Social Psychological and Personality Science
2014, Vol. 5(3) 301-309

DOI: 10.1177/1948550613494226
spps.sagepub.com
SAGE

Performance and Inflammation Outcomes are Predicted by Different Facets of SES Under Stereotype Threat

Neha A. John-Henderson[1], Michelle L. Rheinschmidt[1], Rodolfo Mendoza-Denton[1], and Darlene D. Francis[2]

Abstract

We experimentally tested whether negative stereotypes linked to lower socioeconomic status (SES), in addition to impairing academic performance (Croizet & Claire, 1998), instigate inflammation processes that are implicated in numerous disease processes. In Study 1, verbal test performance and activation of inflammation processes (measured by levels of an inflammatory protein, Interleukin-6 [IL-6]) varied as a function of SES and test framing (i.e., diagnostic vs. nondiagnostic of intellectual ability), with low SES students underperforming and exhibiting greater IL-6 production in the "diagnostic" condition. In Study 2, students expected their verbal exam performance to be compared to peers of higher or lower SES. Low SES students in the upward comparison condition displayed the greatest inflammatory response and worst test performance. Across both studies, different facets of SES predicted vulnerability to negative outcomes, such that low early life SES predicted heightened inflammation responses, while low current SES predicted impaired academic performance.

Keywords

health, social comparison, social neuroscience, socioeconomic status, stress and coping

Individuals from lower socioeconomic status (SES) backgrounds face not only low rates of admission at 4-year universities but also significant obstacles once enrolled in college, including financial pressures, social exclusion, and stereotypes of low intellectual ability (e.g., Ostrove & Long, 2007; Walpole, 2003). As such, being from a lower SES background can fuel academic competency concerns in college settings (Johnson, Richeson, & Finkel, 2011) and contribute to SES-based *stereotype threat*—that is, the threat of confirming negative stereotypes associated specifically with one's SES (Croizet & Claire, 1998; Spencer & Castano, 2007). In this article, we report two studies that examined the parallel effects of SES-based stereotype threat on (1) academic test performance and (2) the activation of inflammation processes, which indexes an aspect of immune system functioning that is relevant to numerous disease processes.

The health implications of SES-based stereotype threat are an important, albeit understudied, area of inquiry, particularly given that a relationship between SES and health has been documented across the full range of SES for a wide variety of health outcomes (Adler & Snibbe, 2003; Operario, Adler, & Williams, 2004). We chose to focus here on inflammatory cytokines, given recent research documenting a direct relationship between SES and inflammation (John-Henderson, Jacobs, Mendoza-Denton, & Francis, 2013; Ratner, Halim, & Amodio, 2013), and given the links between inflammation and health.

Inflammation is orchestrated by a class of immune system proteins called inflammatory cytokines and is adaptive and integral to the body's defense against infection and injury. However, elevated levels of inflammatory cytokines are implicated in the onset and progression of several chronic diseases including diabetes, cardiovascular disease, and depression (Cesari, Penninx, & Newman, 2003; Liu, Ho, & Mak, 2011; Wellen & Hotamisligil, 2005). Even in relatively young populations (e.g., college students), differences in inflammatory responses can predict vulnerability to negative health outcomes later in life (Stowe, Peek, Cutchin, & Goodwin, 2010).

Acute stressors, specifically ones that involve social evaluation, are particularly powerful activators of inflammation responses (Dickerson, Gable, Irwin, Aziz, & Kemeny, 2009; Slavich, Way, Eisenberger, & Taylor, 2010). However, not everyone experiences evaluative contexts in the same way. Research suggests that individuals under stereotype threat may

[1] Department of Psychology, University of California, Berkeley, CA, USA
[2] School of Public Health and Helen Wills Neuroscience Institute, University of California, Berkeley, CA, USA

Corresponding Author:
Neha A. John-Henderson, Department of Psychology, University of California, 3210 Tolman Hall #1650, Berkeley, CA 94720, USA.
Email: nehajohn@berkeley.edu

experience evaluative contexts as particularly stressful (Schmader, Johns, & Forbes, 2008; Steele & Aronson, 1995). As such, the experience of stereotype threat should lead to both academic underperformance and stress-related inflammation responses (Schmader et al., 2008), an idea we directly test in the two studies reported here.

Predicting Performance and Inflammation Responses to Stereotype Threat

In this research, we employed two different experimental manipulations to examine the effects of SES-based stereotype threat. Study 1 was an exact replication of Croizet and Claire (1998) who manipulated test diagnosticity. Study 2 adopted a social comparison manipulation (Johnson et al., 2011; Mendes, Blascovich, Major, & Seery, 2001) to elicit SES-based social comparisons. Consistent with Schmader and colleagues (2008), we hypothesized that the threat of negative evaluation across these manipulations would yield differences in performance and inflammation responses as a function of SES.

At the same time, however, prior research suggests that *different facets* of SES may predict academic performance and inflammation outcomes. For performance outcomes, the extant prior research on SES-based stereotype threat, albeit scarce, has specifically found that measures of *current* SES predict academic performance under purportedly diagnostic testing conditions (Croizet & Claire, 1998; Spencer & Castano, 2007). Manipulations that place one's SES under suspicion should most naturally make one's current SES standing salient; as such, we expected current SES to interact with our manipulations specifically in predicting performance.

Interestingly, however, a body of literature on SES and reactions to stress suggests that a person's *early* SES, rather than their current SES, should be the stronger predictor of the inflammatory responses observed here. Early and current life measures of SES are increasingly recognized in the literature as having independent effects on health outcomes (e.g., Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Miller & Chen, 2007; Miller et al., 2009). Miller and Chen (2007) found that early SES, independent of current SES, predicted future activity of two genes critical to the regulation of inflammation. Consistent with this notion, Carroll, Cohen, and Marsland (2011) found early childhood SES predicted adult serum concentrations of the inflammatory cytokine Interleukin-6 (IL-6), again independently of current SES. Together, these findings suggest that SES in early childhood may "program" the body's physiological response to subsequent stressful situations by influencing the expression of genes critical to the regulation of inflammation.

Although early childhood SES is often assessed retrospectively, the literature suggests that parental homeownership versus nonhomeownership in early life is an effective index of early SES because it can be reported retrospectively with a high degree of accuracy (Miller & Chen, 2007). This index, which we adopt here, has specifically been found to predict inflammatory profiles as well as physical health outcomes in adulthood (Cohen, Doyle, Turner, Alper, & Skoner, 2004; Miller & Chen, 2007; Miller et al., 2009; Saxton, John-Henderson, Reid, & Francis, 2011).

Based on the above research, then, we expected that parental homeownership in early life would be the stronger predictor of changes in levels of inflammation in response to the stress associated with stereotype threat, while current SES would predict academic performance under stereotype threat.

The Present Research

We conducted two studies to test the hypothesis that SES-based stereotype threat would affect inflammation processes as well as impair test performance. We also tested, based on our review of the literature, whether early SES would predict inflammation responses while current SES would predict test performance. Our physiological and behavioral performance outcomes represent a subset of the co-occurring physiological and psychological processes brought upon by stereotype threat (Schmader et al., 2008).

In Study 1, we adopted a classic stereotype threat paradigm that manipulated the purported diagnosticity of a test for intellectual ability, examining differences in performance as well as inflammation as a function of SES. These procedures have been shown to reliably elicit stereotype threat concerns as a function of SES (Croizet & Claire, 1998). In Study 2, we experimentally induced *relative social comparisons* (Johnson et al., 2011; Mendes et al., 2001) to people higher versus lower in SES than oneself. We expected that upward social comparisons would mirror the performance and health outcomes associated with diagnostic tests particularly for low SES students, but that downward social comparisons would attenuate these effects, presumably by removing the threat of underperformance relative to a higher SES group.

Study 1

Building upon Croizet and Claire (1998), who observed differences in performance on a verbal exam as a function of SES and test frame, we asked whether the experience of SES-based stereotype threat would affect activation of inflammatory processes. We attempted to improve on Croizet and Claire, who examined students at the extreme tertiles of the SES distribution, by including participants from the full spectrum of SES available at the University of California (UC), Berkeley, which is one of the most socioeconomically diverse campuses in the United States (Sacks, 2007).

Method

Participants and Procedure

A total of 90 undergraduate students (65 female) at UC Berkeley participated for partial course credit. We excluded three participants with pre- to post-stressor IL-6 changes greater than 3 standard deviations (*SD*) above the mean. The sample was 52.8% Asian, 25.8% White, 14.6% Latino, 5.6% other, and 1.1% African American. Participants provided a sample of oral

Table 1. Frequency of Parental Homeownership and Outcomes by Diagnostic Condition for Study 1.

	Nondiagnostic	Diagnostic
Homeownership/total *n*	26/46	26/44
Raw IL-6 baseline	0.84 (0.78)	1.08 (0.87)
Raw IL-6 post-stressor	1.24 (1.14)	1.97 (1.41)
Verbal performance	15.33 (1.71)	11.84 (4.22)

Note. IL-6 ¼ interleukin-6. Values are expressed as mean (standard deviation).

Table 2. Frequency of Parental Homeownership and Outcomes by SES-Based Social Comparison Condition for Study 2.

	Downward	Upward
Homeownership/total *n*	37/52	29/46
Raw IL-6 baseline	0.56 (0.58)	0.76 (0.84)
Raw IL-6 post-stressor	0.98 (0.96)	1.69 (1.75)
Verbal performance	14.98 (2.63)	13.04 (3.07)

Note. IL-6 ¼ interleukin-6; SES ¼ socioeconomic status. Values are expressed as mean (standard deviation).

mucosal transudate (OMT) for analysis of baseline levels of the inflammatory cytokine IL-6 (see the section on Inflammation Measures). Next, participants completed measures of SES before completing a "verbal task." This verbal task was a graduate entrance (i.e., graduate record examinations type) verbal examination to which we applied Croizet and Claire's (1998) exact manipulation. Specifically, the test was framed as either "diagnostic of intellectual ability" or a "problem-solving exercise" (i.e., nondiagnostic; e.g., Steele & Aronson, 1995) according to random assignment. Thirty minutes after beginning the examination, a second sample of OMT was taken to examine the levels of IL-6 in response to the diagnosticity (i.e., evaluative stressor) manipulation.

Measures

Early Life SES. Participants reported whether their parents owned or rented their home when they were in kindergarten (see Table 1). This index of early life SES (Cohen et al., 2004; Miller & Chen, 2007) was not significantly correlated with our measure of current SES, $r(90) = .17$, $p = .12$.

Current SES. We standardized self-reported parental income and social class self-categorization, $r(89) = .54$, $p < .001$, and combined them into a single composite index of current SES ($M = .01$, $SD = .89$, $\alpha = .69$). Participants reported their parental income on a scale from 1 (*US$20,000 and below*) to 6 (*US$110,000 and above*) over the past year ($M = 4.43$, $SD = 1.61$; Mendoza-Denton, Downey, Purdie, Davis, & Pietrzak, 2002). Social class was indexed on a scale from 1 (*poor*) to 7 (*upper class*; $M = 4.47$, $SD = 1.30$).

Test Performance. Performance was measured by the number of correct responses of the 20 questions included on the verbal exam.

Inflammation Measures. We assessed IL-6 levels in OMT. While IL-6 can exert both inflammatory and anti-inflammatory effects (Scheller, Chalaris, Schmidt-Arras, & Rose-John, 2011), prior research characterizes increases in IL-6 specifically in response to stressors as indicative of an inflammatory response (Dickerson et al., 2009; Slavich et al., 2010). In line with the pre- to post-stressor design used in these studies, we examine changes in levels of IL-6 in response to stereotype threat. Participants provided a baseline sample for IL-6 measurement. An Orasure collective device (Epitope, Beaverton, OR) was placed between the lower cheek and gum for 2 min. After completion of the verbal exam (30 min), participants provided a second sample of OMT for measurement of post-stressor IL-6 levels ($M = 1.59$ pg/mL, $SD = 1.33$). The samples were frozen and stored at −80°C. IL-6 concentrations were determined by an enzyme-linked immunosorbent assay using commercially available kits (R&D systems, Minneapolis, MN). As in previous research (John-Henderson et al., 2013; Kielcot-Glaser et al., 2003), raw IL-6 baseline (skewness = 1.34, standard error [*SE*] = .25) and activation (skewness = .98, *SE* = .25) values were normalized by log transformation.

Body Mass Index (BMI). Participants reported their height and weight, from which we calculated their BMI, using the formula: (weight in pounds × 703/[height in inches]^2). We used BMI as a covariate for the analyses specifically related to inflammation, given its relationship with baseline levels of IL-6 in previous research (Khaodhiar, Ling, Blackburn, & Bistrian, 2004).

Previous Verbal Skills. We assessed possible preexperiment differences in verbal skills using self-reported scores on the scholastic assessment test (SAT) verbal exam ($M = 659.52$, $SD = 86.51$). Self-reported SAT scores have been shown to be highly correlated with official SAT score reports (Rheinschmidt & Mendoza-Denton, 2013).

Analytic Strategy

We ran two parallel regression analyses; one for post-test IL-6 levels that controlled for both pre-test IL-6 levels and BMI, and one for performance that controlled for SAT scores. In the first step, each of these regression analyses included all main effects (condition, early SES, and current SES) and interaction terms. SAT scores did not account for a significant amount of variance in the models across studies ($ps > .57$). We report our analyses without this covariate because it did not change the main pattern of results and limited our sample sizes. Three-way interactions (Condition × Early SES × Current SES) across Studies 1 and 2 were also not significant and will not be discussed further. In what follows, we report the results from the simultaneous regressions that include all two-way terms.[1] This analytic strategy effectively allows us to see whether a significant proportion of the variance in a given outcome is accounted for by early versus current SES while controlling for each other's effect in the model. All continuous variables were standardized for analyses.

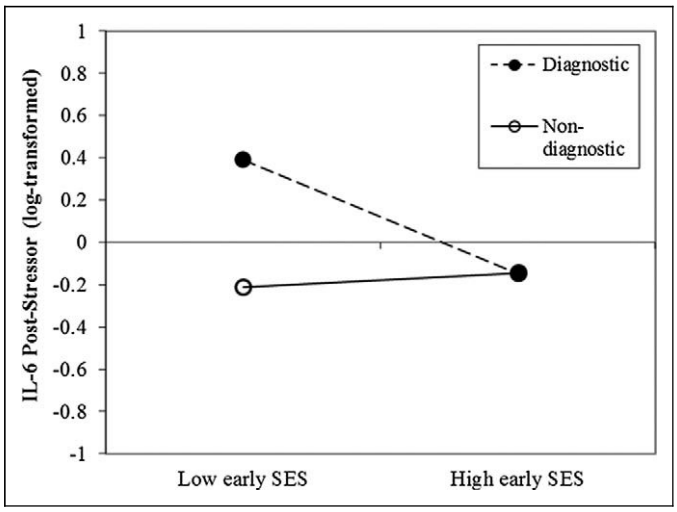

Figure 1. Post-stressor activation of IL-6 in Study 1 as a function of early SES and diagnostic condition, controlling for baseline IL-6 levels and BMI, with all continuous variables standardized. BMI ¼ body mass index; IL-6 ¼ interleukin-6; SES ¼ socioeconomic status.

Results

We observed a significant zero-order correlation between our dependent variables, indicating the expected relationship between performance impairments and higher levels of post-stressor inflammation, r ¼ □.44, $t(90)$ ¼ 4.57, $p < .001$. The multiple linear regression analyses described above were then used to examine both independent and interactive relationships among diagnosticity condition (0 ¼ *nondiagnostic* and 1 ¼ *diagnostic*) and each measure of SES (i.e., early life and current) on performance and post-stressor IL-6.

Inflammation Response

Controlling for baseline levels of inflammation (M ¼ .96, SD ¼ .83) and BMI (M ¼ 22.41, SD ¼ 2.55), we observed a main effect of diagnostic condition, b ¼ .60, $t(80)$ ¼ 3.66, $p < .001$, suggesting that the stereotype threat manipulation led to the expected stress response (Schmader et al., 2008). Consistent with hypotheses, the Condition □ Current SES interaction was not significant, b ¼ □.004, $t(80)$ ¼ □.03, p ¼ .98; however, the Condition □ Early SES interaction on post-stressor IL-6 levels was statistically significant, b ¼ □.61, $t(80)$ ¼ □2.85, $p < .01$. Figure 1 shows predicted values of post-stressor IL-6 from this latter interaction. Simple slope analyses revealed a negative relationship between activation of inflammation and early life SES only when the test was framed as diagnostic (diagnostic: b ¼ □.54, t ¼ □3.52, p ¼ .001 and non-diagnostic: b ¼ .07, t ¼ .46, p ¼ .64).

Test Performance

Regression analyses revealed the predicted main effect of condition, b ¼ □.93, $t(83)$ ¼ □3.88, $p < .001$, indicating that performance, like inflammation response, also suffered as a function of the manipulation. Critical to our analyses, however, the interaction between early SES and condition was not significant, b ¼ □.07, $t(83)$ ¼ □.23, p ¼ .82, while the interaction between current SES and condition was significant, b ¼ .65, $t(83)$ ¼ 3.66, $p < .001$. Figure 2 shows the predicted test scores at 1 *SD* above and below the mean for current SES. Simple slope analyses revealed that, under diagnostic threat, higher current SES predicted better performance, b ¼ .79, t ¼ 5.55, $p < .001$. In the nondiagnostic condition, performance did not vary significantly as a function of current SES, b ¼ .13, t ¼ .84, p ¼ .40.

Discussion

Findings from Study 1 revealed that early life SES predicted inflammatory responses in a task that invoked stereotype threat, confirming prior research documenting the association between early life SES and biological responses to stressors (Miller & Chen, 2010). Conversely, current SES measures predicted performance under threat, consistent with findings from Croizet and Claire. The findings suggest that both past and current measures of SES are important in the experience of SES-based stereotype threat and, in this study, were differentially predictive of performance and immune health outcomes.

Study 2

In Study 2, we asked will encouraging downward social comparisons mitigate the negative performance and health outcomes we observed in Study 1 in response to stereotype threat among low SES participants? And importantly, would upward social comparisons have the opposite effect?

People compare themselves to others along various dimensions (e.g., attractiveness and SES) to navigate their social environments (Fiske, 2012). Research shows that social comparisons can affect *both* performance and health outcomes. Mendes and colleagues (2001) manipulated upward versus downward comparison direction through random assignment to an ostensible interaction partner with higher or lower relative task performance, respectively. This comparison manipulation affected people's perceived resources to complete the task and their cardiovascular reactivity. More specifically, participants interacting with upward comparison partners exhibited less adaptive patterns of cardiovascular response relative to participants interacting with downward comparison partners. Similarly, in Johnson, Richeson, and Finkel (2011), downward (vs. upward) comparison buffered relatively lower income students from cognitive resource depletion following a self-presentation task.

We experimentally tested the role of relative comparison group (i.e., higher or lower current SES) on test performance and inflammation processes. We expected the upward comparison condition in Study 2 to show analogous results to the "diagnostic" condition in Study 1, in the sense that this condition should place lower SES participants under the threat of underperforming relative to a (now relatively) higher SES

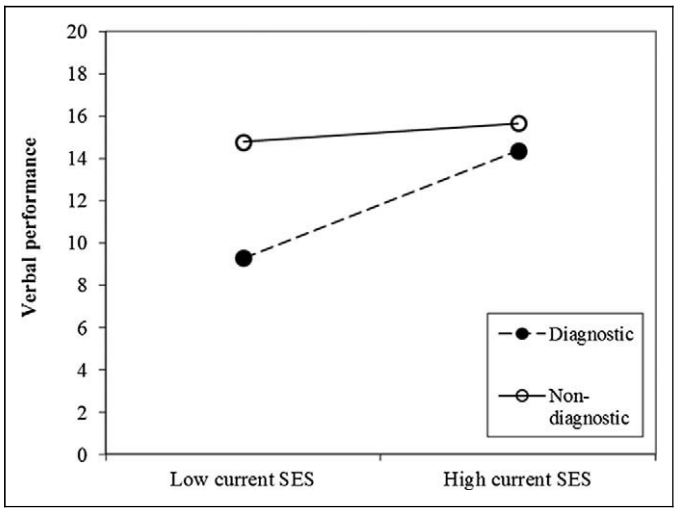

Figure 2. Verbal test performance in Study 1 as a function of current SES and diagnostic condition. Markers represent predicted test performance by condition at 1 standard deviation above and below the standardized mean for current SES. SES ¼ socioeconomic status.

group. By contrast, we expected the downward comparison condition to be analogous to the "nondiagnostic" condition in Study 1, in the sense that the threat of negative evaluation is attenuated when one is in the higher status group in a given domain (i.e., the "default" context for higher SES individuals; Mendoza-Denton, Kahn, & Chan, 2008; Stone, Lynch, Sjomeling, & Darley, 1999; Walton & Cohen, 2003).

Accordingly, our predictions follow from those of Study 1: We expected lower performance and greater inflammation among lower SES individuals in the upward relative to the downward comparison condition. Paralleling Study 1, however, we expected a significant Early SES □ Condition interaction in predicting inflammation responses and a significant Current SES □ Condition interaction in predicting test performance.

Method

Participants and Procedure

A total of 98 undergraduate students (53 female) at UC Berkeley participated for partial course credit. We excluded three participants with pre- to post-stressor IL-6 changes greater than 3 *SD* above the mean. The sample was 54.1% Asian, 27.6% White, 13.3% Latino, 4.1% other, and 1% African American.

On arrival, participants completed the same measures as in Study 1. These included baseline IL-6 levels, BMI, self-reported SAT scores, early life SES, and the current SES composite. In this study, we observed a moderate correlation between early and current measures of SES, r ¼ .30, $t(95)$ ¼ 3.08, $p < .01$.

Participants were assigned to one of the two experimental conditions: upward SES comparison (i.e., performance was compared to that of higher SES individuals) or downward SES comparison (i.e., performance was compared to that of lower SES individuals). Participants in the upward comparison condition were told that their performance would be compared to individuals "two full scale points" above them on an SES index based on parental income, education, and occupational prestige. Participants in the downward comparison condition were told that they would be compared to individuals "two full scale points" below them on this same index. We did not mention minimum and maximum scores on the artificial SES index so that no one believed themselves to be immune to either upward or downward comparison. To ensure that participants understood the manipulation, we asked them to report their comparison group at the end of the study.

Following the comparison manipulation, participants completed the same verbal exam used in Study 1. Upon completion of the exam, they provided a second OMT sample to assess post-stressor levels of IL-6 (M ¼ 1.31, SD ¼ 1.42; see Table 2). Once again, to normalize baseline and post-stressor IL-6 levels, we applied a log transformation to these values.

Results

The analytic strategy remained the same as in Study 1. Downward comparisons were coded as 0 and upward comparisons as 1. We observed a negative relationship between performance and levels of post-stressor inflammation, r ¼ □.32, $t(97)$ ¼ □3.26, $p < .01$, replicating the correspondence between less favorable outcomes observed in Study 1.

Inflammation Response

Controlling for baseline levels of IL-6 (M ¼ .65, SD ¼ .72) and BMI (M ¼ 22.37, SD ¼ 3.38), we again observed a main effect of comparison condition, b ¼ .62, $t(88)$ ¼ 2.70, $p < .01$, on post-manipulation IL-6. Replicating Study 1, the results revealed that the Condition □ Current SES interaction was not statistically significant, b ¼ □.27, $t(88)$ ¼ □1.78, p ¼ .08. Again, however, the Condition □ Early SES interaction on post-stressor IL-6 levels was statistically significant, b ¼ □.67, $t(88)$ ¼ □2.28, $p < .03$. Simple slope analyses revealed a negative relationship between activation of inflammation and early life SES only in the upward comparison condition (upward: b ¼ □.56, t ¼ □2.69, $p < .01$; downward: b ¼ .10, t ¼ .49, p ¼ .63; see Figure 3).

Test Performance

Regression analyses revealed a main effect of comparison condition on performance, b ¼ □.88, $t(91)$ ¼ □3.01, $p < .01$. Replicating Study 1, in the model for test performance, the interaction between early SES and condition was not significant, b ¼ .25, $t(91)$ ¼ .68, p ¼ .50, while the interaction between current SES and condition was significant, b ¼ .53, $t(91)$ ¼ 2.82, $p < .01$. Simple slope analyses revealed that, under the threat of an upward social comparison, current SES predicted performance positively, b ¼ .86, t ¼ 4.96, $p < .001$. In the downward comparison condition, we observed a marginally significant relationship between performance and

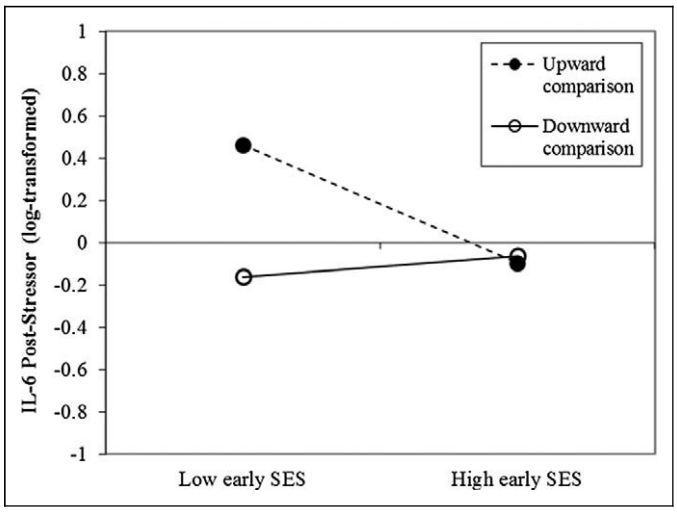

Figure 3. Post-stressor activation of IL-6 in Study 2 as a function of early SES and social comparison condition, controlling for baseline IL-6 levels and BMI, with all continuous variables standardized. BMI ¼ body mass index; IL-6 ¼ interleukin-6; SES ¼ socioeconomic status.

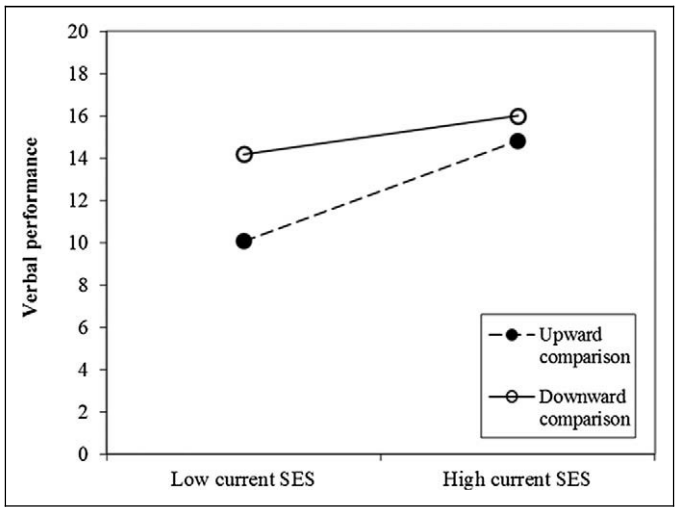

Figure 4. Verbal test performance in Study 2 as a function of current SES and social comparison condition. Markers represent predicted outcomes at 1 standard deviation above and below the standardized mean for current SES. SES ¼ socioeconomic status.

current SES, b ¼ .33, t ¼ 2.03, p ¼ .05 (see Figure 4). Thus, consistent with our predictions, the relationship between SES and performance was more pronounced in the upward than downward social comparison condition.

General Discussion

In line with prior research and theory (e.g., Schmader et al, 2008), two studies showed that the experience of SES-based stereotype threat led to both performance impairments and inflammation responses. Integrating literatures on stereotype threat on one hand and SES and health outcomes on the other, however, we found that performance and inflammation were differentially predicted by current versus early SES. Consistent with prior research on SES-based stereotype threat (e.g., Croizet & Claire, 1998), we expected decrements in performance to be predicted by participants' current SES. At the same time, findings indicating that early SES is a strong indicator of adult stress responses independently of current SES led us to expect that early SES would be a more powerful predictor of inflammation responses in our own studies.

Findings confirmed our expectations across two studies. Given that early and current SES can be correlated (as in our own Study 2), the current findings suggest that even though both performance and health decrements may result from SES-based stereotype threat, the etiology of these decrements may not be the same. The types of mental processes that affect performance, which include rumination about others' evaluations and intrusive ideation about one's standing relative to others (Steele & Aronson, 1995), may be more directly linked to identities that describe us in the *present* than to those that described us in the past. More concretely, a test that is framed as elucidating intellectual differences as a function of SES may more naturally lead people to think about (and worry over) their current SES than their childhood SES. By contrast, a growing literature suggests that early SES leaves a biological residue manifested by increased proinflammatory signaling later in life (Miller & Chen, 2007, 2010; Miller et al., 2009), possibly by influencing the expression of genes associated with the regulation of inflammatory responses. Our findings are consistent with this view, in that inflammatory responses to the stressors in Study 1 (test diagnosticity) and Study 2 (upward social comparisons) were predicted by early SES and were independent of current SES.

Measuring Inflammation in OMT

It must be noted that measurement of inflammation in OMT is not a surrogate for systemic levels of inflammation and that the majority of research linking inflammation to health outcomes relies on assessment of levels of inflammation in blood. Studies that have explored the relationship between levels of inflammation in OMT and circulating levels of inflammation in blood have found inconsistent relationships between the two measurements (Fernandez-Botran, Miller, Burns, & Newton, 2011). However, given that laboratory-based social stressors produce localized expression of inflammatory markers in the mouth (Weik, Herforth, Kolb-Bachofen, & Deinzer, 2008), it is not surprising that we observed changes in levels of inflammation in this study as a function of SES and manipulations of psychological threat. Importantly, studies have shown that levels of inflammatory markers in OMT are related to measures of SES (John-Henderson et al., 2013; Ratner et al., 2013; Saxton et al., 2011), are affected by social evaluative stress (Dickerson et al., 2009), and are related to psychosocial variables (Sjogren, Leanderson, Kristenson, & Ernerudh, 2006). As such, while OMT measures should not be interpreted as a reflection of levels of inflammatory markers in blood, they are nevertheless important, given the above associations.

Early and Current Measures of SES

An individual's SES is different from other identities (e.g., race) in that it can change over the lifetime (Miller et al., 2009). As such, early SES and current SES are not perfectly correlated. This inconsistency is further reflected in how people conceptualize their own and others' SES, namely as a static or malleable aspect of the self (Rheinschmidt & Mendoza-Denton, 2013). It is important to understand the ways in which early SES "stays" with people (e.g., their psychological and biological functioning) and to separate the effects of early SES from those of current SES.

Our findings suggest that the inclusion of both early life and current measures of SES may help elucidate the relationship between SES and performance and health outcomes. Consistent with Miller and Chen (2010), we find evidence that early life SES programs inflammatory responses later in life. It is important to note that homeownership is not a perfect proxy of early life SES; for instance, homeownership may not hold the same meaning for individuals coming from urban areas due to an increased likelihood of renting. Homeownership may also covary with other important aspects of one's early life environment, such as a sense of stability (Haurin, Parcel, & Jean Haurin, 2002). In future research, a more comprehensive survey of early life environment and adversity should be included to uncover the specific components of early life that are implicated in programming of biological responses.

Future Directions

Our findings suggest that stereotype threat can elicit a more pronounced inflammatory response for individuals from low SES backgrounds, which could increase vulnerability to negative health outcomes. Members of ethnic minority and other negatively stereotyped groups report more instances of negative social evaluation than majority group members (Mendoza-Denton et al., 2002), and this greater perceived discrimination is associated with adverse health outcomes (Ratner et al., 2013). In addition, repeated experience of acute social evaluative stressors may increase existing vulnerabilities to ill health (see a literature on allostatic load; e.g., McEwen, 1998). Thus, while our current focus is SES, we expect that our inflammation findings would hold for other stigmatized social identities and, in addition, that low early life SES in combination with other stigmatized social identities would further predispose people to exaggerated inflammatory responses.

In this research, the experimental conditions that elicited poor performance also elicited greater activation of inflammatory processes. As described in Mendes and Jamieson (2011), psychological responses to stereotype threat may trigger physiological (e.g., neurobiological) changes which then influence cognitive and behavioral outcomes. Though we find convergence across behavioral and physiological outcomes, our goal was to establish them as parallel, rather than causally related, outcomes. Our measure of inflammation suggests a larger stress arousal response that affects several body systems (e.g., immune, neuroendocrine) and interacts with cognitive mediators of stereotype threat (e.g., vigilance) to inhibit performance outcomes, as described in the integrated process model of stereotype threat (Schmader et al., 2008). Simultaneous consideration of both the cognitive and the physiological mechanisms of stereotype threat will further pave the way for interventions that boost the achievement and health of negatively stereotyped individuals.

One such intervention may involve teaching lower SES students about stereotype threat effects, following research attesting to the benefits of educating women in math settings about stereotype threat (Johns, Schmader, & Martens, 2005). We expect that such interventions may also buffer negatively stereotyped students from the physiological effects of stereotype threat. Attenuating these negative physiological responses could be a step toward reducing SES-based health disparities.

Acknowledgments

We are grateful for data collection assistance from Hardev Chhokar, Leeran Baraness, and Martha Heredia and article feedback from colleagues in the Berkeley Writing Workshop.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by an NSF Graduate Research Fellowship awarded to Michelle L. Rheinschmidt.

Note

1. Across studies, the two-way Early Life □ Current SES interactions were not significant; however, given that they are at the same level as the other two-way interactions of interest (those by condition), we opted to keep them in the model. The results remain unchanged when this interaction is removed from the models.

References

Adler, N. E., & Snibbe, A. C. (2003). The role of psychosocial processes in explaining the gradient between socioeconomic status and health. *Current Directions in Psychological Science*, *12*, 119–123. doi:10.1111/1467-8721.01245

Carroll, J. E., Cohen, S., & Marsland, A. L. (2011). Early childhood socioeconomic status is associated with circulating interleukin-6 among mid-life adults. *Brain Behavior and Immunity*, *25*, 1468–1474.

Cesari, M., Penninx, B. W., & Newman, A. B. (2003). Inflammatory markers and cardiovascular disease (The Health, Aging and Body Composition [Health ABC] Study). *American Journal of Cardiology*, *92*, 522–528. doi:10.1016/S0002-9149(03)00718-5

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2004). Childhood socioeconomic status and host resistance to infectious illness in adulthood. *Psychosomatic Medicine*, *66*, 553–558. doi:10.1097/01.psy.0000126200.05189.d3

Cohen, S., Janicki-Deverts, D., Chen, E., & Matthews, K. A. (2010). Childhood socioeconomic status and adult health. *Annals of the New York Academy of Sciences*, *1186*, 37–55. doi:10.1111/j.1749-6632.2009.05334

Croizet, J. C., & Claire, T. (1998). Extending the concept of stereotype threat to social class: The intellectual underperformance of students from low socioeconomic backgrounds. *Personality and Social Psychology Bulletin*, *24*, 588–594. doi:10.1177/0146167298246003

Dickerson, S. S., Gable, S. L., Irwin, M. R., Aziz, N., & Kemeny, M. E. (2009). Social-evaluative threat and proinflammatory cytokine regulation: An experimental laboratory investigation. *Psychological Science*, *20*, 1237–1244. doi:10.1111/j.1467-9280.2009.02437.x

Fernandez-Botran, R., Miller, J. J., Burns, V. E., & Newton, T. L. (2011). Correlations among inflammatory markers in plasma, saliva, and oral mucosal transudate in post-menopausal women with past intimate partner violence. *Brain Behavior and Immunity*, *25*, 314–321. doi:10.1016/j.bbi.2010.09.023

Fiske, S. T. (2012). *Envy up, scorn down: How status divides us*. New York, NY: Russell Sage.

Haurin, D. R., Parcel, T. L., & Jean Haurin, R. (2002). Does homeownership affect child outcomes? *Real Estate Economics*, *30*, 635–666. doi:10.1111/1540-6229.t01-2-00053

John-Henderson, N., Jacobs, E. G., Mendoza-Denton, R., & Francis, D. D. (2013). Wealth, health and the moderating role of implicit social class bias. *Annals of Behavioral Medicine*, *45*, 173–179. doi:10.1007/s12160-012-9443-9

Johns, M., Schmader, T., & Martens, A. (2005). Knowing is half the battle: Teaching stereotype threat as a means of improving women's math performance. *Psychological Science*, *16*, 175–179. doi:10.1111/j.0956-7976.2005.00799.x

Johnson, S. E., Richeson, J. A., & Finkel, E. J. (2011). Middle class and marginal? The influence of socioeconomic status on the self-regulatory resources of students at an elite university. *Journal of Personality and Social Psychology*, *100*, 838–852. doi:10.1037/a0021956

Khaodhiar, L., Ling, P. R., Blackburn, G. L., & Bistrian, B. R. (2004). Serum levels of interleukin-6 and C-reactive protein correlate with body mass index across the broad range of obesity. *Journal of Parental and Enteral Nutrition*, *28*, 410–415. doi:10.1177/0148607104028000410

Kielcot-Glaser, J. K., Preacher, K. J., MacCallum, R. C., Atkinson, C., Malarkey, W., & Glaser, R. (2003). Chronic stress and age-related increases in the proinflammatory cytokine IL-6. *Proceedings of the National Academy of Sciences of the United States of America*, *100*, 9090–9095. doi:10.1073/pnas.1531903100

Liu, Y., Ho, R. C., & Mak, A. (2011). Interleukin (IL)-6, tumor necrosis factor alpha (TNF-a) and soluble interleukin-2 receptors (sIL-2R) are elevated in patients with major depressive disorder: A meta-analysis and meta-regression. *Journal of Affective Disorders*, *139*, 230–239. doi:10.1016/j.jad.2011.08.003

McEwen, B. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *84*, 33–44. doi:10.1111/j.1749-6632.1998.tb09546.x

Mendes, W. B., Blascovich, J., Major, B., & Seery, M. D. (2001). Challenge and threat responses during downward and upward social comparisons. *European Journal of Social Psychology: Special Issue on Social Comparisons*, *31*, 477–497. doi:10.1002/ejsp.80

Mendes, W. B., & Jamieson, J. (2011). Embodied stereotype threat: Exploring brain and body mechanisms underlying performance impairments. In M. Inzlicht & T. Schmader (Eds.), *Stereotype threat: Theory, process, and application* (pp. 51–68). New York, NY: Oxford University Press.

Mendoza-Denton, R., Downey, G., Purdie, V., Davis, A., & Pietrzak, J. (2002). Sensitivity to status-based rejection: Implications for African-American students' college experience. *Journal of Personality and Social Psychology*, *83*, 896–918. doi:10.1037//0022-3514.83.4.896

Mendoza-Denton, R., Kahn, K., & Chan, W. (2008). Can fixed views of ability boost performance in the context of favorable stereotypes? *Journal of Experimental Social Psychology*, *44*, 1187–1193. doi:10.1016/j.jesp.2008.03.005

Miller, G. E., & Chen, E. (2007). Unfavorable socioeconomic conditions in early life presage expression of pro-inflammatory phenotype in adolescence. *Psychosomatic Medicine*, *69*, 402–409. doi:10.1097/PSY.0b013e318068fcf9

Miller, G. E., & Chen, E. (2010). Harsh family climate in early life presages the emergence of pro-inflammatory phenotype in adolescence. *Psychological Science*, *21*, 848–856. doi:10.1177/0956797610370161

Miller, G. E., Chen, E., Fok, A. K., Walker, H., Lim, A., & Nicholls, E. F., . . . Kobor, M. S. (2009). Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proceedings of the National Academy of Sciences of the United States of America*, *106*, 14716–14721. doi:10.1073/pnas.0902971106

Operario, D., Adler, N. E., & Williams, D. R. (2004). Subjective social status: Reliability and predictive utility for global health. *Psychology & Health*, *19*, 237–246. doi:10.1080/08870440310001638098

Ostrove, J. M., & Long, S. M. (2007). Social class and belonging: Implications for college adjustment. *The Review of Higher Education*, *30*, 363–389.

Ratner, K. G., Halim, M. L., & Amodio, D. M. (2013). Perceived stigmatization, in group pride, and immune and endocrine activity: Evidence from a community sample of Black and Latina women. *Social Psychological and Personality Science*, *4*, 82–91. doi:10.1177/1948550612443715

Rheinschmidt, M. L., & Mendoza-Denton, R. (2013). Social class and college outcomes: The interplay of rejection sensitivity and entity beliefs. *Manuscript submitted for publication*.

Sacks, P. (2007). *Tearing down the gates: Confronting the class divide in American education*. Berkeley: University of California Press.

Saxton, K. B., John-Henderson, N., Reid, M. W., & Francis, D. D. (2011). The social environment and IL-6 in rats and humans. *Brain, Behavior, & Immunity*, *25*, 1617–1625. doi:10.1016/j.bbi.2011.05.010

Scheller, J., Chalaris, A., Schmidt-Arras, D., & Rose-John, S. (2011). The pro- and anti-inflammatory properties of the cytokine Interleukin-6. *Biochimica et Biophysica Acta*, *1813*, 878–888. doi:10.1016/j.bbamcr.2011.01.034

Schmader, T., Johns, M., & Forbes, C. (2008). An integrated process model of stereotype threat effects on performance. *Psychological Review*, *115*, 336–356. doi:10.1037/0033-295X.115.2.336

Sjogren, E., Leanderson, P., Kristenson, M., & Ernerudh, J. (2006). Interleukin-6 levels in relation to psychosocial factors: Studies on serum, saliva and in vitro production by blood mononuclear cells. *Brain Behavior and Immunity*, *20*, 270–278. doi:10.1016/j.bbi.2005.08.001

Slavich, G. M., Way, B. M., Eisenberger, N. I., & Taylor, S. E. (2010). Neural sensitivity to social rejection is associated with inflammatory responses to social stress. *Proceedings of the National Academy of Sciences of the United States of America*, *107*, 14817–14822. doi:10.1073/pnas.1009164107

Spencer, B., & Castano, E. (2007). Social class is dead. Long live social class! Stereotype threat among low socioeconomic status individuals. *Social Justice Research*, *20*, 418–432. doi:10.1007/s11211-007-0047-7

Steele, C. M., & Aronson, J. (1995). Stereotype threat and the intellectual test performance of African Americans. *Journal of Personality and Social Psychology*, *69*, 797–811. doi:10.1037/0022-3514.69.5.797

Stone, J., Lynch, C. I., Sjomeling, M., & Darley, J. M. (1999). Stereotype threat effects on black and white athletic performance. *Journal of Personality and Social Psychology*, *77*, 1213–1227.

Stowe, R. P., Peek, M. K., Cutchin, M. P., & Goodwin, J. S. (2010). Plasma cytokine levels in a population-based study: Relation to age and ethnicity. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, *65*, 429–433. doi:10.1093/gerona/glp198

Walpole, M. (2003). Socioeconomic status and college: How SES affects college experiences and outcomes. *Review of Higher Education: Journal of the Association for the Study of Higher Education*, *27*, 45–73. doi:10.1353/rhe.2003.0044

Walton, G. M., & Cohen, G. L. (2003). Stereotype lift. *Journal of Experimental Social Psychology*, *39*, 456–457. doi:10.1016/S0022-1031(03)00019-2

Weik, U., Herforth, A., Kolb-Bachofen, V., & Deinzer, R. (2008). Acute stress induces proinflammatory signaling at chronic inflammation sites. *Psychosomatic Medicine*, *70*, 906–912. doi:10.1097/PSY.0b013e3181835bf3

Wellen, K. E., & Hotamisligil, G. S. (2005). Inflammation, stress, and diabetes. *Journal of Clinical Investigation*, *115*, 1111–1119. doi:10.1172/JCI200525102

Author's Biographies

Neha A. John-Henderson is a doctoral candidate in psychology at the University of California, Berkeley. Her research focuses on how psychosocial factors and experiences interact to moderate the relationship between socioeconomic status and health.

Michelle L. Rheinschmidt is a doctoral candidate in social/personality psychology at the University of California, Berkeley. Her research focuses on the topics of stigma, social groups/identities, and strategies for fostering diversity and inclusion.

Rodolfo Mendoza-Denton is an associate professor of psychology at the University of California, Berkeley. Information on him can be found at the following URL: http://psychology.berkeley.edu/people/rodolfo-mendoza-denton

Darlene D. Francis is an associate professor of public health and neuroscience at the University of California, Berkeley. Her research explores how biological, psychological, and social processes interact over a lifetime to influence health and vulnerability to disease.