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Individual and Neighborhood Socioeconomic Status and Inflammation in Mexican-American Women: What is the Role of Obesity?

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Abstract

Objective—Inflammation may represent a biological mechanism underlying associations of socioeconomic status (SES) with cardiovascular disease (CVD). The current study examined relationships of individual and neighborhood SES with inflammatory markers in Mexican-American women and evaluated contributions of obesity and related health behaviors to these associations.

Methods—A random sample of 284 Mexican-American women (mean age 49.74 years) was recruited from socioeconomically diverse South San Diego communities. Women completed measures of sociodemographic characteristics and health behaviors, and a physical examination with fasting blood draw for assay of plasma C-reactive-protein (CRP), interleukin-6 (IL-6), and soluble intercellular adhesion molecule-1 (sICAM-1). Neighborhood SES was extracted from the US Census Bureau 2000 database.

Results—In multilevel models, a one-standard deviation (SD) higher individual and neighborhood SES related to a 27.35% and 23.56% lower CRP ($p < .01$), a 7.04% and 5.32%

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lower sICAM-1 ($p < .05$), and a 10.46% ($p < .05$) and 2.40% lower IL-6 level (*NS*), respectively. Controlling for individual SES, a one-SD higher neighborhood SES related to a 18.05% lower CRP ($p = .07$); there was no unique effect of neighborhood SES for IL-6 or sICAM-1. Differences in body mass index, waist circumference, and dietary fat consumption contributed significantly to SES-inflammation associations.

Conclusions—The findings support a link between SES and inflammatory markers in Mexican-American women, and implicate obesity and dietary fat in these associations. Additional effects of neighborhood SES were not statistically significant. These findings should be viewed tentatively because the relatively small sample size limits the evaluation of multiple contextual factors.

Keywords

Cardiovascular Disease; Hispanic; Inflammation; Obesity; Socioeconomic Status

INTRODUCTION

A growing body of research suggests that in addition to individual (or household) socioeconomic status (SES), neighborhood SES predicts cardiovascular disease (CVD) etiology and progression (1–5) (for discussion, 6). The factors contributing to individual and neighborhood SES effects are likely to overlap and to share final behavioral and physiological conduits, although neighborhood SES may also exert unique contextual influences on cardiovascular risk (6–8).

In examining physiological pathways that connect SES with CVD, inflammation represents an important focus. Substantial evidence indicates that inflammation is fundamental to all stages of atherosclerotic CVD (9, 10). Inflammatory markers including pro-inflammatory cytokines (e.g., interleukin-6; IL-6), acute phase reactants (especially C-Reactive Protein; CRP), and cellular adhesion molecules (e.g., soluble intracellular adhesion molecule-1; sICAM-1), predict CVD morbidity and mortality (11, 12), although it remains unclear whether they are *causally* implicated in CVD etiology (13, 14). Individuals of lower SES also display greater systemic inflammation than their higher SES counterparts (15, 16), and persons who reside in lower SES neighborhoods have elevated inflammation relative to those in more affluent communities (17–21). However, not all studies have explicitly examined the unique role of neighborhood SES after accounting for individual SES (17, 19).

Several factors may contribute to SES-inflammation associations. First, low individual or neighborhood SES may promote exposure to psychological stress across multiple domains and contexts (22, 23). Individuals with lower personal SES often report a greater number of stressful life events, everyday stressors, and chronic stressors (22). Residence in lower SES communities may promote exposure to additional social and ambient stressors, such as crowding, violence, and traffic (24). In turn, neuroendocrine pathways linked with stress-induced activation of the hypothalamic adrenal medullary and sympathetic nervous systems foster up-regulation of innate inflammatory responses (25). Physical challenges, such as noise, environmental toxins, or poor air quality, can further activate inflammatory processes (26).

Additional research suggests that obesity and body composition play key roles in the SES-inflammation association (27, 28). Specifically, stress-induced neuroendocrine activation fosters the accumulation of excess body weight, especially visceral abdominal fat (29). In turn, metabolically active adipose cells secrete pro-inflammatory cytokines (e.g. IL-6), which trigger production of cellular adhesion molecules (i.e., sICAM-1) and acute phase reactants (e.g., CRP) (30, 31). Additionally, unfavorable behaviors associated with stress and/or reduced resources can increase SES-related obesity risks (32, 33). Individuals with

lower personal SES may be unable to afford fresh produce or may lack information about healthy diet and activity patterns; lower SES neighborhoods may have fewer food stores that offer healthy choices and may hold limited opportunities for safe, low-cost physical activity. In addition, healthier diet and activity behaviors have been related to reduced inflammation even after accounting for obesity (34–36).

Prior research suggests that associations of inflammation with individual and neighborhood SES vary across demographic groups, with several studies showing inconsistent associations in ethnic minorities (18, 37–39). However, few studies have examined these associations in Latinos, who form a large and growing segment of the US population (40), and who are at disproportionate risk for certain CVD risk factors including obesity and type 2 diabetes (41, 42). In the National Health and Nutrition Examination Survey III, individual education and income related inversely to an inflammatory composite in Mexican-Americans (43). Among Latino participants (of varied ancestry) in the Multi-Ethnic Study of Atherosclerosis (MESA), higher family income related to lower IL-6, but not CRP; participant education was unrelated to either marker (38). Another report from MESA established inverse, cross-sectional associations of neighborhood SES with CRP and IL-6 and a longitudinal inverse association with IL-6, after accounting for individual SES (21); however, ethnicity-stratified analyses were not reported. In general, SES-health gradients are inconsistent in Latinos, sometimes showing a flattened or even reversed pattern (i.e., higher SES, worse health) that may vary by national origins, nativity, or acculturation (44–47). Additional research is needed to examine SES-inflammation associations in well-defined Latino subgroups.

The current study examined relationships between individual SES and inflammatory markers in middle-aged Mexican-American women from SES-diverse communities in Southern San Diego. We predicted that higher scores on a multi-faceted personal SES composite would relate to lower levels on CVD-relevant inflammatory indicators (CRP, IL-6, and sICAM-1). In addition, the current study examined the unique predictive utility of a neighborhood SES composite after accounting for the same indicators at the individual/family level. These analyses were viewed as exploratory, given the relatively small sample size to examine contextual effects and because sampling was not stratified by census tract. Finally, we sought to examine the specific contributions of obesity and related behavioral pathways (diet, physical activity) to SES-inflammation associations. Because prior studies have identified ethnic and gender differences in biobehavioral mechanisms underlying SES-inflammation relationships (38, 48), examining these pathways in a discrete gender-ethnic group is an important contribution of the present study.

Methods

Participants and Recruitment

Between 2006 and 2009, participants were recruited using targeted telephone and mail procedures from socioeconomically-varied South San Diego neighborhoods with high densities of Latino residents (49). A commercial database (Haines and Company, North Canton, OH) was used to generate lists of Latino (based on surname) female residents between 40–65 year old; women were then randomly selected for recruitment. Sample characteristics were monitored to ensure that the household income distribution was representative of that of the Mexican-American female population in the selected communities. Women were eligible if self-identified as Mexican-American, literate, and free of physician-diagnosed major health conditions (CVDs, treated type 2 diabetes, liver disease, kidney disease, chronic inflammatory conditions, cancer other than non-malignant skin cancers, major mental health problems) and medications with autonomic effects (e.g., stimulants, steroids, major tranquilizers). Of 656 women screened, 363 (55.3%) were eligible and 321 (88.4% of eligible women) enrolled. The current study excluded 17 women

without physical data, 15 without census tract SES information, and 5 with evidence of acute illness (by diary report, $n = 2$, or elevated leukocyte counts, $n = 3$), for a final sample of 284. Women resided in 65 different census tracts.

Procedures

Trained bilingual research technicians gathered self-report and clinical data during two home visits. Participants were asked to refrain from anti-inflammatory medications for at least 48 hours, alcohol for 24 hours, food or beverages (besides water) for 9 hours, and caffeine and tobacco for 30 minutes before the fasting blood draw. All participants provided written informed consent, and Institutional Review Boards at San Diego State University and the University of California San Diego (UCSD) approved study procedures.

Inflammatory markers—Blood samples were transferred on ice, processed, and stored at -80°C until assayed at UCSD laboratories. Circulating levels of plasma CRP, IL-6, and sICAM-1 were determined by multiplex enzyme-linked immunosorbent assay with two sets of internal controls (Meso Scale Discovery Pro-Inflammatory I Panel, with Sector Imager 2400, Gaithersburg, Maryland). Inter- and intra-assay coefficients of variation were $< 10\%$ (50).

Obesity and related behavioral pathways—Trained technicians obtained weight, height, and waist circumference (WC) while participants wore light indoor clothing and no shoes. Weight was measured in the standing position to the nearest 0.1 kg using a calibrated digital scale and height was measured to the nearest 0.1 cm with a standard stadiometer. Waist circumference was measured using a flexible, firm tape measure, to the nearest 0.1 cm at the narrowest point between lower end of the rib cage and iliac crest (51). Waist circumference was regressed on BMI and the residuals were used to represent the independent influence of abdominal adiposity. Dietary fat (% of total calories) and fruit and vegetable consumption (servings/week) were assessed with two screening instruments (52), which generate macronutrient scores that correlate closely with those from the validated, full-length Block Food Frequency Questionnaire (53). Spanish translations of these scales were developed using forward and backward translation for a prior study of Mexican-Americans (54). Physical activity was assessed with the Leisure Time Exercise Questionnaire (LTEQ) (55), which estimates total Metabolic Equivalent of Task Units (METs) achieved through moderate and strenuous exercise in an average week. The English version has demonstrated acceptable reliability ($\alpha = 0.74$) and moderate associations with relevant objective indicators [e.g., maximum oxygen consumption (VO_2max) and body fat (55)]. The LTEQ was translated into Spanish for the current study (forward and back translation with reconciliation by committee).

Socioeconomic status—Indicators of resources and status (household income, educational attainment), wealth (home ownership), and deprivation (public assistance) were assessed. At the individual level, participants reported their highest educational attainment ($<9^{\text{th}}$ grade, 9^{th} – 11^{th} grade, high school diploma or equivalent, some college, bachelors degree, or graduate/professional degree), total gross household income ($<\$15,000$, $\$15,000$ – $24,999$, $\$25,000$ – $34,999$, $\$35,000$ – $49,999$, $\$50,000$ – $74,999$, or $\$75,000+$), and whether they received public assistance and owned their homes. Missing income values were imputed for five participants using the expectation-maximization algorithm (56) based on age and education. Year 2000 census-tract educational attainment and household income and the percentage of individuals who reported receiving public assistance and owning their homes were extracted at <http://factfinder.census.gov>. Single scores for education and income were calculated using the same categories as for individual SES, based on a previously defined algorithm (57). Individual and neighborhood SES composites were created by standardizing

and summing the four SES items at each level. Exploratory factor analyses suggested a single factor underlying the individual (eigenvalue = 1.66, 41.43% variance explained; factor loadings > 0.47) and neighborhood SES indicators (eigenvalue = 3.33; 83.33% variance explained; factor loadings > 0.88). Bivariate associations among individual ($r_s = 0.25$ to 0.52) and neighborhood SES ($r_s = 0.71$ to 0.91) indicators and between the individual and neighborhood SES composites ($r = 0.52$) were all statistically significant ($p_s < .05$). Moreover, patterns of associations of individual and neighborhood SES indicators with a given inflammatory marker were similar (as described below), supporting the composite approach.[†]

Covariates—All analyses controlled for demographic factors that could confound associations of individual or neighborhood SES with inflammation, specifically, age (in years), and language preference (Spanish versus English) and nativity (birthplace reported as Mexico or the US) as proxy markers for acculturation (59, 60). Duration at current residence (in years) was included in analyses examining neighborhood SES effects. Finally, since lower individual (61) and neighborhood SES (62) relate to higher smoking prevalence, and smoking to inflammation (63), current smoking status (regular smoker or not) was controlled in all models.

Statistical analyses

The distributions of inflammatory markers and physical activity were markedly positively skewed. For inflammatory outcomes, extreme values (> 3 SDs above the mean, $n = 2$ for CRP and sICAM-1, $n = 3$ for IL-6) were excluded, and distributions were natural log transformed. Physical activity was categorized into quintile groups to accommodate frequent “0” scores. All predictors were standardized for analysis.

Multilevel analyses using Hierarchical Linear Modeling (HLM) 6.06 (Scientific Software International, Lincolnwood, IL) tested associations of individual and neighborhood SES with each inflammatory marker. The unique effects of individual and neighborhood SES (both grand-mean centered) were first examined in separate models. Subsequently, both predictors were entered in joint models to investigate the added effects of neighborhood SES after accounting for individual SES, using approaches outlined by Ender and Tofighi (64). (i.e., Individual SES was group-mean centered at level-1; neighborhood SES and mean individual SES were both grand-mean centered at level-2).

Covariates and biobehavioral pathways were entered in steps, with each model building on the previous. Model 1 controlled for covariates [age, language, nativity, smoking, and, for models addressing neighborhood SES, duration of residence at current address]; Model 2 added BMI and residualized WC; Model 3 added dietary fat, fruit and vegetable servings, and physical activity. All models were performed for individual SES, neighborhood SES, and for both SES composites jointly. Where obesity or related behavioral factors were statistically significant predictors at the final model step, tests for indirect effects were performed using procedures established for multilevel models (65, 66). Each potential mediator was tested individually, controlling for all covariates; tests for diet and activity also controlled for BMI and residualized WC.[‡]

[†]In response to a suggestion from an anonymous reviewer, American Community Survey (ACS) 2005–2009 average data were also considered for analyses. Census tract SES composites created from the 2000 Census and from the 2005–2009 ACS averages were found to be highly correlated at $r(63) = .91$. Consistently, the overall pattern of findings was not substantively altered in analyses using the 2005–2009 ACS data; however, minor differences were observed such that some previously statistically significant findings were attenuated to marginal or non-significance using the newer data. This suggests that the earlier census measures are a more robust predictor of inflammatory levels and associated mechanisms for the current study. This may in part reflect the residential stability of the sample (more than 75% of participants had resided at their current addresses for at least 5 years at the time of enrollment) as well as the “incubation period” that exists before the full effects of risk factor exposure manifest (58).

Results

Participant characteristics

Table 1 shows the sociodemographic characteristics of the sample, and descriptive statistics and correlations for all study variables.

Primary analyses

Unconditional multilevel models showed that 4%, <1%, and 16% ($p < .05$) of the variance in CRP, IL-6, and sICAM-1, respectively, was attributable to the census tract level. Although this was statistically significant only for sICAM-1, multilevel models were used for all analyses for consistency and to maximize the accuracy of regression estimates. Results described below are summarized in Table 2. Coefficients reflect the estimated percent difference in each inflammatory marker, given a one-SD increase in the SES composite.

CRP—In demographically-adjusted models, a one-SD higher individual SES was associated with a 27.35% lower CRP level ($p < .01$). The effect was reduced by approximately half with control for adiposity. Little further reduction in the association occurred when behavioral pathways were entered, and individual SES still approached significance ($p = .07$). BMI (+44.54%, $p < .01$), residualized WC (+25.66%, $p < .01$), and dietary fat (+19.17%, $p < .01$) were significant predictors of CRP in the final model. Indirect effects of individual SES on CRP via BMI (−6.60%, $p = .05$) and residualized WC (−2.99%, $p = .05$) approached statistical significance. At the census tract level, CRP was 23.56% lower among individuals living in more advantaged neighborhoods ($p < .01$). The neighborhood SES effect was reduced by about half after control for adiposity and was non-significant when behavioral pathways were controlled. Indirect effects of neighborhood SES via BMI (−5.60%, $p = .06$), residualized WC (−3.68%, $p = .04$), and dietary fat (−2.88%, $p = .04$) reached or approached statistical significance. After accounting for individual SES, a one-SD higher neighborhood SES was associated with a 18.05% lower CRP ($p = .07$); entry of BMI and residualized WC attenuated this effect to non-significance. Predictors in the final model were nearly identical to those for individual SES. Controlling for individual SES, only the indirect effect of neighborhood SES on CRP via BMI (−10.12%, $p = .03$) and dietary fat (after accounting for adiposity; −3.42%, $p = .09$) reached or approached statistical significance.

IL-6—A one-SD higher individual SES predicted a 10.46% lower IL-6 level ($p < .05$); the effect was non-significant when indicators of adiposity were controlled. In the final model, BMI (+26.12%, $p < .01$), residualized WC (+13.92%, $p < .01$), dietary fat (−7.95, $p = .09$), and fruit and vegetable consumption (−11.20%, $p < .05$) reached or approached statistical significance. However, only the indirect pathway from individual SES to IL-6 via BMI was statistically significant (−3.10%, $p < .01$). Neighborhood SES did not predict IL-6 levels in any model.

sICAM-1—A one-SD higher individual SES predicted a 7.04% lower sICAM-1 level ($p < .05$). This effect remained statistically significant after indicators of adiposity were included, and approached significance in the final model ($p = .05$). Significant predictors in this model included BMI (+4.00%, $p < .05$) and dietary fat (+4.52%, $p < .05$); however, neither indirect

[‡]Studies in Latinos (38, 46), including prior analyses in the current sample (67), suggest that SES-gradients in physiological risk profiles could vary by nativity or acculturation. However, exploratory analyses (not reported) revealed that individual SES by language (a proxy for acculturation; 68) and SES by nativity interaction effects were non-statistically significant across all inflammatory outcomes. We did not explore interactions of neighborhood SES by individual acculturation or nativity levels, because statistical power was inadequate for testing cross-level interaction effects.

effect was statistically significant. A one-SD increase in neighborhood SES was associated with a 5.32% lower sICAM-1 level ($p < .05$). The effect was marginally significant ($p = .06$) when BMI and residualized WC were entered and non-significant in the final model. Biobehavioral predictors were identical to those for individual SES (Table 2). The indirect effect via dietary fat (-0.96% , $p = .045$) reached statistical significance. Neighborhood SES did not relate to sICAM-1 beyond individual SES in any model.

Discussion

Inflammatory processes are an important component of atherosclerotic CVD (9, 69) and a plausible biological pathway connecting SES with CVD (70). The current study examined cross-sectional associations of individual and neighborhood SES with inflammation in Mexican-American women of diverse SES levels and explored the contributions of obesity and related health behaviors to these associations. Individual SES was inversely associated with all inflammatory markers, although the relationship with IL-6 was least robust. To our knowledge, the present study is the first to demonstrate an inverse association between SES and sICAM-1 in Latinos, adding to the few studies that have identified an association in other sociodemographic groups (71–73). Effect sizes for individual SES-inflammation associations were small-to-moderate in magnitude. The strongest SES-gradient was observed for CRP, such that a one-SD increase in individual SES predicted a 1.32 mg/L lower CRP level. Comparatively, a large national study of initially healthy women showed that, relative to women at low risk according to CRP level (<1 mg/L), women with medium (>1 but <3 mg/L) and high (>3 mg/L) CRP levels had relative risks of 1.7 and 3.1, respectively, for a first cardiovascular event across nine years (74). Thus, given the effect magnitude, the CRP-related findings in the current study appear to be clinically significant.

In additional analyses, we examined effects of neighborhood SES in relation to inflammatory markers, with and without accounting for individual SES indicators. Neighborhood SES was associated with lower CRP and sICAM-1 only. However, control for individual SES attenuated all neighborhood effects to non-significance. The handful of prior studies addressing neighborhood SES-inflammation associations have yielded mixed results that have varied across ethnic groups (18, 20, 21). Notably, the magnitude of the neighborhood SES-CRP relationship in the current study (18%, $p < .10$, controlling for individual SES) was similar to those observed previously. By way of comparison, Pollitt and colleagues (18) found that White individuals residing in high SES census tracts had 24% lower CRP levels than those in low SES neighborhoods, after controlling for occupational class and other demographics. In MESA, a one-SD increase in neighborhood SES predicted a 5.5% lower CRP after controlling for individual SES, age, and sex (21). Due to the relatively small sample size, tests of neighborhood effects in the current study should be viewed tentatively, and effect sizes should be given more weight.

Pathways connecting individual and neighborhood SES with health are certain to overlap, although neighborhood SES is posited to have unique, contextual influences on health enhancing and degrading resources (6, 7). Moreover, individuals with low SES often reside in less advantaged neighborhoods, which constrain opportunities for socioeconomic advancement (75). In the current study, the individual and neighborhood SES composites were related, but not redundant. Importantly, the analyses used several SES indicators that could be represented in a conceptually equivalent manner at both levels. In prior studies where socioeconomic measures were notably more inclusive at the area level (18, 21), residual confounding may have contributed to apparent neighborhood SES effects (76, 77). The ethnic composition of the current sample may also be relevant to the observed findings. Some research in Latinos suggests that neighborhoods characterized as “ethnic enclaves” may confer health advantages that partially offset the deleterious effects of low

neighborhood SES, which would weaken neighborhood SES-health gradients (78, 79). Additional research with larger samples of Latinos, using comprehensive measures of both individual and neighborhood SES, are needed to better understand how neighborhood SES shapes inflammatory pathways in this population.

The relatively weak associations of individual and neighborhood SES with IL-6 deserve additional comment. Consistent with this pattern of findings, a recent review identified a fairly consistent association of SES with CRP levels (16), whereas SES and IL-6 associations were more mixed (70). A possible explanation for the variability in these findings may be the sensitivity of IL-6 to recent physical or emotional perturbations. For example, a meta-analysis of 30 studies examining inflammatory responses to acute psychological stress identified consistent transient increases in circulating IL-6 in healthy samples exposed to laboratory stressors; CRP responses were less consistent and only marginally significant (80). In addition, IL-6 is characterized by a diurnal rhythm, whereas CRP is without diurnal rhythm and has a longer half-life than IL-6 (81). Moreover, although all participants were evaluated in the morning in a fasting state, the timing of the blood draw relative to their sleep and wake schedules was not controlled.

Consistent with prior research (27, 28), obesity played a prominent role in SES-inflammation associations in the current study. Body mass index contributed to the associations of individual SES with CRP and IL-6; the indirect pathway from individual SES to CRP via abdominal obesity also approached significance. At the area level, BMI and dietary fat (marginally significant indirect path) contributed to the unique association of neighborhood SES with CRP (controlling for individual SES). Overall, these findings provide additional support that obesity and related health behaviors form modifiable targets for prevention efforts that seek to reduce CVD and other health risks associated with low SES individual/familial or residential environments.

Several limitations of the current research warrant discussion. First, the cross-sectional design precludes causal conclusions. Prospective research examining SES gradients in inflammation is sparse (21) and additional evidence, particularly in studies with objective health outcomes, is needed. Since the association between obesity and inflammation is likely reciprocal (82), untangling directionality of associations will require prospective research. In addition, consideration of other potential mediating pathways (e.g., other health behaviors, psychosocial factors) was beyond the scope of the current study and represents an important direction for future research. We examined composite measures of SES as a parsimonious approach to simultaneously modeling multiple aspects of the construct while reducing type 1 error risk. However, this approach also obscures information about the relative predictive utility of the indicators and the unique pathways that might explain their associations with inflammatory markers. The findings should also be interpreted with the number of statistical comparisons in mind, since no family-wise type 1 error correction was imposed. In addition, although examining a homogeneous demographic sample can sharpen the lens through which socioeconomic influences are examined, this approach limits generalizability of the findings, and the current results may not apply to other Latino subgroups.

It is also important to emphasize that this study was limited in its ability to examine effects of neighborhood SES. The sample size was modest and women were recruited from a circumscribed region, though with known socioeconomic diversity. The study also lacked the statistical power necessary to identify cross-level interaction effects. We relied on census tract indicators as a convenient approach to characterizing residential environments. However, these and other administratively defined boundaries (e.g., zip code areas, school districts) may not optimally represent residents' definitions of their "neighborhoods" (83). Household-centered approaches may be more sensitive in capturing neighborhood

contextual influences on health (83, 84). Finally, like most prior studies, the current research conceptualized neighborhood SES as a proxy for social and physical environmental characteristics posited to underlie area SES effects on health, rather than studying these processes directly (85).

In conclusion, the current study provides evidence for an individual SES gradient in CRP, IL-6, and sICAM-1, and is suggestive of a neighborhood SES gradient in CRP, in middle-aged Mexican-American women. Obesity (and to a lesser extent, dietary fat) contributed to associations of SES with inflammatory indicators examined. Additional research is needed to determine whether inflammatory elevations associated with low SES are predictors of future atherosclerotic CVD or other health risks in Latinas.

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Acronyms

BMI	body mass index
CRP	C-reactive protein
CVD	cardiovascular disease
HLM	hierarchical linear modeling
IL-6	interleukin-6
MESA	Multi-Ethnic Study of Atherosclerosis
METs	metabolic equivalent of task units
SD	standard deviation
SES	socioeconomic status
sICAM-1	soluble intercellular adhesion molecule-1
UCSD	University of California = San Diego
WC	waist circumference.

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Table 1

Descriptive Statistics and Correlations Among Study Variables.

	Mean (SD) ^a	CRP (r) ^b	IL-6 (r) ^b	sICAM-1 (r) ^b	Individual SES (r)	Neighborhood SES (r)
Sociodemographic Characteristics						
Age (years)	49.74 (6.50)	0.06	0.07	0.19**	-0.01	0.13
Born in Mexico, N(%)	214 (75.35)	-0.07	0.05	-0.04	0.29**	0.17**
Completed Survey in Spanish, N(%)	169 (59.50)	0.08	0.02	0.11#	-0.47**	-0.38**
Regular Smoker, N(%)	25 (8.80)	-0.01	-0.09	0.14*	-0.10#	-0.07
Educational Attainment, N(%)		-0.21**	-0.08	-0.18**	0.67**	0.48**
< 9 th grade	50 (17.61)					
Some High School	53 (18.66)					
High School Diploma or equivalent	34 (11.97)					
Some College	88 (30.99)					
Bachelors Degree	44 (15.49)					
Graduate/Professional Degree	15 (5.28)					
Annual Household Income, N (%)		-0.18**	-0.08	-0.24**	0.81**	0.71**
<\$15,000	30 (10.56)					
\$15,000–24,999	41 (14.44)					
\$25,000–34,999	51 (17.96)					
\$35,000–49,000	56 (19.72)					
\$50,000–74,999	52 (18.31)					
\$75,000+	54 (19.01)					
Own Home, N(%)	207 (72.89)	-0.09	-0.04	-0.14*	0.76**	0.49**
Receive Public Assistance, N(%)	31 (10.92)	-0.14*	-0.07	-0.10	0.72**	0.49**
Inflammatory Markers						
CRP (mg/L)	3.76 (4.87)	-	-	-	-0.21**	-0.35**
IL-6 (pg/mL)	0.67 (0.61)	0.25**	-	-	-0.09	0.09
sICAM-1 (ng/mL)	274.93 (89.57)	0.42**	0.01	-	-0.22**	-0.15
Obesity Pathways						

	Mean (SD) ^a	CRP (r) ^b	IL-6 (r) ^b	sICAM-1 (r) ^b	Individual SES (r)	Neighborhood SES (r)
BMI (weight in kg/height in m ²) ^b	28.96 (6.45)	0.44**	0.27**	0.21**	-0.11 [#]	-0.31*
Residualized WC (measured in cm)	-0.01 (0.99)	0.23**	0.14*	0.06	-0.16**	-0.23 [#]
Behavioral Pathways						
Dietary Fat (% of calories consumed)	33.41 (5.36)	0.21**	-0.03	0.16**	-0.05	-0.38**
Fruits and vegetables (servings/week)	13.30 (5.51)	-0.05	-0.10 [#]	-0.02	0.07	0.02
Physical Activity (METs/week) ^b	2.90 (1.46)	-0.08	-0.01	-0.08	0.13*	0.17

BMI = body mass index; CRP = C-reactive protein; IL-6 = interleukin-6; SD = standard deviation; SES = socioeconomic status; sICAM-1 = soluble intercellular adhesion molecule-1; WC = waist circumference.

[#] $p < 0.10$;

* $p < 0.05$;

** $p < 0.01$.

^a Means and standard deviations (SD) are presented unless otherwise specified.

^b Analyses performed with transformed data.

N = 284. Sample sizes are reduced due to removal of outliers(CRP and sICAM-1, n = 2; IL-6, n = 1) and missing data(individual SES n = 1;residualized WC, n = 2; physical activity, n = 5)

Table 2

Results of Multilevel Modeling Analyses Evaluating the Percent Difference in Inflammatory Markers Associated With a One Standard Deviation Increase in Individual SES, Neighborhood SES, and Neighborhood SES After Accounting for Individual SES.

	CRP		IL-6		sICAM-1	
	% Difference	95% CI	% Difference	95% CI	% Difference	95% CI
Effect of Individual SES (n = 281)						
In model 1 (covariates ^d)	-27.35**	[-44.63; -10.07]	-10.46*	[-19.59; -1.33]	-7.04*	[-12.75; -1.33]
In model 2 (model 1 + obesity ^b)	-14.03*	[-27.75; -0.31]	-5.84	[-14.87; 3.19]	-5.72*	[-11.29; -0.15]
In model 3 (model 2 + behavioral ^c)	-12.72#	[-25.96; 0.52]	-4.30	[-13.54; 4.94]	-5.12#	[-10.39; 0.15]
BMI	44.54**	[33.28; 55.80]	26.12**	[16.51; 35.73]	4.00*	[0.13; 7.87]
Residualized WC	25.66**	[13.66; 37.66]	13.92**	[4.09; 23.75]	0.02	[-2.77; 2.81]
Dietary Fat	19.17**	[6.79; 31.54]	-7.95#	[-17.34; 1.44]	4.52*	[0.53; 8.51]
Fruits and Vegetables	-6.92	[-17.55; 3.71]	-11.20*	[-21.61; -0.79]	-1.74	[-6.63; 3.15]
Physical Activity	4.06	[-7.23; 15.35]	5.33	[-6.94; 17.60]	0.34	[-3.64; 4.32]
Effect of Neighborhood SES (n = 282)						
In model 1 (covariates ^d)	-23.56**	[-36.47; -10.65]	-2.40	[-11.47; 6.67]	-5.32*	[-9.14; -1.50]
In model 2 (model 1 + obesity ^b)	-12.98*	[-25.85; -0.11]	3.11	[-5.42; 11.64]	-4.03#	[-9.58; 1.52]
In model 3 (model 2 + behavioral ^c)	-9.00	[-21.25; 3.25]	2.42	[-4.88; 9.72]	-3.18	[-7.50; 1.14]
BMI	44.75**	[33.45; 56.05]	26.74**	[17.23; 36.25]	4.65*	[0.48; 8.83]
Residualized WC	25.61**	[12.81; 38.41]	14.85**	[5.03; 24.67]	0.41	[-2.65; 3.47]
Dietary Fat	19.21**	[7.34; 31.08]	-7.02	[-16.66; 2.62]	4.99*	[0.94; 9.04]
Fruits and Vegetables	-7.89	[-18.44; 2.66]	-11.97*	[-22.57; -1.37]	-1.90	[-7.10; 3.30]
Physical Activity	4.98	[-6.38; 16.34]	5.65	[-6.64; 17.94]	0.62	[-3.52; 4.76]
Effect of Neighborhood SES Accounting for Individual SES (n = 281)						
In model 1 (covariates ^d)	-18.05#	[-37.40; 1.30]	0.40	[-11.72; 12.52]	-2.08	[-7.89; 3.73]
In model 2 (model 1 + obesity ^b)	-6.91	[-27.92; 14.10]	5.88	[-6.16; 17.92]	-0.82	[-6.86; 5.22]
In model 3 (model 2 + behavioral ^c)	-2.04	[-21.82; 17.74]	5.83	[-4.95; 16.61]	0.48	[-5.64; 6.60]

	CRP			IL-6			sICAM-1		
	% Difference	95% CI		% Difference	95% CI		% Difference	95% CI	
BMI	43.61**	[32.12; 55.10]		26.01**	[16.60; 35.42]		4.47*	[0.25; 8.69]	
Residualized WC	25.15**	[12.71; 37.59]		14.63**	[4.91; 24.35]		0.21	[-2.74; 3.16]	
Dietary Fat	19.15**	[6.95; 31.31]		-6.96	[-16.73; 3.01]		5.07*	[1.06; 9.08]	
Fruits and Vegetables	-6.54	[-17.17; 4.09]		-11.04*	[-21.25; -0.83]		-1.42	[-6.44; 3.60]	
Physical Activity	4.33	[-7.18; 15.84]		5.18	[-7.32; 17.68]		0.60	[-3.62; 4.82]	

BMI = body mass index; CRP = C-reactive protein; IL-6 = interleukin-6; SES = socioeconomic status; sICAM-1 = soluble intercellular adhesion molecule-1; WC = waist circumference. Values reflect the effects of SES in each model (non-italicized), and the effects of pathway variables in model 3 (italicized).

$p < 0.10$;
* $p < 0.05$;
** $p < 0.01$.

^a Age, language, nativity, smoker (yes/no), duration of current residence (neighborhood SES models only)

^b BMI(weight in kg/height in m²) and residualized WC (measured in cm; missing data, n = 2).

^c Dietary fat (% of total calories), fruit and vegetable servings/week, and physical activity (METs/week, in quintiles; missing data, n = 5). Sample sizes are reduced due to outliers (CRP, sICAM-1: n = 2; IL-6: n = 1) and missing data (individual SES n = 1).