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Exposure to neighborhood-level racial residential segregation in young adulthood to midlife and incident subclinical atherosclerosis in Black adults: The Coronary Artery Risk Development in Young Adults Study:

Reddy, Segregation and Subclinical Atherosclerosis

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Abstract

Background: Neighborhood-level racial residential segregation has been linked to several cardiovascular disease risk factors and outcomes in Black adults, but its impact on subclinical atherosclerosis remains unknown. In addition, although the impact of segregation on health may vary over the life course, most studies have examined segregation exposure at a single point in time. This paper takes a life course approach by examining associations of exposure to neighborhood-level racial residential segregation in young adulthood and patterns of exposure from young adulthood to midlife with coronary artery calcification (CAC) incidence.

Methods: We used data on 1,125 Black Coronary Artery Risk Development in Young Adults (CARDIA) study participants free of CAC. Residential segregation was assessed using the Gi* statistic and measured when participants were young adults (18-30 years old, in 1985-1986) and as the pattern from young adulthood to midlife (15 years later). Poisson regression with generalized estimating equations models was used to measure CAC incidence.

Results: We found participants living in low segregation neighborhoods in young adulthood had 0.52 (rate ratio [RR]; 95% confidence interval [CI]: 0.28, 0.98) times lower risk of developing CAC compared to high segregation after adjusting for young adulthood sociodemographic characteristics and neighborhood poverty. Associations were attenuated and no longer statistically significant with adjustment for midlife CAC risk factors hypothesized to be on the causal pathway (RR: 0.56; 95% CI: 0.29, 1.09). Findings for patterns of segregation over time suggest participants

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None.

living in low segregation neighborhoods in young adulthood were less likely to develop CAC than those who started out in medium/high segregation neighborhoods, regardless of where they lived in midlife (RR for increase from low to medium/high: 0.42; 95% CI: 0.19, 0.95; RR for continuously low vs. continuously medium/high segregation neighborhoods: 0.75; 95% CI: 0.31, 1.83).

Conclusions: We found that participants living in more segregated neighborhoods in young adulthood were more likely to develop CAC due at least in part to differences in CAC risk factor burden accumulated over follow-up.

Keywords

African Americans; atherosclerosis; epidemiology; incidence; racism; residence characteristics; risk factors

Introduction

There are large racial disparities in the US for most cardiovascular disease (CVD) risk factors and outcomes.[1, 2] A mounting body of evidence suggests structural forms of racism like racial residential segregation may play a role.[3–8] Racial residential segregation is the physical separation of race groups into different residential areas. It was fueled by discriminatory housing and lending practices that Black adults faced between the 1930s and 1970s and remains high today.[9, 10] Racial residential segregation is hypothesized to impact health by limiting the available resources necessary for optimal cardiovascular health, such as access to healthcare and healthy food options.[5, 11] In addition, racial residential segregation may limit access to health promoting resources (e.g., pharmacies or physical activity facilities) as well as opportunities for upward socioeconomic mobility.

Studies of racial residential segregation have traditionally operationalized segregation at the metropolitan area level,[9] but interest in assessing the health impact of segregation at the neighborhood level has grown over the last decade.[5, 12] When measured at the metropolitan area level, researchers posit that living in a segregated metropolitan area would be harmful for a Black individual's health regardless of where they lived within that area. In contrast, studies at the neighborhood level are analogous to other studies of neighborhood conditions and examine the health impact of living in neighborhoods that are racially segregated.

While previous studies have linked neighborhood-level racial residential segregation to various CVD risk factors, incident CVD, and CVD mortality, no studies to our knowledge have examined associations of segregation with subclinical atherosclerosis. Coronary artery calcification (CAC) scoring has been identified as a potentially useful way of improving assessments of CVD risk in asymptomatic individuals and as a guide to support preventive treatment decisions.[13] Population-based studies suggest White adults may have more calcified plaque than Black adults, but CAC has been shown to have similar predictive capabilities for both groups.[14] In addition, a study of African Americans found adding CAC to the Framingham Risk Score improved the net reclassification index by over 28% in those without CVD.[15] Thus, investigating subclinical atherosclerosis has the potential

to improve our understanding of the mechanisms by which segregation influences adverse cardiovascular outcomes.

Most studies of segregation and CVD risk have assessed segregation at a single timepoint. This limits our understanding of the impact of segregation on CVD risk at different points in the life course. Few studies have examined the timing of exposure to segregated neighborhoods, but it is conceivable that exposure to segregation earlier in the life course could have a more profound impact on health outcomes. For example, living in segregated neighborhoods in childhood impacts the quality of the education one receives which could have a lasting impact on a variety of CVD risk factors by limiting future earning potential in adulthood.[16] Living in a segregated neighborhood earlier in the life course could also shape eating and physical activity behaviors that persist into older ages and increase CVD risk.

In this study we utilized longitudinal data from the Coronary Artery Risk Development in Young Adults (CARDIA) study to examine associations of racial residential segregation in young adulthood and segregation patterns over time with the development of subclinical atherosclerosis in Black adults. We hypothesized that Black adults living in high segregation neighborhoods in young adulthood would be more likely to develop coronary artery calcification (CAC) than Black adults living in low segregation neighborhoods. Findings from this study will help us better understand the impact of segregation on subclinical CVD over the life course.

Methods

Study Population

Individuals interested in accessing the data set and/or study materials can do so by sending a request to the CARDIA Study Coordinating Center via contact information found on the CARDIA website (www.cardia.dopm.uab.edu). Some anonymized data have been made publicly available at the Biologic Specimen and Data Repository Information Coordinating Center (BioLINCC) and can be accessed at https://biolincc.nhlbi.nih.gov/home/.

The CARDIA study is an observational multicenter prospective cohort study examining the development and determinants of subclinical and clinical CVD risk factors and outcomes.[17] Between 1985 and 1986, 5,115 Black and White men and women between the ages of 18 and 30 were recruited from four different field centers: Chicago, IL, Birmingham, AL, Minneapolis, MI, and Oakland, CA. Institutional Review Board approval for the CARDIA study was obtained at each field center. Participants were selected to obtain approximately equal representation across age, sex, race and education. After baseline, the study population was evaluated 2, 5, 7, 10, 15, 20, 25, and 30 years later with high retention rates across examinations (91, 86, 81, 79, 74, 72, 72, and 71%, respectively). At each evaluation, all participants provided written informed consent for the study.

The underlying factors that sort individuals into racially segregated neighborhoods are different for Black Americans than for White Americans. Given our goal of examining the impact of racial residential segregation as a form of structural racism, we restricted our analyses to self-identified Black men and women free of CAC at the year 15 follow-up

examination who also completed computed tomography (CT) scans during the year 20 or 25 examinations (n=1125). Of the 1730 Black participants who completed the year 15 examination, 354 were excluded for not having a CAC measurement. Another 110 were excluded for missing both the year 20 and year 25 examinations, and 2 more were excluded for missing data on neighborhood measures. An additional 139 participants were excluded for having evidence of CAC at the year 15 examination.

Exposure

Neighborhood-level racial residential segregation was measured using the G_i* statistic,[18] based on geocoded addresses of CARDIA participants linked to U.S. Census data closest to the exam year. Census tracts were used as proxies for neighborhoods. We chose the G_i* statistic because it reflects both the contextual and spatial aspects of segregation. Most studies of neighborhood-level racial residential segregation use racial composition, or the proportion of a race group in a neighborhood, as a proxy for segregation.[5, 12, 19] However, this is limited in that it does not incorporate information on the racial composition of the larger area in which the neighborhood is embedded. Living in a neighborhood that is 30% Black means something different in a city that is 30% Black compared with a city that is 5% Black, and this distinction is captured with the G_i* statistic. This is particularly useful for multi-site studies like CARDIA. In addition, while aspatial measures are based on the racial composition of the neighborhood participants live in, the G_i* statistic also incorporates information on the surrounding neighborhoods. This is important when using administrative boundaries to define neighborhoods, as we are in this study.

The G_i* statistic returns a z-score for each neighborhood (census tract), indicating the extent to which the racial composition (e.g., percent Black) in the tract of residence and neighboring tracts deviates from the mean racial composition of some larger areal unit surrounding the tract (in this case the surrounding metropolitan area or county). A positive z-score indicates higher racial segregation or larger representation/clustering of Black individuals than the mean in the metropolitan area or county. Scores near 0 indicate racial integration, or representation of Black individuals similar to that in the metropolitan area or county, and negative scores suggest lower Black representation/clustering, compared to the larger areal unit.[18]

Segregation was measured in two ways: in young adulthood and as a pattern from young adulthood to midlife. Segregation was modeled categorically in order to assess potentially meaningful changes in exposure over time rather than small fluctuations in G_i*. Consistent with previous studies, segregation levels were grouped into three categories: high (G_i* > 1.96), medium (G_i* 0 - 1.96), and low (G_i* < 0).[7, 8] The cutpoint of 1.96 corresponds to the critical z-score value when using a 95% confidence level (α=0.05). Few participants lived in areas with G_i* <−1.96, so we used a cutpoint of 0 for the low category. A cutpoint of 0 is indicative of a z-score equal to or below the mean racial composition of the surrounding metropolitan area or county.

Segregation categories at the baseline exam were used to operationalize exposure in young adulthood. Segregation patterns over time were measured based on the categories participants fell in at baseline and year 15. They were grouped as follows based on the

distribution of the categories in the study population: Continuously High/Medium (G_i* > 0 maintained over measurements), Declined from High/Medium to Low (G_i* > 0 to G_i* < 0 over measurements), Increased from Low to High/Medium (G_i* < 0 to G_i* > 0 over measurements), and Continuously Low (G_i* < 0 maintained over measurements).

Outcome

CAC was determined by non-contrast CT as previously described in detail.[20, 21] During years 15 and 20, CAC was measured by electron beam CT (Chicago and Oakland) and multidector helical CT (Birmingham and Minneapolis). Helical CT was used for all locations in year 25. At years 15 and 20, duplicate scans were performed , 1-2 minutes apart, whereas a single scan was performed at year 25. Presence of CAC was defined as a lesion with a measure greater than zero Agatston units. Agatston units were defined as a lesion 1.87mm^2 or greater with a density equal or greater than 130 Hounsfield Units.[22] CAC was calculated for all the main coronary arteries (right main, left main, left circumflex, left anterior descending) and was totaled together. Incident CAC was defined as any CAC >0 detected at years 20 or 25 in study participants that were free of CAC at year 15.

Covariates

Baseline (young adulthood) measurements of several individual-level sociodemographic characteristics and neighborhood poverty were adjusted for as potential confounders. The sociodemographic covariates included were age, gender, education (less than high school degree, high school degree, and greater than high school degree), and field center. Neighborhood poverty was defined as the mean percentage of census tract population below the US Census-defined poverty threshold.

Traditional CVD risk factors measured at the year 15 examination (midlife) including smoking status, body mass index, systolic blood pressure, fasting glucose, and total cholesterol were adjusted for as factors that are hypothesized to be on the causal pathway between segregation status and CAC development. Smoking status was defined as current versus not current. Body mass index (BMI) was calculated as kg/m^2. Resting systolic and diastolic blood pressure were measured at three 1-minute intervals using random zero sphygmomanometers and reported as the average of the second and third measurements. Serum glucose was measured at each exam using hexokinase coupled to glucose-6-phosphate dehydrogenase. Total cholesterol was measured enzymatically within 6 weeks of collection.[7, 8 17]

Statistical Analysis

We modeled the associations of two indicators of residential segregation with CAC: 1) segregation in young adulthood (comparing participants living in low and medium segregation neighborhoods to those living in high segregation neighborhoods); and 2) segregation patterns between young adulthood and midlife (comparing participants whose neighborhood segregation score increased from low to high/medium, declined from high/medium to low, or remained low in both time points to those with medium/high segregation at both time points).

We assessed associations between these segregation measures and CAC incidence in year 20 or 25 among participants who did not have CAC in year 15 using Poisson regression with a logarithm link function and generalized estimating equations (GEE) to control for correlation by census tract. We used Poisson rather than logistic regression because CAC incidence was not a rare outcome in this cohort, and thus the odds ratio would tend to overestimate the true relative risk.[23] Model 1 adjusted for baseline age, sex, field center, and educational attainment (at baseline for the young adulthood segregation models and at year 15 for the segregation pattern models). Model 2 additionally adjusted for neighborhood poverty (at baseline for the young adulthood segregation models and at year 15 for segregation pattern models). Model 3 additionally adjusted for CAC risk factors in year 15 (smoking status, alcohol use, body mass index, systolic blood pressure, total cholesterol, and total plasma fasting glucose) to determine whether these risk factors explained associations between segregation and CAC. We used multiple imputation with chained equations to impute missing CAC risk factor values (10 imputed datasets). Authors Mayne, Pool, and Kershaw had access to all the CARDIA study data used in these analyses.

Results

The majority of Black CARDIA participants lived in neighborhoods that fell into the high segregation category in young adulthood (Table 1). Those living in low segregation neighborhoods at baseline had higher levels of education and lived in lower poverty neighborhoods compared with those in high segregation neighborhoods. Participants who lived in low segregation neighborhoods at baseline were less likely to be current smokers and more likely to drink alcohol in midlife. They also had lower systolic blood pressure and higher total cholesterol.

For segregation patterns, participants who continuously lived in low segregation neighborhoods were slightly older and had higher levels of educational attainment compared with the other groups (Table 2). Those who continuously lived in high/medium segregation neighborhoods were most likely to have less than a high school degree at baseline. Participants who lived in high/medium segregation neighborhoods in young adulthood lived in higher poverty neighborhoods than those who lived in low segregation neighborhoods, regardless of the level of segregation in their neighborhoods in midlife. Current smoking prevalence was highest and alcohol use was lowest among those living in continuously high/medium segregation neighborhoods. Fasting glucose and systolic blood pressure were lowest among those living in continuously low segregation neighborhoods.

Participants who developed CAC over follow-up had similar individual-level sociodemographic characteristics and levels of neighborhood poverty in young adulthood as those who did not develop CAC (Table 3). Participants who did not develop CAC were less likely to be current smokers in midlife. They also had the lowest levels of fasting glucose, systolic blood pressure, and total cholesterol.

Participants living in low segregation neighborhoods in young adulthood had a 0.50 (rate ratio [RR]; 95% confidence interval [CI], 0.27, 0.92) times lower risk of developing CAC after adjusting for sociodemographic covariates when compared to participants in the

high segregation category (Table 4). These associations persisted with further adjustment for neighborhood poverty (RR: 0.52; 95% CI: 0.28, 0.98) but were attenuated and no longer statistically significant with adjustment for midlife CAC risk factors (RR: 0.56; 95% CI: 0.29, 1.09). There were no differences in incident CAC between participants living in medium segregation neighborhoods and high segregation neighborhoods in young adulthood.

Participants living in low baseline segregation neighborhoods and then medium or high segregation neighborhoods later on had a 0.43 (95% CI: 0.19, 0.99) times lower risk of developing CAC over follow-up after adjustment for sociodemographic characteristics and neighborhood poverty, compared to participants living in high/medium segregation neighborhoods. This association was slightly attenuated and lost statistical significance with further adjustment for midlife CAC risk factors. While not statistically significant, those living in continuously low segregation neighborhoods had a 0.77 (95% CI: 0.31, 1.92) lower risk of developing CAC after adjustment for sociodemographic characteristics and neighborhood poverty; this was further attenuated with adjustment for CAC risk factors.

Discussion

We found that participants who started out in low segregation neighborhoods as young adults were less likely to develop CAC than those who started out in high segregation neighborhoods after adjusting for sociodemographic characteristics and neighborhood poverty. The nature of the findings were similar when we looked at patterns of segregation over time, such that those living in low segregation neighborhoods in young adulthood (the baseline examination) were less likely to develop CAC over follow-up regardless of the type of neighborhood they lived in later (low segregation or medium/high segregation neighborhoods). Our results also demonstrated that associations between racial residential segregation and CAC in young adulthood were attenuated with adjustment for traditional CAC risk factors, suggesting this may be a key underlying mechanism.

This is the first study to our knowledge to examine associations of racial residential segregation with CAC incidence, but our results were consistent with some previous research on racial residential segregation and cardiovascular outcomes. A study using data from the Multi-Ethnic Study of Atherosclerosis found Black adults living in more segregated neighborhoods were more likely to develop CVD over follow-up.[6] Other studies of segregation and CVD have focused on cardiovascular mortality, with mixed findings.[4, 24, 25] A cross-sectional study across metropolitan areas in the U.S. found that Black adults living in more segregated areas were more likely to die from heart disease.[4] In contrast, a Texas study found that Black adults who lived in more segregated (i.e., predominantly Black) neighborhoods lost fewer years of life to heart disease compared with those who lived in neighborhoods with fewer Black residents.[25] In addition, a New York City study found age-adjusted annual CVD mortality rates were higher in Black adults who lived in predominantly White zipcodes (≥ 75% White individuals) compared with Black adults who lived in predominantly Black zipcodes (≥ 75% Black individuals).

Differences in findings could be due to several factors including heterogeneity in how segregation was measured, location of the studies, and age differences between the study populations.

A comparison of our findings for patterns of segregation versus segregation exposure in young adulthood suggests that living in a low segregation neighborhood in young adulthood was protective, even after moving to medium or highly segregated neighborhoods. This suggests neighborhood segregation could act as a catalyst for the premature onset of various cardiovascular disease risk factors, leading to the development of CAC. This is consistent with our findings that associations were attenuated with adjustment for traditional CAC risk factors measured at the year 15 examination.

One pathway that could account for this relationship is the lasting impact segregation can have on earning potential and health behaviors, both of which are associated with the traditional CAC risk factors included in this study.[26–28] Schools in more segregated neighborhoods tend to be under-resourced and often perform more poorly than schools in less segregated areas.[29] Housing values in segregated neighborhoods also tend to be lower, resulting in lower family wealth accumulation.[30] Poorer quality education and opportunities for wealth accumulation could have an adverse impact on a young person's future educational attainment and potential job opportunities.[11, 31] They could also influence the types of foods and recreational activities participants were exposed to as children and/or young adults, which could shape their future health behaviors.

Another pathway that could account for this relationship is early activation of stress pathways, and the resulting biological and behavioral effects that could lead to premature CAC risk factor burden and CAC development.[32, 33] For example, violent crime rates and fatal police shootings are higher in racially segregated neighborhoods compared with non-segregated neighborhoods.[34–36] Higher exposure to violence and other psychosocial stressors may result in dysregulation of the body's stress response systems, including the hypothalamic-pituitary adrenal axis and immune response systems.[37] Multiple manifestations of this dysregulation, such as chronic inflammation and visceral fat accumulation, are associated with CAC risk factors.[38, 39] Stressful experiences can also increase CAC risk through the behaviors adopted as coping mechanisms, such an unhealthy eating or cigarette smoking.[40, 41]

Differential access and exposure to certain environmental factors could play a role as well. For example, studies have shown marginalized communities are more likely to be exposed to ambient air pollution,[42, 43] which has been associated with CAC risk, possibly due to increased oxidative stress and inflammation.[44] Further work is needed to better understand environmental pathways linking segregation to CAC in particular, and CVD risk more broadly. This study is not without limitations. One is that, due to small numbers, we needed to combine medium and highly segregated categories when looking at patterns over time. In addition, the continuously low segregation group was quite small. These issues may have limited our ability to discern specific relationships between segregation over time although outcomes were similar to the young adulthood findings. Another is that we do not have residential history information, so we do not know whether or not participants grew

up in the neighborhoods they lived in as young adults during the baseline examination. In addition, although we adjusted for hypothesized confounders at the individual and neighborhood levels, residual confounding remains a potential concern. Lastly, the CARDIA population is not necessarily representative of the national population, which decreases the generalizability of our findings.

Future studies are needed to better understand causal relationships between segregation and CVD risk. Cohort studies that sample participants to ensure variation in neighborhood characteristics, along with race/ethnicity and socioeconomic status, will better enable researchers to disentangle the inter-related impact of race/ethnicity, place, and class on health.[45] In addition, natural experiments that leverage the exogenous changes in segregation exposure due to policy changes, such as those that generate affordable housing opportunities, will also help strengthen causal inferences around the impact of segregation on health.[46] Finally, agent-based or other dynamic simulation modeling approaches can account for the complex interactions among people and between people and their environments that could impact the relationship between segregation and CVD risk in a way that traditional regression-based approaches cannot.[47]

Conclusions

The results of this study add to the growing literature that racial residential segregation is a key contributor to Black-White cardiovascular health disparities. [6–8, 48] Further, the multiple pathways through which residential segregation could increase CAC risk highlight the critical need to tackle structural racism in the United States. Our findings suggest policies that address root causes of social and economic inequities like racial residential segregation have the potential to alleviate the unequal burden of CVD risk factors and outcomes that plague Black Americans.

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Non-standard Abbreviations and Acronyms

CAC	coronary artery calcification
CVD	cardiovascular disease
CARDIA	Coronary Artery Risk Development in Young Adults
CT	computed tomography
BMI	body mass index
GEE	generalized estimating equations

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Table 1.

Selected Characteristics of Study Participants by Segregation Category in Young Adulthood

	High Segregation N=909	Medium Segregation N=143	Low Segregation N=73
Sociodemographic Characteristics in Young Adulthood			
Age (years) – mean (standard error)	24.3 (0.1)	25.3 (0.3)	24.8 (0.4)
Male Sex – %	37.8	39.2	45.2
Field Center – %			
Birmingham	28.5	28.7	9.6
Chicago	24.3	19.6	49.3
Minneapolis	17.2	13.3	5.5
Oakland	30.0	38.5	35.6
Educational Attainment – %			
<High School Degree	9.6	6.3	5.5
High School Degree	36.4	34.3	27.4
>High School Degree	54.0	59.4	67.1
Percent of census tract population below poverty threshold – mean (standard error)	26.3 (0.4)	16.6 (0.7)	12.6 (1.1)
Coronary Artery Calcification Risk Factors in Midlife			
Current Smoking – %	23.3	22.4	21.9
Current Alcohol Use – %	69.2	73.4	84.9
Body mass index – mean (standard error)	30.3 (0.2)	30.5 (0.6)	28.8 (0.7)
Fasting glucose – mean (standard error)	93.6 (0.7)	97.4 (2.9)	91.5 (2.4)
Systolic blood pressure – mean (standard error)	116.1 (0.5)	117.2 (1.3)	113.1 (1.5)
Total cholesterol – mean (standard error)	181.0 (1.1)	186.0 (3.0)	186.8 (3.8)

Table 2.

Selected Characteristics of Study Participants by Segregation Pattern between Young Adulthood and Midlife

	Continuously High/ Medium Segregation N=853	Declined from High / Medium to Low Segregation N=199	Increased from Low to High / Medium Segregation N=52	Continuously Low Segregation N=21
Sociodemographic Characteristics in Young Adulthood				
Age (years) – mean (standard error)	24.5 (0.1)	24.3 (0.2)	24.5 (0.5)	25.7 (0.8)
Male Sex – %	38.7	35.2	50.0	33.3
Field Center – %				
Birmingham	29.9	22.6	5.8	19.1
Chicago	23.8	23.1	55.8	33.3
Minneapolis	16.4	17.6	7.7	0.0
Oakland	29.9	36.7	30.8	47.6
Educational Attainment – %				
<High School Degree	9.9	6.0	5.8	4.8
High School Degree	37.0	32.2	32.7	14.3
>High School Degree	53.1	61.8	61.5	80.9
Percent of census tract population below poverty threshold – mean (standard error)	25.1 (0.5)	24.2 (0.9)	13.4 (1.4)	10.6 (1.6)
Coronary Artery Calcification Risk Factors in Midlife				
Current Smoking – %	24.2	20.7	23.1	19.1
Current Alcohol Use – %	68.6	74.9	86.5	81.0
Body mass index – mean (standard error)	30.5 (0.2)	29.6 (0.4)	28.7 (0.9)	29.0 (1.2)
Fasting glucose – mean (standard error)	94.1 (0.7)	94.3 (1.7)	92.6 (3.2)	88.8 (2.0)
Systolic blood pressure – mean (standard error)	116.5 (0.5)	115.0 (1.0)	114.5 (1.8)	109.6 (2.7)
Total cholesterol – mean (standard	181.3 (1.2)	183.6 (2.3)	186.7 (4.5)	187.0 (7.1)

Table 3.

Selected Characteristics of Study Participants by Coronary Artery Calcification Development

	Developed coronary artery calcification during follow-up N=257	Did not develop coronary artery calcification during follow-up N=868
Characteristics in Young Adulthood		
Age – mean (standard error)	25.3 (0.2)	24.2 (0.1)
Male Sex	49.0	35.4
Field Center		
Birmingham	24.5	28.1
Chicago	28.8	24.3
Minneapolis	14.8	16.2
Oakland	31.9	31.3
Educational Attainment		
<High School Degree	9.3	8.8
High School Degree	36.2	35.4
>High School Degree	54.5	55.8
Percent of census tract population below poverty threshold – mean (standard error)	24.8 (0.8)	23.9 (0.5)
Coronary Artery Calcification Risk Factors in Midlife		
Current Smoking – %	32.6	20.5
Current Alcohol Use – %	69.7	71.1
Body mass index – mean (standard error)	31.9 (0.5)	29.8 (0.2)
Fasting glucose – mean (standard error)	99.3 (2.1)	92.4 (0.6)
Systolic blood pressure – mean (standard error)	121.5 (1.2)	114.4 (0.5)
Total cholesterol – mean (standard error)	188.4 (2.1)	180.1 (1.1)

Table 4.

Associations of Exposure to Neighborhood-Level Racial Residential Segregation with Incident Coronary Artery Calcification*

Segregation in Young Adulthood+			
	Model 1 Rate Ratio (95% Confidence Interval)	**Model 2 Rate Ratio (95% Confidence Interval)**	**Model 3 Rate Ratio (95% Confidence Interval)**
High Segregation in Young Adulthood	Reference	Reference	Reference
Medium Segregation in Young Adulthood	1.01 (0.75, 1.36)	1.04 (0.75, 1.42)	0.96 (0.66, 1.39)
Low Segregation in Young Adulthood	0.50 (0.27, 0.92)	0.52 (0.28, 0.98)	0.56 (0.29, 1.09)
Segregation Patterns from Young Adulthood to Midlife			
	Model 1 Rate Ratio (95% Confidence Interval)	**Model 2 Rate Ratio (95% Confidence Interval)**	**Model 3 Rate Ratio (95% Confidence Interval)**
Continuously High/Medium Segregation	Reference	Reference	Reference
Declined from High / Medium to Low Segregation	1.17 (0.89, 1.55)	1.17 (0.89, 1.55)	1.29 (0.94, 1.77)
Increased from Low to High / Medium Segregation	0.42 (0.19, 0.95)	0.43 (0.19, 0.99)	0.47 (0.20, 1.07)
Continuous Low Segregation	0.75 (0.31, 1.83)	0.77 (0.31, 1.92)	1.00 (0.36, 2.74)

*Estimated using Poisson regression with generalized estimating equations to account for clustering by census tract in young adulthood (baseline; 1985-1986). This model included participants who did not have coronary artery calcification in midlife (year 15 follow-up examination; N=1,125). Those who developed coronary artery calcification by year 20 or 25 were considered to have incident coronary artery calcification.

+Models were progressively adjusted: Model 1: baseline age, sex, field center, and baseline education. Model 2: Model 1 + baseline neighborhood poverty. Model 3: Model 2 + year 15 potential individual-level CAC risk factors (body mass index, smoking, alcohol use, fasting glucose, systolic blood pressure, and total cholesterol).