ethnic group, included baseline segregation categories, follow-up time (in years), and an interaction between segregation level and follow-up time. The segregation main effect estimates reflect cross-sectional differences in CMR index scores at baseline by segregation category. The follow-up time main effect estimates reflect one-year change in CMR among participants in the low segregation category (reference group). The interaction tested whether CMR trajectories differed by baseline segregation level. Models were progressively adjusted for covariates. Model 1 adjusted for follow-up time in years, baseline age, sex, country of birth, field center, neighborhood residence duration, and time-varying use of antihypertensive medication, lipid-lowering medication, or insulin. Model 2 additionally adjusted for individual-level SES (baseline education and time-varying income-wealth index). Model 3 additionally adjusted for time-varying neighborhood SES. Interactions were tested between baseline covariates and time for each model and included when significant at the $p<0.05$ level in order to allow time trends in CMR index scores to vary across levels of baseline covariates. All analyses were completed in Stata version 14.2 (StataCorp, College Station, Texas).

## RESULTS

Table 1 presents baseline CMR index z-scores and socio-demographic characteristics by segregation category for each race/ethnic group among the 5,015 included participants. Whites who lived in high own-group segregation neighborhoods had the lowest CMR scores, were more likely to have a bachelor's degree, and have higher neighborhood SES than those in lower own-group segregation categories. Those in medium or high-segregation neighborhoods had higher income-wealth scores and had lived in their neighborhood for less time on average than those in low segregation neighborhoods. In contrast, blacks living in medium and high own-group segregation neighborhoods had a higher baseline CMR score than those in low-segregation neighborhoods. They were less likely to have a bachelor's degree, had lower income-wealth scores and neighborhood SES, had lived in their neighborhood for longer on average, and were more likely to have been born in the U.S. Patterns for Hispanics were similar to those of blacks, with the exception that Hispanics in highly Hispanic segregated neighborhoods were more likely to have been born outside of the U.S.

Table 2 presents results from mixed effects models. Among whites, living in a neighborhood with high white segregation was associated with lower CMR scores at baseline after adjusting for demographics and medication use (difference relative to those living in a low white segregation neighborhood: −0.14 SD units of CMR, 95% confidence interval (CI): −0.28, −01) (Model 1). The association was attenuated to non-significance upon adjustment for individual-level and neighborhood-level SES (Models 2 and 3). For black participants, living in a medium or high black segregation neighborhood was associated with higher CMR scores at baseline (0.22, 95% CI: 0.07, 0.37 for medium versus low and 0.24, 95% CI: 0.09, 0.38 for high versus low). Results were slightly attenuated but still significant upon adjustment for individual SES, and unchanged with further adjustment for neighborhood SES (final differences of 0.18, 95% CI: 0.03, 0.33 for medium versus low and 0.17, 95% CI: 0.02, 0.32 for high versus low). For Hispanics, living in a high Hispanic segregation neighborhood was associated with higher CMR at baseline (0.22, 95% CI: 0.09, 0.35) compared to living in a low segregation neighborhood, but the association was attenuated and non-significant upon adjustment for individual and neighborhood SES. Predicted mean CMR index scores at baseline and after 10 years of follow-up for each segregation category, stratified by race/ethnicity, are shown in Figure 1.

The annual change in CMR among those in the reference category was positive (i.e. higher biological risk) for all three race/ethnic groups in initial models which adjusted for demographics and medication use. This remained unchanged after adjustment for SES for blacks and Hispanics (annual increases of 0.04, 95% CI: 0.02, 0.05 for both black and Hispanic participants, Table 2). Baseline own-group segregation category was not associated with changes in CMR over time for any of the three racial/ethnic groups.

## DISCUSSION

Higher baseline own-group racial residential segregation was associated with higher CMR index scores at baseline among blacks and Hispanics, reflecting greater cumulative

biological risk, and with lower CMR scores among whites after adjusting for demographics and medication use. Adjustment for individual and neighborhood-level SES attenuated associations for whites and Hispanics, suggesting social disadvantage as a pathway through which segregation may influence health. For black participants, adjustment of individual-level SES reduced the magnitude of associations, but segregation remained associated with higher CMR. Baseline segregation was not associated with differences in the rate of CMR change over time for any race/ethnic group.

This study is among the first to examine associations of segregation with a multi-system risk index. Examining multi-system dysregulation has been recommended as a way to investigate the biological pathways by which neighborhood environments "get under the skin."[31, 32] These pathways involve the concept of allostatic load, also known as cumulative biological risk, which is the cumulative wear and tear on the body due to repeated physiological adaptation to social, physical, or chemical stressors.[33] Allostatic load is typically measured as an index using biomarkers reflecting multiple physiological systems.[34] Only one prior study has examined allostatic load in the context of segregation.[35] Bellatore et al found that both unequal distribution of minority groups and the degree of potential contact between minority and majority group members within an MSA were associated with higher allostatic load, using an index which included cardiometabolic and inflammatory factors. However, only MSA-level averages were estimated, which implicitly assumes that the association for individuals across different neighborhoods within an MSA is the same.[35] Although our index was limited to two physiological systems (cardiovascular and metabolic), our study contributes to the literature on segregation and physiological dysregulation by using a neighborhood-level segregation measure that incorporates contextual information on the overall racial-ethnic composition of the MSA and racial clustering in adjacent census tracts. Our segregation measures, by being operationalized at a more localized and proximal level, may be able to capture more relevant contextual attributes than using MSA-level measures. Our results suggest that within an MSA, living in a more racially segregated neighborhood is related to greater CMR for blacks and Hispanics and lower CMR for whites, although the magnitude of associations was small.

The protective association of living in a predominantly white neighborhood for whites seen in our initial models is consistent with findings that whites living in majority white neighborhoods have lower cardiovascular disease incidence[36] and mortality.[37] As in prior work,[36] results were attenuated upon adjustment for SES, suggesting socially constructed advantage and disadvantage as a potential pathway through which neighborhood racial/ethnic composition may influence health. Higher levels of black segregation, in contrast, were associated with higher CMR scores even after adjusting for individual-level and neighborhood-level SES. Most prior studies of segregation and CVD have found higher black segregation to be associated with higher CVD risk.[10] The results of our study are consistent with the idea that racial residential segregation is an important determinant of black-white disparities in health. Black racial residential segregation is considered a fundamental cause of health disparities,[4] as a social exposure that limits access to opportunities for socioeconomic advancement and may be upstream to other aspects of the physical and social environment that impact health, such as access to health care and health promoting resources, and psychological stressors. In a longitudinal cohort study of young to

middle-aged adults, a decrease in exposure to racial residential segregation was associated with within-person reductions in systolic blood pressure among black participants.[38] Our results, in conjunction with prior research, suggest that social policies that reduce segregation, such as affordable housing policies, and improve access to resources of residents of segregated neighborhoods may improve health.

For Hispanics, results of prior studies on segregation and health have been mixed,[10] likely due to the heterogeneous correlates of Hispanic segregation in the United States. Hispanic segregation in immigrant enclaves has been linked to health-reinforcing factors such as access to healthier foods.[39] However, Hispanics have also been found to have greater exposure to neighborhood poverty compared to whites,[40] which makes associations of Hispanic segregation with health challenging to disentangle.

Our finding that baseline segregation was associated with baseline CMR, but not with changes in CMR over time, may reflect the fact that the study population included older adults who had lived in their neighborhoods for an average of 20 years at baseline. It is possible that segregation may primarily influence the accumulation of risk factors earlier in life, but is less impactful on the trajectory of CMR among older adults. This is supported by results of a prior study examining black-white differences in CMR over the lifecourse, which indicated that most of the widening of the gap between blacks and whites occurred prior to age 45 years old.[41] Similar patterns were seen for poverty-income ratio, where differences between individuals with a lower and higher poverty-income ratio widened until approximately age 45 and then remained constant.[41]

Strengths of this study include longitudinal data from a diverse multi-ethnic sample of older adults, which enabled us to examine associations of racial residential segregation and CMR among three different race/ethnic groups. The availability of extensive biomarker data at multiple time points allowed us to examine longitudinal changes in CMR. Also, we used a neighborhood-level segregation measure that incorporated information on surrounding neighborhoods and contextualized segregation scores relative to the overall composition of the larger MSA. An important limitation of our study was that our analyses do not allow us to draw conclusions regarding whether associations of segregation with CMR reflect psychosocial processes or behavioral processes (especially given the fact that components of CMR are related to diet and physical activity behaviors). Another limitation is that our outcome measure did not include inflammatory and neuroendocrine markers or hemostatic markers such as fibrinogen, which have been used previously in studies of cumulative biological risk,[42, 43] as these variables were not available longitudinally. Participants with higher baseline CMR scores were more likely to be lost to follow-up, as were black and Hispanic participants who lived in high segregation neighborhoods, which may have biased associations of segregation and change in CMR toward the null. We were unable to include Chinese participants given lack of variation in segregation, which may limit generalizability. Finally, results may be influenced by residual confounding by SES.

## Conclusion

The link between neighborhood-level own-group racial segregation and CMR varies by race. After adjustment for individual-level and neighborhood-level SES, baseline own-group racial residential segregation was not associated with CMR for whites and Hispanics. However, higher segregation was associated with higher CMR scores among blacks, net of individual-level and neighborhood-level adjustments. No association was observed between segregation and CMR change over time.

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## Abbreviations:

| | |
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| **CMR** | cardiometabolic risk |
| **CI** | confidence interval |
| **HDL** | high-density lipoprotein |
| **LDL** | low-density lipoprotein |
| **MESA** | Multi-Ethnic Study of Atherosclerosis |
| **MSA** | metropolitan statistical area |
| **SD** | standard deviation |
| **SES** | socioeconomic status |

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**What is known on this subject?**

- Racial residential segregation has been described as a fundamental cause of health disparities, but associations between segregation and health may operate through multiple pathways and vary by race/ethnicity.
- Because segregation may affect health through multiple mechanisms, it may be informative to examine associations with an index of wear and tear on the body reflecting both cardiovascular and metabolic systems.
- No prior studies have examined associations of neighborhood-level racial segregation with an index combining multiple cardiometabolic factors.

**What this study adds?**

- Using longitudinal data from 5,015 black, white and Hispanic adults in the Multi-Ethnic Study of Atherosclerosis, we found that living in a neighborhood with higher own-group racial residential segregation was associated cross-sectionally with a higher cardiometabolic risk index score among blacks and Hispanics but lower risk among whites after adjustment for socio-demographics and medication use.
- Socioeconomic status explained associations for white and Hispanic participants, but not for black participants.
- Segregation was not associated with changes in cardiometabolic risk index scores over time for any race/ethnic group.

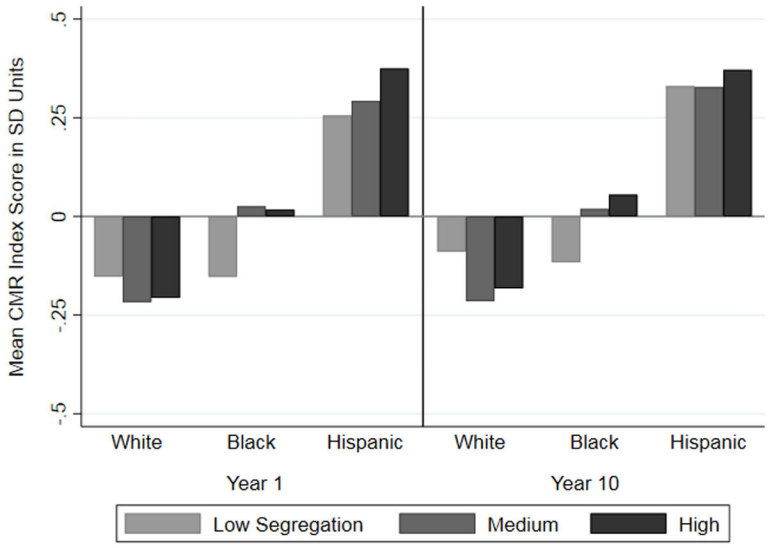


**Figure 1. Predicted mean cardiometabolic risk index scores at baseline and after 10 years of follow-up, by own-group segregation category and race/ethnicity.**

CMR: cardiometabolic risk; SD: standard deviation. CMR scores were converted to z-scores to present results in SD units. A score of 0 indicates the mean CMR score across all participants. Results are marginal predicted mean CMR index scores from race/ethnicity-stratified, adjusted mixed effects linear regression models adjusted for baseline age (centered at 45 years old), baseline age*time interaction, sex, field center, nativity, duration of neighborhood residence, use of antihypertensive medication, lipid-lowering medication, or insulin, education, income-wealth index, and neighborhood socioeconomic disadvantage.

**Table 1.**

Baseline Socio-demographic Characteristics, by Baseline Own-Group Segregation Category and Race, the Multi-Ethnic Study of Atherosclerosis, United States, 2000-2012 (n=5015)[a]

| | Own-Group Racial Residential Segregation Category[e] | | | |
|---|---|---|---|---|
| | Low | Medium | High | *P* value[f] |
| | No. (%) | No. (%) | No. (%) | |
| White participants (n=2291) | | | | |
| No. observations | 1004 | 986 | 301 | |
| Cardiometabolic risk index score[a, b] | −0.14 (1.03) | −0.16 (1.02) | −0.25 (1.02) | 0.2 |
| Age, years[a] | 60.8 (10.2) | 63.3 (9.9) | 63.0 (9.9) | <0.0001 |
| Male gender | 473 (47.1) | 484 (49.1) | 150 (49.8) | 0.6 |
| Education | | | | <0.0001 |
| <High school | 50 (5.0) | 37 (3.8) | 13 (4.3) | |
| High school degree | 176 (17.5) | 163 (16.5) | 29 (9.6) | |
| Some college | 306 (30.5) | 265 (26.9) | 57 (19.0) | |
| Bachelor's degree | 472 (47.0) | 521 (52.8) | 202 (67.1) | |
| Income-wealth index[a,c] | 4.7 (1.8) | 5.4 (1.9) | 5.5 (2.0) | <0.0001 |
| Born outside of United States | 72 (7.2) | 57 (5.8) | 27 (9.0) | 0.1 |
| On antihypertensive medication | 243 (24.2) | 265 (26.9) | 81 (26.9) | 0.3 |
| On lipid-lowering medication | 178 (17.7) | 188 (19.1) | 57 (18.9) | 0.7 |
| On insulin | 11 (1.1) | 5 (0.5) | 3 (1.0) | 0.3 |
| Neighborhood socioeconomic Disadvantage[a,d] | −0.16 (1.19) | −1.37 (1.40) | −2.05 (1.42) | <0.0001 |
| Years lived in neighborhood[a] | 23.2 (15.5) | 18.2 (14.0) | 19.6 (14.1) | <0.0001 |
| Black participants (n=1470) | | | | |
| No. observations | 261 | 355 | 854 | |
| Cardiometabolic risk index score[a,b] | −0.20 (1.10) | 0.01 (1.03) | 0.05 (1.00) | 0.004 |
| Age, years[a] | 60.2 (9.2) | 60.2 (9.5) | 62.1 (10.1) | 0.0008 |
| Male gender | 105 (40.2) | 147 (41.4) | 406 (47.5) | 0.04 |
| Education | | | | 0.02 |
| <High school | 17 (6.5) | 34 (9.6) | 93 (10.9) | |
| High school degree | 42 (16.1) | 70 (19.7) | 164 (19.2) | |
| Some college | 84 (32.2) | 127 (35.8) | 317 (37.1) | |
| Bachelor's degree | 118 (45.2) | 124 (34.9) | 280 (32.8) | |
| Income-wealth index[a,c] | 4.3 (2.2) | 3.8 (2.1) | 3.8 (2.1) | 0.003 |
| Born outside of United States | 35 (13.4) | 55 (15.5) | 50 (5.9) | <0.0001 |
| On antihypertensive medication | 109 (41.8) | 151 (42.5) | 416 (48.7) | 0.05 |
| On lipid-lowering medication | 55 (21.1) | 55 (15.5) | 119 (13.9) | 0.02 |

| | Own-Group Racial Residential Segregation Category[e] | | | |
|---|---|---|---|---|
| | Low | Medium | High | *P* value[f] |
| | No. (%) | No. (%) | No. (%) | |
| On insulin | 4 (1.5) | 13 (3.7) | 17 (2.0) | 0.1 |
| Neighborhood socioeconomic Disadvantage[a,d] | −0.58 (1.31) | −0.06 (1.17) | 0.28 (0.75) | <0.0001 |
| Years lived in neighborhood[a] | 15.0 (12.1) | 17.7 (12.4) | 21.7 (13.9) | <0.0001 |
| Hispanic participants (n=1254) | | | | |
| No. observations | 237 | 218 | 799 | |
| Cardiometabolic risk index score[a,b] | 0.22 (1.07) | 0.37 (1.00) | 0.41 (1.03) | 0.04 |
| Age, years[a] | 61.2 (10.4) | 62.0 (10.4) | 61.3 (10.2) | 0.7 |
| Male gender | 117 (49.4) | 113 (51.8) | 372 (46.6) | 0.3 |
| Education | | | | <0.0001 |
| <High school | 45 (19.0) | 80 (36.7) | 399 (50.0) | |
| High school degree | 56 (23.6) | 37 (17.0) | 171 (21.4) | |
| Some college | 84 (35.4) | 71 (32.6) | 176 (22.0) | |
| Bachelor's degree | 52 (22.0) | 30 (13.7) | 53 (6.6) | |
| Income-wealth index[a,c] | 3.4 (2.3) | 3.1 (2.0) | 2.1 (1.7) | <0.0001 |
| Born outside of United States | 141 (59.5) | 121 (55.5) | 575 (72.0) | <0.0001 |
| On antihypertensive medication | 73 (30.8) | 67 (30.7) | 237 (29.7) | 0.9 |
| On lipid-lowering medication | 37 (15.6) | 27 (12.4) | 113 (14.1) | 0.6 |
| On insulin | 5 (2.1) | 6 (2.8) | 22 (2.8) | 0.9 |
| Neighborhood socioeconomic Disadvantage[a,d] | −1.01 (1.29) | 0.11 (0.68) | 0.94 (0.61) | <0.0001 |
| Years lived in neighborhood[a] | 17.9 (13.0) | 19.1 (13.6) | 19.5 914.4) | |

[a] Values are mean (standard deviation)

[b] Cardiometabolic risk was calculated as an index score combining the following: waist-hip ratio, triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, glucose, systolic blood pressure, pulse pressure, and resting heart rate where higher values indicate higher risk. Scores were transformed into z-scores by subtracting the mean CMR value across the study sample and dividing by the standard deviation.

[c] Ranging 0-8 and defined based on income quintile (0-4) + number of assets (0-4) including home ownership, car ownership, investments, or land ownership. Higher values indicate higher income/wealth.

[d] Based on US census and American Community Survey data. Factor score included % adult residences with bachelor degree, % with management/ professional occupations, median household income, % with interest, dividends, or rental income, and median housing value. A higher value indicates lower socioeconomic status.

[e] Segregation was calculated using the $G_i$* statistic (see Methods for details). Cutpoints for segregation categories were as follows: low: <0; medium: 0-1.96; high: >1.96.

[f] *P* values from chi-squared tests and one-way analysis of variance.

**Table 2.**

Mean Differences in Cardiometabolic Risk (CMR) at Baseline, and Mean Differences in Annual Change Over Follow-up Associated with Own Group Segregation at Baseline, the Multi-Ethnic Study of Atherosclerosis, United States,2000-2012 (n=5015)[a,b,c]

| | Mean Difference in CMR score | | |
|---|---|---|---|
| | **Model 1 β (95% CI)** | **Model 2 β (95% CI)** | **Model 3 β (95% CI)** |
| White participants (N=2291) | | | |
| Association of baseline segregation with baseline CMR | | | |
| Low segregation (reference) | -- | -- | -- |
| Medium segregation | −0.08 (−0.17, 0.08) | −0.07 (−0.16, 0.01) | −0.06 (−0.15, 0.03) |
| High segregation | −0.14 (−0.28, −0.01) | −0.07 (−0.20, 0.06) | −0.05 (−0.19, 0.08) |
| Annual change in CMR for participants in the low segregation Category[d] | 0.03 (0.02, 0.04) | 0.01 (−0.02, 0.03) | 0.01 (−0.02, 0.03) |
| Difference in annual change by baseline segregation category[e] | | | |
| Low segregation (reference) | -- | -- | -- |
| Medium segregation | −0.01 (−0.01, 0.00) | −0.01 (−0.01, 0.00) | −0.01 (−0.01, 0.00) |
| High segregation | 0.00 (−0.01, 0.01) | 0.00 (−0.02, 0.01) | 0.00 (−0.02, 0.01) |
| Black participants (N=1470) | | | |
| Association of baseline segregation with baseline CMR | | | |
| Low segregation (reference) | -- | -- | -- |
| Medium segregation | 0.22 (0.07, 0.37) | 0.18 (0.03, 0.33) | 0.18 (0.03, 0.33) |
| High segregation | 0.24 (0.09, 0.38) | 0.17 (0.03, 0.32) | 0.17 (0.02, 0.32) |
| Annual change in CMR for participants in the low segregation Category[d] | 0.04 (0.02, 0.05) | 0.04 (0.02, 0.05) | 0.04 (0.02, 0.05) |
| Difference in annual change by baseline segregation category[e] | | | |
| Low segregation (reference) | -- | -- | -- |
| Medium segregation | 0.00 (−0.02, 0.01) | 0.00 (−0.02, 0.01) | 0.00 (−0.02, 0.01) |
| High segregation | 0.00 (−0.01, 0.01) | 0.00 (−0.01, 0.01) | 0.00 (−0.01, 0.01) |
| Hispanic participants (N=1254) | | | |
| Association of baseline segregation with baseline CMR | | | |
| Low segregation (reference) | -- | -- | -- |
| Medium segregation | 0.09 (−0.08, 0.26) | 0.03 (−0.13, 0.20) | 0.04 (−0.13, 0.20) |
| High segregation | 0.22 (0.09, 0.35) | 0.11 (−0.03, 0.25) | 0.12 (−0.03, 0.27) |
| Annual change in CMR for participants in the low segregation category[d] | 0.04 (0.02, 0.05) | 0.04 (0.02, 0.05) | 0.04 (0.02, 0.05) |
| Difference in annual change by baseline segregation category[e] | | | |
| Low segregation (reference) | -- | -- | -- |
| Medium segregation | 0.00 (−0.02, 0.01) | 0.00 (−0.02, 0.01) | 0.00 (−0.02, 0.01) |
| High segregation | −0.01 (−0.02, 0.01) | −0.01 (−0.02, 0.01) | −0.01 (−0.02, 0.01) |

CMR, cardiometabolic risk; CI, confidence interval

[a] From mixed effects linear regression with subject-specific random intercepts and time slopes. All models included baseline segregation category, follow-up time in years, and baseline segregation category*follow-up time interaction (to test the hypothesis that change in cardiometabolic risk index scores over time varied by baseline segregation category). Coefficients are presented in units of a standard deviation difference of CBR.

[b] Segregation was calculated for each race/ethnic group using the $G_i^*$ statistic (see Methods for details). Cutpoints for segregation categories were as follows: low: <0; medium: 0-1.96; high: >1.96.

[c] Models were progressively adjusted as follows: Model 1: adjusted for baseline age (centered at 45 years old), baseline age*time since baseline, sex, field center, nativity (foreign-born versus born in United States), duration of residence in neighborhood, and time-varying use of antihypertensive medication, lipid-lowering medication, or insulin. Model 2: Model 1 + education and time-varying income-wealth index. Model 3: Model 2 + time-varying neighborhood socioeconomic disadvantage. In models for white participants only, significant interactions of other baseline covariates with time were present and retained in models: sex*time, field center*time, nativity*time, neighborhood duration*time, education*time.

[d] From the main effect of follow-up time; this is for participants aged 45 years. Due to significant interactions of baseline covariates with time for white participants, this reflects participants in the reference category of each covariate in models for white participants (female, U.S. born, less than high school education, living in neighborhood for 19.9 years at baseline)

[e] Coefficient from the baseline segregation*follow-up time interaction term.