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Neighbourhood Socioeconomic Status and Biological "Wear & Tear" in a Nationally Representative Sample of US Adults

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Abstract

Objective—To assess whether neighbourhood socioeconomic status (NSES) is independently associated with disparities in biological "wear and tear"—measured by allostatic load (AL)—in a nationally representative sample of U.S. adults.

Design—Cross-sectional study.

Setting—Population-based U.S. survey, the Third National Health and Nutrition Examination Survey (NHANES III), merged with U.S. Census data describing respondents' neighbourhoods.

Participants—13,184 adults from 83 counties and 1,805 census tracts who completed NHANES III interviews and medical examinations and whose residential addresses could be reliably geocoded to census tracts.

Main Outcome Measures—A summary measure of biological risk, incorporating nine biomarkers that together represent AL across metabolic, cardiovascular, and inflammatory subindices.

Results—Being male, older, having lower income, less education, being Mexican-American, and being both Black and female were all independently associated with worse AL. After adjusting for

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these characteristics, living in a lower SES neighbourhood was associated with worse AL (coeff. = −0.46; CI −0.079, −0.012). The relationship between NSES and AL did not vary significantly by gender or race/ethnicity.

Conclusions—Living in a lower SES neighbourhood in the United States is associated with significantly greater biological wear and tear as measured by AL, and this relationship is independent of individual SES characteristics. Our findings demonstrate that where one lives is independently associated with AL, thereby suggesting that policies that improve NSES may also yield health returns.

Keywords

Adaptation; physiological Neighbourhood; Socioeconomic factors; Gender; Race/Ethnicity

Introduction

Despite decades of research on socioeconomic status (SES) gradients in health, the underlying causes remain only partially understood.[1–4] Place, or where one lives, has been proposed as contributing to such health disparities, beyond individual socioeconomic characteristics.[5–20] Multiple studies have documented independent effects of place on overall health and mortality, using data from nationally-representative surveys.[11, 12, 17, 21, 22] Yet few studies have examined how these effects get under the skin.

Research on cardiovascular risk factors links living in a socioeconomically disadvantaged neighbourhood with: (a) specific risk factors for cardiovascular disease (e.g., elevated cholesterol and blood pressure[7, 22–26]), (b) increased coronary heart disease incidence[24–27], and (c) 1-year case fatality[24, 27], adjusting for individual characteristics. However, except for Sundquist et al.[25] who examined Swedes age 40–64 years, these studies are based on clustered geographic samples, typically from one or a small number of cities; thus, their generalisability has not been demonstrated.

Assessing neighbourhood effects on physiologic dysregulation provides a more cohesive view than studying individual risk factors. Cumulative biologic risk is a way to consider how life experiences (e.g., educational attainment, employment, and neighbourhood socioeconomic environments) contribute to “wear and tear” in multiple biological regulatory systems—first described by Geronimus[28] as “weathering” and subsequently elaborated by McEwen and Stellar[29] as allostatic load (AL). As outlined by McEwen and Stellar[29], stress (in various forms) can result in dysregulation in multiple biological systems (i.e. AL), thereby increasing risk for an array of health outcomes. Indices of cumulative AL are similar to other summary risk indices (e.g., the Framingham risk score[30] or the Acute Physiology And Chronic Health Evaluation (APACHE) score[31]) developed to summarize information from multiple physiologic variables to predict health outcomes.

AL summary indices are meant to capture the multiple and interrelated dysregulations in physiologic systems that develop over time as they are called up to respond to environmental demands. The value of examining cumulative AL indices rather than individual regulatory systems is the hypothesized ability of such indices to better capture the cumulative extent of the interrelated feedback loops among physiological systems. Consistent with the idea that cumulative AL indices provide a measure of the overall burden of physiological dysregulation, prior work using various AL indices has shown such cumulative indices predict mortality, functional decline, and new or recurrent cardiovascular events[33–37].

A multi-systems perspective on biological risks has particular advantages for considering health impacts of broad social or environmental influences because these influences likely impact multiple physiological regulatory systems simultaneously. Indeed, recent analyses of a nationally representative sample of U.S. adults have demonstrated lower individual SES is associated with significantly higher (worse) AL and its system-level subcomponents.[38] Further, this relationship was shown to hold for all major race/ethnic groups.[38] To date, however, no studies have examined the *independent* contribution of neighbourhood SES (NSES) to AL in a nationally representative sample. Thus, we studied whether NSES is associated with cumulative biologic wear and tear, or dysregulation (measured by AL), in a nationally representative sample of U.S. adults, after adjusting for individual-level demographic and SES characteristics.

Methods

Data

Analysis data come from: (1) individual-level data from the geocoded Third National Health and Nutrition Examination Survey, 1988–1994 (NHANES III); and (2) Census data characterizing NSES for each individual NHANES respondent. NHANES data include interview, clinical examination, and laboratory data for a representative sample of U.S. residents. Blacks and Mexican-Americans were over-sampled. Respondents' residential addresses were geocoded to 1990 census tracts, using either address or closest street intersection. Fourteen percent of the sample (predominantly rural residents) could not be geocoded to a census tract and were excluded. We used census tracts to represent neighbourhoods.[14] Neighbourhood characteristics were derived from the 1990 and 2000 decennial Censuses and linked to NHANES III respondents through census-tract identifiers based on 1990 census-tract boundary definitions and the year the respondent was surveyed. Neighbourhood attributes for years between decennial Censuses were estimated by linear interpolation, assuming a constant rate of change in neighbourhood conditions.

Our study sample included 13,184 adults 20 and over who reside in 1,805 census tracts within 83 U.S. counties who completed surveys and medical examinations, who identified themselves as non-Hispanic White, non-Hispanic Black, or Mexican-American, who were not missing key outcome measure components, and whose residential census tract could be geocoded. The final study samples comprised about 76.4 percent of the geocoded NHANES adult sample. Sensitivity analyses revealed that excluded subjects were significantly more likely than those included to be younger, U.S.-born, non-Hispanic White, to have lower educational attainment and family income, to reside in the South or Midwest, and to live in poorer neighbourhoods with fewer minorities.

Measures

Outcome Measures—We followed the methods described by Seeman et al.[38] for constructing an overall AL index based on NHANES III data, including subindices that reflect metabolic, cardiovascular, and inflammatory biomarkers (see Table 1): *metabolic* biomarkers include total cholesterol, HDL cholesterol, glycosylated hemoglobin, and waist/hip ratio; *cardiac* biomarkers are systolic and diastolic blood pressure, and resting heart rate; and *inflammatory* biomarkers are c-reactive protein (CRP) and serum albumin.

We created a dichotomous indicator for each of the nine biomarkers, reflecting those with "high risk" values (assigned a "1") and "lower risk" values (assigned a "0") based on clinically accepted "high risk" criteria.[39–46] Exclusion criteria are detailed elsewhere[38]; however, individuals reporting a current infection were excluded from the CRP measure. Table 1 reports the percent meeting high-risk criteria, the criteria values, and mean values

for the three subindices. The multi-systems AL score was created by summing the subindex scores.

Independent Variables—The individual-level predictor variables include age; gender; race/ethnicity, categorized as non-Hispanic White (hereafter, "White"), non-Hispanic Black (hereafter, "Black"), and Mexican-American; nativity, categorized as U.S.-born or foreign-born; a ratio of household income relative to the U.S. federal poverty line determined by area of residence and household size (hereafter. the income to poverty ratio, or IPR); educational attainment, categorized as grade-school only, some high school, high school graduate, or post-high school; and marital status (single, married, or other).

To construct the NSES index, we identified 12 theoretically relevant census-tract-level variables and assessed their relationships using factor analysis.[47] We selected the six that loaded highly on the factor that most represented NSES: (1) percent of adults older than 25 with less than a high school education; (2) percent male unemployment; (3) percent of households with income below the poverty line; (4) percent of households receiving public assistance; (5) percent of female-headed households with children; and (6) median household income. We transformed the variables so higher values corresponded to higher NSES and standardized each variable to a mean of zero and standard deviation of one. Finally, we added the six variables and standardized the sum; the resulting NSES index has a zero mean and standard deviation of one (Cronbach's alpha = 0.93). An index value greater than zero denotes a tract with an NSES level above the sample mean.

Analysis

To examine the effect of NSES on total AL, we estimated three-level hierarchical linear models (HLM), with random intercepts at both the county and tract level. To confirm the anticipated parallel NSES gradients for multiple biologic pathways—represented by various AL components—we also estimated HLM models for the three major subindices reflecting metabolic, cardiovascular, and inflammatory components. To assess whether a cumulative index is necessary to capture NSES effects, we further examined the nine dichotomous individual AL biomarkers using generalized linear mixed models[48] (i.e., multilevel logistic models), with random intercepts at tract and county levels.

Analyses adjusted for non-independence of observations at tract and county levels and partitioned the variance of the dependent variables into individual, tract, and county components. Models controlled for all individual-level variables. Based on prior literature, we assessed whether there were gender differences in the relationships between other individual demographic characteristics and AL.[18, 49] Because there were significant gender differences in race/ethnicity and in IPR, we included two interactions (gender*black and gender*IPR) in our model-building process. All models estimating NSES effects control for individual age (and age-squared to capture potential nonlinearity in age effects), race/ethnicity, and individual SES (education and IPR). To address a potential source of selection bias, we conducted secondary analyses excluding individuals who reported cancer, stroke, or heart disease. All analyses were weighted to account for sampling design and survey nonresponse.

RAND Corporation's Institutional Review Board (IRB) approved the study, and the National Center for Health Statistics' (NCHS's) IRB approved the NHANES III survey. Analyses were performed at the NCHS's secure Research Data Center in Hyattsville, Maryland, and conducted using SAS Version 9.2.

Results

The weighted sample was 51.9 percent male and 80.9 percent White, 12.9 percent Black, and 6.1 percent Mexican-American (see Table 1). Subjects ranged from **20** to 90 (range of 22 to 77 between the 5th and 95th percentile; mean = 45), and the mean level of education was 12.6 years. The mean IPR was 3.19 (standard deviation = 1.91). NSES was a standardized index of the six component items (mean = 0). AL ranged from 0 to 9 (range of 0 to 4 between the 5th and 95th percentile. Mean AL score was 1.56 (standard deviation = 1.35).

Table 2 shows NSES was negatively associated with total AL, controlling for individual-level SES and other demographic characteristics (i.e., education, IPR, age, age-squared, gender, race/ethnicity). Specifically, an estimated AL difference of −0.046 (CI −0.079, −0.013) occurs for each standard deviation difference in NSES. Examining the three major AL subindices revealed each was negatively related to NSES, although only associations for the metabolic and cardiovascular indices were statistically significant.

More detailed analyses of associations between NSES and the dichotomous risk indices for the individual AL biomarkers revealed NSES had an independent and statistically significant negative association with HDL cholesterol (OR=0.863; CI 0.802,0.929) and both systolic blood pressure (OR= 0.892;CI 0.821,0.969) and diastolic blood pressure (OR=0.884;CI 0.803,0.974). While the independent relationship between NSES and the other biomarkers was not statistically significant, the effect was in the expected direction for each, with lower NSES associated with greater likelihood of higher risk levels.

While AL score distributions differed for race, gender, and IPR subgroups, tests for interactions between NSES and gender, race/ethnicity, and IPR were not significant (analyses not shown). Thus, the independent relationship between NSES and AL was consistent across all these major population subgroups.

In secondary analyses excluding subjects who reported cancer, stroke, or heart disease, NSES was significantly associated with AL (coef. = −0.048; CI -.086, −0.010). Table 2 shows the same subindices and individual biomarkers were significant; however, the association between NSES and resting heart rate also became significant (OR=0.858;CI 0.760,0.969).

Discussion

NSES was negatively associated with total and major AL subindices in a nationally representative sample of U.S. adults—effects independent of individual-level characteristics, such as race/ethnicity, gender, and household IPR. Indeed, strong findings for the summary AL index, and weaker but consistently negative associations for individual biologic parameters, provide compelling evidence NSES impacts health status through its simultaneous and cumulative impact on multiple interrelated biologic systems. Use of a cumulative index allowed us to better capture the sum total of the more modest differences in some of the biomarkers that contribute to significant differences in overall biological risks by NSES.[37]

Additionally, our findings indicate consistent effects across race/ethnic, gender, and IPR subgroups, suggesting health benefits of living in a higher SES neighbourhood accrue to individuals regardless of these individual characteristics, and indicate that, on average, members of these major population groups do not differ in their vulnerability to the greater biological wear and tear associated with living in lower SES neighbourhoods. However, a

new generation of epigenetic studies may further shed light on how adverse life conditions impact gene expression, which, in turn, may be manifest in measures such as AL.

Our findings have important population health implications. In considering the health impact of NSES, both the size of NSES effects on AL and the substantial variation in NSES nationally are important. For example, disparities in health are illuminated by differences in neighbourhoods within the metropolitan areas of Washington, DC and Detroit, Michigan. In the three-mile drive from Washington's Capitol Hill neighbourhood to the nearby Anacostia neighbourhood, NSES decreases by 65 percent. Our findings indicate such a change in NSES is associated with an average of 0.12 point higher (worse) AL and is even starker when we consider individuals living in the two Detroit communities divided by Alter Road. Travelling from Grosse Point to the adjoining East Detroit, NSES drops 86 percent. Our findings indicate this would entail a 0.26 point difference in AL between the individuals living in these two communities.

Seeman[38] provides further relevance for these AL differences, finding that, on average, a one-unit higher AL is associated with a 17 percent increase in mortality in the NHANES III cohort. Applying these results to our findings, we predict a 2 percent increase in mortality for people living in Anacostia versus those in the Capitol Hill neighbourhood and a 4 percent increase for individuals living in East Detroit versus Grosse Point. Moreover, the relationship between AL and mortality is stronger in younger adults[38]; in these two examples, adults 25–35 would experience increased mortality of 7 percent and 16 percent, respectively. These examples do not represent the full range of NSES in the nation. Thus, comparing well-known, severely disadvantaged, and highly affluent neighbourhoods would represent a larger difference in NSES and in estimated mortality risk. We also expect health trajectories and outcomes might differ by NSES, just as they do by individual-level SES. If so, these illustrative examples may be conservative because they assume health trajectories do not differ by NSES beyond the AL impact. Finally, such estimates likely understate the full impact of differences in NSES on AL or mortality, because they reflect "point-in-time" estimates; actual neighbourhood effects are likely cumulative over the life course, whether their NSES has changed or varied over time.

Others have suggested NSES "gets under the skin" and contributes to health disparities through social networks and social support, health behaviours, and hypervigilance in response to neighbourhood safety concerns or other stressors, such as unemployment and discrimination.[4, 15] Also, built environment characteristics highly correlated to NSES are thought to play a role, including residential crowding, walkability, and access to high-quality food. Thus, the environments we live in can enhance or constrain the opportunity to pursue a healthy life, thereby contributing to socioeconomic disparities in health and mortality.[18] Furthermore, as one reviewer noted, we also need research on individual biologic pathways through which NSES affects health.

The study has several strengths, including the use of a nationally representative sample of U.S. adults and biological data and the ability to control for multiple individual-level socioeconomic and demographic characteristics, thus allowing us to assess NSES's independent contribution. However, this study has some important limitations. First, using cross-sectional data means we cannot determine whether the NSES and AL relationship is causal. For example, we cannot control for neighbourhood self-selection (i.e., those in poor health end up in poor neighbourhoods). However, using cumulative biological risk measures, rather than overt disease, may help minimize the potential effects of such self-selection, if they exist. Unlike overt disease, which may spur some individuals to make major life changes, individuals are far less likely to be aware of, much less to have made decisions about, whether (and where) to move based on their AL. Moreover, the relationship

between NSES and AL held even when individuals with overt conditions (including cancer, stroke, and heart disease) were excluded. This secondary analysis reduces the possibility our findings reflect movement of individuals with health conditions into poorer neighbourhoods. However, NSES and AL may also have reciprocal effects over time or across generations. We need longitudinal studies to address these questions.

Second, operationalising AL using NHANES measures also leads to some potential limitations. For example, NHANES's inflammation measures are limited. Moreover, by excluding CRP levels in calculating AL for individuals with a current infection, we may have excluded some with chronically high inflammation levels, thereby leading to underestimating the relationship between NSES and inflammation.[50]

Third, research on "neighbourhoods" is limited by the need to conceptualize and operationalise geographic spaces. It is difficult to apply geographic boundaries nationally that are meaningful on an individual and programmatic or policy level.[6, 16] While somewhat imprecise as measures of neighbourhood context, census-tract characteristics have been used in most neighbourhood studies.[14, 15] However, the resulting measurement error when applied to the broader construct of "neighbourhood" suggests our findings are likely conservative. This possibility is increased because excluded subjects were more likely to have lower educational attainment and family income and live in poorer neighbourhoods. Also, because addresses that could not be geocoded were primarily from rural residents, our results may not be generalisable to more rural populations.

Finally, NHANES III data allow us to assess the relationship between NSES and AL nationally from 1988 to 1994 and to estimate its impact on mortality based on cohort survival. The NCHS is now geocoding subsequent NHANES data, but to our knowledge, NHANES III data are the only national population data that include AL measures and for which geocoding has already been completed. Examining additional geocoded NHANES data will allow us to assess whether the relationship between NSES and AL has changed over time, independent of individual-level characteristics.

Despite these limitations, our findings have important policy implications. They demonstrate that beyond individual-level socioeconomic factors, where one lives is independently associated with AL, thereby suggesting policies that improve NSES may also yield health returns. Williams et al.[4] argue that the disproportionate distribution of Blacks and Mexican-Americans in low-NSES neighbourhoods is largely due to segregation. Hence, rather than limiting the discussion about policies that can address disparities to those focusing on individual-level health improvements, our findings suggest improving neighbourhood socioeconomic conditions may have significant long-term impacts on improving health and reducing health disparities.

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What is already known on this subject?

People with low socioeconomic status (SES) experience increased physiologic dysregulation as indicated by cumulative, multi-system indices of biological parameters, such as allostatic load (AL).

Living in a neighbourhood with low socioeconomic status (NSES) is associated with poor overall health and increased mortality risk, independent of individual-level SES.

What does this study add?

People who live in disadvantaged neighbourhoods experience increased physiologic dysregulation, irrespective of their own SES.

The relationship between NSES and dysregulation is stronger for a composite index than individual components of AL, providing compelling evidence that NSES impacts health status through multiple biologic systems. Moreover, the relationship between NSES and AL holds even when individuals with overt conditions (including cancer, stroke and heart disease) are excluded from the sample.

Table 1

Weighted Characteristics of Study Participants (n=13,184)

Characteristic	Value
Female (%)	51.9%
Age (yr)	
Mean	45.0
Range (1 – 99 percentiles, see note)	20 – 86
Race/ethnicity (%)	
Non-Hispanic White	80.9%
Non-Hispanic Black	12.9%
Mexican-American	6.1%
Married (%)	64.8%
Nativity - Foreign Born (%)	9.1%
Education (yrs)	
Mean	12.6
Range (1 – 99 percentiles, see note)	2 – 17
Income to Poverty Ratio (IPR)	
Mean	3.19
Range (1 – 99 percentiles, see note)	0.2 – 8.5
Neighborhood SES Index (NSES)	
Mean	0.00
Range (1 – 99 percentiles, see note)	−3.18 – 1.62
Allostatic Load (Total)(mean)	
Mean	1.56
Range (1 – 99 percentiles, see note)	0 – 5
Inflammation subindex (mean)	0.25
Albumin (%<3.8 g/dL)[45]	7%
C-reactive protein [CRP] (% ≥ 0.3 mg/dL)[46]	20%
Metabolic subindex (mean)	1.09
Waist to Hip ratio (% > 0.90 for men; % > 0.85 for women)[39]	61%
Total cholesterol (% ≥ 240 mg/dL)[41]	19%
HDL cholesterol (% < 40mg/dL)[41]	23%
Glycosolated Hemoglobin (% ≥ 6.4%)[42, 43]	6%
Cardiovascular subindex (mean)	0.28
Resting Heart Rate (% ≥ 90bt/min)[44]	6%
Systolic blood pressure (% ≥ 140mm Hg)[40]	15%
Diastolic blood pressure (% ≥ 90mm Hg)[40]	6%

Note: As a data security measure to prevent the examination of small cells in the data, the National Research Data Center limits reporting to the 1st to the 99th percentiles.

Table 2

Relationship of AL and Its Components and NSES, Adjusting for Gender, Racial/Ethnic Group and Individual Characteristics

	Independent Effect of a One Standard Deviation Difference in NSES			
	β coefficient	**95% CI**	**OR**	**95% CI**
Total AL	−0.046	[−0.079, −0.013]		
Inflammation	−0.002	[−0.017, 0.014]		
Albumin			0.954	[0.855, 1.065]
CRP			0.931	[0.850, 1.021]
Metabolic	−0.031	[−0.053, −0.010]		
Waist to Hip Ratio			0.965	[0.897, 1.039]
Total Cholesterol			0.984	[0.929, 1.043]
HDL Cholesterol			0.863	[0.802, 0.929]
Glycosylated Hemoglobin			0.955	[0.867, 1.052]
Cardiovascular	−0.019	[−0.034, −0.004]		
Resting Heart Rate			0.898	[0.801, 1.007]
Systolic Blood Pressure			0.892	[0.821, 0.969]
Diastolic Blood Pressure			0.884	[0.803, 0.974]

Notes: Each measure represents a separate regression model. Sample sizes ranged from 10,981 for CRP to 13,184 for total AL. The models all include gender, race/ethnicity, age, age squared, IPR, nativity, education, marital status, and the interactions of gender with black and with IPR.

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APPENDIX Table 1

Weighted Characteristics of Study Participants (n=13,184)

Characteristic	Full Sample	Low NSES Quartile	High NSES Quartile
Female (%)	51.9%	53.9%	50.8%
Age (yr)			
Mean	45.0	44.4	46.6
Range (1 – 99 percentiles, see note)	20 – 86	20 – 86	20 – 84
Race/ethnicity (%)			
Non-Hispanic White	80.9%	53.7%	94.9%
Non-Hispanic Black	12.9%	32.7%	2.9%
Mexican-American	6.1%	13.6%	2.2%
Married (%)	64.8%	54.3%	75.5%
Nativity - Foreign Born (%)	9.1%	12.2%	7.4%
Education (yrs)			
Mean	12.6	11.0	14.2
Range (1 – 99 percentiles, see note)	2 – 17	0 – 17	7 – 17
Income to Poverty Ratio (IPR)			
Mean	3.19	2.13	4.39
Range (1 – 99 percentiles, see note)	0.2 – 8.5	0.1 – 6.3	0.4 – 9.4
Neighborhood SES Index (NSES)			
Mean	0.00	−1.38	1.02
Range (1 – 99 percentiles, see note)	−3.18 – 1.62	−3.95 – −0.50	0.70 – 1.77
Allostatic Load (Total)(mean)			
Mean	1.56	1.70	1.48
Range (1 – 99 percentiles, see note)	0 – 5	0 – 5	0 – 5
Inflammation subindex (mean)	0.25	0.30	0.24
Albumin (%<3.8 g/dL)[44]	7%	8%	6%
C-reactive protein [CRP] (% ≥ 0.3 mg/dL)[45]	20%	25%	19%
Metabolic subindex (mean)	1.09	1.15	1.03
Waist to Hip ratio (% > 0.90 for men; % > 0.85 for women)[38]	61%	64%	60%

Characteristic	Full Sample	Low NSES Quartile	High NSES Quartile
Total cholesterol (% ≥ 240 mg/dL)[40]	19%	19%	19%
HDL cholesterol (% < 40mg/dL)[40]	23%	24%	20%
Glycosolated Hemoglobin (% ≥ 6.4%)[41, 42]	6%	9%	5%
Cardiovascular subindex (mean)	0.28	0.34	0.24
Resting Heart Rate (% ≥ 90bt/min)[43]	6%	8%	5%
Systolic blood pressure (% ≥ 140mm Hg)[39]	15%	18%	13%
Diastolic blood pressure (% ≥ 90mm Hg)[39]	6%	8%	6%

Note: As a data security measure to prevent the examination of small cells in the data, the National Research Data Center limits reporting to the 1st to the 99th percentiles; “Low NSES” refers to individuals in the first NSES quartile; “High NSES” refers to those in the fourth NSES quartile.

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APPENDIX Table 2

Showing all Coefficients and CIs for Total AL and Its System-level Subcomponents. (MODEL 4)

	Total AL	Inflammatory	Metabolic	Cardiovascular
Gender (female = 1)	−0.2879** [−0.4222, −0.1536]	0.0208 [−0.0365, 0.0781]	−0.3225** [−0.4218, −0.2232]	−0.0028 [−0.0555, 0.0499]
Race/Ethnicity				
Mexican-American	0.2394** [0.1583, 0.3206]	0.0665** [0.0338, 0.0991]	0.1525** [0.0874, 0.2176]	0.0198 [−0.0187, 0.0583]
Non-Hispanic Black	−0.1566** [−0.2554, −0.0579]	0.0936** [0.0542, 0.1331]	−0.3361** [−0.4028, −0.2695]	0.1367** [0.0804, 0.1931]
Non-Hispanic White	*Ref.*	*Ref.*	*Ref.*	*Ref.*
Age	0.0827** [0.0706, 0.0948]	0.0087** [0.0044, 0.0130]	0.0659** [0.0566, 0.0752]	0.0092** [0.0047, 0.0138]
Age Squared	−0.0005** [−0.0006, −0.0004]	−0.00003+ [−0.0001, 0.0000]	−0.0005** [−0.0005, −0.0004]	0.00002 [−0.0000, 0.0000]
Black-Gender Interaction	0.4246** [0.3150, 0.5343]	0.1368** [0.0916, 0.1820]	0.3701** [0.2803, 0.4599]	−0.0256 [−0.0810, 0.0297]
IPR	−0.0202+ [−0.0446, 0.0043]	−0.0204** [−0.0294, −0.0113]	0.0025 [−0.0156, 0.0206]	−0.0058 [−0.0174, 0.0058]
U.S. Born	0.0578 [−0.0300, 0.1455]	0.0301+ [−0.0061, 0.0662]	0.0049 [0.0636, 0.0735]	0.0207 [−0.0212, 0.0626]
Education				
No High School	0.1678** [0.0783, 0.2573]	0.0329+ [−0.0052, 0.0709]	0.1540** [0.0830, 0.2250]	−0.0010 [−0.0394, 0.0374]
High school	0.1512** [0.0685, 0.2338]	0.0449** [0.0091, 0.0806]	0.1089** [0.0501, 0.1677]	−0.0006 [−0.0427, 0.0415]
Some college	0.0950* [0.0005, 0.1895]	0.0345+ [−0.0058, 0.0748]	0.0340 [−0.0423, 0.1103]	0.0161 [−0.0337, 0.0659]
College+	*Ref.*	*Ref.*	*Ref.*	*Ref.*
Marital Status				
Single	0.0022 [−0.0875, 0.0918]	0.0073 [−0.0290, 0.0437]	−0.0407 [−0.1107, 0.0293]	0.0360+ [−0.0015, 0.0734]
Other	−0.0739+ [−0.1493, 0.0015]	0.0015 [−0.0335, 0.0366]	−0.0872** [−0.1399, −0.0345]	0.0159 [−0.0169, 0.0486]
Married	*Ref.*	*Ref.*	*Ref.*	*Ref.*
IPR-Gender Interaction	−0.0518** [−0.0881, −0.0156]	0.0250** [0.0077, 0.0422]	−0.0568** [−0.0815, −0.0320]	−0.0141+ [−0.0291, 0.0009]
NSES	−0.0459** [−0.0792, −0.0127]	−0.0017 [−0.0166, 0.0133]	−0.0313** [−0.0525, −0.0101]	−0.0188** [−0.0343, −0.0034]
Sigma(County)	0.0144**	0.0068**	0.0040*	0.0020**
Sigma(Tract)	0.1463**	0.0171**	0.0868**	0.0251**
Sigma(Residual)	1.2173**	0.2132**	0.5832**	0.2494**

APPENDIX Table 3

Showing Progressive Adjustment of the models for Total AL.
Model 1 includes all variables except IPR, its interaction with gender, and NSES. Model 2 is Model 1 with the addition of IPR and its interaction with gender. Model 3 is Model 1 with NSES. Model 4 is the full model as presented in Table 2.

	Model 1	Model 2	Model 3	Model 4
Gender (female = 1)	−0.4595 ** [−0.5225, −0.3965]	−0.2873 ** [−0.4216, −0.1529]	−0.4593 ** [−0.5221, −0.3965]	−0.2879 ** [−0.4222, −0.1536]
Race/Ethnicity				
Mexican-American	0.2995 ** [0.2169, 0.3822]	0.2616 ** [0.1802, 0.3430]	0.2640 ** [0.1812, 0.3469]	0.2394 ** [0.1583, 0.3206]
Non-Hispanic Black	−0.1129 * [−0.2050, −0.0207]	−0.1202 * [−0.235, −0.0268]	−0.1678 ** [−0.2644, −0.0713]	−0.1566 ** [−0.2554, −0.0579]
Non-Hispanic White	*Ref.*	*Ref.*	*Ref.*	*Ref.*
Age	0.0783 ** [0.0665, 0.0901]	0.0827 ** [0.0706, 0.0947]	0.0787 ** [0.0668, 0.0906]	0.0827 ** [0.0706, 0.0948]
Age Squared	−0.0004 ** [−0.0005, −0.0003]	−0.0005 ** [−0.0006, −0.0004]	−0.0004 ** [−0.0005, −0.0003]	−0.0005 ** [−0.0006, −0.0004]
Black-Gender Interaction	0.4944 ** [0.3930, 0.5957]	0.4276 ** [0.3178, 0.5373]	0.4891 ** [0.3871, 0.5911]	0.4246 ** [0.3150, 0.5343]
IPR		−0.0234 + [−0.0475, 0.0007]		−0.0202 + [−0.0446, 0.0043]
U.S. Born	0.0561 [−0.0314, 0.1437]	0.0601 [−0.0272, 0.1474]	0.0537 [−0.0345, 0.1419]	0.0578 [−0.0300, 0.1455]
Education				
No High School	0.2501 ** [0.1630, 0.3373]	0.1793 ** [0.0913, 0.2674]	0.2271 ** [0.1372, 0.3171]	0.1678 ** [0.0783, 0.2573]
High school	0.1966 ** [0.1141, 0.2791]	0.1562 ** [0.0736, 0.2389]	0.1856 ** [0.1031, 0.2682]	0.1512 ** [0.0685, 0.2338]
Some college	0.1199 * [0.0166, 0.2232]	0.0964 * [0.0021, 0.1907]	0.1157 * [0.0126, 0.2188]	0.0950 * [0.0005, 0.1895]
College +	*Ref.*	*Ref.*	*Ref.*	*Ref.*
Marital Status				
Single	0.0163 [−0.0723, 0.1048]	0.0064 [−0.0829, 0.0958]	0.0094 [−0.0795, 0.0984]	0.0022 [−0.0875, 0.0918]
Other	−0.0324 [−0.1064, 0.0416]	−0.0706 + [−0.1451, 0.0040]	−0.0396 [−0.1148, 0.0357]	−0.0739 + [−0.1493, 0.0015]
Married	*Ref.*	*Ref.*	*Ref.*	*Ref.*
IPR-Gender Interaction		−0.0521 ** [−0.0884, −0.0159]		−0.0518 ** [−0.0881, −0.0156]
NSES			−0.0652 ** [−0.0970, −0.0334]	−0.0459 ** [−0.0792, −0.0127]

+ Coefficient significant at the 0.10 level,

* Coefficient significant at the 0.05 level,

** Coefficient significant at the 0.01 level.

APPENDIX Table 4

Table 2 showing analyses excluding subjects with self-reported history of cancer, heart disease, or stroke.

	Independent Effect of a One Standard Deviation Difference in NSES			
	β coefficient	**95% CI**	**OR**	**95% CI**
Total AL	−0.048	[−0.086, −0.010]		
Inflammation	0.000	[−0.016, 0.016]		
Albumin			0.949	[0.841, 1.071]
CRP			0.941	[0.854, 1.038]
Metabolic	−0.032	[−0.055, −0.010]		
Waist to Hip Ratio			0.950	[0.883, 1.023]
Total Cholesterol			0.976	[0.917, 1.040]
HDL Cholesterol			0.872	[0.805, 0.944]
Glycosolated Hemoglobin			0.949	[0.854, 1.054]
Cardiovascular	−0.023	[−0.040, −0.006]		
Resting Heart Rate			0.858	[0.760, 0.969]
Systolic Blood Pressure			0.862	[0.786, 0.944]
Diastolic Blood Pressure			0.873	[0.783, 0.973]

NOTE: Sample sizes ranged from 9,906 for CRP to 11,893 for total AL.