Neighborhood-Level Racial/Ethnic Residential Segregation and Incident Cardiovascular Disease: The Multi-Ethnic Study of Atherosclerosis

Running title: Kershaw et al.; Residential segregation and cardiovascular disease

Kiarri N. Kershaw, PhD¹; Theresa L. Osypuk, PhD²; D. Phuong Do, PhD³;
Peter J. De Chavez, MS¹; Ana V. Diez Roux, MD PhD⁴

¹Dept of Preventive Medicine, Northwestern University Feinberg School of Medicine, Chicago, IL; ²Division of Epidemiology & Community Health, University of Minnesota School of Public Health, Minneapolis, MN; ³Depts of Public Health Policy & Administration, and Epidemiology, University of Wisconsin-Milwaukee, Milwaukee, WI; ⁴Dept of Epidemiology and Biostatistics, Drexel University School of Public Health, Philadelphia, PA

Address for Correspondence:
Kiarri N. Kershaw, PhD
Northwestern University
680 N Lake Shore Drive, Suite 1400
Chicago, IL 60611
Tel: 312-503-4014
Fax: 312-908-9588
E-mail: k-kershaw@northwestern.edu

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Abstract

Background—Previous research suggests neighborhood-level racial/ethnic residential segregation is linked to health, but it has not been studied prospectively in relation to cardiovascular disease (CVD).

Methods and Results—Participants were 1,595 non-Hispanic Black, 2,345 non-Hispanic White, and 1,289 Hispanic adults from the Multi-Ethnic Study of Atherosclerosis free of CVD at baseline (ages 45-84). Own-group racial/ethnic residential segregation was assessed using the Gini statistic, a measure of how the neighborhood racial/ethnic composition deviates from surrounding counties’ racial/ethnic composition. Multivariable Cox proportional hazards modeling was used to estimate hazard ratios (HR) for incident CVD (first definite angina, probable angina followed by revascularization, myocardial infarction, resuscitated cardiac arrest, CHD death, stroke, or stroke death) over 10.2 median years of follow-up. Among Blacks, each standard deviation increase in Black segregation was associated with a 12% higher hazard of developing CVD after adjusting for demographics (95% Confidence Interval (CI): 1.02, 1.22). This association persisted after adjustment for neighborhood-level characteristics, individual socioeconomic position, and CVD risk factors (HR: 1.12; 95% CI: 1.02, 1.23). For Whites, higher White segregation was associated with lower CVD risk after adjusting for demographics (HR: 0.88; 95% CI: 0.81, 0.96), but not after further adjustment for neighborhood characteristics. Segregation was not associated with CVD risk among Hispanics. Similar results were obtained after adjusting for time-varying segregation and covariates.

Conclusions—The association of residential segregation with cardiovascular risk varies according to race/ethnicity. Further work is needed to better characterize the individual- and neighborhood-level pathways linking segregation to CVD risk.

Key words: epidemiology, cardiovascular events, race and ethnicity, neighborhoods
Introduction

The spatial distribution of racial/ethnic groups in U.S. metropolitan areas is highly patterned as a consequence of a complex set of social, cultural, and economic forces that also lead to the differential distribution of and exposure to resources and opportunities across space by race/ethnicity.\textsuperscript{1,2} Despite discrimination and other exclusionary housing practices having been outlawed for over 50 years, racial/ethnic residential segregation in U.S. metropolitan areas remains high; the average White individual lives in a neighborhood that is 75% White while the average Black and Hispanic individuals, who make up approximately 13 and 16% of the U.S. population, respectively,\textsuperscript{3} live in neighborhoods that are only 35% White.\textsuperscript{4} Predominantly minority neighborhoods are disproportionately higher in poverty than predominantly White neighborhoods.\textsuperscript{5} It has been proposed that racial/ethnic residential segregation may lead to adverse cardiovascular (CVD) outcomes for minorities through multiple mechanisms, including limiting opportunities for socioeconomic mobility, access to health-promoting resources, exposure to safe areas, and access to quality health care.\textsuperscript{2,6} However, as the ethnic density hypothesis posits, segregation may also confer beneficial health outcomes for minorities by fostering strong social networks, reinforcing social control, and shielding minorities from exposure to prejudice and discrimination.\textsuperscript{7}

While studies examining associations of metropolitan-level segregation with mortality have largely found that higher metropolitan area segregation is associated with worse outcomes among Blacks,\textsuperscript{8-12} findings from studies investigating the relationship between segregation and mortality across neighborhoods within cities (herein referred to as neighborhood-level segregation) are less clear.\textsuperscript{13-19} Existing studies of neighborhood-level segregation and cardiovascular outcomes have largely used death certificate data to examine associations of
racial composition with CVD mortality rates, and findings generally indicate higher segregation is related to lower mortality particularly among older Blacks.\textsuperscript{13-15} However, these studies are limited by their inability to capture non-fatal cardiovascular outcomes and minimal adjustment for individual-level confounders.

Moreover, studies of segregation and health have largely focused on Blacks.\textsuperscript{20} Few have examined the impact of segregation on health for other minority groups, and none to our knowledge have examined associations of segregation with cardiovascular outcomes. Given that the Hispanic population is projected to double, to comprise almost one-third of the U.S. population by 2060,\textsuperscript{21} there is a strong imperative to also investigate the impact of residential segregation in Hispanics.

Using data from the Multi-Ethnic Study of Atherosclerosis (MESA), we prospectively examined associations of own-group neighborhood-level racial/ethnic residential segregation with incident cardiovascular disease in non-Hispanic Black, non-Hispanic White, and Hispanic adults. We then assessed whether these relationships were explained by individual socioeconomic position (SEP), neighborhood characteristics, and traditional individual behavioral and biological CVD risk factors.

\section*{Methods}

\section*{Study population}

MESA is an observational prospective cohort study designed to examine the determinants of subclinical cardiovascular disease in adults aged 45-84 years. Self-identified Black, Chinese, Hispanic, and White participants free of clinical cardiovascular disease at baseline were recruited from six sites (New York, New York; Baltimore City and County, Maryland; Forsyth County,
North Carolina; St. Paul, Minnesota; Chicago, Illinois; and Los Angeles County, California) between 2000 and 2002. Random population samples were selected at each site using various population-based approaches. Additional details are provided elsewhere. Of the selected persons deemed eligible after screening, 59.8% participated in the study. Institutional review board approval was obtained at each site and all participants gave informed consent. Four additional examinations have been completed since baseline: exam 2 (2002-2004), exam 3 (2004-2005), exam 4 (2005-2007) and exam 5 (2010-2012). Due to the small number of cardiovascular events among Chinese participants, our analyses were restricted to Blacks, Hispanics, and Whites.

**Racial/ethnic residential segregation**

Neighborhood-level racial/ethnic residential segregation was measured separately for Blacks, Whites, and Hispanics using the local $G^*_i$ statistic, based on the geocoded addresses of MESA participants linked to U.S. Census data. The $G^*_i$ statistic returns a Z-score for each neighborhood (census tract), indicating the extent to which the racial/ethnic composition in the focal tract and neighboring tracts deviates from the mean racial composition of some larger areal unit surrounding the tract (in our case the set of counties represented in each MESA site). Higher positive $G^*_i$ Z-scores indicate higher racial/ethnic segregation or clustering (over-representation), scores near 0 indicate racial integration, and lower negative scores suggest lower racial/ethnic representation (under-representation), compared to the racial composition of the larger areal unit.

Most studies of neighborhood-level racial/ethnic residential segregation use racial/ethnic composition, or the proportion of a race/ethnic group in a neighborhood, as a proxy for segregation. However, this measure is limited in that it does not incorporate any information on the racial composition of the larger area in which the neighborhood is embedded or on the
distribution of groups in space.\textsuperscript{1} The \( G_{i}^* \) statistic, in contrast, better reflects both the contextual and spatial aspects of segregation. A given neighborhood will have a higher \( G_{i}^* \) statistic the larger the difference between its racial composition and the composition of the larger areal unit. In addition, a neighborhood surrounded by similarly segregated areas will have a higher \( G_{i}^* \) statistic than those surrounded by less segregated areas. Further details on the \( G_{i}^* \) statistic are available in the Supplemental Methods.

**Fatal and nonfatal incident CVD**

Incident CVD was defined as first definite angina, probable angina followed by revascularization, myocardial infarction, resuscitated cardiac arrest, coronary heart disease (CHD) death, stroke, or stroke death. Incident CHD, defined as first myocardial infarction, resuscitated cardiac arrest, or CHD death, was also assessed as a secondary outcome. MESA uses a standard adjudication protocol to classify events.\textsuperscript{22} Every 9-12 months, participants (or when necessary their proxies) are contacted to inquire about hospital admissions, cardiovascular diagnoses, and deaths. Possible vascular events are abstracted from hospital records and sent for review and classification by an independent adjudication committee. Outcome follow-up data were available for events occurring on or before and adjudicated through December 31, 2011 (median follow-up of 10.2 years).

**Covariates**

Sociodemographic covariates included age, sex, education (categorized as less than high school graduate, high school graduate, some college, and college degree or higher), health insurance status, and income (specified in quartiles). Baseline income was available and used for 80.0% of Black participants, 85.0% of Hispanic participants, and 88.3% of White participants. When baseline income was missing, data from Exam 2 were used (4.4% of Black participants, 1.2% of
Hispanic participants, and 1.2% of White participants).

Neighborhood (census tract) characteristics were adjusted for included neighborhood poverty, the neighborhood social environment, and the neighborhood physical environment. Neighborhood poverty was defined as the percent of neighborhood residents who were below the U.S. Census Bureau-defined poverty threshold, based on baseline address linked to Census 2000 data. Survey-based information on the neighborhood social and physical environments was collected as part of the MESA Neighborhood study, an ancillary study designed to assess neighborhood conditions of potential relevance to cardiovascular disease. In addition to MESA participants, a sample of 5,988 individuals (recruited between January and August 2004) residing in the same neighborhoods as MESA participants were asked to rate several aspects of their neighborhood via a telephone survey. Supplementing the neighborhood survey with participants other than those in MESA reduced the potential for same source bias, increased the within-neighborhood sample size for constructing more reliable subjective neighborhood variables, and provided a more representative measure of neighborhood conditions.26

The neighborhood social environment measure was generated by summing 3 scales, including neighborhood safety (a composite of three self-reported questions of safety and violence), neighborhood social cohesion (a composite of five self-reported questions on feelings of mutual trust and solidarity with neighbors), and aesthetic quality (a composite of three self-reported questions relating to noise, presence of trash and litter, and overall neighborhood attractiveness). Possible scores ranged from 3 to 15, with higher scores indicating a better social environment (Cronbach’s alpha: 0.89). The neighborhood physical environment measure was constructed by summing responses of neighborhood walking environment (4 items) and neighborhood food environment (2 items) scales. Possible scores ranged from 2 to 10
(Cronbach’s alpha: 0.75). For each neighborhood scale, conditional empirical Bayes estimates were derived to increase reliability of the measures. Additional details on the development of the scales are provided elsewhere.26 Both the neighborhood social and physical environment measures were categorized into tertiles.

We also adjusted for traditional behavioral and biological CVD risk factors, including cigarette smoking, physical activity, diabetes, systolic blood pressure, total cholesterol, HDL cholesterol, and BMI. Physical activity was categorized as high (≥1000 MET-minutes per week of energy expenditure from recreational activity), intermediate (>0 and < 1000 MET-minutes per week), or physically inactive based on the 2008 Physical Activity Guidelines for Americans.27 Cigarette smoking was categorized as current, former, and never; alcohol use was categorized as heavy (>14 drinks per week for men and >7 drinks per week for women), moderate, or none. Diabetes was defined as having a fasting glucose ≥ 126 mg/dl or being on insulin or oral hypoglycemic medications.28 Resting seated blood pressure was measured three times at a single visit by trained and certified clinic staff using a Dinamap PRO 100 automated oscillometric device (Critikon, Tampa, FL), and the average of the last two measurements was used.29 Plasma HDL and total cholesterol were measured by the cholesterol-oxidase method.30

**Statistical analysis**

All analyses were stratified by race/ethnicity. Descriptive analyses compared baseline participant characteristics by levels of residential segregation score (high: \( G_i^* > 1.96 \); medium: \( G_i^* 0 – 1.96 \); and low: \( G_i^* < 0 \)). The high category corresponds to statistically significant clustering at the \( \alpha=0.05 \) level, and the low category corresponds to both the absence of any clustering or areas in which the group is significantly under-represented (\( G_i^* < -1.96 \)). These categories were combined because the number of Blacks and Hispanics living in areas where they were
significantly under-represented was very small. Baseline characteristics were also compared in those who did and did not develop incident CVD/CHD.

A series of marginal Cox proportional hazards regression modeling were estimated using a robust sandwich estimator to examine the relationship between baseline continuous segregation and CVD/CHD event while accounting for any residual correlations among individuals residing in the same census tract.31,32 Time was measured from date of the baseline exam and subjects were censored at the last contact date for outcome information if a CVD/CHD event was not observed. The proportional hazards assumption was tested by evaluating interactions of analysis time with the full complement of covariates; there was no evidence of violation. Model 1 adjusted for demographics including age, sex, and MESA study site (nativity was adjusted for as well for the analysis of Hispanics). We did not find evidence suggesting associations of segregation with incident CVD varied by MESA study site (P for interaction ≥ 0.5), so findings are presented pooled across the sites. Model 2 additionally adjusted for neighborhood-level characteristics through which segregation may impact CVD/CHD, including neighborhood poverty, the neighborhood physical environment, and the neighborhood social environment. The Black (r=0.31), Hispanic (r=0.31), and White (r=-0.59) segregation scores were correlated with neighborhood poverty, but not strongly enough to preclude us from assessing their independent associations. Model 3 further adjusted for individual factors that may have been influenced by segregation: education and income. Model 4 additionally adjusted for baseline CVD risk factors including hypercholesterolemia, hypertension, diabetes, body mass index, physical activity, current alcohol use, and current cigarette smoking as potential individual-level mediators.

We estimated three additional models to leverage our longitudinal data structure and more precisely operationalize our constructs. We first modeled the behavioral and biological
CVD risk factors as time-varying covariates (Model 5), and then we specified both residential segregation and CVD risk factors as time-varying (Model 6). As a secondary analysis, we also examined associations of change in segregation score with incident CVD. This was done by calculating the difference in the segregation score between each exam and baseline and including this variable as a time varying covariate in the Cox regression.

For analyses of Hispanic segregation, we also tested whether the association between segregation and incident CVD varied between those of Mexican origin and those not of Mexican origin (including Dominican, Puerto Rican, Cuban, and other Hispanics) by incorporating a segregation*Mexican origin interaction term. This was motivated by prior research that has found associations between residential environment and health to differ by Hispanic subgroup.33, 34 Small sample sizes precluded assessing this relationship separately in the other Hispanic subgroups. Based on previous research,35 we also tested for effect modification by nativity and acculturation. Nativity was dichotomized as foreign-born vs. US-born. Acculturation was assessed using a summary score based on length of time lived in the US (3=US born; 2=foreign born and lived in the US ≥20 years; 1=foreign-born and lived in the US 10-19 years; and 0=foreign born and lived in the US <10 years) and language spoken at home (2=English; 1=English and Spanish; and 0=Spanish).36 Scores ranged from 0 (least acculturated) to 5 (most acculturated) and were categorized into three groups (0-1, 2, and 3-5).

Analyses were based on the 5,227 participants free of cardiovascular disease with complete data on all covariates at baseline. Of the total 6,011 Black, Hispanic, and White MESA participants, 576 were excluded for missing address data and 4 were excluded for having prevalent CVD at baseline (as determined after recruitment via surveillance for events that occurred prior to baseline). Another 204 were missing data on one or more of the other study
covariates measured at baseline. All analyses were conducted using SAS 9.4 (SAS Institute, Inc., Cary, North Carolina).

Results

Baseline Black segregation scores ranged from -2.85 to 11.58 for Blacks (median: 2.47; interquartile range (IQR): 0.63 to 3.92). Median segregation scores remained similar across the follow-up examinations. The distribution of baseline Hispanic segregation for Hispanics was similar, with scores from -3.20 to 12.12 (median: 3.16; IQR: 0.67 to 4.82). Median scores decreased steadily across the follow-up examinations. For Whites, baseline White segregation scores ranged from -4.86 to 5.02 (median: 0.44; IQR: -1.34 to 1.26). Median segregation scores over the follow-up examinations for all groups (not shown).

Overall, the patterning of individual- and neighborhood-level characteristics by level of segregation was similar in Black and Hispanic participants (Table 1). With few exceptions, Black and Hispanic participants living in less segregated tracts had higher socioeconomic position (SEP) and better neighborhood conditions than their counterparts in more segregated areas. However, there were few significant differences in behavioral or biological CVD risk factors by level of segregation. In contrast, in Whites, higher White segregation was generally associated with higher individual and neighborhood SEP, better neighborhood physical and social environments, and lower current smoking, physical inactivity, and BMI.

Table 2 presents baseline characteristics by race/ethnicity and incident CVD status. Among all race/ethnic groups, those who developed CVD were older, and more likely to be male, hypertensive, and diabetic. Participants who developed CVD were more likely to have low education and income, though this difference was only significant for Whites. There were no
significant differences in neighborhood poverty by CVD status for Blacks and Hispanics. In contrast, Whites who developed CVD lived in higher poverty neighborhoods. Blacks who developed CVD were less likely to live in poor (lowest tertile) social environments but more likely to live in poor physical environments. Mean segregation scores were higher for Blacks who developed CVD than those who did not, but this difference was not statistically significant (P=0.07). Segregation scores were lower for Hispanics and Whites who developed CVD, but again these differences were not statistically significant (P=0.45 and 0.1, respectively). Patterns were similar when the outcome was restricted to CHD (not shown).

In Blacks, higher segregation was significantly associated with higher incident CVD (Table 3). Each unit increase in Black segregation was associated with a 12% increase (Model 1 HR=1.12, 95% CI: 1.02, 1.22) in the hazard of developing CVD after adjusting for age, sex, and study site. This association remained unchanged with further adjustment for individual- and neighborhood-level SEP, and measures of the neighborhood physical and social environments (Models 2 and Model 3). Adjustment for potential mediating behavioral and biological CVD risk factors (Model 4) had no effect on this association (Model 4: HR: 1.12; 95% CI: 1.02, 1.23). Point estimates for hazard ratios associated with segregation were slightly larger among Blacks when the outcome was restricted to CHD (Model 1 HR: 1.18; 95% CI: 1.05, 1.33); associations remained essentially unchanged in fully adjusted models (Model 4 HR: 1.17; 95% CI: 1.03, 1.32).

Hispanic segregation levels were not associated with CVD incidence for Hispanics across all adjusted models (e.g., Model 1 HR: 1.00; 95% CI: 0.95, 1.05). There was some evidence suggesting higher segregation was related to higher CVD incidence among foreign-born and less acculturated Hispanics, but the tests for interaction were not statistically significant.
(Supplemental Table 1; P for interaction in demographic-adjusted models = 0.09 and 0.1, respectively). The association between segregation and incident CVD did not vary significantly by country of origin (P for interaction in demographic-adjusted models = 0.14). Findings were similar when analyses were restricted to CHD (Model 1 HR: 1.03; 95% CI: 0.95, 1.12).

Higher White segregation among Whites was associated with 12% lower CVD incidence in demographic-adjusted models (Table 3, Model 1 HR: 0.88; 95% CI: 0.81, 0.96). This association was attenuated slightly and lost statistical significance with adjustment for neighborhood characteristics (Model 2 HR: 0.91; 95% CI: 0.80, 1.02). This association remained unchanged with further adjustment for individual SEP and CVD risk factors. As with incident CVD, segregation was inversely associated with incident CHD for Whites in models adjusted for demographics (Model 1 HR: 0.88; 95% CI: 0.79, 0.99) but was attenuated after adjusting for neighborhood characteristics (Model 2 HR: 0.94; 95% CI: 0.80, 1.10).

All associations between segregation and CVD were essentially unchanged when CVD behavioral and biological risk factors were modeled as time-varying covariates as well as when both segregation and risk factors were modeled as time-varying (not shown). In secondary analyses, change in segregation score was not significantly associated with incident CVD among Whites (HR: 0.85; 95% CI: 0.69, 1.05), Blacks (HR: 1.01; 95% CI: 0.84, 1.22), or Hispanics (HR: 0.99; 95% CI: 0.86, 1.14).

Discussion

We found that higher neighborhood-level own-group racial residential segregation was associated with higher CVD incidence among Blacks and with lower CVD incidence among Whites after adjusting for individual-level demographic characteristics. The magnitude of these
associations persisted for Blacks with further adjustment for individual SEP, neighborhood characteristics, and traditional CVD risk factors. Associations were attenuated for Whites with adjustment for neighborhood characteristics. We found no evidence linking segregation to CVD incidence for Hispanics. All findings were similar when the outcome was restricted to CHD and when segregation and risk factors were modeled as time-varying.

Our results for Whites are consistent with previous research, which has shown CVD mortality rates are lower for Whites living in predominantly White neighborhoods compared to those living in predominantly minority neighborhoods.\textsuperscript{15} In our study, the association for Whites was attenuated after adjusting for neighborhood characteristics, suggesting this was a key pathway linking segregation to cardiovascular risk. This is consistent with the segregation literature which suggests neighborhood poverty is a predominant pathway by which racial/ethnic segregation influences health and health disparities, especially since exposure to high neighborhood poverty for Black and Hispanic Americans is so much higher than for White Americans.\textsuperscript{2,5} In addition, neighborhood poverty and neighborhood racial/ethnic composition are correlated, particularly for Blacks and Whites.\textsuperscript{2,37} The correlations between own-group segregation and poverty were higher in whites than in Blacks, which may explain why adjusting for neighborhood characteristics attenuated associations of segregation with CHD more for Whites than Blacks.

Prior work on the relationship between neighborhood-level segregation and CVD in Blacks has been less consistent. In contrast to our results, a Texas study found that Blacks living in predominantly Black areas lost fewer years of life to heart disease,\textsuperscript{13} and studies in New York City and Atlanta showed older Blacks (≥ 65 years) living in predominantly Black neighborhoods had lower mortality rates (CHD and stroke, respectively) than those living in predominantly
White neighborhoods.\textsuperscript{14,15} However, consistent with our findings, stroke mortality rates in the Atlanta study were higher for younger Blacks living in predominantly Black vs. predominantly White neighborhoods.\textsuperscript{14} Differences across these studies may be due to a variety of factors including the cities included in the analyses, the way segregation was measured (tract racial composition in other studies vs. $G_i$ statistic in our study, the latter which incorporates spatial dependence of neighboring tracts), and the type of events included (e.g., fatal in other studies vs. a combination of fatal and non-fatal in our study). Age differences across studies may also play a role, particularly if among older populations mortality selection is operating to cull Blacks such that only hearty Blacks had survived to participate in the sample. Since mortality selection may be especially operative in predominantly Black neighborhoods,\textsuperscript{9,11} this could explain the apparent protective effect of racial composition with CVD mortality seen in previous studies of adults ages 65 and older.

Although we did not directly examine the contribution of segregation to Black-White differences in CVD, our finding that higher segregation is associated with higher CVD risk in Blacks is consistent with the notion that racial residential segregation may be a fundamental cause of Black-White health disparities.\textsuperscript{2,38} Segregation has been called a fundamental cause of disparities because it determines access to opportunities for socioeconomic attainment and influences exposure to other health-harming and health-promoting conditions in the social and physical environment.\textsuperscript{2} In our study, adjusting for socioeconomic conditions and characteristics of the neighborhood social and physical environments did not attenuate associations of segregation with incident CVD for Blacks. Our findings also persisted with adjustment for several mediating behavioral and biological risk factors. One reason for this may be that other pathways we did not test, such as stress-related neuroendocrine and sympathetic nervous system
pathways, or experiences of prejudice and discrimination\textsuperscript{29,40} may link segregation to increased CVD risk.\textsuperscript{29} Differences in access to quality healthcare may be another unmeasured pathway linking segregation to increased CVD risk. For example, a study using national Medicare data found Black patients are significantly more likely to undergo coronary artery bypass grafting and abdominal aortic aneurysm repair procedures in low-quality hospitals than Whites living in medium and high segregation areas, but they found no significant Black-White differences in low segregation areas.\textsuperscript{6}

It may also be that persistent associations of segregation with CVD among Blacks may be due to residual confounding. For example, our baseline neighborhood measures may not adequately reflect the life course neighborhood-level exposures that influence CVD risk. However, MESA participants were asked in the study at baseline how long they lived in their current address, and these data show participants lived in their baseline addresses for an average of nearly 20 years (20.9 years for Whites, 19.7 years for Blacks, and 18.2 years for Hispanics). In addition, when we operationalized segregation as time-varying over 10 years of follow up, we found similar results as a baseline operationalization. Another possibility is that error in the measurement of the behavioral and biological CVD risk factors could have led to an underestimate of the impact of risk factors, even though our time-varying operationalization of CVD risk factors across almost a decade improves upon much prior longitudinal CVD evidence.

No studies to our knowledge have examined associations of neighborhood-level residential segregation with cardiovascular outcomes among Hispanics. Findings from studies of neighborhood-level segregation and other health outcomes among Hispanics are mixed.\textsuperscript{19,34,41} As with Blacks, research suggests Hispanics face housing discrimination in rental and sales markets.\textsuperscript{40} However, the continuous influx of Hispanic immigrants into the U.S. promotes the
development and maintenance of immigrant enclaves that ease the transition into the U.S. labor market and facilitate retention of potentially health-promoting social and cultural ties, which may help to counterbalance the disadvantaged context Hispanics face.\textsuperscript{38,39} Our null findings among Hispanics may reflect heterogeneity in the social and economic contexts of Hispanic enclaves.

Because Hispanics reflect a very heterogeneous population, previous research suggests that nativity or Hispanic country of origin may modify the impact of Hispanic segregation on health. For example, an examination of segregation and birthweight among Mexican-origin mothers showed living in ethnic enclaves was associated with lower birthweight for infants born to US-born Mexican-origin mothers but not foreign-born Mexican-origin mothers.\textsuperscript{35} In addition, a study of Puerto Rican and Mexican American Chicago residents found that higher segregation was associated with more frequent acute physical symptoms (e.g., headaches and pains in chest or heart) in Puerto Ricans.\textsuperscript{34} In contrast, higher segregation in that Chicago study was not associated with physical symptoms in first-generation (foreign born) Mexican Americans, but it was associated with better health in US-born (second- or higher generation) Mexican Americans. However, we did not find significant differences in the Hispanic segregation-CVD association by acculturation, nativity, or country of origin. It is possible that the Mexican vs. non-Mexican categorization was too broad, but unfortunately, small sample sizes of other non-Mexican Hispanic subgroups limited our ability to examine neighborhood segregation-CVD associations among those groups. Further work is needed to better characterize the possible differing relationships between segregation and CVD risk among Hispanic subgroups. Studies in larger, more diverse (geographically and/or ethnically) Hispanic cohorts will help us better disentangle the impact of segregation on CVD risk in this heterogeneous population.

In secondary analyses, we found change in segregation was not related to CVD risk. One
An explanation is that there was not enough change in segregation levels across examinations, because few participants moved and/or segregation levels did not change substantially over time in the neighborhoods where participants resided. Another explanation is that since many of the exposures associated with segregation that influence cardiovascular risk are experienced over the life course, later life moves may not be sufficient to mitigate the adverse health effects of living in segregated neighborhoods. Further work is needed in younger cohorts to better understand how segregation patterns over the life course impact cardiovascular risk.

Important strengths of our study include the prospective study design over 10 years of follow up, multi-ethnic study population, adjudicated health outcomes, inclusion of multiple levels of potential confounders and mediators relevant for CVD, and our modeling of time-varying segregation and CVD covariates. One limitation of our study is that we did not have sufficient power to examine cardiovascular outcomes in Chinese MESA participants or to assess relationships of segregation with specific cardiovascular outcomes other than CHD (e.g., stroke). Another limitation is that MESA participants are not necessarily representative of the national population or even the cities in which they reside, which may reduce the generalizability of our findings.

No prospective study to our knowledge has investigated the connection between neighborhood-level racial/ethnic residential segregation and incident CVD. Our findings suggest associations of segregation with CVD risk vary by race/ethnic group, reflecting differences in the processes that lead to segregation across these different groups and in the consequences of segregation for CVD relevant exposures. One proposed strategy for reducing the adverse impact of segregation on health is to address characteristics of segregated neighborhoods that are unhealthy. Thus, a better understanding of the impact of segregation on CVD risk as well as the
individual- and neighborhood-level pathways linking segregation to CVD may help guide efforts to prevent CVD and reduce racial/ethnic disparities in cardiovascular outcomes.

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**Conflict of Interest Disclosures:** None.

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Table 1. Selected baseline MESA participant characteristics by race/ethnicity and segregation category*.

<table>
<thead>
<tr>
<th></th>
<th>Blacks (n=1595)</th>
<th></th>
<th></th>
<th>Hispanics (n=1289)</th>
<th></th>
<th></th>
<th>Whites (n=2345)</th>
<th></th>
<th></th>
<th>p-value</th>
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<tbody>
<tr>
<td></td>
<td>Low (n=280)</td>
<td>Medium (n=388)</td>
<td>High (n=927)</td>
<td>p-value</td>
<td>Low (n=238)</td>
<td>Medium (n=226)</td>
<td>High (n=823)</td>
<td>p-value</td>
<td>Low (n=1039)</td>
<td>Medium (n=1006)</td>
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<td>Age, mean years (SD)</td>
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<td>60.7 (9.4)</td>
<td>62.5 (10.2)</td>
<td>0.0001</td>
<td>61.3 (10.4)</td>
<td>62.2 (10.5)</td>
<td>61.2 (10.3)</td>
<td>0.42</td>
<td>60.9 (10.2)</td>
<td>63.4 (9.9)</td>
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<td>Sex, % male</td>
<td>40.4 (41.5)</td>
<td>41.5 (47.7)</td>
<td>70.3 (50.4)</td>
<td>0.03</td>
<td>50.4 (52.7)</td>
<td>46.9 (49.9)</td>
<td>0.25</td>
<td>46.9 (49.2)</td>
<td>49.7 (5.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Education &lt; high school, %</td>
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<td>11.9 (19.4)</td>
<td>0.006</td>
<td>20.6 (36.7)</td>
<td>49.9 (19.7)</td>
<td>&lt;0.0001</td>
<td>5.1</td>
<td>4.0</td>
<td>5.7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Income &lt; $25,000, %</td>
<td>17.5 (27.6)</td>
<td>35.0 (35.1)</td>
<td>0.001</td>
<td>35.5 (41.2)</td>
<td>53.8 (19.7)</td>
<td>&lt;0.0001</td>
<td>19.7</td>
<td>12.2</td>
<td>10.0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No health insurance, %</td>
<td>3.2 (3.6)</td>
<td>7.9 (10.1)</td>
<td>0.001</td>
<td>10.1 (17.3)</td>
<td>17.0</td>
<td>0.03</td>
<td>4.0</td>
<td>1.4</td>
<td>0.7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Current smoking, %</td>
<td>16.4 (16.8)</td>
<td>18.7 (12.2)</td>
<td>0.27</td>
<td>12.0 (12.0)</td>
<td>13.5 (12.5)</td>
<td>0.53</td>
<td>12.5</td>
<td>10.2</td>
<td>7.0</td>
<td>0.03</td>
</tr>
<tr>
<td>Heavy alcohol use, %</td>
<td>2.5 (2.3)</td>
<td>3.0 (2.9)</td>
<td>0.3</td>
<td>4.0 (2.7)</td>
<td>0.21</td>
<td>9.5</td>
<td>9.4</td>
<td>10.3</td>
<td>0.08</td>
<td></td>
</tr>
<tr>
<td>Physical inactivity, %</td>
<td>22.1 (22.9)</td>
<td>23.2 (46.6)</td>
<td>0.77</td>
<td>40.3 (35.4)</td>
<td>&lt;0.0001</td>
<td>18.4</td>
<td>15.2</td>
<td>13.3</td>
<td>0.0004</td>
<td></td>
</tr>
<tr>
<td>Body mass index, mean kg/m² (SD)</td>
<td>29.9 (5.5)</td>
<td>30.4 (6.0)</td>
<td>30.0 (5.7)</td>
<td>0.44</td>
<td>29.2 (5.3)</td>
<td>29.3 (4.5)</td>
<td>29.6 (5.2)</td>
<td>0.46</td>
<td>28.1 (5.3)</td>
<td>27.4 (4.8)</td>
</tr>
<tr>
<td>Systolic blood pressure, mean mm Hg</td>
<td>128.9 (20.8)</td>
<td>130.4 (21.5)</td>
<td>131.9 (21.3)</td>
<td>0.1</td>
<td>124.3 (20.2)</td>
<td>127.4 (18.0)</td>
<td>126.7 (22.7)</td>
<td>0.21</td>
<td>120.9 (19.7)</td>
<td>123.3 (20.2)</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>15.0 (18.6)</td>
<td>16.4 (13.5)</td>
<td>0.45</td>
<td>17.7 (13.5)</td>
<td>18.2</td>
<td>0.22</td>
<td>5.6</td>
<td>6.0</td>
<td>4.7</td>
<td>0.69</td>
</tr>
<tr>
<td>Total cholesterol, mean mg/dl</td>
<td>189.8 (33.6)</td>
<td>190.1 (35.3)</td>
<td>189.0 (36.5)</td>
<td>0.87</td>
<td>196.1 (34.9)</td>
<td>197.8 (37.1)</td>
<td>198.2 (36.7)</td>
<td>0.74</td>
<td>197.9 (37.6)</td>
<td>193.2 (32.3)</td>
</tr>
<tr>
<td>HDL cholesterol, mean mg/dl</td>
<td>53.9 (15.3)</td>
<td>51.9 (14.5)</td>
<td>51.9 (15.1)</td>
<td>0.13</td>
<td>48.6 (12.6)</td>
<td>47.3 (13.4)</td>
<td>47.8 (13.1)</td>
<td>0.51</td>
<td>51.3 (15.2)</td>
<td>52.9 (16.2)</td>
</tr>
<tr>
<td>Neighborhood poverty, mean (SD)</td>
<td>13.0 (13.1)</td>
<td>20.4 (14.8)</td>
<td>21.4 (10.7)</td>
<td>&lt;0.0001</td>
<td>11.9 (10.1)</td>
<td>15.6 (10.3)</td>
<td>25.6 (11.3)</td>
<td>&lt;0.0001</td>
<td>14.5 (8.0)</td>
<td>7.1 (4.2)</td>
</tr>
<tr>
<td>Poor neighborhood social environment, %†</td>
<td>21.8 (37.1)</td>
<td>35.2 (13.1)</td>
<td>&lt;0.0001</td>
<td>8.0 (10.1)</td>
<td>16.8 (10.3)</td>
<td>45.2 (11.3)</td>
<td>&lt;0.0001</td>
<td>63.1 (8.0)</td>
<td>7.6 (4.2)</td>
<td>16.3 (3.6)</td>
</tr>
<tr>
<td>Poor neighborhood physical environment, %†</td>
<td>24.3 (28.6)</td>
<td>38.0 (20.6)</td>
<td>&lt;0.0001</td>
<td>20.6 (13.1)</td>
<td>29.2 (13.3)</td>
<td>38.2 (13.1)</td>
<td>&lt;0.0001</td>
<td>43.6 (10.1)</td>
<td>28.7 (4.2)</td>
<td>11.3 (3.6)</td>
</tr>
</tbody>
</table>

Abbreviation: SD=standard deviation

*Only the most relevant categories of each baseline characteristic are displayed. Segregation is measured using the G² statistic. For all race/ethnic groups, low segregation=G² <0; medium segregation =G² 0-1.96; and high segregation =G² >1.96

†Poor neighborhood social and physical environments is defined as the lowest tertile of the social and physical environment scores, respectively
Table 2. Selected baseline MESA participant characteristics by race/ethnicity and incident cardiovascular disease status*.

<table>
<thead>
<tr>
<th></th>
<th>Blacks</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes (n=136)</td>
<td>No (n=1459)</td>
<td>p-value</td>
<td>Yes (n=120)</td>
<td>No (n=1167)</td>
<td>p-value</td>
<td>Yes (n=241)</td>
<td>No (n=2104)</td>
<td>p-value</td>
</tr>
<tr>
<td>Age (years), mean (SD)</td>
<td>66.0 (10.0)</td>
<td>61.3 (9.8)</td>
<td>&lt;0.0001</td>
<td>67.3 (9.4)</td>
<td>60.8 (10.2)</td>
<td>&lt;0.0001</td>
<td>67.7 (9.3)</td>
<td>61.7 (10.0)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Sex, % male</td>
<td>58.8</td>
<td>43.6</td>
<td>0.0006</td>
<td>65.0</td>
<td>46.9</td>
<td>0.0002</td>
<td>61.0</td>
<td>46.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Education &lt; high school, %</td>
<td>15.4</td>
<td>10.4</td>
<td>0.35</td>
<td>46.7</td>
<td>41.7</td>
<td>0.16</td>
<td>12.0</td>
<td>3.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Income &lt; $25,000, %</td>
<td>35.3</td>
<td>29.6</td>
<td>0.3</td>
<td>56.7</td>
<td>47.3</td>
<td>0.12</td>
<td>24.9</td>
<td>14.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No health insurance, %</td>
<td>8.8</td>
<td>5.8</td>
<td>0.15</td>
<td>10.8</td>
<td>16.3</td>
<td>0.12</td>
<td>4.2</td>
<td>2.2</td>
<td>0.07</td>
</tr>
<tr>
<td>Current smoking, %</td>
<td>17.7</td>
<td>17.8</td>
<td>0.59</td>
<td>15.8</td>
<td>12.7</td>
<td>0.03</td>
<td>14.5</td>
<td>10.4</td>
<td>0.15</td>
</tr>
<tr>
<td>Heavy alcohol use, %</td>
<td>2.2</td>
<td>2.8</td>
<td>0.46</td>
<td>3.3</td>
<td>2.9</td>
<td>0.96</td>
<td>7.5</td>
<td>9.8</td>
<td>0.006</td>
</tr>
<tr>
<td>Physical inactivity, %</td>
<td>27.9</td>
<td>22.5</td>
<td>0.29</td>
<td>36.7</td>
<td>29.7</td>
<td>0.29</td>
<td>19.5</td>
<td>16.0</td>
<td>0.02</td>
</tr>
<tr>
<td>Body mass index, mean kg/m² (SD)</td>
<td>30.1 (5.4)</td>
<td>30.1 (5.8)</td>
<td>0.94</td>
<td>29.8 (5.1)</td>
<td>29.4 (5.1)</td>
<td>0.4</td>
<td>28.6 (4.7)</td>
<td>27.6 (5.1)</td>
<td>0.003</td>
</tr>
<tr>
<td>Systolic blood pressure, mean mm Hg</td>
<td>137.4 (20.7)</td>
<td>130.4 (21.2)</td>
<td>0.0002</td>
<td>137.5 (24.2)</td>
<td>125.2 (20.9)</td>
<td>&lt;0.0001</td>
<td>132.8 (22.2)</td>
<td>121.7 (19.5)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>30.2</td>
<td>15.4</td>
<td>&lt;0.0001</td>
<td>30.0</td>
<td>15.9</td>
<td>0.0001</td>
<td>8.3</td>
<td>5.3</td>
<td>0.06</td>
</tr>
<tr>
<td>Total cholesterol, mean mg/dl</td>
<td>190.0 (39.4)</td>
<td>189.4 (35.3)</td>
<td>0.83</td>
<td>201.5 (40.0)</td>
<td>197.3 (36.0)</td>
<td>0.24</td>
<td>194.6 (34.8)</td>
<td>195.7 (35.1)</td>
<td>0.65</td>
</tr>
<tr>
<td>HDL cholesterol, mean mg/dl</td>
<td>48.7 (14.6)</td>
<td>52.6 (15.0)</td>
<td>0.004</td>
<td>44.2 (11.0)</td>
<td>48.2 (13.2)</td>
<td>0.0003</td>
<td>47.8 (13.9)</td>
<td>52.9 (15.8)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>G-statistic segregation score, mean (SD)</td>
<td>2.94 (3.02)</td>
<td>2.46 (2.90)</td>
<td>0.07</td>
<td>3.04 (2.98)</td>
<td>3.26 (3.19)</td>
<td>0.45</td>
<td>-0.17 (1.69)</td>
<td>0.03 (1.80)</td>
<td>0.1</td>
</tr>
<tr>
<td>Neighborhood poverty (%), mean (SD)</td>
<td>18.2 (10.5)</td>
<td>19.9 (12.8)</td>
<td>0.08</td>
<td>20.3 (12.4)</td>
<td>21.4 (12.3)</td>
<td>0.32</td>
<td>11.0 (7.3)</td>
<td>10.0 (7.3)</td>
<td>0.04</td>
</tr>
<tr>
<td>Poor neighborhood social environment, %†</td>
<td>24.3</td>
<td>34.1</td>
<td>0.01</td>
<td>30.0</td>
<td>33.7</td>
<td>0.37</td>
<td>34.9</td>
<td>33.1</td>
<td>0.85</td>
</tr>
<tr>
<td>Poor neighborhood physical environment, %†</td>
<td>45.6</td>
<td>32.2</td>
<td>0.005</td>
<td>30.8</td>
<td>33.6</td>
<td>0.67</td>
<td>34.4</td>
<td>32.9</td>
<td>0.04</td>
</tr>
</tbody>
</table>

Abbreviation: SD=standard deviation

*The category “Yes” is used to indicate participants who developed cardiovascular disease over the follow-up period. The category “No” is used to indicate those who did not.
†Poor neighborhood social and physical environments is defined as the lowest tertile of the social and physical environment scores, respectively
Table 3. Adjusted hazard ratios of cardiovascular disease and coronary heart disease (and 95% confidence intervals) associated with each standard deviation increase in baseline racial/ethnic residential segregation.

<table>
<thead>
<tr>
<th>No. of events</th>
<th>Model 1*</th>
<th>Model 2†</th>
<th>Model 3‡</th>
<th>Model 4§</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incident cardiovascular disease</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blacks</td>
<td>136</td>
<td>1.12 (1.02, 1.22)</td>
<td>1.12 (1.02, 1.23)</td>
<td>1.11 (1.02, 1.22)</td>
</tr>
<tr>
<td>Hispanics</td>
<td>120</td>
<td>1.00 (0.95, 1.05)</td>
<td>1.00 (0.94, 1.08)</td>
<td>1.00 (0.93, 1.08)</td>
</tr>
<tr>
<td>Whites</td>
<td>241</td>
<td>0.88 (0.81, 0.96)</td>
<td>0.91 (0.81, 1.02)</td>
<td>0.91 (0.81, 1.01)</td>
</tr>
<tr>
<td><strong>Incident coronary heart disease</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blacks</td>
<td>55</td>
<td>1.18 (1.05, 1.33)</td>
<td>1.16 (1.02, 1.31)</td>
<td>1.15 (1.02, 1.30)</td>
</tr>
<tr>
<td>Hispanics</td>
<td>60</td>
<td>1.03 (0.95, 1.12)</td>
<td>1.03 (0.91, 1.16)</td>
<td>1.03 (0.92, 1.15)</td>
</tr>
<tr>
<td>Whites</td>
<td>103</td>
<td>0.88 (0.79, 0.99)</td>
<td>0.94 (0.80, 1.10)</td>
<td>0.94 (0.81, 1.09)</td>
</tr>
</tbody>
</table>

*Adjusted for demographics (age, sex, study site, and, for Hispanics, nativity).
†Adjusted for all covariates in Model 1 plus neighborhood covariates (neighborhood poverty, neighborhood social environment, and neighborhood physical environment).
‡Adjusted for all covariates in Model 2, plus socioeconomic position (education, income, and health insurance status).
§Adjusted for all covariates in Model 3, plus baseline CVD risk factors (systolic blood pressure, total cholesterol, HDL cholesterol, diabetes, BMI, cigarette smoking, current alcohol use, and physical activity).
Neighborhood-Level Racial/Ethnic Residential Segregation and Incident Cardiovascular Disease: The Multi-Ethnic Study of Atherosclerosis
Kiarri N. Kershaw, Theresa L. Osypuk, D. Phuong Do, Peter J. De Chavez and Ana V. Diez Roux

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Data Supplement (unedited) at:
http://circ.ahajournals.org/content/suppl/2014/12/01/CIRCULATIONAHA.114.011345.DC1
SUPPLEMENTAL MATERIAL
Supplemental Methods: Description of the local $G_i^*$ statistic

Developed by Getis and Ord,23 the local $G_i^*$ statistic is calculated as:

\[
G_i^* = \frac{\sum_{j=1}^{n} w_{ij} x_j - \bar{x} \sum_{j=1}^{n} w_{ij}}{S} \sqrt{\frac{N \sum_{j=1}^{n} w_{ij}^2 - (\sum_{j=1}^{n} w_{ij})^2}{N - 1}},
\]

where $i$ is the focal census tract for which the local $G^*$ index is estimated, $x_j$ is the proportion of a specific race/ethnic group $x$ in tract $j$ (which is a neighboring tract when it does not equal the focal tract), $w_{ij}$ is a spatial weight between focal tract $i$ and neighboring tract $j$ (used to weight the contribution of the racial/ethnic composition of each tract $j$ to the estimation of $G_i^*$), $\bar{x}$ is the mean proportion of a race/ethnic group across all $N$ tracts in the surrounding larger areal unit, and

\[
S = \sqrt{\frac{\sum_{j=1}^{n} x_j^2}{N} - (\bar{x})^2}.
\]

In these analyses, a first order rook spatial weight matrix was used. This means that the focal tract is given a weight of 1, all tracts that share a border with the focal tract are considered adjacent tracts and given a weight of 1/n (where n is the number of adjacent tracts), and all non-adjacent tracts are given a weight of 0.

In this study the larger areal units are as follows: Forsyth County, NC for the Forsyth site; Queens, Kings, New York, and Bronx Counties, NY for the New York site; Baltimore City and County, MD for the Baltimore site; Ventura, Los Angeles, Orange, Riverside, and San Bernadino Counties, CA for the Los Angeles site; Kane, DuPage, Cook and Will Counties, IL for the Chicago site; and Anoka,
Hennepin, Ramsey, Washington, Carver, Scott, and Dakota Counties, MN for the St. Paul site. These groupings of counties were selected to be consistent with the areas used for calculating the built environment variables. These groupings of counties are generally centered on the largest city or cities near the tract, but they are generally smaller than the US Census Bureau-defined metropolitan areas. Sensitivity analyses were conducted using baseline segregation measures calculated using the entire metropolitan area as the larger areal unit instead of the selection of counties described above, and findings were similar (not shown).

We used baseline address linked to Census 2000 data for baseline segregation measures. To operationalize time-varying segregation, we linked addresses at each follow up exam to tract-level data derived from the Census 2000 (for Exam 2 and those who participated in Exam 3 in 2004) or the American Community Survey 2005-2009 (for Exam 3 participants who were interviewed in 2005 and all Exam 4 participants.
Supplemental Table 1: Adjusted hazard ratios and 95% confidence intervals associated with a one unit increase in baseline Hispanic residential segregation with incident cardiovascular disease among Hispanics by acculturation, nativity, and country of origin

<table>
<thead>
<tr>
<th></th>
<th>Model 1*</th>
<th>Model 2†</th>
<th>Model 3‡</th>
<th>Model 4§</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acculturation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>1.16 (0.93, 1.45)</td>
<td>1.16 (0.93, 1.47)</td>
<td>1.15 (0.92, 1.44)</td>
<td>1.12 (0.86, 1.45)</td>
</tr>
<tr>
<td>Medium</td>
<td>1.08 (0.96, 1.21)</td>
<td>1.08 (0.95, 1.22)</td>
<td>1.07 (0.94, 1.21)</td>
<td>1.05 (0.91, 1.21)</td>
</tr>
<tr>
<td>High</td>
<td>0.95 (0.90, 1.01)</td>
<td>0.95 (0.88, 1.02)</td>
<td>0.95 (0.88, 1.03)</td>
<td>0.97 (0.89, 1.05)</td>
</tr>
<tr>
<td>P for interaction</td>
<td>0.1</td>
<td>0.09</td>
<td>0.1</td>
<td>0.4</td>
</tr>
<tr>
<td><strong>Nativity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foreign-born</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>US-born</td>
<td>0.95 (0.89, 1.02)</td>
<td>0.96 (0.88, 1.04)</td>
<td>0.96 (0.89, 1.04)</td>
<td>0.98 (0.89, 1.07)</td>
</tr>
<tr>
<td>P for interaction</td>
<td>0.09</td>
<td>0.1</td>
<td>0.14</td>
<td>0.3</td>
</tr>
<tr>
<td><strong>Country of Origin</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mexican</td>
<td>0.97 (0.92, 1.03)</td>
<td>0.98 (0.90, 1.05)</td>
<td>0.97 (0.90, 1.05)</td>
<td>0.98 (0.90, 1.07)</td>
</tr>
<tr>
<td>Non-Mexican</td>
<td>1.07 (0.95, 1.21)</td>
<td>1.08 (0.95, 1.24)</td>
<td>1.08 (0.94, 1.25)</td>
<td>1.09 (0.94, 1.26)</td>
</tr>
<tr>
<td>P for interaction</td>
<td>0.14</td>
<td>0.16</td>
<td>0.17</td>
<td>0.21</td>
</tr>
</tbody>
</table>

*Adjusted for demographics (age, sex, study site, and, for country of origin, nativity).
†Adjusted for all covariates in Model 1 plus neighborhood covariates (neighborhood poverty, neighborhood social environment, and neighborhood physical environment).
‡Adjusted for all covariates in Model 2, plus socioeconomic position (education and income).
§Adjusted for all covariates in Model 3, plus baseline CVD risk factors (systolic blood pressure, total cholesterol, HDL cholesterol, diabetes, BMI, cigarette smoking, current alcohol use, and physical activity).