Not So Black and White: The Association Between Allostatic Load and Residential Racial/Ethnic Segregation in a Multi-Ethnic Cohort

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Abstract

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This study investigates the association between residential segregation and allostatic load (AL), a measure of cumulative biological risk using the Multi-Ethnic Study of Atherosclerosis. Racial segregation was measured for each racial/ethnic group using the local G_i* statistic, a spatial measure that reflects the level at which racial minorities are clustered together in contiguous neighborhoods. Two measures of AL were utilized, a restricted measure incorporating cardio-metabolic indicators, and a comprehensive measure incorporating neuroendocrine and inflammatory biomarkers. For both scores, standardized scores were calculated to indicate where the individual's value placed them (in standard deviation units) relative to accepted clinical thresholds for higher risk, and summed to create an overall AL score. Analysis consisted of generalized estimating equations, ordinary least squares, and mixed effects models. Higher levels of segregation were associated with better AL scores for whites, but with worse AL scores for non-whites. This analysis concludes that residential segregation is a significant predictor of AL, even after adjustment for individual demographic characteristics, but segregation impacts the AL scores of different racial/ethnic groups in distinct ways.

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1. Introduction

More than fifteen years ago, racial residential segregation was declared by Williams and Collins as a fundamental cause of racial disparities in health[1]. Although not the first to link segregation and health, following this publication more studies began to identify the mechanisms through which segregation influences health trajectories and outcomes. At both the individual and neighborhood level, this literature proposes that segregation dramatically impacts the physical, social, and economic environment of residents[35,36]. Empirically, segregation has been shown to concentrate poverty[30], diminish employment opportunities, and limit access to quality education[12]. Residents of highly segregated neighborhoods typically endure reduced public services, low quality housing, and high levels of property and violent crime[11]. There are commonly fewer options for affordable healthy food, but higher than average numbers of retail stores selling alcohol and tobacco[34]. Segregated neighborhoods typically experience high resident turnover, social disorganization, and low levels of social capital. Finally, residents may experience chronic stress[16] – whether through racial discrimination, social isolation, or exposure to violence – which over time may accelerate cellular aging[2]. Furthermore, it is likely that these mechanisms operate interdependently, to compound the effect of segregation on health beyond any single factor.

Despite these robust associations, the literature linking residential segregation to health is not as consistent as expected. Contradictory results across health outcomes and racial/ethnic groups have led to the conclusion that segregation's influence may not be uniformly positive or negative, and that to clarify this association it may be best to examine the effect of segregation separately for different racial/ethnic groups.

Given the history of segregation in the United States, it is unsurprising that the overwhelming majority of literature linking segregation to health has focused on African

Americans. However, as an influx of immigrants in the late 20th century began to change the racial composition of American cities, more research has emerged on Hispanic (and occasionally Asian) segregation[3,4]. It is typically research on non-black minorities that has provided evidence of the potentially protective effects of segregation on health, buttressing the notion that the effects of segregation may vary by race/ethnicity. Some argue that the clustering of a particular racial/ethnic group may, for example, limit exposure to interpersonal discrimination and may also allow for the maintenance of strong social networks[5-8].

As to why segregation may operate differently across racial/ethnic groups, it has been suggested that the different sorting processes that create segregation for each racial/ethnic group – whether residents are sorted through overtly discriminatory mechanisms as with African Americans or the immigrant driven creation of enclaves in the case of Hispanics and Asians- may explain contradictory findings across minority populations[9]. Segregation may also operate differentially across racial/ethnic groups by benefiting those with high levels of capital (typically the ethnoracial majority group) and harming those with low levels of capital (disadvantaged minority groups) through the concentration of neighborhood affluence or poverty[10].

In addition, this literature has been complicated by measurement issues – namely, which dimension of segregation is causally responsible for improving or impairing health. Most studies on segregation and health use either the index of dissimilarity or racial/ethnic composition as a proxy measure. Neither of these measures, however, accounts for characteristics like neighborhood boundaries or proximity to other neighborhoods. Rather, both reflect the degree to which members of racial/ethnic groups are over or underrepresented in a neighborhood (evenness), but not the extent to which members of these groups live disproportionately in contiguous areas (clustering). Additional studies occasionally employ Metropolitan Statistical Area (MSA) segregation values,

which provide a single segregation value for the city under study. Unfortunately, this measure obscures heterogeneity in the levels of segregation experienced by different racial/ethnic groups throughout the metropolitan area. Each of these measures are aspatial in nature in the sense that they do not provide information about the racial/ethnic composition of contiguous neighborhoods[11, 12]. Given that it is theorized that segregation results in the clustering of numerous social, economic, and political constraints within a city[13,14], a spatial measure of neighborhood-level segregation, that captures the clustering of racial/ethnic groups within an MSA, may be a more appropriate representation of segregation.

To capture how segregation may affect health through numerous mechanisms, with widely varied physiological effects, this study examines a health outcome that reflects a cumulative, multi-system approach to conceptualizing health. In addition, it is not reliant on symptomatic presentation or diagnosis. Allostatic load (AL) is the "wear and tear" on the body due to repeated physiological adaptation to stressors (whether they are social, physical, or chemical in nature)[15]. Although the stress response is an adaptive mechanism designed to protect the body from perceived threats, chronic stress or persistent exposure to stressors requires constant adjustment to these systems, eventually reducing the ability of these systems to respond to future stressors (physiological dysregulation). Furthermore, AL was specifically chosen by Massey as the ideal biological mechanism linking residential segregation, concentrated poverty, and racial health disparities in his biosocial model of stratification[16].

Due to the involvement of multiple physiological systems in the stress response, AL is typically measured as an index using biomarkers reflecting the cardiovascular, metabolic, neuroendocrine and immune systems[17]. It has been consistently shown to be predictive of poor

health outcomes, including premature mortality, cardiovascular disease, and decline in physical and cognitive function[18].

The present study adds to the existing literature on segregation and health in several ways. First, there is a limited empirical work on the association between segregation and biological measures of health[12]. Second, it uses a spatial neighborhood measure that accounts for both the racial/ethnic composition of the focal neighborhood and that of the contiguous neighborhoods. In addition, this analysis uses two distinct AL measures, including one that incorporates neuroendocrine markers, which are considered the primary mediators linking chronic stress and disease[15], but which are typically not available in other studies. Finally, it also examines the interaction between segregation and change in AL with age, using longitudinal data to ask whether segregation is positively associated with age-related increases in AL. As each segregation measure is specific to one racial/ethnic group in the sample, it will be possible to determine if these associations vary by group membership as well.

2. Methods

2.1 *Study Population*

The Multi-Ethnic Study of Atherosclerosis (MESA) is a longitudinal epidemiological study designed to monitor the progression of clinical and subclinical cardiovascular disease. Non-random population samples of adults aged 45 to 84 years were selected from six sites across the United States (Winston Salem, NC; New York, NY; Baltimore, MD; St. Paul, MN; Chicago, IL; and Los Angeles, CA), for a total of 6,814 participants at the baseline exam. Consisting of both an interviewer-assisted survey and a clinic visit, the first exam took place between July 2000 and August 2002. Four follow-up exams took place over the next ten years, with 4,716 individuals (69% of original sample) retained by the final exam (2010-2012). The racial/ethnic groups

represented in the sample include White, Black, Hispanic, and Chinese. Their sample proportions and demographic profiles can be found in Table 1, and have been reported elsewhere[19]. An ancillary study of MESA was conducted during exams three and four (between July 2004 and October 2006) to collect clinical data on stress hormones. Known as MESA Stress, the substudy enrolled an approximately random sample of 1,002 White, Black, and Hispanic participants from the MESA study population at two sites (New York, NY; and Los Angeles, CA). Additional details about exclusion criteria and participation in MESA stress, as well as sample characteristics have been published elsewhere[20].

2.2 *Allostatic Load*

Our study uses both a restricted and a more "complete" measure of allostatic load. The restricted measure consists of eight cardiovascular and metabolic measures available at all five exams. For each of these indicators, standardized scores were calculated to indicate where the individual's value placed them (in standard deviation units) relative to accepted clinical thresholds for higher risk. These cut points are 0.90 hip-waist ratio for men and 0.85 for women, 200 mg/dL for triglycerides, 160 mg/dL for low density lipoprotein (LDL) cholesterol, 40 mg/dL high density lipoprotein (HDL) cholesterol, 4.84 log of glucose (126 mg/dL of glucose), 140 mmHg for systolic blood pressure, 60 mmHg for pulse pressure, and 90 beat/min for heart rate. Higher values for each indicator indicate a higher risk profile. Although only available for on exam, the comprehensive AL score incorporates the cardio-metabolic markers of the restricted measure plus neuroendocrine and inflammatory markers available for the MESA stress sub-sample including D-dimer, interleukin-6, salivary cortisol, tumor necrosis-factor alpha, and factor VIII clotting protein. Without standard clinical cut points for most of these measures, the threshold for high risk is defined as the highest quartile. For both AL measures, individual parameters were summed across

all systems to create an overall AL score – with higher values indicating higher (worse) AL. This operationalization follows the lead of recent scholarship[21] and improves upon previous work that used the sum of dichotomized individual indicators denoting either high or low risk. Standardizing rather than dichotomizing individual scores allows for more nuances in predicting outcomes.

2.3 *Segregation*

Racial segregation is measured separately for each racial/ethnic group using a spatial measure known as the local G_i^* statistic, that reflects the level at which racial minorities are clustered together in contiguous neighborhoods (tracts). Census tracts for participants were determined according to the residential addresses provided at the baseline exam.

The local G_i^* statistic yields a z-score for each census tract[22], which indicates the extent to which the racial composition of that tract (and neighboring tracts) deviates from the mean racial composition of the entire MSA. A greater positive score reflects greater clustering or segregation of that particular racial/ethnic group within a census tract (overrepresentation) in relation to the MSA, while a greater negative score reflects under-representation of that group. Unlike measures such as the index of dissimilarity, the G_i^* statistic expands the scale of the neighborhood beyond the single tract a study participant resides in, to include contiguous tracts.

To determine which contiguous tracts are included as neighboring tracts, our analysis employs the spatial weight matrix known as "first order rook". Using the rook measure, tracts are considered neighbors if they share a border. Tracts that touch the index tract at one point are not recognized. While some conceptualizations rely on a fixed distance (such as 1 or two miles) from the centroid of the index tract, unfortunately some census tracts of MESA participants have no neighbors by this definition. It also ignores that while a one mile radius in New York may be meaningful for social interactions or infrastructure, one mile in North Carolina may not. The

number of neighbors also varies with the size of the census tract, whereas with a rook measure all tracts have at least one neighbor, and the number of neighbors is more consistent across MESA sites.

2.4 *Covariates*

Potential cofounders in the cross-sectional analysis include age, gender, individual socioeconomic status (SES), neighborhood SES, and site of exam. The first model controls for age (a continuous variable) as well as gender, with male as reference category. In the second model, we also adjust for individual SES, which in our analysis includes highest level of education completed, gross family income, and a composite variable measuring wealth. The wealth index assigns a score of 0-4 according to participant ownership of the following assets: owning a home, owning one or more cars, owning land, or owning an investment (such as stocks, bonds, mutual funds, retirement investments). Participants received a zero score for wealth when they did not report any assets and four when they owned all four types of assets. This operationalization reflects previous published work[27]. In the third model, neighborhood SES is added to the previous covariates. Neighborhood SES is represented by an index variable composed of tract data matched from the 2000 US Census. After selecting sixteen census variables from the 2000 US Census, principal component analysis selected six variables that were developed into a summary index. Following previous work[20], the final index includes the following indicators: median household income, percentage with household income <$50,000, median value of owner occupied homes, percentage with at least a high school degree, percentage with at least a bachelors degree, and percentage with managerial/professional occupations. Each indicator was scaled so that higher values indicate higher SES, and transformed into a z-score. All six indicator z-scores were then summed together. Finally, in order to capture any variability in segregation and AL across site as

well as any unmeasured variables associated with site of exam, it is included as a potential cofounder in the last model. In the longitudinal analysis, we adjust for time (a continuous measure), measured as years since the date of participant's baseline exam, as well as an interaction term between segregation and time.

2.5 *Analytical Strategy*

To account for clustering of multiple AL measures within participants, we used generalized estimation equations (GEE) to regress race/ethnicity specific segregation values on allostatic load for a population averaged analysis. Further covariates were included in each additional model, as described above. Additionally, we examine the association between segregation and change in AL over time using mixed effects linear models. In addition to being robust to missing data, mixed effects regression analysis allows for a fixed portion of the model to represent the population average while using random intercepts and slopes to increase the precision of the regression formula for each individual observation. The model contains fixed effects for segregation and covariates (including an interaction term for segregation and time since baseline exam), as each individual will experience an approximately linear trend in change in AL[33]. However, because overall AL scores will vary from person to person, between-person variability is modeled as a random effect and time since first exam is modeled as a random slope. For all mixed effects models, likelihood-ratio tests indicated if the inclusion of random intercepts and slopes was warranted. Finally, multiple ordinary least squares regression (OLS) was used for analysis of the MESA Stress sample, as data from this sample was collected at only one time point. Analysis for mixed effects and OLS followed the same four model structure as the population averaged analysis.

3. Results

Outcomes and covariates stratified by race/ethnicity are presented in Table 1. Mean segregation scores for each group indicate low levels of clustering among white participants within their own census tracts, while Black, Hispanic, and Chinese participants display increasing higher average levels of segregation, although there is also high variability within groups. Mean scores for both outcomes, the restricted AL and comprehensive AL score, are highest in Hispanic participants, followed by Black, Asian (in the restricted score only), and white participants. As noted previously, higher values indicate worse AL. Hispanic participants also experience the greatest mean change in AL between first and last examination, compared to relatively equal levels of change among other groups. Table 2 presents coefficients of the multivariate regression analysis for both the full MESA sample and the MESA Stress subsample, the latter being cross-sectional.

To examine how different racial/ethnic groups experience distinct associations with segregation, and to reflect the race-specific segregation measures, all models are stratified by race/ethnicity. Results suggest important distinct associations between segregation and AL for whites versus non-whites. For white participants, after adjustment for age and gender, greater tract-level clustering of White residents was associated with a lower (better) AL score in the first model using either the restricted AL or comprehensive AL measure (B=-0.129, SE=0.028; B=-0.235, SE=0.039, Table 2 Model 1). Greater clustering of White residents in a neighborhood remained advantageous, though slightly attenuated, for the health of White residents after adjusting for individual SES in both samples. In the full sample, however, the inverse association between segregation and AL is reversed for whites after adjustment for neighborhood socioeconomic status.

The association between matched-race/ethnicity segregation and AL was uniformly positive for the remaining racial/ethnic groups in MESA. For black participants, greater clustering

of black residents was positively associated with a higher (worse) AL score across both samples. This association was still present after adjustment for all covariates in the stress subsample (B=0.082, SE=0.023; Table 2, Model 4) but lacked significance in the population averaged models. Greater segregation of Hispanic residents was positively associated with the restricted measure of AL for Hispanic participants in our initial model (B=0.058, SE=0.021; Table 2, Model 1) but this association disappeared after adjustment for individual SES. As with Black participants, greater clustering of Hispanics was positively associated with our comprehensive AL measure even after adjustment for all covariates (B=0.087, SE=0.032; Table 2, Model 4). Finally, for Chinese participants, greater clustering of Chinese residents is positively associated with a higher AL score in our first model (B=0.055, SE=0.017; Table 2, Model 1) and after adjustment for individual SES (B=0.047, SE=0.019; Table 2, Model 2).

Results in Table 3 display coefficients of our mixed effects models. When including time since baseline exam as both a fixed effect and a random slope, we find greater clustering of white residents is still significantly associated with a better AL score for whites (B=-0.052, SE=0.028; Table 3, Model 1) after adjustment for age, gender, and individual socioeconomic status. This positive association disappears once neighborhood SES is introduced in Model 3. As in our population averaged and cross-sectional models, greater clustering of non-whites within a census tract is consistently associated with a poorer AL score, although significance only holds for Chinese participants (B=0.044, SE=0.017; Table 3, Model 1) when adjusting for gender and age. Lastly, Table 4 investigates whether segregation is associated with time-related increases in AL. Although the patterning of positive vs. negative association remains consistent for whites and non-whites, we find the interaction term between segregation and time elapsed since baseline exam non-significant for all racial/ethnic groups.

4. Discussion

4.1 *Relevance to Existing Literature*

Our results lend themselves to two main conclusions: residential segregation is a significant predictor of AL, even after adjustment for multiple demographic characteristics, and segregation impacts the AL scores of different racial/ethnic groups in distinct ways. Our results are consistent with previous health research that has reported higher white segregation to be associated with lower risk for cardiovascular disease and higher black segregation as associated with higher CVD risk[24]. Furthermore, several studies[24, 25] have also found these associations attenuated by adjusting for neighborhood characteristics. Although previous literature has found mixed impact of segregation on health for Hispanics,[12,23,16] we find Hispanic clustering to exert negative effects on health. It is possible Hispanic segregation may vary in its effects on resident health according to whether the neighborhood consists of foreign or native-born Hispanics. Although prior research has suggested the effect of Hispanic segregation may vary by generational status[26], examining the role of neighborhood clustering by nativity should be a priority for future research. Previous literature on the relationship between Asian segregation and health outcomes is quite limited, although our findings correspond to a recent study linking segregation among Asian Americans with an increased risk of exposure to carcinogenic agents[27].

Our conclusions also align with the significant association found between segregation and inflammatory markers in the sole study to date examining residential segregation and AL[18]. However, in contrast to our findings, they report the deleterious effect of segregation to be similar in magnitude between racial/ethnic groups and no significant interaction between segregation and race with AL. We suggest this discrepancy may reflect the distinct operationalization of both segregation and AL between our studies.

We propose these results have important implications for understanding the relationship between residential segregation and health, foremost that segregation should not be viewed as a uniformly positive or negative influence, but rather one that exerts itself distinctly across racial/ethnic groups. Given the detrimental effects associated with segregation for non-whites in our sample, it appears residential clustering may not confer shelter from racial discrimination or provide stress buffers such as social support[28, 29].

The results of the longitudinal analysis also suggest that individual socioeconomic status plays an important role in determining the effect of segregation on health for White and Hispanic participants. This is consistent with previous work[10,16,30] that identifies the spatial concentration of poverty as a product of segregation and a key pathway through which segregation influences health.

The association between Black clustering and AL is only significant in the Stress subsample with the comprehensive measure of AL, which sensitivity analysis (see Appendix) indicates may be a result of the additional biomarkers rather than the size or composition of the sample. This result is also demonstrated amongst Hispanic participants. The divergent results between the comprehensive AL and restricted AL measures in both Black and Hispanic participants begs the question of what the additional biomarkers of the comprehensive measure are capturing compared to the restricted measure. Although the two measures have a correlation of 0.63, they are designed to capture different physiological systems. As mentioned, the comprehensive measure of AL in this study attempts to improve on previous AL indexes by including additional biomarkers representing the neuroendocrine and immune systems that are also involved in the biological stress response. As they are recognized as the proximate (or immediate) effects, rather than the more accumulative (or downstream) measures of stress, it is possible that

the additional markers of the comprehensive AL measure are sensitive to recent stressors – and thus reflecting recent experiences of stress more prevalent among African Americans and Hispanics. One possible source of daily stress, not controlled for in this analysis, is the experience of discrimination. This would be consistent with research indicating both groups typically report higher levels of discrimination daily than non-Hispanic whites. However, as the literature on segregation and exposure to discrimination is also mixed, it is difficult to say whether these recent stressors are correlated with or causally related to the experience of segregation or not – and if so, then to what degree.

Segregated neighborhoods are also purportedly the source of numerous social, economic, and political stressors[1,31,32]. Given the empirical relationship between stress exposure and accelerated cellular age[2], we expected to see that those residing in highly segregated minority neighborhoods age "faster" compared to those in White neighborhoods. Unfortunately given our results we are unable to determine if segregation has a significant moderation on the relationship between time and AL.

4.*2 Strengths and Limitations*

Several limitations of our analysis should be noted. Neither the MESA sample nor the MESA Stress sample is representative of the national population of the United States. In addition, there is always the potential for a mortality selection effect when studying an older population such as MESA. For inclusion in MESA, participants were also required to be free of cardiovascular disease at baseline, which may further bias our results, and in particular may be an issue when cardiovascular disease becomes prevalent in black populations at earlier ages than non-Hispanic whites. It is also possible we are unable to see the full effects of segregation on AL without examining a younger cohort, or without reference to levels of segregation experienced at earlier

points in the life course. Future research should endeavor to assess levels of segregation at potential critical development periods such as early childhood.

Another important limitation concerns the measure of segregation in this analysis. While this paper has argued that clustering reflects a more important dimension of segregation than evenness with regards to health effects, an absolute measure of clustering – one that expressed the average number of minority residents in nearby areal units as a proportion of the total population in these nearby units – might provide different results than the relative measure used in this analysis. This may be particularly important when comparing MSAs with substantially large minority populations (such as majority minority cities) and MSAs with predominantly white populations – as the clustering of minority groups in majority minority cities may be depressed by a relative measure. Of the six sites where MESA participants were sampled – only one (St. Paul, Minnesota) does not have a significant high proportion of minority residents. The caveat, however, is that this likely matters most for the racial/ethnic group that contributes most to majority minority status – such as African Americans in Baltimore or Hispanics in LA – but less so for Asians in any American city. It also may miss on how the everyday experiences of living in a segregated neighborhood differs between MSA's with large minority populations and those with predominantly white populations.

While most studies on segregation and health rely on a black/white dichotomy, we used a multi-ethnic study to examine variations in the relationship across four racial/ethnic groups. Furthermore, no studies to our knowledge have tested the relationship between segregation and AL using a spatial measurement of segregation, nor using multiple distinct measures of AL. Overall, our findings linking segregation to AL are consistent with the hypothesis that racial

residential segregation is a fundamental cause of health disparities, and further support the idea that AL is a key mechanism by which the social context of segregation "gets under the skin".

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TABLES

Table 1: Sociodemographic and health characteristics by race/ethnicity, Multi-Ethnic Study of Atherosclerosis 2000-2010 (n=6,814)

	NH White	NH Black	Hispanic	Chinese
No. of participants, MESA	2,622	1,892	1,496	804
No. of participants, MESA Stress	197	276	529	N/A
Age, Years Mean (SD)	62.6(10.2)	62.1(10.1)**	61.3(10.3)***	62.3(10.3)
Male	48	44.5	48.2	48.5
Education[a]				
< High school	4.7	11.4	42.1	21.4
High school	17.0	19.0	21.3	16.1
Some College/Technical	27.5	34.7	26.1	21.4
College/Graduate degree	50.9	34.9	10.4	41.2
Household Income[a]				
<24,999	15.2	26.7	46.5	45.4
25,000-49,000	26.3	29.8	33.4	23.3
50,000-74,999	19.9	19.3	10.5	12.3
>75,000	36.3	16.9	7.8	18.5
Wealth Index Mean (SD)	2.76(1.2)	2.13(1.4)***	1.45(1.3)***	1.9(1.4)***
NSES Index Mean (SD)	-4.0(6.2)	1.2(4.7)***	2.0(5.3)***	-2.2(6.2)***
Site				
Winston Salem, NC	21.9	26.4	0.2	0
New York, NY	8.5	20.1	33.2	0.2
Baltimore, MD	20.3	29.3	0	0
St Paul, MN	23.1	0	30.8	0
Chicago, IL	21.2	16.0	0	37.9
Los Angeles, CA	5.0	8.2	35.8	61.8
Segregation Mean (SD)	0.016(1.79)	2.52(2.89)	3.24(3.18)	6.05(4.47)
AL-Restricted Mean (SD)	-8.26(3.86)	-7.67(3.82)***	-6.38(3.91)***	-7.71(3.65)***
AL-Comprehensive[bc] Mean (SD)	-4.74(2.8)	-3.37(2.9)***	-3.33(2.9)***	
Mean Change in AL-Restricted[d]	-0.45	-0.36	-0.74	-0.40

Notes:

Abbreviations - NH: Non-Hispanic, SD: Standard Deviation, NSES: Neighborhood socio-economic status, AL: Allostatic Load

Results are percentages within variable categories and race/ethnicity, unless otherwise indicated

*p<0.05, **p<0.01, ***p<0.001 for comparisons between each racial/ethnic group and non-Hispanic White group using one way ANOVA tests

[a] Categories listed do not reflect level of measurement in analysis

[b] Sample size for AL-Comprehensive is reduced to 1,002

[c] AL-Stress measured at Exam 3 (2004-2006)

[d] Reflects mean change in AL between Exam 1 and Exam 5

Table 2. Coefficients Associated With Each Unit Increase in Baseline Racial/Ethnic Segregation

	Model 1[a]		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Restricted AL								
White	-0.129***	0.028	-0.049	0.029	0.029	0.035	0.003	0.037
Black	0.024	0.021	0.009	0.023	0.002	0.024	0.013	0.027
Hispanic	0.058**	0.021	0.029	0.023	0.017	0.024	0.024	0.025
Chinese	0.055***	0.017	0.047*	0.019	0.038	0.019	0.040	0.019
Comprehensive AL								
White	-0.235***	0.039	-0.140**	0.042	-0.001	0.067	-0.010	0.068
Black	0.043*	0.017	0.064***	0.017	0.069***	0.017	0.082***	0.023
Hispanic	0.153***	0.022	0.111**	0.023	0.076*	0.032	0.087**	0.032

Notes:
*p<0.05; **p<0.01;***p<0.00
[a] Model 1 adjusted for age and gender, Model 2 adjusted for age, gender, and individual socioeconomic status, Model 3 adjusted for age, gender, individual socioeconomic status, and neighborhood socioeconomic status, and Model 4 adjusted for age, gender, individual socioeconomic status, neighborhood socioeconomic status, and site of exam

Table 3. Coefficients Associated With Each Unit Increase in Baseline Racial/Ethnic Segregation, Mixed Effects Analysis

	White		Black		Hispanic		Chinese	
	b	SE	b	SE	b	SE	b	SE
Model 1[a]								
Segregation	-0.099***	0.028	0.024	0.021	0.034	0.021	0.045*	0.017
Time[b]	-0.083***	0.009	-0.055***	0.011	-0.119***	0.013	-0.077***	0.016
Model 2[c]								
Segregation	-0.081*	0.033	0.035	0.024	0.015	0.023	0.039*	0.019
Time[b]	-0.083***	0.009	-0.048***	0.014	-0.137***	0.016	-0.086***	0.023
Segregation*Time	-0.004	0.004	0.003	0.003	0.007	0.004	0.001	0.003

Notes:
*p<0.05; **p<0.01;***p<0.00
[a] Adjusted for age at baseline, gender, and time since baseline exam
[b] Measured as time since baseline exam (years)
[c] Adjusted for age at baseline, gender, time since baseline exam, and an interaction term of segregation by time since baseline exam