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Socioeconomic Status and Social Support: Social Support Reduces Inflammatory Reactivity for Individuals Whose Early-Life Socioeconomic Status Was Low

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Abstract

Low socioeconomic status (SES) during childhood confers risk for adverse health in adulthood. Accumulating evidence suggests that this may be due, in part, to the association between lower childhood SES and higher levels of pro-inflammatory cytokines. Drawing from literature showing that low childhood SES predicts exaggerated physiological reactivity to stressors and that lower SES is associated with a more communal, socially attuned orientation, we hypothesized that inflammatory reactivity would be more greatly affected by cues of social support among individuals whose childhood SES was low than among those whose childhood SES was high. In two studies, we found that individuals with lower subjective childhood SES exhibited greater reductions in pro-inflammatory cytokine reactivity to a stressor in the presence of a supportive figure (relative to conditions with an unsupportive or neutral figure). These effects were independent of current SES. This work helps illuminate SES-based differences in inflammatory reactivity to stressors, particularly among individuals whose childhood SES was low.

Keywords

early-life socioeconomic status, subjective socioeconomic status, social support, interleukin-6, social evaluation, inflammatory reactivity

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Socioeconomic status (SES) is a consistently powerful predictor of health and disease in human populations. This relationship has been documented for a striking variety of mental- and physical-health outcomes (e.g., Adler & Ostrove, 1999; Clark, DesMeules, Luo, Duncan, & Wielgosz, 2009). A growing body of research provides evidence that these SES-based health disparities take root early in life. Early-life socioeconomic disadvantage predicts increased risk for illness in adulthood, even after socioeconomic factors in adulthood are accounted for (Shonkoff, Boyce, & McEwen, 2009). These enduring effects of early-life experiences provide support for life-course theories of health disparities, which emphasize the critical role of early-life experiences in influencing health trajectories and affecting risk for illness in adulthood (Matthews & Gallo, 2010; Pollitt, Rose, & Kaufman, 2005).

In particular, inflammation and dysregulation of inflammatory pathways within the immune system are proposed to be key mechanisms through which experiences in childhood may affect health across the life span. In this view, early experience is biologically embedded (Hertzman, 1999; Miller, Chen, & Parker, 2011), such that early-life adversity associated with low-SES environments gives rise to behavioral and biological tendencies that affect health and vulnerability to disease in adulthood. As predicted by this model, low childhood SES is associated

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with an immune-system phenotype characterized by elevated levels of pro-inflammatory cytokines (Carroll, Cohen, & Marsland, 2011; Saxton, John-Henderson, Reid, & Francis, 2011). Less clear, however, is whether low childhood SES is associated with exaggerated inflammatory reactivity to acute stressors. Given evidence that early-life environments program biological reactivity to later stressors, we hypothesized that individuals whose childhood SES was low would show markedly greater increases in pro-inflammatory cytokines in response to a stressor relative to individuals whose childhood SES was high.

Ultimately, an individual's health outcomes depend on both his or her sensitivity to the context and the characteristics of the surrounding environment (Ellis & Boyce, 2005). Given that low childhood SES is associated with a biological sensitivity to stressors, we manipulated an important feature of the surrounding environment—the availability of social support—and then assessed inflammatory reactivity. We hypothesized that cues associated with social support (e.g., nodding, eye contact) would be particularly effective in reducing inflammatory reactivity to stressors among individuals whose childhood SES was low. We drew from emerging theories of social class, which suggest that low social class, compared with high social class, is associated with a more socially attuned and communal orientation (e.g., Kraus, Piff, Mendoza-Denton, Rheinschmidt, & Keltner, 2012). Recent evidence demonstrates that lower social class predicts more attentiveness to other individuals (Kraus, Côté, & Keltner, 2010) and a more communal view of making choices (Stephens, Fryberg, & Markus, 2011).

On the basis of this work, we predicted that in a stressful context, the inflammatory responses of low-childhood-SES individuals would be more greatly affected by the presence or absence of social support than the responses of high-childhood-SES individuals. Specifically, we hypothesized that participants whose childhood SES was low would exhibit significantly higher poststressor levels of pro-inflammatory cytokines in the absence of social support, compared with participants whose childhood SES was high, but that this difference would be eliminated in the presence of social support. Given the large body of research documenting relationships among an individual's current SES, health, and physiological responses (e.g., Adler et al., 1994; Adler & Newman, 2002; Derry et al., 2013), we tested whether observed relationships among social support, childhood SES, and inflammatory responses were independent of current SES, and whether the relationships among social support, current SES, and inflammatory responses showed a similar pattern.

Research suggests that subjective measures of SES predict important health outcomes independently of objective measures of SES (e.g., Cohen et al., 2008; Singh-Manoux, Marmot, & Adler, 2005). Subjective measures may allow individuals to cognitively average a broad array of socioeconomic indicators, only some of which are commonly assessed using traditional objective measures. These subjective measures may capture components of social rank (e.g., relative status) that are only loosely tied to objective measures of SES. With regard to early-life socioeconomic factors, subjective measures may be particularly informative because individuals are less likely to remember specific details about socioeconomic factors from their early childhood. Recent research has found that subjective measures of childhood SES are significant predictors of biobehavioral responses to threat (Gianaros et al., 2008; Yanagisawa et al., 2013).

The Present Research

We tested our hypothesis across two contexts in which we manipulated cues of social support. In Study 1, participants discussed a negative emotional experience from their past with a confederate, who was trained to be either supportive and engaged or unsupportive and disengaged. In Study 2, participants took part in the Trier Social Stress Test (TSST), an explicitly stress-evoking paradigm that utilizes social evaluation as a known and reliable elicitor of inflammatory responses (Dickerson, Gable, Irwin, Aziz, & Kemeny, 2009). Again, evaluators were trained to be either supportive or unsupportive, or were not present (control condition).

In both studies we measured baseline and poststressor levels of the inflammatory marker interleukin-6 (IL-6). We chose poststressor IL-6 as our outcome on the basis of a meta-analysis on the effects of acute psychological stress on inflammatory markers, which revealed particularly robust effects for IL-6 (Steptoe, Hamer, & Chida, 2007). We measured levels of IL-6 in oral mucosal transudate (OMT), a filtrate of blood plasma. Although not a surrogate for systemic levels of inflammation in plasma, levels of inflammatory markers in OMT are related to measures of SES (e.g., Saxton et al., 2011) and are affected by acute stressors (Chiang, Eisenberger, Seeman, & Taylor, 2012; Dickerson et al., 2009; John-Henderson, Rheinschmidt, & Mendoza-Denton, 2014; Slavich, Way, Eisenberger, & Taylor, 2010).

Study 1

Method

Participants. Sixty-three undergraduate students (42 females, 19 males, 2 whose gender was unreported) participated in this study and received partial course credit. We aimed to collect data from roughly 60 participants, with 30 participants in each condition, which would fall within the norm of studies examining the effects of acute

stressors on inflammatory reactivity (e.g., Dickerson et al., 2009; John-Henderson, Rheinschmidt, Mendoza-Denton, & Francis, 2013). Participants were 19.34 years old on average (range = 18–23, *SD* = 1.25). Sixty-three percent of participants identified themselves as Asian American, 21% as Caucasian, 7% as Latino, 5% as African American, 3% as Middle Eastern, and 1% as "other." Participants were randomly assigned to the supportive and unsupportive conditions.

Procedure. We contacted participants the evening before data collection to remind them to refrain from eating or drinking anything (with the exception of water) for 1 hr prior to the lab session. When participants arrived at the lab, they provided baseline samples of OMT. They were then introduced to their partner, one of three female confederates, with whom they would interact for the next two tasks. The first task was a fast-friends task (Aron, Melinat, Aron, Vaollone, & Bator, 1997; Page-Gould, Mendoza-Denton, & Tropp, 2008); the participant and confederate, who was blind to experimental condition, conducted a conversation using a series of note cards with questions designed to facilitate rapport building. To standardize the experience for all participants, the confederate responded to questions with previously scripted answers. After 3 min, the participant, and ostensibly the confederate, went to separate rooms to complete a set of surveys on their initial impressions of one another.

For the second task, participants were told that they would be randomly selected to discuss a positive or negative experience with their partner. However, so that the interaction would be stressful, all participants were told to recount a negative experience. They read the instructions for this task and then were asked to briefly describe the negative experience they planned to share. Next, the experimenter reminded them of the instructions and told them that they would share their experience first and then would be asked to listen to their partner's experience. Although the confederate never shared an experience, these instructions were given to decrease suspicion. Participants were instructed to speak for the full 3 min allotted. During the subsequent interaction with the confederate, if a participant stopped before this time, the confederate reminded him or her of the instructions, and if a participant notified the experimenter that he or she was done prematurely, the experimenter asked the participant to talk for the full duration of time.

The confederates were trained how to act supportive and engaged or unsupportive and disengaged during this interaction. They were trained by the experimenter, practiced with a mock participant, received feedback from the experimenter regarding their behaviors during the mock interaction, and were given additional feedback during their first three sessions of the study, to help them create standardized supportive and unsupportive responses.

Prior to the interaction, the participant was given a sheet of paper with specific prompts to help aid the discussion (e.g., "discuss how you felt during the experience"). The confederate was also given a sheet of paper with a list; although it looked like it was a list of the same prompts participants had been given, in reality it was a list of behaviors the confederate had to enact during the interaction (i.e., behaviors the confederate had been taught during training). The list of behaviors in the supportive condition included maintaining eye contact with the participant as long as it was socially acceptable, nodding when the participant spoke, asking the participant one question to show that the confederate was listening and engaged, making at least two validating comments (e.g., "I'm so sorry,"), leaning forward, and making noises or facial expressions indicating compassion (these were taught to the confederates during training). The list of behaviors in the unsupportive condition included looking away from the participant every few seconds (e.g., looking at the paper or a watch), abstaining from head nodding, refraining from asking the participant any questions, shifting weight and fidgeting, leaning backward, and making noises and facial expressions indicating distress.

After the interaction, the participant, and ostensibly the confederate, went to separate rooms to fill out surveys. Items assessing the participant's perceptions of the confederate were embedded among scales assessing other variables of interest. Twenty-five minutes after the beginning of the second social interaction, the experimenter returned and obtained a second OMT sample, so that poststressor levels of IL-6 could be assayed.

Self-report measures

Subjective measures of SES. Participants were asked to rate their family's social class when they were children. They were asked to base this rating on the family's income, education, and occupational prestige, using a scale ranging from 1 (*lower class*) to 5 (*upper class; M* = 2.73, *SD* = 1.08; John-Henderson et al., 2013). Similarly, participants were asked to indicate their perception of their current social class on a scale ranging from 1 (*lower class*) to 5 (*upper class; M* = 3.15, *SD* = 1.04; John-Henderson et al., 2013; Stellar, Manzo, Kraus, & Keltner, 2012).

Objective measures of SES. Participants were asked to report whether, at the time they were in kindergarten, their parents owned their home (Saxton et al., 2011). Fifty-five percent of our sample reported that their parents were homeowners. This objective measure of childhood SES was significantly related to our subjective measure

of childhood SES (r = .43, p = .001). Participants also reported the annual household income of their parents over the past year, on a scale from 1 (*US$20,000 and below*) to 6 (*US$110,000 and above*; M = 4.27, SD = 1.90; Kraus, Adler, & Chen, 2013; Mendoza-Denton, Downey, Purdie, Davis, & Pietrzak, 2002; Stellar et al., 2012). This objective measure of current SES was significantly related to our subjective measure of current social class (r = .73, p = .001).

Subjective emotions. Participants indicated the extent to which they felt "annoyed," "sad," "anxious," "ashamed," "relaxed," "failed," "self-conscious," "afraid," "happy," "angry," and "embarrassed" after the second interaction. The rating scale ranged from 1 (*not at all*) to 10 (*as much as I've ever felt*). Using the same scale, they also indicated how much they felt the following emotions toward their partner after the interaction: annoyed, surprised, angry, upset, warm, and compassionate. One participant did not respond to the questions measuring emotions felt toward the partner and was omitted from all analyses that included these emotions.

Inflammation measures. To assess baseline and poststressor IL-6 levels in OMT, we used an OraSure collection device (Epitope, Beaverton, OR), which was placed between the lower cheek and gum for 2 min. The samples were frozen and stored at −80 °C. IL-6 concentrations were determined by an enzyme-linked immunosorbent assay (ELISA) using commercially available kits (R&D Systems, Minneapolis, MN). The inter- and intra-assay coefficients of variation were less than 9% and 4.5%, respectively. We examined poststressor IL-6 levels (M = 1.36 pg/ml, SD = 1.44) controlling for baseline levels (M = 0.62 pg/ml, SD = 0.69). To adjust for nonnormal distributions, we applied a log-transformation to these variables. We report analyses excluding 1 participant whose baseline IL-6 level was more than 3 standard deviations above the mean. Our results remained the same regardless of whether this outlier was included.

Covariates. We controlled for gender, race, smoking status (current smoker or nonsmoker), and baseline levels of IL-6 in our analyses predicting poststressor IL-6 levels.[1]

Results

A one-way analysis of variance confirmed that which confederate participated in the interaction did not have a significant effect on poststressor IL-6 levels, $F(3, 50)$ = 0.10, p = .96. We also wanted to ensure that neither SES, condition, nor their interaction was associated with the severity of the experience shared with the confederate. Analyses of experience severity, as rated by coders on a scale from 1 (*least severe*) to 5 (*most severe*), revealed no significant main effect or interaction, Fs < 0.87, ps > .18.

Manipulation checks. To examine the effect of the experimental manipulation on participants' affect, we created composite scores for participants' reported negative emotions and positive emotions, as well as the negative emotions and the positive emotions they felt toward their partner after the interaction (αs ≥ .70). Participants in the unsupportive condition reported feeling significantly more negative affect (M = 3.37, SD = 2.06) compared with those in the supportive condition (M = 2.18, SD = 1.10), $t(62)$ = 3.62, p = .001, and significantly less positive affect (unsupportive: M = 2.90, SD = 1.75; supportive: M = 3.90, SD = 1.88), $t(62)$ = 2.19, p = .03. Furthermore, participants in the unsupportive condition reported feeling significantly more negative emotion toward their partner (M = 3.23, SD = 2.06) compared with those in the supportive condition (M = 1.30, SD = 0.71), $t(62)$ = 5.08, p < .001, and significantly less positive emotion toward their partner (unsupportive: M = 2.37, SD = 1.81; supportive: M = 3.98, SD = 2.07), $t(62)$ = 3.31, p = .002. These findings confirmed that the manipulation affected participants' emotional responses to the stressor.

Social support, SES, and inflammation. We tested for support of our main hypothesis—a significant interaction between early-life subjective SES and social-support condition in predicting poststressor IL-6 levels. We entered early-life subjective SES, current subjective SES, social-support condition, gender, race (Caucasian or other), smoking status, baseline IL-6 level, and a term for the interaction between early-life subjective SES and social-support condition into a regression predicting poststressor IL-6 levels. We entered early subjective SES and current subjective SES into our regression at the same time because we wanted to understand the unique contribution of each to IL-6 reactivity. The analysis revealed a marginally significant main effect of early subjective SES, β = −0.20, $t(50)$ = −1.84, p = .07, and a similar marginally significant main effect of social-support condition, β = −0.17, $t(50)$ = −1.79, p = .08. These main effects were qualified by a significant interaction between social-support condition and early-life subjective SES, β = 0.20, $t(50)$ = 2.10, p = .04 (see Fig. 1).[2] A simple-slopes analysis revealed that early-life subjective SES negatively predicted poststressor IL-6 level in the unsupportive condition, β = −0.22, $t(50)$ = 1.83, p = .07, but not in the supportive condition, β = 0.004, $t(50)$ = 0.03, p = .98. In addition, participants whose early-life subjective SES was low had significantly greater poststressor IL-6 levels in the unsupportive condition than in the supportive condition, β = −0.40, $t(50)$ = 2.72, p < .001, whereas those

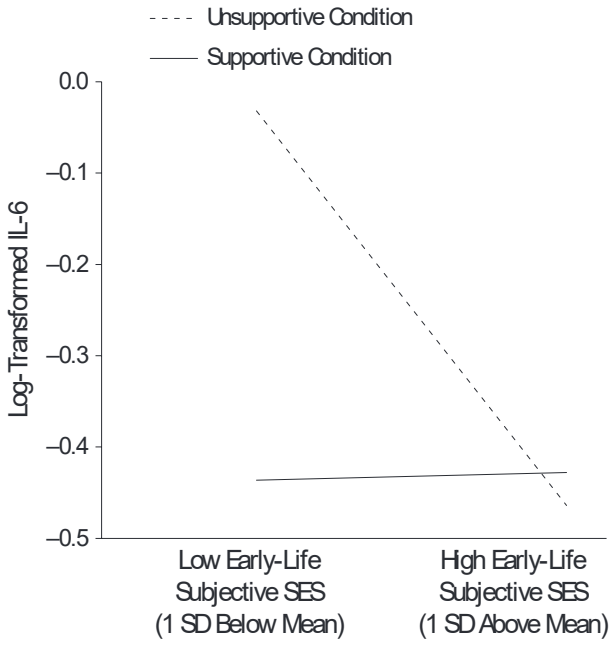

Fig. 1. Results from Study 1: poststressor level of interleukin-6 (IL-6) as a function of early-life subjective socioeconomic status (SES) and social-support condition. Results are from a linear regression analysis controlling for baseline level of IL-6, race, gender, current subjective SES, and cigarette use.

whose early-life subjective SES was high showed no difference in poststressor IL-6 levels between the conditions, $\beta = 0.04$, $t(50) = 0.24$, $p = .80$.

In a separate regression, we tested for evidence of a similar interaction between current subjective SES and social-support condition in predicting poststressor levels of IL-6. There was no main effect for current subjective SES, $\beta = -0.04$, $t(50) = -0.33$, $p = .74$, and the effect of condition was marginal, $\beta = -0.17$, $t(50) = -1.74$, $p = .09$. Further, the coefficient for the interaction term (Current Subjective SES × Condition) was not significant, $\beta = 0.01$, $t(50) = 0.09$, $p = .93$.

We also assessed whether objective measures of early-life and current SES interacted with social-support condition to predict IL-6 reactivity. The interaction between early-life objective SES and social-support condition was not statistically significant, $\beta = -0.21$, $t(50) = -1.13$, $p = .27$, nor was the interaction between current objective SES and social-support condition, $\beta = 0.15$, $t(50) = 1.50$, $p = .14$.

Overall, in Study 1, we found support for our main hypothesis that early-life subjective SES is an important predictor of inflammatory reactivity to stressful contexts, but only when no social support is provided. When social support was provided, early-life subjective SES was no longer a significant predictor of poststressor IL-6 level. Participants whose early-life subjective SES was low exhibited significantly lower poststressor IL-6 levels in the presence of social support than in its absence, whereas participants whose early-life subjective SES was high showed no difference between conditions. We did not find evidence that current subjective SES or objective measures of SES interacted with our social-support manipulation in a similar manner to predict variance in poststressor IL-6.

Study 2

In our second study, we tested whether we could replicate the interactive pattern we identified in Study 1. We used a well-validated social evaluative task, the TSST, a known elicitor of psychological and physiological stress responses. In addition to supportive and unsupportive conditions, we included a control condition, which allowed us to compare IL-6 reactivity in the presence and absence of social support.

Method

Participants. Ninety-seven undergraduate students (58 females, 39 males) participated for partial course credit. In keeping with the reasoning for Study 1, and in order to have approximately 30 participants in each of the three conditions, we aimed to collect data from roughly 90 participants. Participants were 19.56 years old on average (range = 18–24, *SD* = 1.25). Forty-four percent of participants identified themselves as Asian American, 30% as Caucasian, 10% as Latino, 3% as Middle Eastern, 7% as African American, 1% as Native American, and 5% as "other."

Procedure. As in Study 1, we contacted participants the evening before data collection to remind them to refrain from eating or drinking anything (with the exception of water) for 1 hr prior to the lab session. Participants provided baseline samples of OMT and completed measures of current and early SES, along with covariate measures, before the TSST. The TSST consisted of three components. First, each participant had 5 min to prepare a speech focusing on the qualities that made him or her a desirable candidate for a position as lab manager. The responsibilities and duties of the lab manager were described by the experimenter. Second, after the preparation period, the participant delivered a 5-min speech in one of three social-support conditions. Third, following the speech, the participant performed an oral task involving difficult mathematical operations for 5 min.

In all conditions, the participant gave the speech and performed the math task facing a video camera. In the control condition, these tasks were performed in the presence of the experimenter, who sat off to the side, out of direct view of the participant. In the unsupportive and supportive conditions, two confederates, ostensibly

serving as evaluators, watched the participant in full view. The confederates, one male and one female, gave nonverbal indications of frustration in the unsupportive condition and nonverbal indications of support in the supportive condition. Six confederates (three male, three female) had attended training sessions (like those described for Study 1) to learn standardized supportive and unsupportive behaviors. Each confederate was given a piece of paper, ostensibly for the purpose of taking notes during the participant's speech. In reality, the paper listed the behaviors (taught during training) that the confederate had to enact or refrain from enacting. The list in the supportive condition included smiling, nodding (especially when the participant made a good point), making at least one validating noise (e.g., "uh huh"), leaning forward, refraining from crossing arms, writing notes when the participant made a good point, looking at the other evaluator approvingly, and making compassionate, understanding facial expressions during the math task. The list in the unsupportive condition included keeping a neutral facial expression without smiling, refraining from nodding, sighing once to show exasperation, pinching the eyebrows together so as to appear skeptical a few times, writing notes while looking judgmental when the participant made a bad point, looking at the other evaluator disapprovingly, and looking around as if bored or distracted; in addition, one confederate leaned backward once, and the other crossed arms (who enacted each behavior was predetermined).

Twenty-five minutes after beginning the TSST, participants completed measures of the support they felt that they had received from their evaluators, provided a second sample of OMT so that poststressor levels of IL-6 could be assayed, and were debriefed.

Self-report measures

SES. We used the same subjective and objective measures of early-life SES described for Study 1 (early-life subjective SES: $M = 2.99$, $SD = 1.06$; 67% of parents were homeowners). As in Study 1, these measures were significantly related to one another ($r = .30$, $p = .003$). We also used the same subjective and objective measures of current SES as in Study 1 (subjective SES: $M = 3.15$, $SD = 0.98$; objective SES: $M = 4.61$, $SD = 1.72$). Also as in Study 1, these measures were significantly related to one another ($r = .65$, $p = .001$). Both measures of current SES were significantly related to the objective and subjective measures of early-life SES (rs $\geq .32$, ps $\leq .001$).

Perceived support from the evaluators. Participants rated how supportive they thought the evaluators were, on a scale from 1 (*unsupportive*) to 7 (*supportive*; $M = 3.11$, $SD = 1.61$).

Inflammation measures. The procedure for collecting samples and assaying IL-6 levels was the same as described for Study 1. The inter- and intra-assay coefficients of variation were less than 9% and 6.3%, respectively. Again, baseline ($M = 0.98$ pg/ml, $SD = 0.54$) and poststressor ($M = 1.80$ pg/ml, $SD = 0.98$) levels of IL-6 were nonnormally distributed, and therefore we applied a log-transformation. In the reported analyses, we excluded 2 participants who were classified as outliers because their baseline levels of IL-6 were 3 standard deviations above the mean. Our results remained the same when we included these outliers.

Covariates. As in Study 1, we controlled for gender, race, smoking status (current smoker or nonsmoker), and baseline levels of IL-6 in our analyses predicting poststressor IL-6 levels.[3]

Results

As expected, participants perceived the evaluators as significantly more supportive in the supportive condition ($M = 3.90$, $SD = 1.54$) than in the unsupportive condition ($M = 1.91$, $SD = 0.75$), $t(51) = -5.62$, $p < .001$. We did not test for an effect of the identity of the evaluators in this study because the evaluators were randomly paired (one male and one female), so there were nine possible combinations of evaluators, which would render the sample for each combination too small for a meaningful significance test.

Once again, we tested for support of our primary hypothesis that early-life subjective SES would interact with social-support condition to predict poststressor IL-6. We entered social-support conditions (dummy-coded), early-life and current subjective SES, the covariates, and terms for the interactions between early-life subjective SES and the social-support conditions into a regression model predicting poststressor IL-6. Early-life subjective SES was not a significant predictor of poststressor IL-6 level, $\beta = 0.003$, $t(81) = 0.02$, $p = .98$. However, there was an effect of social-support condition for both of our dummy-coded condition variables, βs > 0.31, $t(81)$s ≥ 3.26, ps $\leq .002$. The interaction term for the comparison of the supportive and unsupportive conditions was significant, $\beta = -0.27$, $t(81) = -2.51$, $p = .01$, as was the interaction term for the comparison of the supportive and control conditions, $\beta = -0.31$, $t(81) = -2.58$, $p = .01$.[4] Figure 2 shows poststressor level of IL-6 as a function of early-life SES in all three conditions.

Simple-slopes analyses revealed a significant negative relationship between early-life subjective SES and poststressor IL-6 level in the unsupportive condition, $\beta = -0.30$, $t(81) = 3.30$, $p = .001$, and the control condition, $\beta = -0.28$, $t(81) = 2.84$, $p = .006$, but not in the supportive condition, $\beta = 0.001$, $t(81) = 0.01$, $p = .99$. Participants whose early-life subjective SES was low had significantly lower poststressor IL-6 levels in the supportive condition than in the unsupportive condition, $\beta = 0.73$, $t(81) = 5.89$, $p < .001$, and the control condition, $\beta =$

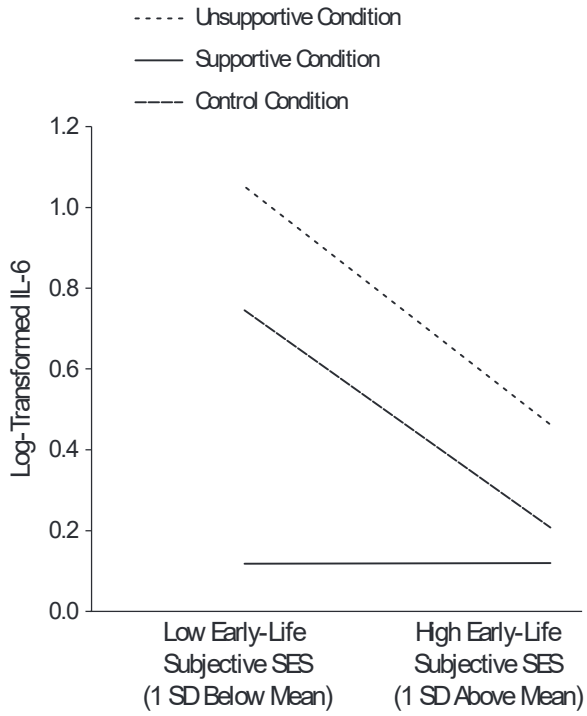

Fig. 2. Results from Study 2: poststressor level of interleukin-6 (IL-6) as a function of early-life subjective socioeconomic status (SES) and social-support condition. Results are from a linear regression analysis controlling for baseline level of IL-6, race, gender, current subjective SES, and cigarette use.

0.63, $t(81) = 4.32$, $p < .001$. Those whose early-life subjective SES was high also had significantly lower poststressor IL-6 levels in the supportive condition than in the unsupportive condition, $\beta = 0.34$, $t(81) = 1.99$, $p = .05$, but not the control condition, $\beta = 0.09$, $t(81) = 0.55$, $p = .58$.

Again, we tested whether there were similar interactions between current subjective SES and social-support condition in predicting poststressor levels of IL-6. We used the same regression equation except that the interaction terms were made with current subjective SES, rather than early-life subjective SES. There was no main effect of current subjective SES, $\beta = 0.21$, $t(81) = 1.32$, $p = .19$. Both of our dummy-coded condition variables yielded significant main effects, βs > 0.32, $t(81)$s ≥ 3.32, ps $\leq .001$. There were marginally significant interaction terms for the comparison of the unsupportive and supportive conditions, $\beta = -0.20$, $t(81) = -1.90$, $p = .06$, and for the comparison of the supportive and control conditions, $\beta = -0.21$, $t(81) = -1.81$, $p = .07$. Simple-slopes analyses did not reveal a significant relationship between current subjective SES and poststressor IL-6 level in any of the conditions, βs ≤ 0.11, $t(81)$s ≤ 1.35, ps $\geq .18$. Participants with low current subjective SES did have significantly lower poststressor IL-6 levels in the supportive condition than in the unsupportive condition, $\beta = 0.48$, $t(81) = 5.33$, $p < .001$, and the control condition, $\beta = 0.56$, $t(81) = 3.80$, $p < .001$. Those with high current subjective SES also had significantly lower poststressor IL-6 levels in the supportive condition than in the unsupportive condition, $\beta = 0.42$, $t(81) = 2.36$, $p = .02$, but not the control condition, $\beta = 0.18$, $t(81) = 1.11$, $p = .27$.

Given that our results suggested that both current subjective SES and early-life subjective SES interacted with social-support condition to predict variance in poststressor IL-6 levels, we entered interaction terms for both current and early-life subjective SES into a simultaneous regression model with the previously used covariates (John-Henderson et al., 2013). The interaction term for early-life subjective SES and the comparison of the unsupportive and supportive conditions remained significant, $\beta = -0.26$, $t(79) = -2.02$, $p = .05$, and the interaction term for early-life subjective SES and the comparison of the control condition and the supportive condition was marginally significant, $\beta = -0.33$, $t(79) = -1.76$, $p = .08$. By contrast, the interactions between current subjective SES and social-support condition were no longer marginally significant and the effect sizes were significantly reduced—unsupportive compared with supportive condition: $\beta = -0.06$, $t(79) = -0.48$, $p = .63$; control condition compared with supportive condition: $\beta = 0.03$, $t(79) = 0.17$, $p = .87$.

We also tested whether objective measures of SES interacted with social-support condition to predict poststressor level of IL-6. The interactions between early-life objective SES (parental homeownership) and social-support condition were not statistically significant, βs ≤ -0.23, $t(79)$s ≤ -1.68, ps $\geq .10$. Similarly, the interactions between current objective SES (parental income) and condition were not significant, βs ≤ -0.17, $t(79)$s ≤ -0.95, ps $\geq .34$.

In this study, we replicated the interaction between social support and early-life subjective SES using a classic social-evaluative task. We found further support for the claim that the interaction between social support and early-life subjective SES predicts inflammatory reactivity to a stressful context. We also found evidence that in a social-evaluative context, current subjective SES interacts similarly with social support to predict variance in poststressor IL-6 levels. The interaction terms involving current subjective SES were marginally significant, though the average effect sizes were comparable to the effect sizes observed for the interaction between social-support condition and early-life subjective SES. Thus, both early-life and current SES interact meaningfully with social support to predict variance in poststressor levels of IL-6. However, the final regression analysis including interaction terms for both current and early-life subjective SES suggests that early-life subjective SES interacted more strongly with our manipulations of social support than current subjective SES did. In addition, the simple-slopes analyses revealed that current subjective SES did not predict poststressor IL-6 levels in any condition. It appears that early-life subjective SES interacts with the presence or absence of social support more strongly and consistently across contexts than does current subjective SES.

Discussion

In two studies, we demonstrated the importance of early-life SES in predicting IL-6 levels following social stressors. Specifically, we found that the presence or absence of social support interacted with early-life subjective SES to predict levels of OMT IL-6 in response to acute social stressors. These effects were independent of measures of current SES. In the unsupportive conditions of both studies, and in the control condition of Study 2, we found a significant negative relationship between early-life subjective social class and poststressor levels of IL-6. These negative (unsupportive) and ambiguously negative (control) conditions elicited greater poststressor IL-6 levels in participants whose subjective SES during childhood had been low rather than high. As hypothesized, however, this negative relationship was eliminated in the positive (supportive) condition. This interactive effect was observed both following an interpersonal interaction (Study 1) and in the context of social-evaluative threat (Study 2).

In these studies, participants received social support from a stranger. Although many social interactions, and social-evaluative stressors in particular, involve strangers or slight acquaintances (e.g., interviewer, audience), it would be interesting to know how these effects would be altered if support could be provided by a close other (e.g., friend or family member).

Our research highlights one antidote to pro-inflammatory reactivity to stressors in individuals whose childhood SES was low. The availability of social support in the context of an otherwise stressful situation eliminated differences in physiological reactivity related to early-life SES. Although prior research indicates that low SES is associated with increased risk for a number of diseases (Galobardes, Lynch, & Smith, 2008), our findings suggest that the greater attunement to the interpersonal environment also associated with low SES (Kraus et al., 2012) could serve as an important protective factor. Further, interventions focused on providing tangible social support may be particularly beneficial in reducing SES-based health disparities. Note that individuals reared in high-SES environments did not consistently exhibit lower levels of poststressor IL-6 in the supportive condition than in the unsupportive conditions. This suggests that such individuals benefit less from the presence of social support than do those reared in low-SES environments. Although this finding is somewhat surprising, it is in line with research showing that higher SES is associated with being more self-focused, independent, and autonomous (e.g., Kraus et al., 2012).

Why might social support minimize IL-6 reactivity among individuals whose early-life subjective SES was low? Individuals from such backgrounds exhibit down-regulation of genes with response elements for the glucocorticoid receptor, an important regulator of the secretion of cortisol, which can exert anti-inflammatory actions on the immune system (Miller et al., 2009). Social stressors are known to generally elicit increases in cortisol, but repeated exposure to stress and cortisol might lead to increased resistance to its anti-inflammatory effects (Gouin, Hantsoo, & Kiecolt-Glaser, 2008). It is plausible that differential output of cortisol and differences in glucocorticoid resistance related to early-life SES could contribute to the effects reported here, and this possibility may provide a fruitful avenue for future research. In addition, the blood pressure reactivity of individuals reared in low-SES environments during childhood, compared with those raised in high-SES environments, is more sensitive to the specific context of a stressor (Chen, 2007). Thus, it is possible that the interactions with early-life subjective SES that we observed are in part a product of differences in the activity of the autonomic nervous system. Consideration of whether and how early-life SES and the availability of social support synergistically affect the activity of multiple physiological systems will improve understanding of mechanistic pathways that may explain the observed interactive patterns. It is also possible that our findings can be explained by sensitivity to threats. For individuals whose early-life social class was low, the presence of social support may counter tendencies to experience strong threat responses in evaluative situations.

Our research suggests important boundaries to the observed effects. First, although early-life SES appeared to interact more consistently with social-support cues than did current SES, future work should differentiate the contexts in which current SES is more or less likely to influence outcomes. For example, current SES may exert a more consistent influence on outcomes when it is made salient (John-Henderson et al., 2013). Second, subjective early-life SES interacted more strongly with cues of social support than did objective measures of SES. These findings are in keeping with past work indicating that subjective measures of SES predict inflammatory reactivity independently of objective measures (e.g., Derry et al., 2013). These subjective assessments of SES may more accurately represent an individual's socioeconomic experience. It is also possible, however, that more detailed examinations of objective socioeconomic factors will identify dimensions of objective SES that are more predictive of acute physiological reactivity to social stressors. Future work should employ multiple measures of objective SES.

There are important limitations to these studies. First, future research should include additional covariates, such as physical exercise and body mass index, which have been linked to markers of inflammation (O'Connor et al., 2009). Second, given our use of OMT to measure levels

of IL-6, it will be important to examine whether the same effects are observed when inflammatory reactivity is measured using markers of systemic inflammation in blood.

In two studies, we demonstrated that the presence or absence of a socially supportive figure (or figures) moderates the relationship between early-life SES and inflammatory reactivity to a stressor. To the degree that inflammatory reactivity contributes to elevated systemic inflammation, and if similar patterns are found when reactivity is assessed using markers in blood, these findings have implications for longer-term health outcomes and vulnerabilities related to differences in SES.

Author Contributions

N. A. John-Henderson developed the study concept. All authors contributed to the study design. N. A. John-Henderson and J. E. Stellar performed the data analysis under the supervision of R. Mendoza-Denton. N. A. John-Henderson drafted the manuscript, and all authors provided critical revisions. All authors approved the final version of the manuscript for submission.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

Notes

1. Participants were asked to report their height and weight, but because too many participants did not provide this information, we did not include body mass index as a covariate. However, body mass index was not correlated with changes in IL-6 in response to our manipulation, $r = -.19$, $p = .19$.
2. This interaction effect held in an analysis predicting IL-6 change while controlling for baseline levels, $\beta = 0.42$, $t(50) = 3.43$, $p = .001$.
3. As in Study 1, we did not include body mass index as a covariate because too many participants did not report their height and weight. Again, body mass index was not correlated with changes in IL-6 in response to our manipulation, $r \leq -.15$, $p = .21$.
4. As in Study 1, these effects held in an analysis predicting IL-6 change while controlling for baseline levels, βs ≥ -0.27, $t(81)$s ≥ -2.06, ps $\leq .04$.

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