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Neighborhood social cohesion is associated with lower levels of interleukin-6 in African American women

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Abstract

Introduction: Social cohesion is a positive neighborhood characteristic defined by feelings of connectedness and solidarity within a community. Studies have found significant associations between social cohesion and cardiovascular disease (CVD) risk factors and outcomes. Inflammation is one potential physiological pathway linking social cohesion to CVD development, but few studies have evaluated the relationship between social cohesion and inflammatory biomarkers. Prior research has also established that race and gender can modify the effects of neighborhood features, including social cohesion, on CVD risk factors and outcomes. This study aimed to examine the association between social cohesion and the inflammatory biomarkers interleukin-6 (IL-6) and C-reactive protein (CRP) in a cohort of African-American and White women and men.

Materials and Methods: Data from the Morehouse and Emory Team Up to Eliminate Health Disparities (META-Health) Study were used to assess the association between social cohesion and inflammation among African American (n=203) and White (n=176) adults from the Atlanta metropolitan area. Social cohesion was measured using the social cohesion subscale from the Neighborhood Health Questionnaire. Inflammatory biomarkers were measured from plasma frozen at −70°C. Multivariable linear regression analyses were conducted, controlling for demographic, clinical, behavioral, and psychosocial factors sequentially. Interaction by race and gender was also examined.

Results: In models adjusted for age, race, gender, and education, social cohesion was significantly associated with lower levels of IL-6 (β=−0.06, p=0.03). There was a significant race-social cohesion interaction (p=0.04), and a marginally significant gender-race-social cohesion

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interaction (p=0.09). In race-stratified models controlling for age, gender, and education, social cohesion was associated with lower IL-6 levels in African Americans (β=−0.11, p=0.01), but not Whites (β=0.01, p=0.91). In fully adjusted race-and gender-stratified models, social cohesion was associated with lower levels of IL-6 in African American women only (β=−0.15, p=0.003). CRP was not associated with social cohesion in fully adjusted models.

Conclusion: The association between social cohesion and lower levels of IL-6 is modified by gender and race, with the strongest association emerging for African American women. Although the pathways through which social cohesion impacts inflammation remain unclear, it is possible that for African American women social cohesion manifests through neighborhood networks.

Keywords

Neighborhood; Social Cohesion; Inflammation

Introduction

A strong and consistent body of research has established the importance of neighborhood context for cardiovascular health (1). Studies have documented linkages between cardiovascular health and a range of neighborhood factors, including neighborhood disadvantage (2, 3), violent crime (4), unemployment (4), and social disorganization (5). However, most of these studies have focused on the detrimental impact of negative neighborhood characteristics on cardiovascular health, with limited attention to the effects of positive, or protective, aspects of “place” on health.

One positive neighborhood factor that may influence cardiovascular health is social cohesion. Social cohesion is defined as feelings of connectedness and solidarity experienced by neighbors, members of a community, or other societal groups (6). A socially cohesive neighborhood is characterized by the presence of strong social bonds that are believed to develop when neighbors share trust and norms of reciprocity with one another (6). Across studies, social cohesion has been associated with reduced cardiovascular disease (CVD) events (7, 8) and a more favorable CVD risk factor profile (9, 10), including higher levels of physical activity (10, 11) and lower levels of smoking (9, 11).

Less is known, however, about the physiological pathways linking social cohesion to CVD risk (12, 13, 14, 15, 16), and inflammation is one plausible mechanism that has been underexplored. Animal models have documented linkages between disruption of the social environment and increased inflammatory responses, providing some support for the notion that non-disruptive, or cohesive, social environments might protect against inflammation (17, 18, 19). In particular, interleukin-6 (IL-6) and C-reactive protein (CRP) are two inflammatory biomarkers with strong and consistent relationships with incident CVD (20, 21).

To our knowledge, only two prior studies have examined the association between neighborhood social cohesion and inflammation. In a study of 151 young, healthy Brazilian adults who migrated to metropolitan Boston, greater social cohesion was associated with lower levels of CRP (22), while in a population-based sample there was no significant

association between social cohesion and a range of inflammatory markers (16). However, neither of these studies considered potential effect modification by demographic characteristics, thus, we sought to examine the association between social cohesion and inflammatory biomarkers in an urban cohort of African American and White women and men with particular attention to interaction by gender and race. A substantial body of research has demonstrated that neighborhood effects may vary by gender (23). In a study of thirteen neighborhood features, characteristics of the socio-political environment, amenities and the physical environment, and economic factors were more consistently associated with self-reported health among women than among men (24). Studies have also found that neighborhood characteristics such as excessive noise, violence, and objectively reported crime may affect CVD risk factors in women, but not in men (13, 25). Furthermore, prior research suggests that the neighborhood social environment in particular may be more important for women's health than for men's health (26, 27, 28). Some hypothesize that the social environment is more critical for women because they spend more time in the neighborhood setting while caring for children and doing domestic work (26), while others suggest that gender differences in the types and uses of social relationships may be responsible (29). Previous studies have consistently shown gender differences in the impact of social cohesion on CVD risk as well, with higher social cohesion reducing the odds of a range of CVD indices, including hypertension, coronary artery calcification, obesity, and depression among women, but not among men (15, 30, 31, 32).

Missing from this literature is an "intersectional" perspective that recognizes social identities are not independent and considers how the intersection of gender and race may modify the association between social cohesion and CVD risk differently than either gender or race alone (33). In the United States (US), African Americans and Whites live in disparate neighborhood contexts (34), and research indicates that race and ethnicity may also modify neighborhood effects (23). Studies have found that associations between neighborhood characteristics and CVD risk factors vary by race, with one study finding a stronger association for African Americans when considering the role of neighborhood crime (25) and another identifying a greater effect for Whites when assessing the impact of neighborhood racial composition (35). For social cohesion, specifically, findings have been less consistent for race, with some studies finding racial differences in the association between social cohesion and CVD risk, while other studies have not (11, 15, 36).

The current study aimed to expand upon previous work by adopting an intersectional approach to evaluate the joint effect of gender and race on the linkage between social cohesion and inflammatory biomarkers relevant for CVD risk. We used data from the Morehouse and Emory Team Up to Eliminate Health Disparities (META-Health) Study, which sampled African American and White individuals from four distinct counties with area incomes above and below the median level at an equal rate—providing some design control for neighborhood SES. Using META-Heath data, we examined cross-sectional associations between neighborhood social cohesion and two inflammatory biomarkers, IL-6 and CRP, controlling for demographic, clinical, behavioral, and psychosocial characteristics. We hypothesized that higher social cohesion would be associated with lower levels of CRP and IL-6, and that gender and/or race might modify these associations. Using an intersectional framework, we also considered the possibility that there might be race

differences within gender and/or gender differences within race; we therefore also examined associations for each gender-race group. Finally, to determine whether any observed linkages between social cohesion and inflammation were independent of detrimental neighborhood factors, we also controlled for neighborhood safety and violence in exploratory analyses.

Materials and Methods

Study Sample

Between 2005 and 2010, the META-Health Study recruited residents from the metropolitan Atlanta area for a two-stage cross-sectional study of traditional and psychosocial CVD risk factors. The first stage sampled African American and White adults 30 to 65 years old (n=3,391) through a random digit dialing survey. Participants were sampled from four distinct counties stratified by county median income within race to ensure the inclusion of an adequate representation of individuals from varying levels of socioeconomic backgrounds. A subset of these participants (n=465) was then invited to an in-person study visit at the Emory or Morehouse School of Medicine. For analyses assessing IL-6 as the outcome, a total of 86 participants were excluded due to missing data on social cohesion and/or IL-6, while 83 study subjects were excluded when CRP was the outcome under consideration. Those missing social cohesion, IL-6, or CRP did not differ by age, race, gender, education, or depressive symptoms when compared to participants included in the analytic sample.

During the in-person clinic visit, clinical and anthropometric data were collected and questionnaires were administered to assess demographics and psychosocial characteristics. Pregnant women and individuals who reported recent acute illness, including cold-like symptoms or pain, were excluded from the META-Health study. The Emory University and Morehouse University Institutional Review Committees approved this study, and all participants provided written informed consent.

Measurement of Interleukin-6 and C-Reactive Protein

Subjects were instructed to fast for 12 hours prior to the study visit, during which venous blood was collected in sodium heparin tubes. Inflammatory biomarkers were measured from plasma frozen at −70°C. IL-6 was quantified by ultrasensitive ELISA (R&D Systems, Minneapolis, Minnesota) and high sensitivity CRP by immunonephelometry (Siemens/Dade Behring). For IL-6, the inter-and intra-assay variation coefficients were 3.7% and 3.4%, respectively, while for CRP they were 5.0% and 4.3%. In the analytic sample, IL-6 and CRP were correlated at 0.39.

Measurement of Social Cohesion

Social cohesion was assessed via four items drawn from the Neighborhood Health Questionnaire (37) based on prior work on neighborhood contexts (11, 12, 14, 16, 30, 31, 36). The social cohesion items were originally developed by Sampson and colleagues as part of the Chicago Project of Human Development (38). The scale has been previously validated within multiethnic cohorts and demonstrates good internal consistency and test-retest reliability (11, 37). Items inquire whether neighbors are willing to help one another, get

along, trust each other, and share the same values (37). Responses are scored using a five point Likert scale (“Strongly Agree” to “Strongly Disagree”, with an additional response option of “Don’t Know/Not Sure”). An overall score for social cohesion was created for each participant by averaging his or her scores on the individual items. Individual items marked “Don’t Know/Not Sure” were removed from the overall calculation and scores were assigned a missing value if three or more items were answered “Don’t Know/Not Sure”. For both the individual items and overall score, higher scores indicated greater perceived social cohesion.

Measurement of Covariates

Demographic (age, race, gender, education), clinical (body mass index (BMI), history of hypertension, history of diabetes or impaired fasting glucose, low-density lipoprotein cholesterol (LDL-C), total cholesterol, fasting glucose, antihypertensive use, statin use), behavioral (history of smoking, leisure time physical activity, sleep quality), and psychosocial (depressive symptoms) characteristics were selected as baseline covariates based on previous literature and the potential for these factors to affect inflammatory biomarker measurement (16, 39, 40).

Demographics—Race was self-reported as “Black or African-American” or “White or Caucasian”. Gender was also self-reported as “Male” or “Female”. Education was considered the highest grade attained and was categorized as high school or less (encompassing elementary school, some high school, and high school/General Equivalency Diploma), some college, and college and more. We selected education as our primary measure of socioeconomic status (SES) at the individual level. Education is a key determinant of SES and frequently has less missing data than measures like income or wealth (41). Additionally, because we sampled from distinct counties stratified by median income within race, we wanted to avoid overcontrolling for income by adding it as an additional indicator of individual SES.

Clinical—At the study visit, participants’ height and weight were measured and BMI (kg/m^2) was calculated. Serum levels of total cholesterol, LDL-C, and fasting glucose were measured by spectrophotometry. Statin and antihypertensive use was distinguished as users versus non-users. History of hypertension, diabetes, and impaired fasting glucose were determined via participant self-report or use of antihypertensive or antidiabetic medications.

Behavioral—A standardized 11-item questionnaire from the Atherosclerosis Risk in Communities (ARIC) Study was utilized to evaluate self-reported smoking history (42, 43), which was dichotomized for analyses as current smoker or former/never smoker. The self-administered Baecke physical activity questionnaire was used to obtain summary scores for sport and non-sport physical activity during leisure time (44). Following the methodology of prior studies, the eight items assessing sport activity were added to the items measuring non-sport activity to create a 16-item summary Baecke leisure time activity index, with higher scores signifying greater physical activity during leisure time (45). The Baecke questionnaire has been validated (46, 47) and previously employed to measure physical activity in both African Americans and Whites (48). Study subjects also completed the19-

item Pittsburgh Sleep Quality Index (PSQI), which has been validated in biracial samples and inquires about overall sleep quality and sleep-related symptoms from the previous month (49, 50). Throughout analyses, the total PSQI score was treated as a continuous variable, though scores above five indicate poor sleep quality.

Psychosocial—The 21-item Beck Depression Inventory (BDI)-II was self-administered to assess depressive symptoms experienced over the past two weeks. Although the BDI-II has been more frequently validated within White populations, small studies have validated this measure among African Americans as well (51, 52). The total BDI-II score was considered as a continuous variable ranging from 0 to 63. Higher scores were indicative of more depressive symptoms, with scores 0–13 representing minimal to no depression, 14–19 mild depression, 20–28 moderate depression, and 29–63 severe depression.

Statistical Methods

Descriptive statistics on variables of interest were summarized as proportions for categorical variables, means±SD for continuous variables, and median (IQR) for unlogged CRP and IL-6, which were heavily skewed. Because we were ultimately interested in examining associations by both gender and race, racial differences were tested within gender groups. Within gender groups, categorical and continuous variables were compared across race via chi-square tests, unpaired two-sample t-tests, and Wilcoxon non-parametric tests.

Pearson and Spearman correlations analyses were conducted for the overall sample and the four gender-race subgroups to examine bivariate associations between the inflammatory biomarkers, social cohesion, and the demographic, clinical, behavioral, and psychosocial covariates of interest. Multivariable linear regression analyses were then conducted to assess the association between social cohesion and the inflammatory biomarkers, adjusting for demographics and relevant covariates. Due to the skewed distribution of CRP and IL-6, natural log transformed levels were used throughout all analyses. In addition to bivariate analyses, four stepwise models were considered: Model 1 contained demographics: age, race, gender, and education; Model 2 added BMI, history of hypertension, history of diabetes and impaired fasting glucose, LDL-C, total cholesterol, fasting glucose, antihypertensive use, and statin use; Model 3 added history of smoking, leisure time physical activity, and sleep quality; and Model 4 added depressive symptoms. To determine whether associations varied by gender, race, or gender and race simultaneously, social cohesion*gender, social cohesion*race, and social cohesion*gender*race interaction terms were tested within the full sample. Models were stratified when significant interactions were observed. Two-tailed tests using a 0.05 significance level were utilized to determine statistical significance for all main effects and two-way interaction terms. We set a more conservative significance level of 0.10 for tests of the three-way interaction. Rouhani (53) has argued that increasing the alpha level may be necessary when conducting intersectional analyses, as some of the variation resulting from the interaction may already be encompassed in the main effect test. Increasing the alpha level also compensates for the lower power of tests of interaction that results when the measurement error of the individual variables involved in the interaction is compounded (53). Statistical analyses were performed using SAS 9.4 (SAS Institute, Cary NC, USA).

Results

Participant Characteristics

Table 1 summarizes demographic, clinical, behavioral, psychosocial, neighborhood, and inflammatory variables for women and men separately, by race. For both women and men, African Americans were less likely than Whites to have completed college (41.8% compared to 71.6% for women; 36.2% compared to 66.7% for men). African American women reported lower social cohesion, greater antihypertensive use, less leisure time physical activity, and poorer sleep quality compared to White women. African American women also had higher BMI and higher levels of CRP than White women. African American men were younger than White men, though there were no racial differences in CRP or IL-6 levels among men.

Bivariate Correlations

Bivariate analyses revealed a significant negative association between social cohesion and IL-6 in the overall sample ($r=-0.14$, $p=0.006$) (Table 2). There were also significant negative associations between IL-6, education, and leisure time physical activity. Significant positive associations were detected between IL-6 and age, history of hypertension, history of diabetes or impaired fasting glucose, fasting glucose, antihypertensive use, BMI, Framingham Risk Score, ASCVD Risk Score, and CRP.

There was also a significant negative association between CRP and social cohesion ($r=-0.11$, $p=0.04$) (Table 3). Education and leisure time physical activity were negatively associated with CRP as well, while history of hypertension, antihypertensive use, BMI, smoking, Framingham Risk Score, ASCVD Risk Score and IL-6 were positively associated with CRP. Across the gender-race groups, the bivariate associations varied, though there was a significant association between IL-6 and social cohesion among African American women.

Social Cohesion and Interleukin-6

In the multivariable linear regression models adjusted for age, race, gender, and education, social cohesion was significantly associated with a 6% decrease in IL-6 levels ($\beta=-0.06$, $p=0.03$) and there was a significant race by social cohesion interaction ($p=0.04$). The gender by social cohesion interaction was non-significant ($p=0.23$).

Race-stratified models controlling for age, gender, and education revealed social cohesion was significantly associated with a 11% decrease in IL-6 levels among African Americans ($\beta=-0.11$, $p=0.01$), but not Whites ($\beta=0.01$, $p=0.91$) (Table 4). For African Americans, the association between social cohesion and IL-6 remained significant in models that were fully adjusted for clinical, behavioral, and psychosocial variables (Table 4).

Because the gender by race by social cohesion interaction was $p=0.09$, and we were using an intersectional approach, we ran additional analyses stratified by the four gender-race groups. Models adjusted for age and education showed a significant association between social cohesion and lower levels of IL-6 in African American women only, and the association remained significant in fully-adjusted models ($\beta=-0.15$, $p=0.003$) (Table 5). There were no

significant associations observed between social cohesion and IL-6 in White women, White men, or African American men (Table 5).

We also considered whether the protective effect of social cohesion could outweigh negative aspects of the neighborhood social environment by running exploratory analyses to determine whether our observed associations in African American women were independent of self-reported neighborhood safety and violence (54, 55). Neighborhood safety and violence were assessed via two additional subscales of the Neighborhood Health Questionnaire (37) and controlled for in each of the four stepwise models. Social cohesion remained significantly associated with lower levels of IL-6 for African American women in both the minimally and fully adjusted models also controlling for safety and violence ($\beta = -0.15$, $p=0.01$ nad $\beta=-0.16$, $p=0.02$, respectively; data available upon request).

Social Cohesion and C-Reactive Protein

Social cohesion was not associated with CRP in minimally or fully adjusted models, though in models controlling for age, race, gender, and education there was significant gender by social cohesion interaction ($p=0.01$). However, there was no significant race by social cohesion ($p=0.95$) or gender by race by social cohesion interaction ($p=0.56$). Upon stratification by gender, the only significant association detected between social cohesion and CRP was for the unadjusted female model ($\beta=-0.09$, $p=0.02$), though this association was attenuated after including additional covariates.

Discussion

In this bi-racial, community-based sample, we found that social cohesion was associated with lower levels of IL-6 in African American, but not White, middle aged adults. The association among African Americans persisted following adjustment for demographic, clinical, behavioral, and psychosocial variables. Although African Americans reported lower levels of social cohesion than Whites, the significant race by social cohesion interaction that we identified indicates that social cohesion may be a more impactful neighborhood factor for African Americans relative to Whites. Our findings also demonstrate that the association between social cohesion and IL-6 may vary by gender-race group. We detected a marginally significant gender by race by social cohesion interaction and found consistently larger cohesion parameter estimates for African American women relative to other groups, which suggests they may drive the association between social cohesion and IL-6 among African Americans.

Although studies examining linkages between negative social factors and inflammation have often found stronger associations among African-Americans compared to Whites (56, 57), the fact that significant associations between social cohesion and IL-6 were only observed among the African-Americans in our cohort was somewhat surprising. Prior studies of social cohesion and CVD conducted among adults living in Chicago found that social cohesion was protective against stroke and cardiovascular mortality among Whites, but not African Americans (58, 59). Our findings do not directly correspond to these results, though differences between Midwestern and Southern African American neighborhoods may account for this discrepancy. Due to racial residential segregation and economic

restructuring, poverty is particularly concentrated in African American neighborhoods in the Upper Midwestern United States (60). It is therefore possible that the negative health effects of poverty overshadow the protective effect of social cohesion for African Americans living in these environments. It is also important to note that the current cohort was specifically chosen to represent both lower-and middle-income communities, which are fairly common in the US South; thus areas with high levels of concentrated poverty were not included.

Social cohesion may be especially influential for African Americans in Southern states due to its relationship to agency and collective efficacy. Throughout the US, and especially in the South, African Americans have historically engaged in collective action in response to structural and economic challenges (61). It is also plausible that the shared experience of these adversities evokes heightened group identification, demonstrating why social cohesion, and the inherent sentiments of connectedness and solidarity, may be a prominent force in the lives of African Americans (62, 63). Our findings suggest that the ongoing necessity for collective action and elevated group identification may contribute to the greater role of social cohesion in positively impacting health among African Americans, but not Whites.

Among African Americans, associations were stronger for African American women than for African American men. Additionally, in exploratory analyses adjusting for neighborhood safety and violence, the association between social cohesion and lower levels of IL-6 remained significant among African American women. These results align with several prior studies, which found that the protective effect of social cohesion on cardiovascular health might be stronger for women than for men (13, 15, 30, 31, 32, 64). Sociological research on women's relationships in the neighborhood setting offers additional insight into these findings. Studies on neighborhood networks demonstrate that women have larger networks, know more of their neighbors by name, and talk or visit with neighbors more frequently (65). In addition to maintaining larger social networks, women are more emotionally involved in the life events occurring within their networks (66). For African American women specifically, neighborhood relationships may operate through fictive kin networks. In African American communities, fictive kin are individuals who are not related by blood or marriage, such as neighbors, but are assigned kinship status (67). Implicit in kinship status is intensified mutual obligation, with expectations for fictive kin to engage in the duties typically assigned to extended family, such as providing childcare, transportation, financial assistance, or emotional support (67, 68, 69, 70). African American women are more likely to report having fictive kin than African American men (67), thus it is possible that for African American women, neighborhood fictive kin networks embody the effects of both African American group identification and female social network integration. This phenomenon may be unique to African American women's intersectional identity (71, 72), and may underlie the heightened effect of social cohesion on IL-6 observed among African American women relative to the other gender-race groups.

To our knowledge, only two prior studies have examined associations between social cohesion and inflammation. Homes and Marcelli found that social cohesion was associated with lower levels of CRP in a sample of young, healthy Brazilian adults living in Boston, but born outside the US (22). Conversely, Nazmi et al. found no significant association between social cohesion and a range of inflammatory markers (16). However, neither of these studies

focused on gender and race as potential effect modifiers of the association between social cohesion and inflammation. Our findings suggest that gender and race may be critically important factors to consider in this relationship. Yet, the exact pathways through which social cohesion impacts inflammation are unclear. For African American women in this study, the association between social cohesion and IL-6 persisted even following adjustment for age, education, BMI, history of hypertension, history of diabetes or impaired fasting glucose, LDL-C, total cholesterol, fasting glucose, antihypertensive use, statin use, smoking history, leisure time physical activity, sleep quality, and depressive symptoms. It is possible that social cohesion experienced at the neighborhood level protects against excessive hypothalamic-pituitary-adrenal (HPA) axis activity, which can drive cortisol elevations and inflammatory responses (73). For example, in a study of neighborhood characteristics and features of the diurnal cortisol curve, higher social cohesion was associated with increased cortisol upon awakening, steeper early decline, and steeper wake-to-bed slope (14). Furthermore, another study examining associations between neighborhood factors and cortisol profiles also identified a relationship between lower social cohesion and decreased cortisol upon awakening (74).

We did not detect significant associations between social cohesion and CRP. Although Holmes and Marcelli observed a significant relationship between social cohesion and CRP, their participants had lower BMIs and lower levels of CRP, suggesting there could be clinically relevant differences in our samples (22). Additionally, CRP and IL-6 were only weakly correlated in our analytic sample, and other studies indicate that these two inflammatory biomarkers may respond differently to social factors (75, 76, 77), and that CRP and IL-6 may be differentially impacted by certain clinical characteristics, such as obesity (78). Future studies are needed to better understand additional factors that might explain this pattern of results.

Recent research also indicates that IL-6 may be a more robust CVD risk factor than CRP. In a large prospective study, IL-6 was associated with major cardiovascular events such as myocardial infarction, heart failure, and cardiovascular mortality even following multivariable adjustments, while no such association was found for CRP (79). Similarly, Fanola et al. recently found that IL-6 was associated with adverse cardiovascular events independently of CRP (80). These findings highlight the importance of IL-6 as a CVD predictor and suggest further exploration of social cohesion’s protective effect against IL-6 is warranted among African American women.

Limitations

Our study is not without limitations. Primarily, the cross-sectional nature of this study precludes causal inference. Longitudinal research on the relationship between social cohesion and inflammation will be necessary to determine causality and to establish the mechanisms through which social cohesion impacts inflammation. Secondly, although demographic, clinical, behavioral, and psychosocial covariates were controlled for, residual confounding may exist. Although participants were sampled from distinct counties stratified by county median income within race, individual level income was not explicitly controlled for during regression analyses to avoid overcontrolling for this factor. However, education

was included in all models as a measure of individual SES. Furthermore, it is possible that SES may also modify the effect of social cohesion on IL-6. Given our sample size we were hesitant to add a four-way interaction term including gender, race, SES, and social cohesion, although this may be worth considering during future studies. Additionally, an association may not have been observed among men due to our study's smaller male sample. Future studies including a larger number of men should confirm our findings that gender modifies the association between social cohesion and IL-6. Finally, although the META-Health study sample is population-based, it consists of non-Hispanic White and non-Hispanic African American individuals residing in four Georgia counties. The results of this study may therefore not be generalizable to other racial/ethnic groups or populations living in other areas.

Conclusion

In this sample of African American and White adults from the metropolitan Atlanta area, neighborhood social cohesion was associated with IL-6 among African Americans, with the strongest and most robust associations observed in African American women. This study adds to the neighborhood effects literature by considering a positive neighborhood characteristic, as previous research has largely concentrated on negative neighborhood factors, and by adopting an intersectional approach. These findings also expand upon the current literature on neighborhood context and cardiovascular health by demonstrating that inflammation represents an additional CVD risk factor that may be impacted by social neighborhood exposures. Future interventions aiming to reduce CVD among African American women might consider incorporating activities designed to foster neighborhood social cohesion.

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Highlights

- Positive aspects of neighborhoods may be linked to CV health, via inflammation
- Evidence suggests that associations may be patterned by race and gender
- We examined linkages between social cohesion and inflammation by race and gender
- Social cohesion was inversely related to IL-6 for African-Americans, not Whites.
- Associations among African-Americans were largely driven by African-American women

Table 1.

Participant Characteristics by Race and Gender.

	White Women (n=116)	African American Women (n=134)	p	White Men (n=60)	African American Men (n=69)	p
Age (years)	52±10	50±9	0.19	53±9	48±9	0.004
Education			<0.001			0.001
Elementary, high school, or GED	7 (6.0%)	39 (29.1%)		7 (11.7%)	21 (30.4%)	
Some college	25 (21.6%)	38 (28.4%)		11 (18.3%)	22 (31.9%)	
College graduate	83 (71.6%)	56 (41.8%)		40 (66.7%)	25 (36.2%)	
Hypertension	48 (41.4%)	72 (53.7%)	0.05	35 (58.3%)	42 (60.9%)	0.77
Diabetes/Impaired	11 (10.4%)	24 (18.9%)	0.07	14 (24.6%)	13 (20.0%)	0.54
Fasting Glucose LDL-C (mg/dL)	119.4±33.1	117.8±36.3	0.72	112.8±32.6	124.4±40.2	0.08
Total Cholesterol (mg/dL)	204.6±40.0	197.6±38.6	0.17	191.1±48.3	195.5±44.8	0.60
Fasting Glucose (mg/dL)	89.8±17.3	93.7±25.9	0.19	93.2±10.5	92.8±12.5	0.85
Statin Use	10 (8.6%)	18 (13.4%)	0.22	14 (23.3%)	11 (15.9%)	0.31
Antihypertensive Use	19 (16.7%)	39 (29.8%)	0.02	15 (25.0%)	23 (33.8%)	0.28
BMI (kg/m^2)	27.4±6.6	31.5±7.9	<0.001	30.2±6.2	30.7±7.1	0.67
Current Smoker	11 (9.5%)	23 (17.2%)	0.07	9 (15.0%)	15 (21.7%)	0.31
Leisure PA	6.6±1.4	5.9±1.3	<0.001	6.4±1.4	6.2±1.6	0.42
Framingham Risk Score	5.9±5.3	6.3±5.9	0.61	12.0±7.0	11.9±9.0	0.25
ASCVD Risk Score	2.8±2.8	4.5±5.9	0.24	7.1±5.8	8.5±6.5	0.10
PSQI	5.2±2.9	6.6±3.9	0.003	5.1±3.7	6.0±3.7	0.15
BDI-II	7.5±6.0	8.3±8.3	0.43	8.3±10.2	8.0±7.3	0.88
Neighborhood Social Cohesion	3.9±0.7	3.5±0.7	0.002	3.8±0.6	3.6±0.6	0.10
CRP (mg/L)	1.61 [0.81–3.59]	2.80 [1.30–6.78]	<0.001	1.61 [1.01–3.75]	1.97 [0.64–5.77]	0.95
IL-6 (pg/ml)	0.81 [0.55–1.19]	0.89 [0.57–1.68]	0.07	0.81 [0.60–1.18]	0.67 [0.47–1.31]	0.44

Numerical values are means±SD or median [IQR]. T-tests, Wilcoxon non-parametric (Kruskal-Wallis) tests, and X^2 tests were performed to compare Whites and African Americans within gender groups. Abbreviations: GED, General Equivalency Diploma; LDL-C, low density lipoprotein cholesterol; BMI, body mass index; PA, physical activity; ASCVD, Atherosclerotic Cardiovascular Disease; PSQI, Pittsburgh Sleep Quality Index; BDI-II, Beck Depression Inventory II; CRP, C-reactive protein; IL-6, interleukin-6.

Table 2.

Bivariate correlations of IL-6 with social cohesion and demographic, clinical, behavioral, and psychosocial covariates.

	Overall (N=376)	White Women (N=116)	African American Women (N=131)	White Men (N=60)	African American Men (N=69)
Age (years)	0.11 *	0.09	0.03	0.06	0.34 **
Education	−0.17 **	−0.12	−0.20 *	−0.30 *	−0.05
Hypertension	0.25 **	0.27 **	0.25 **	0.12	0.31 **
Diabetes/Impaired Fasting Glucose	0.16 **	−0.005	0.19 *	0.18	0.21
LDL-C (mg/dL)	0.02	0.02	−0.05	0.22	0.03
Total Cholesterol (mg/dL)	−0.01	−0.07	−0.08	0.20	0.03
Fasting Glucose (mg/dL)	0.13 *	0.16	0.12	0.28 *	0.05
Statin Use	0.08	0.06	0.15	−0.15	0.19
Antihypertensive Use	0.17 **	0.24 **	0.18 *	−0.09	0.26 *
BMI (kg/m^2)	0.34 **	0.41 **	0.38 **	0.11	0.26 *
Current Smoker	0.08	0.02	0.08	0.18	0.06
Leisure PA	−0.14 **	−0.06	−0.11	−0.03	−0.30 *
Framingham Risk Score	0.16 **	0.20 *	0.14	0.18	0.37 **
ASCVD Risk Score	0.17 **	0.12	0.15	0.31 *	0.35 **
PSQI	0.05	−0.05	0.03	−0.05	0.19
BDI-II	0.03	−0.07	0.01	0.04	0.16
Neighborhood Social Cohesion	−0.14 **	0.04	−0.31 **	−0.06	−0.02
CRP (mg/L)	0.42 **	0.42 **	0.41 **	0.10	0.55 **

* $p < 0.05$

** $p < 0.01$

Values determined using Pearson correlations for continuous covariates and Spearman correlations for categorical covariates.

Table 3.

Bivariate correlations of CRP with social cohesion and demographic, clinical, behavioral, and psychosocial covariates.

	Overall (N=358)	White Women (N=109)	African American Women (N=126)	White Men (N=57)	African American Men (N=66)
Age (years)	0.08	0.21*	−0.11	0.15	0.26*
Education	−0.12*	−0.09	−0.08	−0.02	−0.06
Hypertension	0.25**	0.29**	0.16	0.25	0.29*
Diabetes/Impaired Fasting Glucose	0.08	0.06	0.04	0.11	0.06
LDL-C (mg/dL)	−0.0005	0.10	−0.08	0.02	0.02
Total Cholesterol (mg/dL)	−0.04	0.02	−0.16	0.08	−0.002
Fasting Glucose (mg/dL)	0.09	0.20*	−0.001	0.04	0.17
Statin Use	0.04	0.10	−0.14	0.04	0.23
Antihypertensive Use	0.22**	0.27**	0.15	0.12	0.28*
BMI (kg/m^2)	0.35**	0.43**	0.27**	0.22	0.37**
Current Smoker	0.11*	0.14	0.08	0.09	0.08
Leisure PA	−0.18**	−0.30**	−0.03	−0.05	−0.19
Framingham Risk Score	0.13*	0.34**	−0.03	0.20	0.26*
ASCVD Risk Score	0.15**	0.36**	−0.01	0.25	0.31*
PSQI	0.04	0.08	−0.08	0.04	0.04
BDI-II	0.06	0.22*	−0.12	0.18	0.09
Neighborhood Social Cohesion	−0.11*	−0.13	−0.17	0.07	0.12
IL-6 (pg/ml)	0.42**	0.42**	0.41**	0.10	0.55**

* $p < 0.05$

** $p < 0.01$

Values determined using Pearson correlations for continuous covariates and Spearman correlations for categorical covariates.

Table 4

Multivariable Linear Regression of Social Cohesion and IL-6 by Race.

	White n = 176			African American n = 203		
	β	SE	p	β	SE	p
Model 1						
Neighborhood Social Cohesion	0.005	0.040	0.910	−0.108	0.039	0.006
Adjusted for age, gender, and education						
Model 2						
Neighborhood Social Cohesion	0.059	0.043	0.169	−0.090	0.039	0.020
Adjusted for Model 1 covariates + BMI, HTN, DM/IFG, LDL-C, total cholesterol, fasting glucose, antihypertensive use, and statin use						
Model 3						
Neighborhood Social Cohesion	0.056	0.046	0.220	−0.084	0.039	0.032
Adjusted for Model 2 covariates + smoking history, leisure time physical activity, and sleep quality (PSQI)						
Model 4						
Neighborhood Social Cohesion	0.052	0.047	0.263	−0.087	0.039	0.026
Adjusted for Model 3 covariates + depressive symptoms (BDI-II)						

Abbreviations: IL-6, interleukin-6; BMI, body mass index; HTN, history of hypertension; DM/IFG, history of diabetes or impaired fasting glucose; LDL-C, low density lipoprotein cholesterol; PSQI, Pittsburg Sleep Quality Index; BDI-II, Beck Depression Inventory II

Table 5

Multivariable Linear Regression of Social Cohesion and IL-6 by Gender and Race.

	White Women n = 116			African American Women n = 134			White Men n = 60			African American Men n = 69			Three-Way Interaction*
	β	SE	p	β	SE	p	β	SE	p	β	SE	p	p
Model 1													
Neighborhood Social Cohesion	0.021	0.050	0.669	−0.148	0.045	0.001	−0.034	0.075	0.648	0.010	0.072	0.891	0.089
Adjusted for age and education													
Model 2													
Neighborhood Social Cohesion	0.087	0.052	0.101	−0.126	0.046	0.008	−0.024	0.089	0.788	0.031	0.078	0.689	0.056
Adjusted for Model 1 covariates + BMI, HTN,DM/IFG, LDL-C, total cholesterol, fasting glucose, antihypertensive use, and statin use													
Model 3													
Neighborhood Social Cohesion	0.083	0.056	0.143	−0.134	0.048	0.007	0.005	0.097	0.958	0.073	0.072	0.313	0.033
Adjusted for Model 2 covariates + smoking history, leisure time physical activity, and sleep quality (PSQI)													
Model 4													
Neighborhood Social Cohesion	0.068	0.057	0.232	−0.147	0.048	0.003	0.009	0.098	0.925	0.074	0.072	0.307	0.026
Adjusted for Model 3 covariates + depressivesymptoms (BDI-II)													

Abbreviations: IL-6, interleukin-6; BMI, body mass index; HTN, history of hypertension; DM/IFG, history of diabetes or impaired fasting glucose; LDL-C, low density lipoprotein cholesterol; PSQI, Pittsburg Sleep Quality Index; BDI-II, Beck Depression Inventory II

*Between social cohesion, gender, and race in full models.