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Peer Support as Moderator of Association Between Socioeconomic Status and Low-Grade Inflammation in Adolescents

Tao Jiang[1], Edith Chen[1, 2], Phoebe H. Lam[2], Jungwon Kim[1], Hee Moon[1], and Gregory E. Miller[1, 2]
[1] Institute for Policy Research, Northwestern University
[2] Department of Psychology, Northwestern University

Objective: Individuals who grow up in low-socioeconomic status (SES) families are at an increased risk of health problems across the lifespan. Although supportive social relationships are postulated to be a protective factor for the health of these individuals, the role of friend support in adolescence is not well understood. Given that low-grade inflammation is one key biological mechanism proposed to explain links between family SES and health outcomes, we examined whether adolescents' friend support buffers the association between family SES and low-grade inflammation among adolescents. ***Method:*** 277 dyads of adolescents (63.5% female; 39.4% White, 38.3% Black, and 32.1% Hispanic; $M_{age} = 13.92$ years) and one of their parents participated in this longitudinal study (two waves approximately 2 years apart). Parents reported family objective SES (i.e., income, savings, and education) and family subjective SES (i.e., subjective social status). Adolescents reported perceived friend support. Fasting antecubital blood was drawn from adolescents at both visits. Low-grade inflammatory activity was represented by a composite of inflammatory biomarkers and numbers of classical monocytes. ***Results:*** Adolescents' friend support moderated the associations of family subjective SES with both the inflammation composite and classical monocyte counts across cross-sectional, longitudinal, and prospective change (only significant for the inflammation composite) analyses. Specifically, lower family subjective SES was associated with higher levels of low-grade inflammation only among adolescents lower, but not higher, in friend support. No moderation was observed for objective SES. ***Conclusion:*** Supportive peer relationships buffer the link between family subjective, but not objective, SES and low-grade inflammation in adolescence.

Public Significance Statement
The current study sheds light on the role of adolescents' peer relationships in health disparities. The findings suggest that supportive peer relationships during adolescence may have the potential to play a preventive and protective role in the health of individuals who grow up in families in which parents have low subjective social status.

Keywords: inflammation, peer relationships, adolescents, socioeconomic status, perceived social status

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Family socioeconomic status (SES) during childhood and adolescence (referred to as "family SES" hereafter) is linked to health across the lifespan. Substantial evidence indicates that youth living in low-SES families are more likely to have health problems (e.g., asthma; see Chen et al., 2002; Poulain et al., 2020 for reviews). Furthermore, low family SES during childhood and adolescence is associated with a higher risk of premature mortality, and developing cardiovascular disease, some cancers, and autoimmune conditions during adulthood, independent of adult SES (Kittleson et al., 2006; Poulton et al., 2002; see Cohen et al., 2010; Galobardes et al., 2004, 2008 for meta-analyses and reviews).

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Tao Jiang https://orcid.org/0000-0001-8894-7669
Phoebe H. Lam https://orcid.org/0000-0002-0248-2504
Phoebe H. Lam is now at Department of Psychology, Carnegie Mellon University.
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Correspondence concerning this article should be addressed to Tao Jiang, Institute for Policy Research, Northwestern University, 1801 Maple Avenue, Suite 2450, Evanston, IL 60201, United States. Email: tjiang@northwestern.edu

Most previous research on this topic used indicators such as parents' occupational status, income, and/or education to assess the socioeconomic environments youth are exposed to (termed family objective SES). These types of indicators reflect the economic and social resources accessible to a family (Tan et al., 2020). Nascent research has used parents' subjective social status as another indicator of family SES (termed family subjective SES; Roy et al., 2019; Ursache et al., 2015). Family subjective SES refers to parents' perceptions about their relative socioeconomic position in comparison to others, which are shaped not only by their socioeconomic resources but also by psychological factors, such as social comparison, self-efficacy, and feelings of inequality (Tan et al., 2020). Research has shown that adults who subjectively perceive their SES to be lower than others have higher levels of negative affect and rumination and more mental and physical health issues, independent of the objective indicators of their SES (O'Leary et al., 2021; see Cundiff & Matthews, 2017 for a meta-analysis). Accordingly, family subjective SES may influence parenting behaviors and parent–youth interactions above and beyond the effects of family objective SES, which in turn shape the social environments youth experience and thus their health outcomes. Indeed, empirical evidence shows that lower family subjective SES is associated with more symptoms in children with asthma (Diep et al., 2019) and a slower positive increase in self-reported physical health over time during adolescent development (Kim et al., 2021). Thus, both family objective and subjective SES can be linked to youth's health outcomes.

Low-Grade Inflammation as a Mechanism Linking Family SES and Health

Through what mechanisms might family SES influence health during childhood, adolescence, and even adulthood? Inflammation is postulated to be a key underlying biological mechanism (Miller, Chen, & Parker, 2011). Inflammation represents a response of the body to injury or infection, but psychosocial stressors, including socioeconomic hardships, can also influence how inflammatory responses unfold (Danese & Lewis, 2017; Muscatell et al., 2020). Specifically, socioeconomic disadvantage experienced during childhood or adolescence can shape the way monocytes—cells of the innate immune system—respond to stimuli, resulting in sustained low-grade inflammation across the lifespan (Lam et al., 2022; Miller, Chen, & Parker, 2011). In turn, elevated levels of low-grade inflammation have been shown to predict morbidity and mortality from the types of health problems that have been associated with socioeconomic disadvantage, such as cardiovascular disease and some cancers (see Furman et al., 2019 for a review). Given the mechanistic role of low-grade inflammation in many socially patterned diseases, and in the link between family SES and these diseases, the current study examines whether family objective and subjective SES are associated with low-grade inflammation in adolescents.

Low-grade inflammatory activity is commonly represented by levels of circulating inflammatory biomarkers (e.g., C-reactive protein [CRP], interleukin-6 [IL-6], tumor necrosis factor-α [TNF-α]). Previous research has demonstrated that lower family SES is associated with higher levels of inflammatory biomarkers in children, adolescents, and adults (see Chiang et al., 2022; Milaniak & Jaffee, 2019 for meta-analyses). However, it is unclear to what degree these biomarkers actually represent immunologic processes because they can be released by a variety of tissues, such as adipose and muscle, some of which are not involved in immune responses. Thus, besides inflammatory biomarkers, the current study measures the prevalence of monocyte populations that are involved in initiating and maintaining inflammation (Geissmann et al., 2010). Human monocytes can be divided into three subpopulations, including classical ($CD14^{+}CD16^{-}$), intermediate ($CD14^{+}CD16^{+}$), and nonclassical monocytes ($CD14^{-}CD16^{+}$; Narasimhan et al., 2019). Although the functions of intermediate monocytes are not well understood, it is clear that classical monocytes are the cellular drivers of many inflammatory responses, whereas nonclassical monocytes are responsible for maintaining vascular homeostasis (Narasimhan et al., 2019). Both animal and human research indicate that under conditions of chronic psychological stress, classical monocytes are selectively mobilized into circulation from bone marrow, and are primed to mount aggressive inflammatory responses to subsequent stimuli. Both of these phenomena are largely mediated via adrenergic mechanisms involving the sympathetic nervous system (Finegood et al., 2020; Miller, Murphy, et al., 2014; Powell et al., 2013; see Weber et al., 2017 for a review). Nonclassical monocytes appear to be less affected by stressful conditions, for reasons that are not understood. Regardless, to our knowledge, no research has examined whether family objective and subjective SES are associated with the prevalence of different populations of monocytes involved in inflammation. The current study aims to fill this gap and examine the associations between family SES and classical and nonclassical monocytes in adolescents.

Friend Support in Adolescence as a Protective Factor

Although low family SES is associated with poor health outcomes, many individuals can remain healthy, despite growing up in low-SES environments. Extensive research has documented a host of resilience factors (see Chen & Miller, 2013 for a review), one of which is supportive social relationships (Chen et al., 2017). Supportive relationships during childhood and adolescence can reduce the psychosocial stress experienced by low-SES children or adolescents, which protects them from developing health problems. For example, for individuals who experienced low family SES, supportive parental relationships during childhood are associated with a less pro-inflammatory phenotype (Chen et al., 2011), fewer metabolic-syndrome components (e.g., high blood pressure; Miller, Lachman, et al., 2011), and less susceptibility to the common cold (Cohen et al., 2020) later in life.

Accumulating evidence indicates that supportive parental relationships can buffer against the link between low family SES and poor health, but it is unclear whether other relationships function similarly. During adolescence, peer relationships become increasingly important (Brown & Larson, 2009; Harris, 1995). Adolescents spend more time with their peers as they mature, and peer relationships become more intimate and serve as important coping resources in adolescents' life (Mitic et al., 2021). Furthermore, supportive peer relationships can buffer the effects of negative family environments experienced by adolescents (Gauze et al., 1996). For example, perceived support from friends buffers the effect of negative parenting (e.g., unilateral parental decision making) on adolescents' externalizing behavior at school (e.g., fighting with others; Lansford et al., 2003). Supportive peer relationships also improve adjustment after parental bereavement during adolescence (Dopp & Cain, 2012).

Because peer relationships are crucial for adolescents and can buffer the impacts of negative family environments, it is possible that supportive peer relationships during adolescence might also buffer the association between low family SES and poor physical health outcomes. However, there has been little empirical research on this hypothesis.

Related studies have shown that supportive peer relationships buffer the association between cyber-harassment and physical symptoms (e.g., headache) among adolescent boys (Fridh et al., 2015), and they also buffer the association between racial discrimination during adolescence and allostatic load in early adulthood among African Americans (Brody et al., 2014). To our knowledge, no study has examined whether adolescents' friend support buffers the association between their family SES and low-grade inflammation. The current study aims to fill this gap using a two-wave longitudinal design.

The Present Study

Given the evidence reviewed above, we hypothesized that friend support would buffer adolescents from the low-grade inflammation that generally accompanies socioeconomic disadvantage. To evaluate this hypothesis, we had parents report on family SES using both objective and subjective approaches. Adolescents reported on friend support by questionnaire and had fasting blood drawn at a baseline visit and 2 years later. The blood was used to assess both inflammatory biomarkers and monocyte subpopulations. We predicted the buffering pattern would be apparent for inflammatory biomarkers and classical monocytes, but not for nonclassical monocytes, as classical monocytes are more responsive to psychosocial stress than nonclassical monocytes. We expected similar patterns to emerge for objective and subjective indicators of SES, as both have been related to health outcomes.

Method

Transparency and Openness

This study was not preregistered. The study materials and analysis scripts are available via the following OSF link: https://osf.io/gt49r/?view_only=4c9b1d59b1854d3c83df8807bfc99f4e (Jiang 2023). The study was approved by the Institutional Review Board of Northwestern University. Because the participants who provided the data were informed that their study-related information would be kept confidential, the data for this study are not publicly available. The data can be available upon request with Institutional Review Board approval and a data use agreement.

Participants and Procedure

Adolescents from the Chicago area ($N = 277$; 63.5% female; $M_{age} = 13.92$ years, $SD_{age} = 0.54$ years) were recruited for a two-wave longitudinal study. The study was conducted prior to the COVID-19 pandemic. Table 1 shows the characteristics of the sample. The sample was diverse in race/ethnicity and SES. About 39.4% of the adolescents identified as White, 38.3% as Black, 32.1% as Hispanic, and 9.4% as other races. Furthermore, based on the income-to-needs ratio (INR) using federal poverty thresholds at study entry in 2015 (www.census.gov), 18.1% of the sample were from poor households (INR < 1.0), 21.3% from low-income households (INR < 2.0), 29.9% from middle-income households (INR 2–4), and 31.7% from high-income households (INR > 4). All adolescents were in good health when they were recruited. Specifically, they were not pregnant, had no history of chronic medical or psychiatric conditions, had not been taking prescription medications for the past month, and did not have an acute infectious disease for the past 2 weeks.

Adolescents and one of their parents or guardians participated in the Time 1 visit when adolescents were in eighth grade. In the laboratory, adolescents completed surveys, interviews, a fasting antecubital blood draw, and anthropometric measurements, and their parents or guardians (85.2% mother, 12.3% father, and 2.5% others) completed questions about household demographics and family subjective SES. Two hundred and fifty-seven adolescents (92.4% retention rate; 65.0% female; $M_{age} = 15.98$ years, $SD_{age} = 0.54$ years) participated in the Time 2 visit roughly 2 years later ($M_{interval} = 2.04$ years, $SD_{interval} = 0.12$ years). In the laboratory, they completed surveys, interviews, a fasting antecubital blood draw, and anthropometric measures again. A sensitivity power analysis showed that this sample size provided 80% power to detect effect sizes as small as Cohen's $f^2 = .03$ (i.e., $R^2 = .03$) at $p = .05$.

Measures

Family Objective and Subjective SES

Family objective SES was assessed at Time 1 with family gross income, family savings, and education (i.e., highest degree obtained) reported by parents. We calculated the INR to indicate their economic need (i.e., family gross income divided by the federal poverty threshold, which varies by family size). Following prior research (Finegood et al., 2020), we created a composite score for family objective SES by standardizing and averaging INR (natural log-transformed), family savings, and education (Cronbach's $\alpha = .69$), with higher scores reflecting higher levels of family objective SES.

Family subjective SES was assessed at Time 1 with the MacArthur Scale of Subjective Social Status (Adler et al., 2000). Parents rated two items on a 10-rung ladder to indicate their perceived social standing relative to other people in their communities and in the United States (1 = *lowest standing/worst off*, 10 = *highest standing/best off*). Because participants' ratings on the two items were consistent with each other (Cronbach's $\alpha = .69$), we created a composite score of the two items to indicate family subjective SES, with higher scores reflecting higher levels of family subjective SES.[1]

Perceived Support From Friends

Adolescents' perceived support from friends (shortened as friend support henceforth) was assessed at Time 1 with an adapted version of the Harter Social Support Scale (Harter, 1985). Items began with the phrase, "Please circle the number that indicates how true you feel the following statements are about your friends." Six items were used (e.g., "Some kids have a close friend who they can talk to about things that bother them." and "Some kids have a close friend who really understands them."). Adolescents rated these items on a 4-point Likert scale (1 = *really false for me*, 4 = *really true for me*). The scale had good internal consistency (Cronbach's $\alpha = .83$). We created a composite score with higher scores reflecting higher levels of friend support. This measure captures adolescents' perceived emotional (vs. tangible) support from their friends.

[1] Adolescents' subjective appraisals of their family's SES can be another indicator of family SES. However, the current study focuses on how adolescents' low-grade inflammation is influenced by family socioeconomic circumstances they are exposed to, instead of their perceptions of the circumstances. Thus, adolescents' own subjective SES is not included. The online supplemental materials report the results of all main analyses after controlling for adolescents' subjective SES. The results did not substantially change.

Table 1
Descriptives of the Sample

Descriptives	*N* (%) or mean (*SD*)	*N* of missing data (%)
Adolescent age	13.92 (0.54)	0 (0)
Adolescent sex, female/male	176/101 (63.5%/36.5%)	0 (0)
Adolescent race, White (non-Hispanic)	109 (39.4%)	0 (0)
Adolescent race, Black (non-Hispanic)	106 (38.3%)	0 (0)
Adolescent race, other (non-Hispanic)	26 (9.4%)	0 (0)
Adolescent ethnicity, Hispanic (any race)	89 (32.1%)	0 (0)
Family INR	3.79 (4.73)	0 (0)
Family saving (thousands of dollars)	116.37 (414.89)	17 (6.1)
Parent education (highest degree obtained)	3.28 (1.27)	0 (0)
Puberty status	3.69 (0.73)	0 (0)
BMI (percentile based on age and sex)	71.32 (26.04)	0 (0)
Family objective SES	−0.01 (0.79)	0 (0)
Family subjective SES	6.01 (1.71)	0 (0)
Perceived support from friends	3.42 (0.60)	0 (0)
Inflammation composite at Time 1	0.00 (0.60)	2 (0.7)
Inflammation composite at Time 2	0.00 (0.63)	24 (8.7)
Classical monocytes at Time 1 (counts/μl)	136.65 (56.75)	2 (0.7)
Classical monocytes at Time 2 (counts/μl)	138.23 (67.84)	22 (7.9)
Nonclassical monocytes at Time 1 (counts/μl)	34.51 (17.36)	2 (0.7)
Nonclassical monocytes at Time 2 (counts/μl)	35.14 (22.82)	22 (7.9)

Note. $N = 277$. All variables were measured at Time 1, except indicators of low-grade inflammation, which were measured at both Times 1 and 2. Items for family subjective SES were rated on a 10-point scale (1 = *lowest standing/worst off*, 10 = *highest standing/best off*), and items for perceived support from friends were rated on a 4-point scale (1 = *really false for me*, 4 = *really true for me*); Parent education was coded on a 5-point scale (1 = *less than high school*, 2 = *high school diploma*, 3 = *some college*, 4 = *bachelor's degree*, 5 = *graduate degree*); Pubertal status was coded on a 5-point scale (1 = *prepubertal*, 2 = *early pubertal*, 3 = *midpubertal*, 4 = *late pubertal*, 5 = *postpubertal*). SES = socioeconomic status; INR = income-to-needs ratio; BMI = body mass index.

Inflammatory Biomarker Composite

At both visits, we assessed low-grade inflammation using six biomarkers, including IL-6, IL-8, IL-10, TNF-α, CRP, and soluble urokinase-type plasminogen activator receptor (suPAR). All of these biomarkers are commonly used, except suPAR, which is a newer biomarker of low-grade inflammation (Bourassa et al., 2021) that predicts risk for cardiovascular disease and all-cause mortality (Botha et al., 2015). Biomarkers were assayed in serum collected between 8:00 and 10:00 a.m. after an overnight fast. CRP was measured in duplicate by high-sensitivity immunoturbidimetric assay on a Roche/Hitachi cobas c502 instrument. Cytokines were measured in triplicate by four-plex immunoassay (Aldo et al., 2016) on a microfluidic platform (Simple Plex; Protein Simple). Finally, suPAR was measured in duplicate by immunoassay (Human Quantikine ELISA; R&D Systems). Intra-assay coefficients of variation ranged from 1.6% to 5.0%.

All biomarkers were log-transformed to adjust for skew in the distributions. Consistent with approaches used in previous research (Finegood et al., 2020; Miller et al., 2022), we created an inflammation composite by standardizing and averaging the log-transformed values at each visit, with higher scores reflecting higher levels of low-grade inflammation. The inflammation composite had good internal consistency at both visits (Cronbach's $\alpha = .64$ and .69 at Times 1 and 2).

Classical and Nonclassical Monocytes

At both visits, classical and nonclassical monocytes were assessed using a standardized flow cytometry protocol (Heimbeck et al., 2010). First, fasting antecubital blood was drawn into Sodium-Heparin Vacutainers (Becton-Dickinson). After red blood cells were lysed (Pharm Lyse, Becton-Dickinson), the pelleted cells were washed, blocked with normal human serum, and stained with mouse, anti-human monoclonal antibodies against CD14 (fluorescein isothiocyanate), CD16 (phycoerythrin), HLA-DR (peridinin chlorophyll protein-cyanin 5.5), and CD45 (allophycocyanin), all purchased from Becton-Dickinson. Following a 20-min incubation, the cells were washed and fixed (CytoFix/CytoPerm, Becton-Dickinson), and incubated for another 20 min. Data were acquired on a Guava 6HT2L (Millipore), with 30,000 events collected per specimen, and analyzed using FlowJo software (Tree Star Inc). Populations of classical ($CD14^+/CD16^-$) and nonclassical ($CD14^-/CD16^+$) monocytes were defined by a sequential gating procedure.

Covariates

Following prior research (Miller et al., 2022), a set of covariates were selected a priori and included in all analyses. They were adolescents' age, sex, race/ethnicity (two dummy variables indicating whether they were non-Hispanic White [reference group], Black, or non-Black participants of color), body mass index (BMI; percentile based on age and sex), and pubertal status (5-point scale [1 = *prepubertal*, 5 = *postpubertal*]; Petersen et al., 1988).

Analytic Strategies

A series of moderation analyses with multiple regressions in SPSS (Version 28) were used to examine whether adolescents' friend support moderates the associations between family objective and subjective SES and adolescents' indicators of low-grade inflammation, including

the inflammation composite and classical and nonclassical monocyte numbers. Specifically, in each analysis, we included adolescents' friend support, family objective SES, family subjective SES, and the two interaction terms of friend support with objective and subjective SES as predictors, and one indicator of inflammation as the outcome. Furthermore, we conducted three separate moderation analyses for each indicator of inflammation, with the outcome being measured: (a) at Time 1; (b) at Time 2 (2 years later); and (c) the prospective change in inflammation from Time 1 to Time 2. For analyses of change, Time 2 variables were included as the outcome and Time 1 variables were included as an additional covariate. Analyses also included the set of covariates described above. Significant interactions were plotted, and simple slopes were computed for the lower level (i.e., 1 *SD* below the mean), mean level, and higher level (i.e., 0.97 *SD* above the mean/the highest possible value) of friend support. Using the Johnson–Neyman (J–N) regions of significance analyses, we also calculated the percentage of the sample with significant associations between family SES and the outcome for each significant interaction.

We handled missing data using listwise deletion (missing data only occurred for outcome variables, see Table 1). All continuous variables were standardized prior to conducting analyses. To reduce the influence of outliers on parameter estimation while maximizing statistical power, outliers for all variables were inspected (defined as ± 3 *SD* from the sample mean)[2] and were winsorized to the next highest/lowest value.

Results

Tables 1 and 2 show the means, *SD*s, and zero-order correlations for all variables. Family objective and subjective SES were modestly correlated ($r = .46$, $p < .001$), which is consistent with previous research (Tan et al., 2020), indicating that these two constructs reflect two related but distinct indicators of family SES. Adolescents' friend support was positively correlated with family objective SES ($r = .21$, $p < .001$), but not with family subjective SES. Neither of the SES indicators was associated with measures of inflammatory activity. In addition, the inflammation composite was generally correlated with monocyte subtypes, with the associations being stronger for classical monocytes (*rs* ranged from .13 to .31) than for nonclassical monocytes (*rs* ranged from .03 to .22). The correlations between the inflammation composite and BMI (*rs* ranged from .22 to .29) were stronger than those between monocyte subtypes and BMI (*rs* ranged from .08 to .16), indicating that adipose mass is more linked to inflammatory biomarkers than to monocyte counts.

Inflammation Composite

For the cross-sectional associations with the inflammation composite at Time 1, the interaction between family objective SES and adolescents' friend support was not significant ($\beta = -.05$, 95% confidence interval, CI = [−.19, .09], $p = .460$). In contrast, the interaction between family subjective SES and friend support was significant ($\beta = .20$, 95% CI = [.07, .33], $p = .003$, see Table S1 in the online supplemental materials). Further decomposition of the significant interaction revealed that the lower family subjective SES was associated with higher levels of the inflammation composite at Time 1 only among adolescents lower in friend support (simple slopes for lower support: $\beta = -.27$, 95% CI = [−.46, −.09], $p = .003$; mean support: $\beta = -.08$, 95% CI = [−.20, .04], $p = .205$, and higher support: $\beta = .11$, 95% CI = [−.06, .28], $p = .193$; see Figure 1, Panel A). The J–N regions of significance analyses showed that for those whose friend support was lower than 0.25 *SD* below the mean (35.7% of the sample), family subjective SES was negatively associated with the inflammation composite at Time 1.

For the longitudinal associations with the inflammation composite at Time 2, the interaction between family objective SES and friend support was also not significant ($\beta = -.07$, 95% CI = [−.21, .08], $p = .373$), but the interaction between family subjective SES and friend support was significant ($\beta = .24$, 95% CI = [.10, .37], $p < .001$). The decomposition of the significant interaction revealed that family subjective SES was negatively associated with the inflammation composite at Time 2 only among those lower in friend support, and family subjective SES even became positively associated with the inflammation composite for those higher in friend support (simple slopes for lower support: $\beta = -.25$, 95% CI = [−.45, −.06], $p = .010$; mean support: $\beta = -.02$, 95% CI = [−.14, .11], $p = .786$, and higher support: $\beta = .21$, 95% CI = [.04, .38], $p = .015$; see Figure 1, Panel B). In addition, for those whose friend support was lower than 0.58 *SD* below the mean (30.0% of the sample), family subjective SES was negatively associated with the inflammation composite at Time 2. In contrast, for those whose friend support was higher than 0.70 *SD* above the mean (30.7% of the sample), family subjective SES was positively associated with the inflammation composite at Time 2.

A similar pattern was observed for prospective change in the inflammation composite during the 2 years between Times 1 and 2. Specifically, the interaction between family objective SES and friend support was not significant ($\beta = -.04$, 95% CI = [−.17, .09], $p = .500$), but the interaction between family subjective SES and friend support was significant ($\beta = .15$, 95% CI = [.02, .27], $p = .019$). The negative association between family subjective SES and change in the inflammation composite from Time 1 to Time 2 became statistically nonsignificant when adolescents perceived more friend support, and family subjective SES became positively associated with change in the inflammation composite for those higher in friend support (simple slopes of lower support: $\beta = -.13$, 95% CI = [−.30, .05], $p = .156$; mean support: $\beta = .02$, 95% CI = [−.09, .13], $p = .730$, and higher support: $\beta = .16$, 95% CI = [.01, .32], $p = .040$; see Figure 1, Panel C). In addition, for those whose friend support was lower than 2.24 *SD* below the mean (2.2% of the sample), family subjective SES was negatively associated with change in the inflammation composite. For those whose friend support was higher than 0.85 *SD* above the mean (30.7% of the sample), family subjective SES was positively associated with change in the inflammation composite.

Classical Monocytes

The results for classical monocytes were largely consistent with those for the inflammation composite. For the cross-sectional

[2] The number of outliers for each variable is: $N_{\text{Time 1}} = 2$ and $N_{\text{Time 2}} = 4$ for the inflammation composite; $N_{\text{Time 1}} = 3$ and $N_{\text{Time 2}} = 3$ for classical monocytes; $N_{\text{Time 1}} = 2$ and $N_{\text{Time 2}} = 4$ for nonclassical monocytes; $N = 2$ for family objective SES; $N = 2$ for friend support; $N = 2$ for age; $N = 1$ for pubertal status.

Table 2
Zero-Order Correlations for All Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Family objective SES	—													
2. Family subjective SES	.46***	—												
3. Perceived support from friends	.21***	.001	—											
4. Inflammation composite at Time 1[a]	−.02	−.09	−.01	—										
5. Inflammation composite at Time 2[a]	−.03	−.05	.01	.47***	—									
6. Classical Monocytes at Time 1	−.002	−.03	.06	.31***	.20**	—								
7. Classical Monocytes at Time 2	−.02	−.10	−.05	.13*	.30***	.45***	—							
8. Nonclassical monocytes at Time 1	−.01	−.06	.02	.18**	.14*	.45***	.27***	—						
9. Nonclassical monocytes at Time 2	−.05	−.03	−.08	.03	.22***	.19**	.44***	.28***	—					
10. Adolescent age	−.17**	−.16**	−.06	−.03	.04	−.10	.08	−.10	.08	—				
11. Adolescent sex	−.15*	−.07	.11	−.13*	−.04	−.10	−.08	−.06	−.02	.04	—			
12. Adolescent race, Dummy 1	−.29***	−.06	−.10	−.02	−.10	.04	−.01	.15*	.17**	.02	−.01	—		
13. Adolescent race, Dummy 2	−.22***	−.20***	−.12*	−.003	.05	−.10	.01	−.07	−.08	.11	.01	−.57***	—	
14. BMI	−.13*	−.14*	.02	.29***	.22***	.11	.16**	.08	.14*	.05	.02	.10	.03	—
15. Pubertal status	−.19**	−.11	.04	−.15*	.06	.03	.01	−.02	.04	.26***	.57***	.07	.03	.18**

Note. Sex was coded as 1 for *male* and 2 for *female*. Two dummy variables for race indicate whether participants were White, Black, or non-Black participants of color. Dummy variables 1 and 2 were coded as 0 and 0, respectively, when participants only identified as White and did not identify as any other race; Dummy variables 1 and 2 were coded as 1 and 0, respectively, when participants identified as Black regardless of what other races they identified; Dummy variables 1 and 2 were coded as 0 and 1, respectively, when participants identified as any other non-White race. Pubertal status was coded on a 5-point scale (1 = *prepubertal*, 2 = *early pubertal*, 3 = *midpubertal*, 4 = *late pubertal*, 5 = *postpubertal*). SES = socioeconomic status; BMI = body mass index.
[a] The correlations among individual inflammatory biomarkers and covariates are reported in Table S4 in the online supplemental materials.
* $p < .05$. ** $p < .01$. *** $p < .001$.

associations with classical monocyte counts at Time 1, the interaction between family objective SES and friend support was not significant (β = −.06, 95% CI = [−.21, .10], $p = .470$), but the interaction between family subjective SES and friend support was significant (β = .15, 95% CI = [.004, .29], $p = .043$, see Table S2 in the online supplemental materials). The negative association between family subjective SES and classical monocytes at Time 1 became statistically nonsignificant for adolescents higher in friend support (simple slopes of lower support: β = −.20, 95% CI = [−.40, .003], $p = .054$; mean support: β = −.05, 95% CI = [−.19, .08], $p = .447$, and higher support: β = .09, 95% CI = [−.10, .28], $p = .339$; see Figure 2, Panel A). In addition, for those whose friend support was lower than 1.08 *SD* below the mean (17.3% of the sample), family subjective SES was negatively associated with classical monocytes at Time 1.

For the longitudinal associations with counts of classical monocytes at Time 2, the interaction between family objective SES and friend support was also not significant (β = −.12, 95% CI = [−.28, .04], $p = .136$), but the interaction between family subjective SES and friend support was significant (β = .18, 95% CI = [.04, .33], $p = .013$). As expected, the negative association between family subjective SES and classical monocytes at Time 2 became statistically nonsignificant for those higher in friend support (simple slopes of lower support: β = −.28, 95% CI = [−.49, −.07], $p = .009$; mean support: β = −.09, 95% CI = [−.23, .04], $p = .170$, and higher support: β = .08, 95% CI = [−.10, .27], $p = .370$; see Figure 2, Panel B). In addition, for those whose friend support was lower than 0.28 *SD* below the mean (35.7% of the sample), family subjective SES was negatively associated with classical monocytes at Time 2.

For the prospective change in counts of classical monocytes from Time 1 to Time 2, the interaction between family objective SES and friend support was not significant (β = −.09, 95% CI [−.23, .05], $p = .202$), nor was the interaction between family subjective SES and friend support (β = .11, 95% CI = [−.03, .24], $p = .113$).

Nonclassical Monocytes

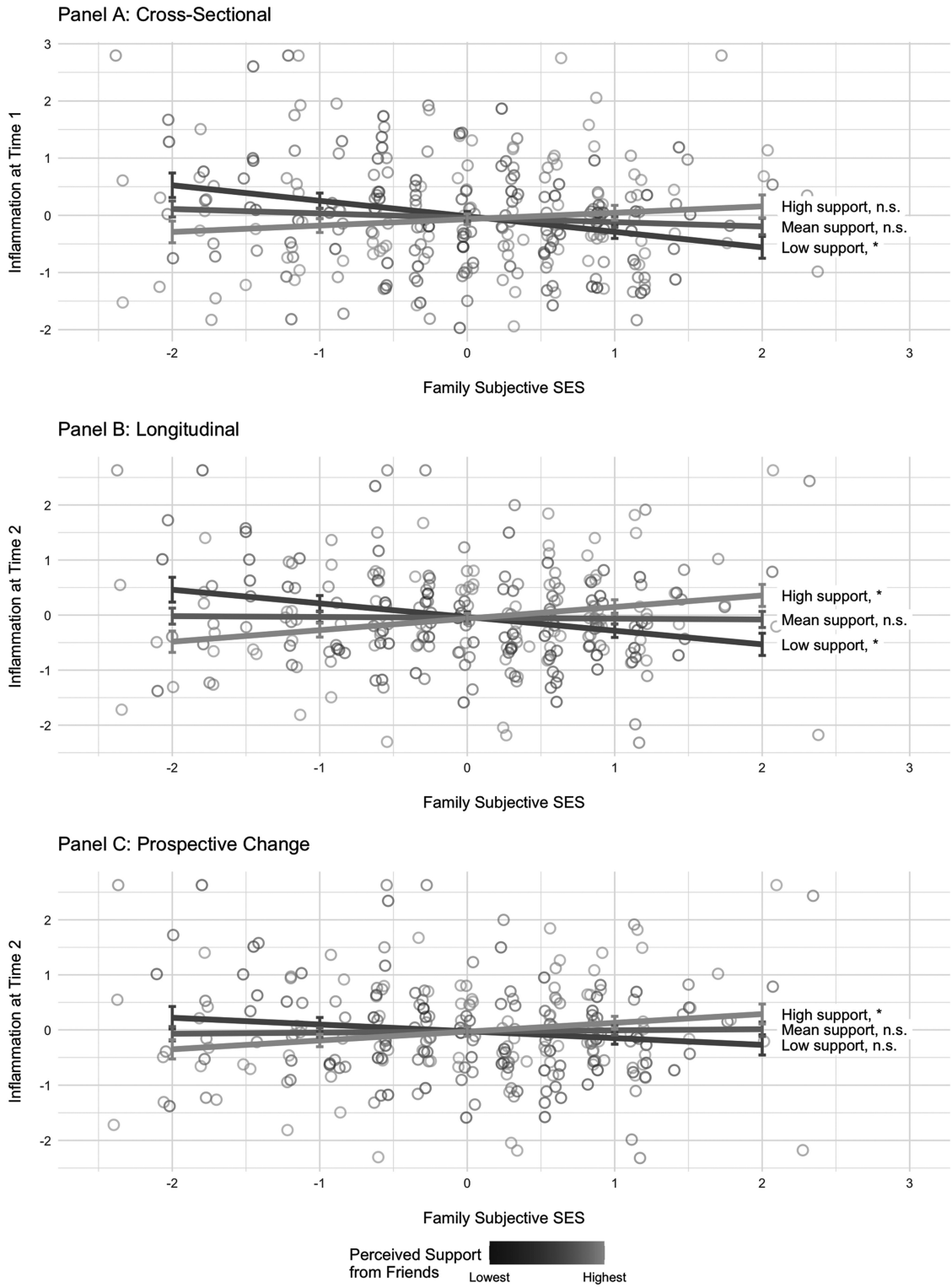
Although we hypothesized that friend support would not moderate the associations between family SES and nonclassical monocytes, one significant interaction effect emerged. For nonclassical monocytes at Time 1, the interaction between family objective SES and friend support was significant (β = −.20, 95% CI = [−.34, −.06], $p = .006$, see Table S3 in the online supplemental materials). Family objective SES was positively associated with nonclassical monocytes at Time 1 among adolescents lower, but not higher, in friend support (simple slopes of lower support: β = .31, 95% CI = [.10, .52], $p = .004$; mean support: β = .11, 95% CI = [−.05, .27], $p = .165$, and higher support: β = −.08, 95% CI = [−.30, .13], $p = .459$). In addition, for those whose friend support was lower than .25 *SD* below the mean (35.7% of the sample), family objective SES was positively associated with nonclassical monocytes at Time 1. All other interactions were not significant ($ps \geq .068$). The full results are reported in the online supplemental materials.

Sensitivity Analyses

We conducted sensitivity analyses to assess the robustness of the results. First, to assess whether the positive skewness of the

Figure 1

Family Subjective SES Predicting Inflammatory Biomarker Composite at Higher, Mean, and Lower Levels of Friend Support

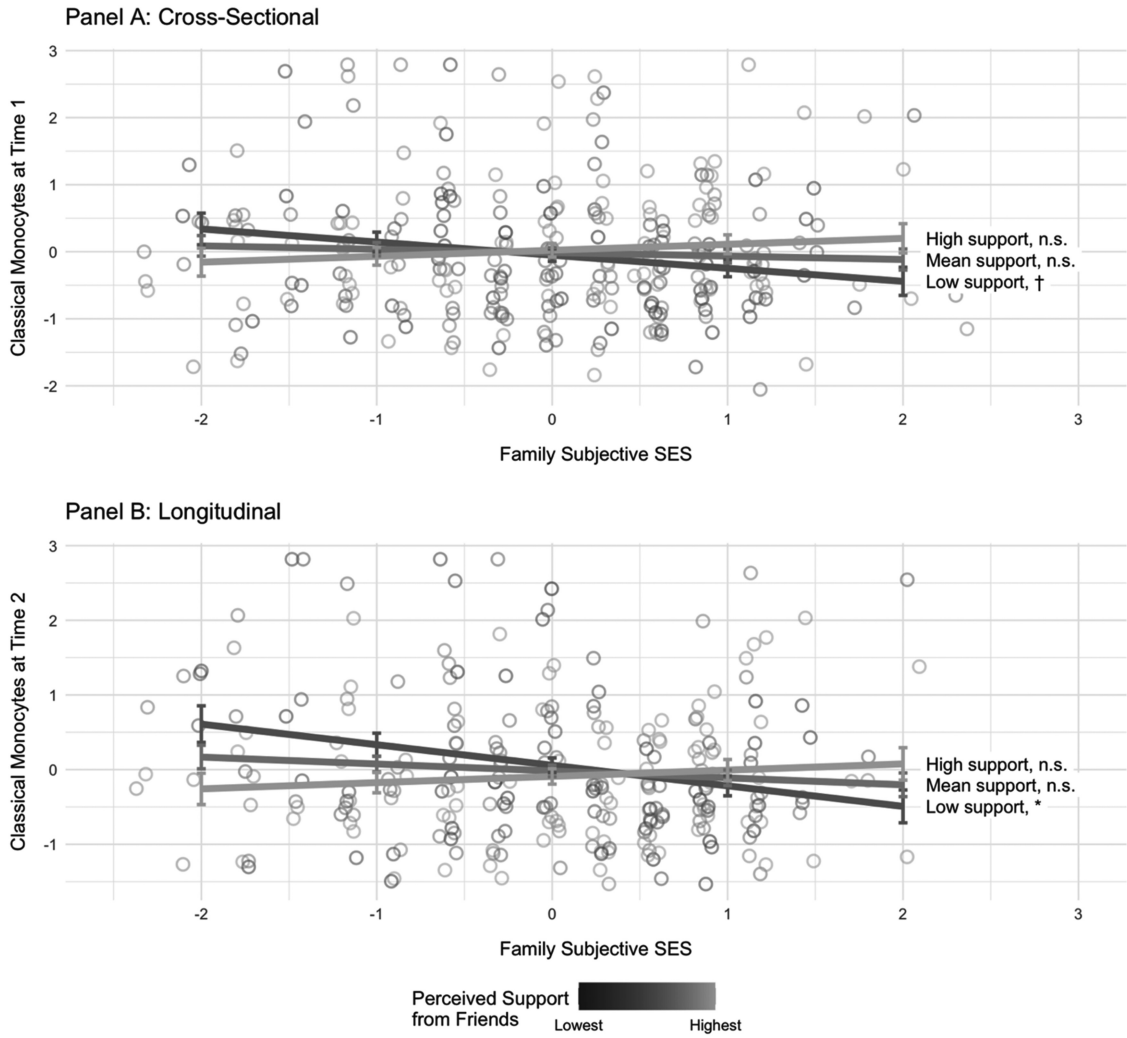


Note. Interactions are plotted at 2.82 (1 *SD* below the mean), 3.42 (at the mean level), and 4 (0.97 *SD* above the mean) on a 1–4 scale of friend support. All variables were standardized, and higher values indicate higher levels of the variables. SES = socioeconomic status.

* $p < .05$. n.s. = $p \geq .10$.

Figure 2

Family Subjective SES Predicting Counts of Classical Monocytes at Higher, Mean, and Lower Levels of Friend Support



Note. Interactions are plotted at 2.82 (1 *SD* below the mean), 3.42 (at the mean level), and 4 (0.97 *SD* above the mean) on a 1–4 scale of friend support. All variables were standardized, and higher values indicate higher levels of the variables. SES = socioeconomic status.

† $p < .10$. * $p < .05$. n.s. = $p \geq .10$.

outcome distributions influenced the results, we reran all main analyses using generalized linear models in SPSS (Version 28). A gamma error distribution and log link function were implemented to account for the positive skewness. Second, to assess the influence of overadjustment bias on the results (van Zwieten et al., 2022), we reran all main analyses without adjusting for covariates. Third, to assess the influence of the extreme values, we reran all main analyses without winsorizing the outliers. In these three sets of analyses, all interaction effects were similar in terms of point estimates, CIs, and *p* values, with the following exceptions. When running the analyses without adjusting for covariates, the interaction between family objective SES and friend support on nonclassical monocytes at Time 2 became significant ($\beta = -.14$, 95% CI = [−.28, −.01], $p = .043$), and the interaction between family subjective SES and friend support on classical monocytes at Time 1 became nonsignificant ($\beta = .13$, 95% CI = [−.02, .27], $p = .081$). Furthermore, the interaction between family subjective SES and friend support on nonclassical monocytes at Time 2 became significant in the analyses without adjusting for covariates ($\beta = .13$, 95% CI = [.01, .26], $p = .039$) and without winsorizing outliers ($\beta = .15$, 95% CI = [.002, .30], $p = .048$; see the online supplemental materials for the full results).

We also conducted additional sensitivity analyses, which were requested during the review process. First, we reran all main analyses after controlling for additional covariates, including parental support and adolescents' subjective SES. Second, we reran all main analyses for the subset of participants who self-identified as persons of color. Third, we reran all main analyses after winsorizing some additional outliers. In all three cases, the results were largely consistent with

those reported in the main analyses (see the online supplemental materials for the full results).[3]

Discussion

Individuals who grow up in low-SES families are at an increased risk of health problems throughout their childhood, adolescence, and even adulthood. Although supportive social relationships are postulated to play a protective role in preventing these health problems, the role of friend support in adolescence as a protective factor is not well understood. The current study aimed to examine whether adolescents' friend support buffers the associations between family SES and indicators of low-grade inflammation across a 2-year period in adolescence, given that low-grade inflammation is one key biological mechanism proposed to explain links between family SES and health outcomes (Miller, Chen, & Parker, 2011). The current study assessed low-grade inflammation using both inflammatory biomarkers and monocyte populations, it also measured two types of family SES indicators (i.e., family objective and subjective SES), and it examined whether friend support buffered the link between SES and low-grade inflammation in an adolescent sample.

As predicted, we observed that friend support moderated the associations between family subjective SES and indicators of low-grade inflammation in adolescents. Specifically, interaction effects were found between friend support and family subjective SES predicting the inflammation composite and classical monocyte counts across different types of temporal analyses, including cross-sectional (at the baseline), longitudinal (2 years later), and prospective change (over 2 years; only significant for the inflammation composite) analyses. These interaction effects could not be accounted for by age, sex, race/ethnicity, BMI, or pubertal status. Because we included both family objective and subjective SES and their interaction terms with friend support in each analysis, these interaction effects also could not be accounted for by the interaction between family objective SES and friend support.

The decomposition of these interactions showed a consistent pattern in which lower family subjective SES was associated with higher levels of low-grade inflammation only among adolescents lower in friend support,[4] and the associations became statistically nonsignificant or even sometimes going in the opposite direction among those higher in friend support. These findings are consistent with a scenario where friend support is a resilience factor that attenuates low-grade inflammation for adolescents with low family subjective SES. This interpretation is strengthened by the fact that similar patterns emerged across cross-sectional, 2-year longitudinal, and prospective change analyses. With that said, we acknowledge these interactions could be decomposed in another way, which would lead to the interpretation that higher friend support is inversely associated with low-grade inflammation for those with lower, but not higher, family subjective SES. In that scenario, subjective SES, rather than friend support, would be the moderator.

In contrast to family subjective SES, friend support did not moderate associations between family objective SES and indicators of low-grade inflammation. These nonsignificant results were found for both the inflammation composite and classical monocyte counts, and for cross-sectional, longitudinal, and prospective change analyses. That is, friend support only buffers the link between low-grade inflammation and family subjective, but not objective, SES, despite the fact that both objective and subjective indicators of family SES are linked to health outcomes in previous literature (Diep et al., 2019; Kim et al., 2021; Kittleson et al., 2006). One explanation for these findings could relate to the different mechanisms underlying the effects of objective versus subjective SES. Whereas family objective SES impacts both socioeconomic resources accessible to youth (e.g., safe housing, healthy food) as well as social environments youth are exposed to (e.g., parenting behaviors, parent–youth interactions; Chen & Miller, 2013), we speculate that family subjective SES primarily impacts the latter. Both socioeconomic resources and social environments within a family are associated with youth's low-grade inflammation (Miller, Brody, et al., 2014; Schmeer & Yoon, 2016). However, having emotionally supportive peer relationships may be especially effective for reducing the stress caused by negative social family environments, both because it entails a social buffer of a social (as opposed to material) stressor and because having support in the peer domain might help compensate for difficult relationships in the family domain. In contrast, adolescent friends are much less likely to be able to provide socioeconomic resources that could help buffer the material deprivation often associated with objectively low family SES. This explanation is consistent with previous research, showing that friend support buffers the link between negative parenting behavior and adolescents' externalizing behavior (Lansford et al., 2003), but it does not buffer the link between family objective SES and adolescents' academic performance (Malecki & Demaray, 2006).

As predicted, the results for classical monocytes largely converged with those for the inflammation composite. These findings align with previous findings from both animal and human research, which have shown that under chronic psychosocial stress, classical monocytes are typically recruited into circulation and are the ones that exhibit a strong pro-inflammatory phenotype that contributes to low-grade inflammation (Miller, Murphy, et al., 2014; Powell et al., 2013; Weber et al., 2017). We included nonclassical monocytes in our analyses in order to test a differential prediction that for nonclassical monocytes, there would be no buffering effect of friend support, as this subpopulation is less responsive to psychosocial stress than classical monocytes. We note that there were occasional isolated findings that emerged for nonclassical monocytes, but these were not consistent across cross-sectional, longitudinal, and prospective change analyses, and their strength varied across sensitivity analyses (see the online supplemental materials for the full results).

Another unexpected finding was that the associations between family subjective SES and the inflammation composite were positive for those higher in friend support in longitudinal and prospective

[3] For participants of color, the interactions between family objective SES and friend support on counts of both classical and nonclassical monocytes became significant in longitudinal and prospective change analyses. However, because race did not moderate any results in the main analyses, we do not detail these results here. The full results are reported and discussed in the online supplemental materials.

[4] Across all significant interactions, the J–N regions of significance analyses indicated that lower family subjective SES was associated with higher levels of low-grade inflammation among 2.2%–35.7% of the sample who had lower friend support. Despite this consistent pattern, the results regarding the percentages of the sample should be interpreted with caution because the regions of significance vary considerably across different samples due to variability in the distribution of observed values and statistical power to detect significant effects.

change analyses. Adolescents who have supportive friendships may face stress in maintaining their friendships when they live in families with high subjective SES. In such families, parents may have specific expectations regarding the types of friends their adolescents should have (see Heimer, 1997). As a result, these adolescents may experience pressure to meet these standards and a fear of parental judgment, which could lead to elevated levels of low-grade inflammation. Future research should test whether this finding replicates in other samples and then test this potential mechanism.

Implications

The current study sheds light on the role of adolescents' peer relationships in health disparities. Despite the importance of peers during adolescence (Brown & Larson, 2009; Harris, 1995; Mitic et al., 2021), there has been limited research on how adolescents' friend support can protect physical health (cf. Brody et al., 2014; Fridh et al., 2015). The current study is the first to document that adolescents' friend support buffers the association between family subjective SES and low-grade inflammation in adolescents. The clinical implications of this buffering effect are yet to be determined, given that this is a healthy and young sample, and epidemiologic studies have not yet addressed how inflammatory biomarkers in childhood relate to cardiovascular disease in adulthood. However, the effect size of family subject SES on low-grade inflammation when friend support is low is comparable to that of BMI in the current study (see Table S1 in the online supplemental materials), but perceiving high friend support eliminates this effect. Given that elevated levels of low-grade inflammation are linked to many health problems across the lifespan (Furman et al., 2019; Kiecolt-Glaser et al., 2010), the findings suggest that positive peer relationships during adolescence may have the potential to play a preventive and protective role in the health of individuals who grow up in families with low subjective SES. Thus, adolescents' peer relationships could plausibly be a target of interventions aiming to protect the health of individuals who grow up in low-SES families, especially those families in which parents have low subjective social status. Previous psychosocial interventions have mainly focused on parent-youth interactions (e.g., Miller, Brody, et al., 2014), but additional intervention components targeting adolescents' peer relationships may be effective in protecting youth's health.

The current study also emphasizes the importance of considering potentially unique effects of family objective and subjective SES on health outcomes. Previous research has predominantly focused on how family objective SES during childhood and adolescence (e.g., parents' income and education) is linked to one's health across the lifespan (see Cohen et al., 2010; Poulain et al., 2020 for reviews). As a result, theoretical frameworks explaining this link have focused on how objective family SES circumstances can shape youth's life experiences (e.g., Chen & Miller, 2013). In contrast, relatively less research exists on family subjective SES, as this research has emerged only more recently. The research that exists though does show that this new indicator uniquely predicts health outcomes beyond family objective SES (Diep et al., 2019; Kim et al., 2021), suggesting the importance of not lumping together all types of SES indicators. In addition, future theoretical work is needed that specifies the distinct versus similar mechanisms through which each of these two indicators of family SES during childhood and adolescence is linked to one's health outcomes (see Highlander & Jones, 2022 for an integrative model explaining the link between family SES and externalizing problem behavior). We propose that family objective SES impacts both socioeconomic resources and social environments, whereas family subjective SES primarily impacts the latter. Accordingly, resilience factors may also function differently for each. Thus, the current study provides one preliminary example of this, but future research is needed to extend our understanding of how family objective versus subjective SES during childhood and adolescence might impact one's health differentially, and how moderating factors might operate differentially for these two indicators of family SES.

Limitations and Future Research

First, the longitudinal design of the current study allowed us to test the hypotheses using different types of temporal analyses, including cross-sectional, longitudinal, and prospective change analyses, the results of which support the plausibility of causal associations. However, this study does not allow us to fully elucidate the causal direction of the hypothesized associations because of its correlational nature. Future research might use a randomized controlled intervention design to test the causal effect of friend support on the link between family SES and low-grade inflammation. Second, only one of the adolescents' parents or guardians (85.2% mother, 12.3% father, and 2.5% others) participated in the study and reported family objective and subjective SES. Although this is a common practice in the clinical and developmental literature (Highlander & Jones, 2022), future research should measure family SES, especially family subjective SES, from a broader set of household members to capture a more comprehensive picture of the family environment for youth. Third, the measure of friend support in the current study only captured perceived emotional (vs. tangible) support from friends. Although we propose that adolescents may be less likely to perceive or receive tangible support, especially socioeconomic resources, from their friends, future research can measure this type of friend support and examine whether it buffers the link between family objective SES and low-grade inflammation. Fourth, the current study only measured concurrent family SES for adolescents. Although family SES is relatively stable (see Goodman et al., 2015), future research can examine whether friend support during adolescence buffers the detrimental effects of low family SES during childhood. Fifth, the sample size of the current study might provide insufficient statistical power to detect small effect sizes for prospective change analyses; larger samples (e.g., $N = 530$ to 1,570 is required to detect Cohen's f^2 between .005 to .015 with .80 power) can be used in future research to gauge prospective associations. Studies with larger samples can also examine whether there are gender or race differences in the associations observed in the current study.

In addition, future research can examine whether the results of the current study hold when using other ways to measure friend support besides self-report questionnaires (e.g., observational or teacher-report measures). Future research can also broaden the age range of participants to better understand the function of friendships at different developmental stages.

Conclusion

Early life socioeconomic disadvantage is linked to poor health outcomes across the lifespan. The current study shows that adolescents'

friend support buffers the link between family subjective, but not objective, SES and low-grade inflammation in adolescence. These findings suggest that positive peer relationships may help to protect the physical health of adolescents who live in families with low subjective SES.

Resumen

Objetivo: Las personas que crecen en familias de bajo nivel socioeconómico (NSE) tienen un mayor riesgo de sufrir problemas de salud a lo largo de su vida. Aunque se postula que las relaciones sociales de apoyo son un factor protector para la salud de estos individuos, no se comprende bien el papel del apoyo de los amigos en la adolescencia. Dado que la inflamación de bajo grado es un mecanismo biológico clave propuesto para explicar los vínculos entre el NSE familiar y los resultados de salud, examinamos si el apoyo de los amigos en adolescentes amortigua la asociación entre el NSE familiar y la inflamación de bajo grado entre los adolescentes. ***Método:*** 277 díadas de adolescentes (63.5% mujeres; 39.4% blancos, 38.3% negros y 32.1% hispanos; edad media = 13.92 años) y uno de sus padres participaron en este estudio longitudinal (dos oleadas con aproximadamente dos años de diferencia). Los padres informaron el NSE objetivo de la familia (es decir, ingresos, ahorros y educación) y el NSE subjetivo de la familia (es decir, el estatus social subjetivo). Los adolescentes informaron haber percibido apoyo de sus amigos. En ambas visitas se extrajo sangre antecubital de los adolescentes mientras en ayuna. La actividad inflamatoria de bajo grado estuvo representada por una combinación de biomarcadores inflamatorios y números de monocitos clásicos. ***Resultados:*** El apoyo de los amigos de los adolescentes moderó las asociaciones del NSE subjetivo familiar tanto con el compuesto de inflamación como con los recuentos de monocitos clásicos en los análisis de cambios transversales, longitudinales y prospectivos (solo significativos para el compuesto de inflamación). Específicamente, un NSE subjetivo familiar más bajo se asoció con niveles más altos de inflamación de bajo grado solo entre los adolescentes con un apoyo de amigos más bajo, pero no más alto. No se observó moderación para el NSE objetivo. ***Conclusión:*** Las relaciones de apoyo entre pares amortiguan el vínculo entre el NSE familiar subjetivo, pero no objetivo, y la inflamación de bajo grado en la adolescencia.

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