# Socioeconomic Disadvantage, Neighborhood Belonging, and Inflammation Among Adolescents

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**Objective:** Childhood socioeconomic disadvantage is associated with a host of adverse health outcomes across the lifespan. However, there is increasing interest in identifying factors that may promote resilience to disadvantage's effects on health. One promising candidate in this regard is a sense of neighborhood belonging, which could offset health risks by providing a sense of connection to others, as well as a sense of belonging to a community larger than oneself.

**Methods:** In a sample of 245 adolescents (age: mean [standard deviation] = 15.98 [0.54] years; sex: 64.1% female; race: 41.6% White, 37.6% Black/African American, 9.8% Other; ethnicity: 68.6% non-Hispanic), we examined neighborhood belonging as a moderator of the relationship between socioeconomic disadvantage (measured on a 0- to 5-point scale, mean [standard deviation] = 1.21 [1.36]) and low-grade inflammation (measured via a composite of circulating inflammatory biomarkers including IL-6, IL-8, IL-10, TNF-α, CRP, and suPAR). Covariates included age, sex, race/ethnicity, and pubertal status.

**Results:** Neighborhood belonging buffered the relationship between socioeconomic disadvantage and low-grade inflammation, a key mechanistic pathway to multiple chronic diseases. Specifically, there was a positive relationship between socioeconomic disadvantage and low-grade inflammation among individuals with low neighborhood belonging, but not among individuals with high neighborhood belonging.

**Conclusions:** These findings suggest that neighborhood belonging is one type of social connection factor that can mitigate the relationship between socioeconomic disadvantage and low-grade inflammation in youth.

**Key words:** neighborhood belonging, socioeconomic disadvantage, inflammation

**Abbreviations: BMI** = body mass index, **CRP** = C-reactive protein, **IL-6** = interleukin 6, **IL-8** = interleukin 8, **IL-10** = interleukin 10, **IPR** = income-to-poverty ratio, **suPAR** = soluble urokinase-type plasminogen activator receptor, **TNF-α** = tumor necrosis factor alpha

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## INTRODUCTION

Childhood socioeconomic disadvantage (a construct that includes low income, low educational attainment, and/or related circumstances such as unemployment) is associated with a host of adverse health outcomes across the lifespan (1–4). Previous research points to life stressors and health practices as major drivers of this relationship (5,6). Disadvantaged individuals are more likely to experience stressors related to finances and lack of resources (7,8), and have ongoing exposure to neighborhood violence (9–11) and family conflict (12–14). Additionally, socioeconomic disadvantage is associated with a higher frequency of behaviors associated with health problems (e.g., smoking, physical inactivity, and calorically dense diets) (15). Biologically, low-grade inflammation serves as a key biological mechanism that may be responsible for connecting stressors and lifestyle with health problems (6,16,17). Multiple meta-analyses have noted the relationship between socioeconomic disadvantage and higher levels of proinflammatory biomarkers (e.g., IL-6, TNF-α, and CRP) (17–20). Inflammation serves as the immune system's response to acute infection and injury. However, under conditions of chronic stress, the immune system can exhibit an exaggerated and sustained inflammatory response, which can lead to a host of health risks and chronic diseases (21,22). Additionally, poor health behaviors are also associated with increased inflammation (e.g., greater smoking, less physical activity, and poorer diets among disadvantaged individuals) (23–25). Among adolescents, elevated low-grade inflammation, measured by quantifying protein biomarkers including interleukin 6 (IL-6), tumor necrosis factor (TNF) α, and C-reactive protein (CRP) in circulating blood, is associated with metabolic syndrome and obesity (26–29). Additionally, elevated low-grade inflammation has been implicated in increased morbidity and mortality due to the development of multiple chronic diseases, including cardiovascular disease, diabetes, and autoimmune diseases (20,30).

There is growing interest in identifying protective factors that can buffer the impact of socioeconomic disadvantage on youth health (31–37). Currently, there is a host of research indicating that various forms of social support can mitigate the negative impact of socioeconomic disadvantage on health. For instance, Woodward et al. (38) found that three different types of social support (i.e., positive social interactions, affectionate support, and tangible support) each individually buffered the negative relationship between socioeconomic status and stress. In other work focusing on adults who experienced early-life socioeconomic disadvantage, those who recalled high childhood maternal warmth exhibited less proinflammatory signaling compared to those with low childhood maternal warmth (39). Additionally, high maternal nurturance buffered the relationship between childhood poverty and metabolic symptoms

in adulthood (40), whereas maternal responsiveness attenuated the role of cumulative risk (related to poverty and other stressors) on allostatic load among youth (41). Another example of social support acting as a buffer includes the impact of role models on youth, such that low socioeconomic status youth with supportive role models exhibited lower inflammation (i.e., IL-6) than low socioeconomic status youth without supportive role models (33).

As these studies illustrate, there has been considerable research on social support as a buffer against socioeconomic disadvantage. However, theory and research in this domain have largely focused on close ties at the individual relationship level (i.e., parent–child relationship, role models). Expanding the scope of the social support concept to also consider support at the neighborhood level may add a more comprehensive picture of how one's social network can buffer health risks associated with socioeconomic disadvantage (42,43). Consistent with this possibility, there is evidence that neighborhood social conditions forecast health outcomes in adulthood. Specifically, higher social capital (i.e., social cohesion, trust, and reciprocity) is associated with lower rates of mortality (44), myocardial infarction, and cardiovascular disease (45,46), and better self-reported health (47).

These observations suggest the possibility that favorable social conditions in the neighborhood may attenuate some of the health consequences of socioeconomic disadvantage. However, this hypothesis needs to be tested directly by examining biological mechanisms associated with poor health risk (i.e., inflammation), especially in the early decades of the lifecourse, when increased inflammation can play a role in the development of chronic health problems (48–50). In that light, here we consider the possibility that a sense of neighborhood belonging serves as a protective factor on the relationship between socioeconomic disadvantage and increased inflammation among youth. Drawing on theory related to the fundamental "need to belong" identified by Baumeister and Leary (51), neighborhood belonging may benefit individuals by tapping into the need for relational bonds with others and being part of a group or social network larger than oneself (52). In particular, neighborhood belonging may promote a sense of social cohesion with one's local community, foster community trust, and contribute to a sense of loyalty and emotional attachment to the community, which may be particularly advantageous for individuals facing socioeconomic challenges (53). Specifically, neighborhood belonging may reduce the impact of socioeconomic-related stressors, mitigating the relationship between socioeconomic disadvantage and stress-related inflammation. For example, individuals with high neighborhood belonging may seek support from neighbors during times of stress or may have role models in their neighborhood who help promote healthy behaviors when facing stress. Although there is some evidence of the positive impact of neighborhood belonging among adolescents (54–57), research is needed to examine whether neighborhood belonging can offset health risks (by way of increased inflammation) associated with socioeconomic disadvantage among youth.

In this study, we examined the role of neighborhood belonging as a potential buffer of the association between childhood socioeconomic disadvantage and low-grade inflammation, a biomarker that forecasts a variety of adverse health outcomes among adolescents and across the lifespan (20,26–30). We hypothesized that among youth who experienced high levels of belonging in their neighborhood, there would be no association between socioeconomic disadvantage and low-grade inflammation among youth. In contrast, among youth who experienced low levels of belonging in their neighborhood, the traditional relationship of high socioeconomic disadvantage with low-grade inflammation would be evident.

# METHODS

## Participants and Procedures

Our sample consisted of 245 adolescents (age: mean [standard deviation {SD}] = 15.98 [0.54] years) who participated in a two-wave longitudinal study on social disparities and cardiovascular risk. The sample was recruited to broadly reflect the demographics of Cook County, Illinois. Recruitment strategies included advertisements in schools, public transportation, and local media. Eligibility criteria included being in eighth grade, having good health (i.e., no infectious disease in the past 2 weeks, without history of chronic illness, free of prescription medications in past month), and not being pregnant. Participants were 64.1% female, 68.6% non-Hispanic (ethnicity), and 41.6% White, 37.6% Black/African American, 9.8% Other (race). Based on the U.S. Census' federal poverty thresholds, 17.6% of the sample lived in poverty (income-to-poverty ratio, IPR < 1.0), 20.8% in low-income homes (IPR 1–2), 24.9% in middle-income homes (IPR 3–4), and 36.7% in high-income homes (IPR > 4).

Participants completed study assessments while in eighth grade, and again 2 years later while in tenth grade. Here we focus on data from tenth grade, when neighborhood belonging was measured. At each visit, participants completed anthropometric measurements and had fasting antecubital blood drawn between 8:00 and 10:00 AM, which was used to measure biomarkers of low-grade inflammation (see below). Additionally, both a parent/guardian and participating youth completed surveys and interviews to assess demographic and psychosocial constructs. Written consent was provided by a parent or legal guardian for their child's participation; youth provided written assent for their participation. All procedures were approved by the Institutional Review Board of Northwestern University. The study was conducted from 2015 to 2019.

## Measures

### Socioeconomic Disadvantage

Parents reported on five domains of household socioeconomic disadvantage: income-to-poverty ratio, parent education attainment, parent unemployment, family structure, and receipt of government assistance. Total socioeconomic disadvantage was determined by a count score with a possible range of 0–5. Participants received one point for each of the following indicators of disadvantage: a household income to poverty ratio less than 1.99, at least one parent with a high school education or less, at least one unemployed parent, living in a single parent household, and receiving government assistance (58,59). We differentiate our use of socioeconomic disadvantage in this study from socioeconomic status, as we also include domains of family structure (i.e., living in a single parent household)

to include additional sources of socioeconomic-related stress (e.g., only having one parent who is able to provide income and who is also responsible for primary caregiving duties) that are not typically captured in measures of socioeconomic status.

## Neighborhood Belonging

Neighborhood belonging was assessed using four self-report items from the *Chicago Youth Development Study Community and Neighborhood Measure* (60). Participants were asked to think about their neighborhood and assess their feelings about it on a scale of 1 (strongly agree) to 5 (strongly disagree). The items were "I feel like I belong to my neighborhood," "I feel loyal to the people in my neighborhood," "I like to think of myself as similar to the people who live in my neighborhood," and "Given the opportunity, I would like to move out of my neighborhood" (Cronbach's $\alpha = .792$). After responses to the first three items were reverse coded, the four items were summed to form a composite, with higher scores indicating higher neighborhood belonging. Neighborhood belonging was measured as a continuous variable. When decomposing simple slopes to examine how neighborhood belonging moderated the relationship between socioeconomic disadvantage and inflammation, low neighborhood belonging was operationalized as 1 SD below the mean, and high neighborhood belonging was operationalized as 1 SD above the mean.

## Interpersonal Social Support

In post-hoc analysis, the widely used and extensively validated Harter Social Support Scale for Children was used as a covariate to assess the unique impact of neighborhood belonging (61). Participants reported the degree to which they perceive social support from others on a four-point scale across six items, with higher scores indicating greater perceived social support. The Cronbach's $\alpha$ for this sample was .873.

## Depressive Symptoms

Depressive symptoms were assessed as a covariate in post-hoc analysis. Depressive symptoms were measured using the Revised Child Anxiety and Depression Scale, an extensively validated self-report measure (62,63). Participants reported depressive symptoms on a four-point scale across 10 items; raw scores were converted to T scores based on reference groups of the corresponding grade level and gender, with higher scores indicating greater depressive symptoms (64). The Cronbach's $\alpha$ for this sample was .865.

## Physical Activity

Physical activity was assessed as a covariate in post-hoc analysis using three items from the Physical Activity and Exercise Questionnaire—Adolescents (65). The first item assessed how many days the individual was very active in the last week after school on a five-point scale (1 = none; 2 = 1 time; 3 = 2 or 3 times; 4 = 4 times; 5 = 5 times). The second item assessed how many times the individual was very active during the last weekend on a five-point scale (1 = none; 2 = 1 time; 3 = 2 or 3 times; 4 = 4 or 5 times; 5 = 6 or 7 times). The third item assessed how often the individual engaged in activities involving physical effort on a five-point scale (1 = All or most of my free time was spent doing things that involve little physical effort; 2 = I sometimes (1–2 times last week) did physical things in my free time (e.g., played sports, went running, swimming, bike riding, did aerobics); 3 = I often (3–4 times last week) did physical things in my free time; 4 = I quite often (5–6 times last week) did physical things in my free time; and 5 = I very often (7 or more times last week) did physical things in my free time). Final physical activity scores were calculated by taking the average of these three items, with higher scores indicating higher physical activity. The Cronbach's $\alpha$ for this sample was .733.

## Inflammation

Inflammation was assessed using a composite index consisting of serum cytokines (IL-6, IL-8, IL-10 and TNF-α), CRP, and soluble urokinase-type plasminogen activator receptor (suPAR) measured in fasting antecubital blood. CRP is a commonly assessed inflammation biomarker that is associated with higher risk of coronary heart disease, myocardial infarctions, stroke, and diabetes (66–69). suPAR is a newer inflammatory biomarker thought to reflect vascular inflammation (70) that is less sensitive to acute changes in diet and health compared to CRP (71,72). Furthermore, suPAR has been associated with cardiovascular disease, diabetes, cancer, and mortality (independent of CRP) (73–75).

CRP was measured in duplicate by high-sensitivity immunoturbidimetric assay on a Roche/Hitachi cobas c502 instrument. The average intra-assay and interassay coefficients of variation were 2.5% and 5.6%. The cytokines were measured in triplicate by four-plex immunoassay (76) on a microfluidic platform (Simple Plex; Protein Simple). Across runs, the intra-assay coefficients of variation for duplicate pairs were 4.0% (IL-6), 4.6% (IL-10), 3.0% (IL-8), and 3.8% (TNF-α). The corresponding interassay coefficients of variation were 6.4%, 7.0%, 8.5%, and 5.8%. suPAR was measured in duplicate by immunoassay (Human Quantikine ELISA; R&D Systems). The intra-assay and interassay coefficients of variation were 1.6% and 1.1%.

The raw values of each biomarker were log-10 transformed to normalize distributions. Subsequently, log-transformed values were *z*-scored and averaged to form the inflammation composite (58). Similarly to previous work, we utilized this composite approach as these cytokines values clustered (see Table S1, Supplemental Digital Content, http://links.lww.com/PSYMED/B47, for correlations between individual inflammatory markers) to correct for skewness, reduce statistical testing, and limit the rate of false positives (77). We included both proinflammatory and anti-inflammatory cytokines in our composite because they both have important roles in immune regulation. Although they have different functional properties, proinflammatory and anti-inflammatory cytokines are generally correlated in a positive direction. This is because anti-inflammatory cytokines (e.g., IL-10) are only expressed under conditions of inflammatory activity (78–81). After assessing the inflammation composite for outliers, one case was removed as its standardized value was over 3.

## Covariates

A priori selected covariates included age (years, continuous), sex (male/female), race/ethnicity, and pubertal status (via five-item Pubertal Developmental Scale, continuous) (82). Pubertal status was included as a covariate based on

 

**TABLE 1.** Means, Standard Deviations, and Zero-Order Correlations With Confidence Intervals of All Variables of Interest

| Variable | *M* or % | SD | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Inflammation | 0.00 | 0.62 | | | | | | | | | | | | |
| 2. Socioeconomic disadvantage | 1.21 | 1.36 | 0.06 [−0.07 to 0.18] | | | | | | | | | | | |
| 3. Neighborhood belonging | 12.23 | 3.33 | −0.09 [−0.21 to 0.03] | −0.23** [−0.34 to −0.10] | | | | | | | | | | |
| 4. Age | 15.98 | 0.54 | 0.05 [−0.07 to 0.18] | 0.16* [0.04 to 0.28] | −0.10 [−0.22 to 0.02] | | | | | | | | | |
| 5. Sex (female) | 64.1% | | −0.05 [−0.17 to 0.08] | 0.10 [−0.03 to 0.22] | 0.01 [−0.11 to 0.13] | 0.01 [−0.11 to 0.13] | | | | | | | | |
| 6. Race (Black) | 37.6% | | −0.09 [−0.21 to 0.03] | 0.23** [0.11 to 0.34] | −0.20** [−0.31 to −0.08] | 0.01 [−0.11 to 0.14] | 0.01 [−0.11 to 0.13] | | | | | | | |
| 7. Ethnicity (Hispanic) | 31.4% | | 0.03 [−0.09 to 0.15] | 0.20** [0.08 to 0.32] | 0.03 [−0.09 to 0.16] | 0.08 [−0.05 to 0.20] | 0.02 [−0.11 to 0.14] | −0.56** [−0.64 to −0.47] | | | | | | |
| 8. Pubertal status | 4.15 | 0.63 | 0.01 [−0.12 to 0.13] | −0.02 [−0.14 to 0.11] | −0.01 [−0.13 to 0.12] | 0.10 [−0.02 to 0.22] | 0.55** [0.46 to 0.63] | −0.00 [−0.12 to 0.12] | −0.04 [−0.16 to 0.08] | | | | | |
| 9. Interpersonal social support | 20.25 | 4.16 | −0.07 [−0.19 to 0.05] | −0.22** [−0.33 to −0.10] | 0.24** [0.12 to 0.35] | −0.10 [−0.22 to 0.02] | 0.09 [−0.04 to 0.21] | −0.10 [−0.22 to 0.02] | −0.03 [−0.15 to 0.10] | 0.06 [−0.06 to 0.18] | | | | |
| 11. Depressive symptoms | 49.93 | 11.29 | 0.05 [−0.08 to 0.17] | 0.10 [−0.02 to 0.23] | −0.34** [−0.45 to −0.23] | 0.09 [−0.04 to 0.21] | 0.07 [−0.05 to 0.19] | −0.06 [−0.18 to 0.06] | 0.13* [0.00 to 0.25] | 0.08 [−0.04 to 0.20] | −0.23** [−0.34 to −0.11] | | | |
| 10. Physical activity | 2.56 | 0.97 | −0.04 [−0.16 to 0.09] | −0.10 [−0.22 to 0.03] | 0.09 [−0.03 to 0.21] | −0.11 [−0.23 to 0.01] | −0.24** [−0.35 to −0.12] | −0.09 [−0.21 to 0.03] | −0.00 [−0.12 to 0.12] | −0.20** [−0.31 to −0.08] | 0.04 [−0.08 to 0.16] | −0.24** [−0.35 to −0.12] | | |
| 12. BMI | 25.07 | 6.53 | 0.28** [0.16 to 0.39] | 0.19** [0.07 to 0.31] | −0.12 [−0.24 to 0.01] | 0.03 [−0.09 to 0.16] | −0.01 [−0.13 to 0.11] | 0.06 [−0.06 to 0.18] | 0.13* [0.01 to 0.25] | 0.12 [−0.00 to 0.24] | −0.04 [−0.16 to 0.09] | 0.10 [−0.02 to 0.22] | −0.07 [−0.19 to 0.06] | |
| 12. Baseline inflammation | 0.00 | 0.60 | 0.47** [0.37 to 0.56] | 0.11 [−0.01 to 0.23] | −0.15* [−0.26 to −0.02] | −0.00 [−0.13 to 0.12] | −0.11 [−0.23 to 0.01] | −0.04 [−0.16 to 0.08] | 0.02 [−0.10 to 0.14] | −0.08 [−0.20 to 0.04] | −0.04 [−0.16 to 0.09] | 0.08 [−0.05 to 0.20] | 0.05 [−0.08 to 0.17] | 0.35** [0.23 to 0.45] |

BMI = body mass index.

The table reflects a sample of 245 participants. *M* and SD are used to represent mean and standard deviation, respectively. Correlation values reflect zero-order correlations. Values in square brackets indicate the 95% confidence interval for each correlation.

* $p < .05$.

** $p < .01$.

previous work showing that a) socioeconomic disadvantage accelerates pubertal development and that b) many immune functions also shift across pubertal development (83–85). In post-hoc analysis, interpersonal support, depressive symptoms, physical activity, body mass index (BMI), and baseline inflammation were included as covariates in subsequent models.

Race/ethnicity was based on parent reports' of whether individuals identified as White, African American, Latino, or Other (i.e., Asian, Native Hawaiian/Pacific Islander, and/or American Indian/Alaskan Native). Multiracial participants who identified as African American as one of their races were categorized as African American. Participants who identified as Latino and did not identify as African American were categorized as Latino. Only participants who exclusively identified as White were categorized as such. Thus, two dummy variables were created with White only as the reference group and included in the model to assess race/ethnicity.

## Statistical Analyses

Descriptive statistics and zero-order correlations were first calculated and observed (Table 1). We then assessed the main effects of a) socioeconomic disadvantage on inflammation and b) neighborhood belonging on inflammation to assess independent effects of these predictors before examining whether they interacted with one another. Next, we used hierarchical regression analysis to conduct the regression models for our primary analysis in steps (Table 2). The first step included covariates only, the second step included the main effects of socioeconomic disadvantage and neighborhood belonging, and the third step included the interaction of socioeconomic disadvantage and neighborhood belonging. Our primary analysis involved the inflammation composite modeled as a function of the interaction between socioeconomic disadvantage and neighborhood belonging including the main effects of socioeconomic disadvantage and neighborhood belonging and all covariates. Simple slopes were calculated for our primary analysis at 1 SD above and below the mean of neighborhood belonging.

Next, post-hoc analyses were conducted to determine whether our results would change if another support indicator (i.e., interpersonal support) was included as a covariate in the model. To do so, we first included interpersonal support as a covariate in the model to rule out the possibility that an effect of interpersonal support confounded our primary results. We then included the interaction between socioeconomic disadvantage and interpersonal support as a covariate to assess whether or not the moderating role of neighborhood belonging on socioeconomic disadvantage was driven by a possible interaction between interpersonal support and socioeconomic disadvantage, given that interpersonal support may have an additional buffering impact on the relationship between socioeconomic disadvantage and inflammation. Subsequently, we examined the interaction between socioeconomic disadvantage and interpersonal support to see whether another type of support independently moderated the relationship between socioeconomic disadvantage and inflammation (without neighborhood belonging in the model). We then included additional covariates, including depressive symptoms, physical activity, and BMI in subsequent post-hoc analysis, as all are known risk factors for inflammatory activity (24,86–88). Next, although our primary analysis was conducted using data from the second wave of our two-wave study (when neighborhood belonging was assessed), we controlled for baseline inflammation during the first wave (2 years prior) in post-hoc analysis to increase robustness of results. Finally, we included sensitivity analyses where we examined each individual inflammatory biomarker (i.e., IL-6, IL-8, IL-10, TNF-α, CRP, and suPAR) as our outcome of interest (rather than the composite) to determine whether our results were driven by specific inflammatory markers.

A sensitivity power analysis showed that this sample size provided 80% power to detect effect sizes as small as Cohen's $f = 0.180$ (i.e., $R^2 = 0.031$) at $p = .05$. All analyses were conducted in *R*.

## Transparency and Openness

The study materials and code used for analysis are available at the following OSF link: https://osf.io/s56n9/?view_only=5d3a5fab777b47c996af44a3bed81005. This study was not preregistered. Because participants were informed that their study data would be kept confidential, the data for this study are not publicly available. The data can be available upon request with Northwestern University IRB approval.

# RESULTS

Descriptive statistics and zero-order correlations of the inflammation composite, socioeconomic disadvantage, neighborhood belonging, and all covariates are reported in Table 1. Neither socioeconomic disadvantage ($b = 0.04$; $p = .211$) nor neighborhood belonging ($b = -0.02$; $p = .128$) was associated with the inflammation composite. However, in line with our primary hypothesis, socioeconomic disadvantage and neighborhood belonging interacted to predict inflammation ($b = -0.02$; $p = .014$) (Figure 1 and Figure S1, Supplemental Digital Content, http://links.lww.com/PSYMED/B47; see results of the hierarchical regression analysis reported in Table 2). Simple slope analyses indicated a positive relationship between socioeconomic disadvantage and inflammation among individuals with low neighborhood belonging ($b = 0.11$; $p = .012$), but not among individuals with high neighborhood belonging ($b = -0.04$; $p = .376$).

In post-hoc analyses, we found that the interaction between socioeconomic disadvantage and neighborhood belonging persisted when another type of support indicator (i.e., interpersonal support) was included as a covariate in the model ($b = -0.02$; $p = .015$). Second, we found that the interaction between socioeconomic disadvantage and neighborhood belonging predicting inflammation also remained significant when we included the interaction between interpersonal social support and socioeconomic disadvantage as a covariate ($b = -0.02$; $p = .044$). Additionally, we examined the interaction between socioeconomic disadvantage and interpersonal support predicting inflammation, which was not significant ($b = -0.01$; $p = .060$).

Next, we examined the interaction between socioeconomic disadvantage and neighborhood belonging predicting inflammation with additional covariates, including a) depressive symptoms, b) physical activity, and c) BMI (known risk factors for

 

**TABLE 2.** Interaction of Socioeconomic Disadvantage and Neighborhood Belonging Predicting Inflammation

| | *b* | SE | CI | *p* |
|---|---|---|---|---|
| Step 1 | | | | |
| (Intercept) | −0.90 | 1.19 | (−3.25 to 1.44) | .448 |
| Age | 0.06 | 0.07 | (−0.09 to 0.21) | .416 |
| Sex (female) | −0.08 | 0.10 | (−0.28 to 0.11) | .403 |
| Race (Black) | −0.14 | 0.09 | (−0.33 to 0.05) | .147 |
| Ethnicity (Hispanic) | −0.04 | 0.11 | (−0.25 to 0.17) | .698 |
| Pubertal status | 0.03 | 0.08 | (−0.12 to 0.18) | .659 |
| $R^2$ | 0.015 | | | |
| Step 2 | | | | |
| Socioeconomic disadvantage | 0.04 | 0.03 | (−0.02. 0.11) | .211 |
| Neighborhood belonging | −0.02 | 0.01 | (−0.04 to 0.01) | .128 |
| $R^2$ | 0.031 | | | |
| Step 3 | | | | |
| Socioeconomic disadvantage by neighborhood belonging | −0.02 | 0.01 | (−0.04 to >−0.00) | .014 |
| $R^2$ | 0.056 | | | |

SE = standard error; CI = confidence interval.

The table reflects hierarchical regression analysis of full sample ($n$ = 245), with first step including covariates only, second step including the main effects of socioeconomic disadvantage and neighborhood belonging, and third step including the interaction of socioeconomic disadvantage and neighborhood belonging. Primary analysis involved the inflammation composite modeled as a function of the interaction between socioeconomic disadvantage and neighborhood belonging including the main effects of socioeconomic disadvantage and neighborhood belonging and all covariates.

inflammation) (24,86–88), in subsequent post-hoc analysis. The interaction between socioeconomic disadvantage and neighborhood belonging persisted when controlling for depressive symptoms ($b$ = −0.02, $p$ = .015), physical activity ($b$ = −0.02, $p$ = .009), and BMI ($b$ = −0.02, $p$ = .006).

Further post-hoc analyses were conducted to further increase robustness of results. Although we analyzed data for our primary analysis in the second wave of our two-wave study (when neighborhood belonging was assessed), we ran a subsequent analysis controlling for baseline inflammation using data from the first wave of the study. Here, we found that the interaction between socioeconomic disadvantage and neighborhood belonging predicting inflammation remained significant ($b$ = −0.02, $p$ = .024). Finally, we included sensitivity analyses to determine whether the moderating effect of neighborhood belonging on the relationship between socioeconomic disadvantage and inflammation was driven by specific inflammatory markers. Neighborhood belonging moderated the relationship between socioeconomic disadvantage and IL-6 ($b$ = −0.03, $p$ = .043) and IL-10 ($b$ = −0.05, $p$ < .001). The moderation effects were the same direction, but below the conventional significance threshold for TNF-α ($b$ = −0.02, $p$ = .094) and CRP ($b$ = −0.02, $p$ = .138). There was no evidence of moderation for IL-8 ($b$ < 0.01, $p$ = .795) and suPAR ($b$ = −0.01, $p$ = .547).

## DISCUSSION

Socioeconomic disadvantage is associated with a range of adverse health outcomes, and there is considerable interest in identifying modifiable protective factors that could mitigate this relationship. Toward that end, we observed evidence that a sense of neighborhood belonging may protect disadvantaged youth from low-grade inflammation. Specifically, we observed a positive relationship between socioeconomic disadvantage and inflammatory biomarkers among youth with low neighborhood belonging, but this association was not apparent among youth with high neighborhood belonging. These findings suggest the possibility that neighborhoods can be a source of social connection for youth from disadvantaged households, which offer protection against physical health risks.

Consistent with this hypothesis, previous research has shown that social support can buffer the relationship between low socioeconomic status and higher inflammatory activity (6). For example, social support buffered the relationship between low early-life subjective socioeconomic status and IL-6

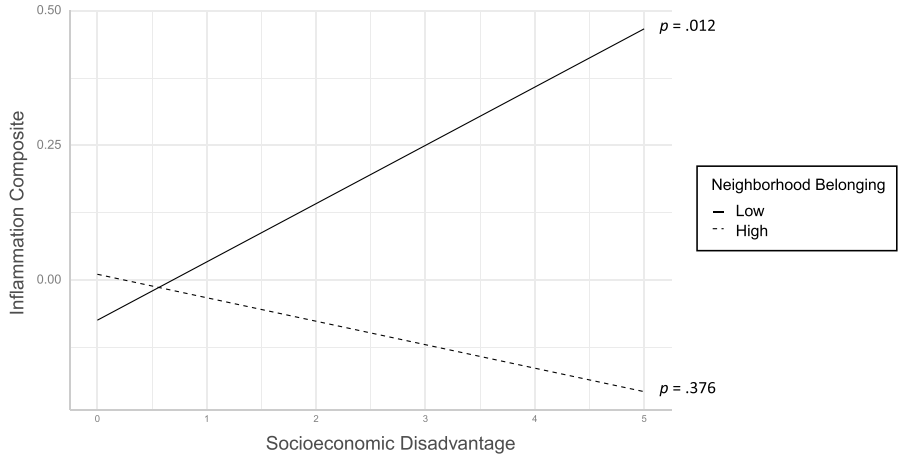


**FIGURE 1.** Adjusted interaction of socioeconomic disadvantage and neighborhood belonging predicting inflammation. *Note:* This figure represents the interaction between socioeconomic disadvantage and neighborhood belonging predicting inflammation (i.e., a composite of log-10 transformed, *z*-scored circulating inflammatory biomarkers (i.e., IL-6, IL-8, IL-10, TNF-α, CRP, and suPAR), adjusting for age, sex, race/ethnicity, and pubertal status. Simple slopes are displayed at 1 standard deviation below and above the mean to represent low and high neighborhood belonging, respectively. IL = interleukin; TNF-α = tumor necrosis factor alpha; CRP = C-reactive protein; suPAR = soluble urokinase-type plasminogen activator receptor.

reactivity following a laboratory stressor (89). In another study, social support from peers buffered the relationship between low family subjective socioeconomic status and high inflammation among adolescents (90). Conversely, loneliness and social isolation have been associated with increased inflammation (91–93). Together, these studies suggest that individuals who are more socially isolated may have greater health risks, whereas supportive social relationships may reduce stress and other negative emotions for disadvantaged individuals, serving as a protective factor for the relationship between socioeconomic disadvantage and health risks. In line with this research, we found support for the hypothesis that a sense of belonging to one's neighborhood buffered the relationship between socioeconomic disadvantage and inflammation. Neighborhood belonging may be able to offset health risks associated with socioeconomic disadvantage by providing adolescents with social resources that may counteract the disadvantages associated with fewer financial resources, as well as providing a sense of security in knowing that they can depend on their community when facing socioeconomic-related stressors. Additionally, neighborhood belonging may provide access to social support beyond an individual's family environment, which may be particularly important given that socioeconomic challenges typically impact the entire family unit.

Why would neighborhood belonging act in this manner? One explanation is that youth with high neighborhood belonging have access to resources that promote healthy coping with stressors related to socioeconomic disadvantage. These resources could include role models in the community that provide examples of successful strategies for coping and emotion regulation more generally. Another explanation may be related to how neighborhood belonging can promote healthy behaviors, such as physical activity, which can offset the association between socioeconomic disadvantage and inflammation. For instance, youth from more cohesive neighborhoods (i.e., ones with stronger social bonds) engage in greater physical activity because of stronger community-based norms and/or greater availability of community-based recreational activities (94). In support of this notion, other studies have found that similar constructs such as high neighborhood collective efficacy (i.e., the belief that individuals in a neighborhood can successfully come together to accomplish shared goals) and high neighborhood social capital (i.e., the social functioning of neighborhoods including the interpersonal relationships, trust, shared norms, and reciprocity of its members) are associated with higher physical activity among youth (95,96), as well as lower BMI (97–99). In future research, it will be important to test these hypotheses directly, and identify pathways through which neighborhood belonging offsets health risks in youth.

Given that our study examines both socioeconomic disadvantage and neighborhood belonging, it is important to note the role that neighborhood quality (e.g., housing conditions, services and facilities, perceived security) may play on these variables. First, neighborhood quality can serve as an indicator of socioeconomic-related stress. Additionally, neighborhood belonging may relate to neighborhood quality in varying ways. For example, individuals with high belonging in neighborhoods with low neighborhood quality may seek support from their neighbors to provide a greater sense of security from physical danger or share resources with one another not available to them through neighborhood services. Conversely, individuals with low belonging in neighborhoods with low neighborhood quality may experience increased fear or stress from neighborhood violence or poor conditions. Taken together, although low neighborhood quality, or related socioeconomic-related stress in one's neighborhood, may negatively impact health, neighborhood belonging may play a specific role in buffering this relationship. One consideration when noting how neighborhood quality may relate to neighborhood belonging is one item of our neighborhood belonging measure, which asks individuals, "Given the opportunity, I would like to move out of my neighborhood." Here, we hope to capture cases where, despite living in communities with lower neighborhood quality, high neighborhood belonging may motivate individuals to stay in the community even if given the opportunity to move. This emphasizes our focus on how neighborhood belonging, rather than a component of neighborhood quality, may serve as a possible protective factor on the relationship between socioeconomic disadvantage and inflammation; however, future work should examine how neighborhood quality may relate to the protective role of neighborhood belonging on this relationship.

The associations observed here may be particularly salient during adolescence, when "fitting in" to social groups becomes increasingly important (100). Research shows that during adolescence, a sense of belonging plays a large role in the development of self-esteem and the ability to cope with hardships, including those related to socioeconomic disadvantage (101,102). Currently, most research in this domain focuses on adolescents' social connectedness at the individual level, emphasizing constructs like peer support, parental support, and peer and school belonging (103–107). However, our results suggest that belonging at the neighborhood level may also be protective against the health risks associated with socioeconomic disadvantage, even when controlling for individual sources of social connection. We speculate that neighborhood belonging provides youth a sense of connectedness to not only peers but also a broader multigenerational community that provides role models, concrete resources, and social support that facilitate coping with disadvantage-related stressors.

Future research is needed to further understand mechanisms that may underlie this relationship. For example, perhaps youth from socioeconomically disadvantaged households who experience high neighborhood belonging find others in their community that can help them engage in healthy behaviors within their neighborhood environments. Or perhaps youth from socioeconomically disadvantaged households who experience high neighborhood belonging turn to others in the community who help teach them emotion regulation or other coping strategies to adaptively face stressors in their lives (108,109). In turn, these healthy behaviors and coping strategies can mitigate the effects of socioeconomic disadvantage on inflammation. Additionally, future work should also consider how the exchange of tangible resources may offset financial stressors among individuals who experience high neighborhood belonging. In sum, future research should explore these explanations to better understand mechanisms underlying how neighborhood belonging may come to buffer the impact of socioeconomic disadvantage on health risk and outcomes in youth.

Limitations of the present study include the cross-sectional, observational study design. Given the cross-sectional study design, we note that we cannot make causal inferences from our results and that conclusions regarding the direction of the association are limited in the present study. Furthermore, there may be unobserved variables that could account for the relationships found in this study, such as other neighborhood factors related to socioeconomic disadvantage (i.e., exposure to violence), or other factors that could shape participants' reports of belonging (e.g., personality traits). Future research should include measures of other potential confounding factors, including factors that may contribute to resilience among youth from socioeconomically disadvantaged households to determine the relative importance of neighborhood belonging in the context of other resilience factors.

In conclusion, findings from the current study contribute to the literature by demonstrating neighborhood belonging as a buffer between socioeconomic disadvantage and low-grade inflammation among adolescents. If replicated, these findings suggest that fostering a stronger sense of community in socioeconomically disadvantaged neighborhoods may represent one avenue to disrupting the link between socioeconomic disadvantage and inflammation in youth. Future research to further a mechanistic understanding of these relationships and to test neighborhood-based interventions would help bolster efforts to mitigate the negative health outcomes related to socioeconomic disadvantage earlier in life.

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