

Self-Esteem Moderates Neuroendocrine and Psychological Responses to Interpersonal Rejection

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In this study, the authors investigated self-esteem as a moderator of psychological and physiological responses to interpersonal rejection and tested an integrative model detailing the mechanisms by which self-esteem may influence cognitive, affective, and physiological responses. Seventy-eight participants experienced an ambiguous interpersonal rejection (or no rejection) from an opposite sex partner in the context of an online dating interaction. Salivary cortisol was assessed at 5 times, and self-reported cognitive and affective responses were assessed. Compared with those with high self-esteem, individuals with low self-esteem responded to rejection by appraising themselves more negatively, making more self-blaming attributions, exhibiting greater cortisol reactivity, and derogating the rejector. Path analysis indicated that the link between low self-esteem and increased cortisol reactivity was mediated by self-blame attributions; cortisol reactivity, in turn, mediated the link between low self-esteem and increased partner derogation. Discussion centers on the role of self-esteem as part of a broader psychobiological system for regulating and responding to social threat and on implications for health outcomes.

Keywords: interpersonal rejection, self-esteem, cortisol, stress hormones, health

Interpersonal rejection is often a painful and threatening experience. Nevertheless, people differ in how they construe and respond to rejection. Previous research shows, for example, that individuals with negative self-views are more likely to construe social interactions as rejecting and to exhibit maladaptive cognitive, emotional, and behavioral responses to rejection, compared with those with more positive self-views (e.g., Downey & Feldman, 1996; Murray, Bellavia, Rose, & Griffin, 2003; Nezlak, Kowalski, Leary, Blevins, & Holgate, 1997; Sommer & Baumeister, 2002). Moreover, there is growing evidence that individuals with negative self-views are at greater risk for difficulties in their close relationships, in part because their concerns about rejection often trigger self-defensive cognitive and social responses that

interfere with their ability to develop and sustain satisfying relationships (Murray, Holmes, & Collins, 2006).

Most of the empirical work on rejection has focused on the adverse consequences of social rejection for emotional well-being, cognitive functioning, and social behavior. Recently, scholars have turned their attention to understanding the physiological consequences and neural processing dynamics of rejection and related social threats (Dandeneau, Baldwin, Baccus, Sakellaropoulou, & Pruessner, 2007; Dickerson & Kemeny, 2004; Eisenberger & Lieberman, 2004; MacDonald & Leary, 2005; Pietrzak, Downey, & Ayduk, 2005; Stroud, Tanofsky-Kraff, Wilfley, & Salovey, 2000). In the current investigation, we build on this line of research by examining the role of self-esteem as part of a broader psychobiological response system for regulating and coping with threats to social acceptance. Our primary theoretical goal is to integrate recent models concerning neurobiological responses to rejection with models from the close relationships literature concerning the role of self-esteem in regulating social threat. In doing so, we test a model that specifies some mechanisms through which low self-esteem (LSE) may activate physiological stress pathways and defensive social action in response to interpersonal rejection.

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Psychological and Physiological Responses to Rejection

Numerous theories in social, developmental, and evolutionary psychology suggest that human beings have a fundamental need for social inclusion that evolved because social ties and interpersonal attachments were critical to the survival and reproductive success of our human ancestors (Baumeister & Leary, 1995; Bowlby, 1982; Dickerson & Kemeny, 2004; MacDonald & Leary, 2005; Williams, 2001). The adaptive value of social acceptance is still vital in contemporary life, in which social ties continue to be an important source of both material and emotional resources that sustain health and well-being. Because social acceptance is such an important human

need, events (such as rejection) that violate the fulfillment of this need lead to a variety of adverse psychological outcomes, including anxiety and depression (Leary, Tambor, Terdal, & Downs, 1995), diminished self-worth (Leary et al., 1995; Zadro, Williams, & Richardson, 2004), a sense of losing control (Williams, Cheung, & Choi, 2000), and impaired cognitive and self-regulatory abilities (Baumeister, DeWall, Ciarocco, & Twenge, 2005; Baumeister, Twenge, & Nuss, 2002).

Although the psychological consequences of rejection are well-documented, scholars have only recently begun to examine the physiological and neural processing dynamics of rejection. This new line of research is guided by a number of theoretical models that are rooted in a common assumption—that because social acceptance is critical to safety and survival, humans evolved physiological and neural processing mechanisms designed for monitoring threats to social acceptance, along with motivational and behavioral response systems for coping with such threats (Eisenberger & Lieberman, 2004; Gruenewald, Dickerson, & Kemeny, 2007; MacDonald & Leary, 2005; Pietrzak, Downey, & Ayduk, 2005). According to these models, social threats (such as rejection) should activate physiological systems that were designed to respond to physical threats, including neuroendocrine, cardiovascular, and immunological systems.

One neuroendocrine system of special interest is the hypothalamic-pituitary-adrenal axis (HPA axis), which is one component of the human physiological stress response. HPA axis activation begins when a stressor triggers a cascade of hormones, ultimately resulting in the release of glucocorticoids, such as cortisol, from the adrenal cortex. Cortisol travels throughout the body and interacts with the cardiovascular and autonomic nervous systems to prepare the body to respond efficiently to a stressor (McEwen, 1998; Sapolsky, 1998).

Although threats to physical safety are the prototypical conditions under which the HPA system is activated, social threats can also activate this system. Dickerson, Gruenewald, and Kemeny (2004) argue in their social self-preservation theory that threats to one's social self—including threats to one's social esteem, status, and acceptance—trigger a coordinated psychobiological response that includes activation of the HPA system as well as changes in social cognition, motivation, and behavior. Consistent with this theory, a small number of studies have shown that rejection from peers is associated with increased HPA activity in children (Gunnar, Sebanc, Tout, Donzella, & Van Dulmen, 2003) and adult women (Stroud, Salovey, & Epel, 2002; Stroud et al., 2000). There is also evidence that situations involving the potential for interpersonal rejection on the basis of performance failure lead to increases in HPA activity. In a meta-analytic review of laboratory studies of acute stressors, Dickerson and Kemeny (2004) found that motivated performance stressors (such as public speaking tasks) lead to heightened cortisol reactivity primarily when performed in the presence of an evaluative audience. This suggests that it is the threat to the social self (a threat to one's social esteem or social standing), not the threat of performance failure per se, which drives heightened HPA activity. This conclusion is supported by studies that show that cortisol levels increase during performance tasks as the degree of social evaluation increases (Dickerson, Mycek, & Zaldivar, 2008; Gruenewald, Kemeny, Aziz, & Fahey, 2004; Rohleder, Beulen, Chen, Wolf, & Kirschbaum, 2007).

Thus, prior research indicates that interpersonal rejection and other social evaluative threats that signal the potential for social exclusion are associated with increased HPA activity. Heightened

HPA activity in such situations is assumed to be an adaptive response to acute social threat; it signals potential danger and motivates protective action. However, chronic or prolonged activation of the HPA axis can result in wear and tear on the body, which can lead to poor health outcomes including cardiovascular disease, strokes, ulcers, and decreased immunity (Cohen, Janicki-Deverts, & Miller, 2007; McEwen, 1998; Sapolsky, 1998). What factors may lead to a maladaptive pattern of HPA activation in response to rejection? Individual differences in the sensitivity of the HPA system to subtle or ambiguous social cues, which are prevalent in everyday life, may be one risk factor. For example, some individuals have a lower threshold for detecting rejection cues or for appraising them as threatening to the self, which can result in more frequent or more potent activation of the HPA system. Indeed, social self-preservation theory assumes that physiological responses to social threats will be modulated by individual differences in protective or vulnerability factors (Dickerson & Kemeny, 2004), but these factors have not yet been empirically identified. In the current investigation, we suggest that trait self-esteem may be one such factor that plays a key role in regulating the social self-preservation system and modulating HPA activation in response to ambiguous interpersonal rejection.

Self-Esteem as a Moderator of Responses to Rejection

There are many reasons to expect that self-esteem will modulate cortisol reactivity in response to interpersonal rejection, but two processes may be especially important: (a) Self-esteem may calibrate the threshold for detecting negative social evaluations, and (b) self-esteem may shape the appraisal processes through which negative social evaluations are translated into negative self-evaluations. There are a number of pathways through which these effects might occur.

Because individuals with LSE rely heavily on social approval to feel good about the self (Baldwin & Sinclair, 1996; Schneider & Turkat, 1975), the goal of feeling valued and discerning whether others truly care for them is likely to be chronically active (Murray, Holmes, & Collins, 2006). In addition, self-doubts and expectations of rejection—such as those reflected in LSE—make rejecting experiences more painful because they pose a greater proportional loss to a more vulnerable sense of worthiness (Leary & Baumeister, 2000; Murray, Holmes, & Collins, 2006). Hence, according to Murray, Holmes, and Collins's (2006) model of risk regulation in close relationships, individuals with LSE are likely to have a prevention-oriented cognitive-motivational system that quickly detects rejection, strongly signals the possibility of hurt, and motivates protective action. As a result, they are more likely to attend to rejection cues and to see rejection in ambiguous social cues (Baldwin, Baccus, & Fitzsimmons, 2004; Baldwin & Sinclair, 1996; Bellavia & Murray, 2003; Dandeneau & Baldwin, 2004; Murray, Holmes, & Griffin, 2000; Murray, Rose, Bellavia, Holmes, & Kusche, 2002; Sommer, Williams, Ciarocco, & Baumeister, 2001; Williams et al., 2000). In contrast, individuals with high self-esteem (HSE) should have less need for a defensively calibrated social alarm system because specific rejections pose a smaller proportional loss to a rich resource (Murray, Holmes, & Collins, 2006). Therefore, we reasoned that individuals with LSE should have a social self-preservation system that is calibrated such that even minor threats to social inclusion, or ambiguous cues to interpersonal rejection, would activate the HPA system.

Not only are they more likely to notice and attend to rejection cues, individuals with LSE are also more likely to process those cues in ways that are threatening to their overall sense of social worth (Baldwin et al., 2004; Leary et al., 1995; Murray et al., 2000; Murray et al., 2002; Nezlek et al., 1997; Williams et al., 2000). LSE individuals are more likely to believe that acceptance by others is contingent on possessing socially desirable qualities (Baldwin & Sinclair, 1996; Baumeister, 1993; Murray, Griffin, Rose, & Bellavia, 2006). Hence, they are likely to see rejection as evidence of a flawed self, which may lead them to make internal, self-blaming attributions for the rejection (Collins, Ford, Guichard, & Allard, 2006) and to question their desirability as a relationship partner. Moreover, because rejection experiences will be interpreted through the lens of their negative self schemas (Markus, 1977), negative aspects of the self will be easily accessible and may result in an overgeneralization of the experience to the self as a whole (Kernis, Brockner, & Frankel, 1989) and an increase in negative appraisals of the self (Kernis et al., 1989; Williams & Zadro, 2001). In contrast, for individuals with HSE, positive self-views and general expectations of acceptance should lead to external attributions for rejection and should buffer the impact of specific rejection experiences on their broader self-concept. Therefore, we hypothesized that individuals with LSE, compared with those with HSE, would experience ambiguous rejection cues as more threatening to their social self, leading them to feel more devalued and less worthy as a romantic partner. We also predicted that these appraisals of social self-threat (perceptions of a flawed or devalued self) would mediate self-esteem differences in cortisol reactivity.

If individuals with HSE and LSE differ in the way they construe interpersonal rejection—and in the degree to which it harms their overall sense of social worth and acceptance—then it is likely that they will also differ in their social responses to rejection. According to the risk-regulation model, individuals with LSE respond to social threat by prioritizing self-protection goals over relationship-promotion goals, resulting in an implicit if–then contingency rule that links feelings of rejection to social distancing behaviors. For example, in intimate relationships, individuals with LSE are more likely to respond to self-threat by derogating their romantic partner and their relationship (Murray et al., 2002). Derogation is thought to be a defensive strategy for protecting the self from additional hurt by distancing oneself from the source of social pain (MacDonald & Leary, 2005) and diminishing a partner's value as a source of social connection (Murray et al., 2002; Romero-Canyas & Downey, 2005). Thus a secondary goal in the current study was to investigate the links between cortisol reactivity and individual differences in social responses to rejection.

How might the social self-preservation system, and HPA activation in particular, shape social responses to rejection? Little is known about the links between cortisol reactivity and social behavior in humans, and there is unlikely to be a simple relationship between HPA activation and specific social actions. Nevertheless, to the extent that the HPA system is implicated in the fight or flight

response and serves to motivate defensive behavior (McEwen, 2002; Sapolsky, 1998), HPA activation may act as an alarm system that signals social threat and motivates the defensive responses to rejection that are characteristic of LSE individuals. These defensive behaviors may take a variety of forms. When individuals have the opportunity to avoid rejection or gain acceptance, they may respond to cues of rejection by attempting to make themselves more desirable as relationship partners (Sommer et al., 2001). However, in cases in which rejection is unavoidable they may engage in other defensive behaviors that allow them to protect the self in the face of uncontrollable rejection. These behaviors may include social withdrawal or distancing behaviors, such as derogation of the rejector (see also MacDonald & Leary, 2005). By derogating the rejector, individuals can create distance between themselves and the source of the social evaluative threat, repairing their damaged ego by perceiving the rejector as a less appealing and less worthy social partner.

In light of prior research and theory linking rejection with defensive behaviors (particularly for those with LSE) we chose to focus on partner derogation as one form of defensive behavior. Although prior research has demonstrated that social evaluative threat is independently associated with both HPA activation and defensive behaviors, researchers have not yet investigated the relationship between HPA activation and defensive behaviors. We sought to bridge this gap by investigating HPA activation as a biological mediator of defensive social behavior. We predicted that individuals with LSE would be more likely than those with HSE to derogate an interaction partner following rejection (rating his/her traits more harshly) and that these differences in partner derogation would be explained, at least in part, by differences in social self-threat and subsequent cortisol reactivity.

The Present Study

In the current investigation, we examined the influence of self-esteem on psychological and neuroendocrine (cortisol) responses to an ambiguous interpersonal rejection by a potential dating partner. We used an intentionally ambiguous rejection manipulation—one that contained a high degree of attributional ambiguity—so that we could examine individual differences in social construal and sensitivity to social threat. We predicted that individuals with LSE (relative to those with HSE) would appraise the rejection as more harmful to the social self (as evidenced by more negative social self-evaluations, more self-blaming attributions, and greater emotional distress), would show greater increases in cortisol reactivity, and would be more likely to engage in defensive social action (as evidenced by higher levels of partner derogation).

Finally, we proposed and tested a mediational model (see Figure 1) to explain the links between self-esteem and psychological and neuroendocrine responses to rejection. According to this model, individuals with LSE would be more likely to appraise the rejection as threatening to the social self, which would then result in increased HPA activation (cortisol reactivity). Increases in HPA



Figure 1. Hypothesized model of responses to rejection.

activation would then predict increases in self-protective action (partner derogation).

Method

Participants. Participants were 78 undergraduate students (39 men, 39 women) between the ages of 17 years and 32 years ($M = 19.6$, $SD = 2.7$).¹ All participants were single (not currently in a romantic relationship) and were screened to rule out conditions or behaviors that might affect cortisol secretion, such as clinical depression, cigarette use, and the use of certain medications (Crook, 1997; Hibel, Granger, Kivlighan, & Blair, 2006; Masharani et al., 2005). Participants received either \$20 or course credit for their participation.

Procedure. Participants were recruited for a two-part study involving investigation of their physiological responses to a variety of activities, including online dating activities.

Session 1: Questionnaire session. Participants completed a background questionnaire that contained demographic and personality measures, including the 10-item Rosenberg Self-Esteem Scale (Rosenberg, 1965). Next, they wrote a personal description of themselves and had their picture taken. They were led to believe that these items would be shown to another participant during the second session of the study. Finally, participants were scheduled to return to the lab 1 week later and were given guidelines to adhere to on the day of their second session. These guidelines were designed to prevent participants from engaging in activities that might interfere with their production of cortisol or contaminate samples of salivary cortisol.

Session 2: Experimental session. Participants reported individually for a 2 hr laboratory session. To control for diurnal variations in cortisol production, all sessions were scheduled between 2:15 p.m. and 6:30 p.m. (Kirschbaum & Hellhammer, 1989).

For the first 20 min, participants relaxed and acclimated to the lab by reading magazines and filling out a short questionnaire that assessed their health behaviors that day. Next, participants provided a sample of saliva, which served as the first baseline cortisol measure. Because the release of cortisol takes approximately 20 min to be expressed in saliva, this sample reflected participants' HPA activity when they arrived at the lab 20 min earlier.

In the next phase of the study, participants were led to believe that they would be participating in a 10-min online chat with an opposite-sex participant who was ostensibly working in a different room. First, they were asked to read about the other participant and view his or her photo, and they were led to believe that the other participant was also reading about them. In reality, participants were presented with a standard set of materials that had been pilot-tested to ensure that the false interaction partner was moderately attractive and that the male and female versions were similar in physical attractiveness and interpersonal appeal. Next participants answered a short questionnaire that assessed premanipulation self-appraisals, impressions of the interaction partner, and mood.

After 10 min (and immediately before the online chat was to begin) the experimenter delivered the rejection manipulation. To create an ambiguously rejecting situation (and also a nonrejecting, control situation), we adapted a methodology used by Ayduk, Downey, Testa, Yen, and Shoda (1999). Participants in the rejection condition were told that there would be no online chat because

the other participant "chose not to continue with the experiment." This information was designed to be ambiguous and to provide room for subjective construal. Participants in the control condition were told that the other participant could not continue with the study because he or she was feeling very sick. This information was designed to be unambiguous by providing a clear external reason for the partner's decision to discontinue with the study.

Next, the experimenter collected the second saliva sample, which reflected participants' HPA activity 15–20 min prior to the manipulation and which served as the second baseline measure. Participants then completed another questionnaire that assessed their postmanipulation self-appraisals, impressions of their interaction partner, and mood. Finally, participants relaxed and read neutral magazines for the remaining hour while three postmanipulation saliva samples were taken at 20 min intervals.

At the end of the study, participants completed a final questionnaire that included an open-ended measure of attributions for the rejection and a manipulation check. Participants were then carefully debriefed.

Session 2: Salivary cortisol measures. Saliva samples were collected with salivettes and were kept frozen at -80° Celsius until they were shipped on dry ice to an independent laboratory (Salimetrics LLC, State College, Pennsylvania) for analysis. Each sample was assayed in duplicate with a high sensitivity enzyme immunoassay. The test had a lower limit of sensitivity of $0.003 \mu\text{g/dl}$, a standard curve range of 0.007 to $1.8 \mu\text{g/dl}$, and average intra- and interassay coefficients of variation 8.45% and 8.2% , respectively. Method accuracy, determined by spike recovery, and linearity, determined by serial dilution, were 103% and 96% .² The average of the duplicate tests was used in all analyses.

Session 2: Self-report measures. Preinteraction expectations. Prior to the manipulation, participants answered two questions to assess how important the upcoming interaction was to them and how well they thought the interaction would go. Both questions were answered on a scale from 1 (*not at all*) to 7 (*extremely*). Participants also rated their partner's physical attractiveness on a 7-point scale.

Self-appraisals. Prior to and following the manipulation, participants rated themselves on a series of interpersonal qualities (items were rated on a 6-point scale). An index of social self-appraisals (popular, fun to be with, well-liked by others, socially incompetent—reverse scored) was computed for each time (T) point ($\alpha_{T1} = .85$, $\alpha_{T2} = .82$).

¹ An additional 7 participants were recruited for this study but were excluded from the present analyses (5 from the experimental group and 2 from the control group). One gay participant was excluded because he reported being uncomfortable responding to the opposite-sex participant. Five participants were excluded due to suspicion, and 1 was excluded due to a failure to properly follow the procedures necessary to ensure a clean saliva sample.

² Tests of spike recovery and linearity are two parameters used in bioanalytic validation of cortisol assays. Spike recovery establishes accuracy of the cortisol measurements by spiking (inserting) a known quantity of cortisol into an existing sample and assaying it. The acceptable range for spike recovery is between 80% and 120% . Linearity involves serially diluting samples of known amounts of cortisol and comparing observed values with expected values. Perfect linearity would be 100% .

Partner derogation. Prior to and following the manipulation, participants rated their interaction partner on a series of interpersonal traits (items were rated on a 7-point scale). To measure partner derogation, we computed an index of negative partner evaluations (critical and judgmental, rude, thoughtless, complaining, controlling and dominant, emotional or moody, immature, anxious, irrational) for each time point ($\alpha_{T1} = .85$, $\alpha_{T2} = .94$).

Mood. Prior to and following the manipulation, participants described how they were feeling on a series of emotions (items were rated on a 5-point scale). On the basis of a principal components analysis, we computed an index of positive mood (happy, enthusiastic, confident, comfortable, relaxed, calm; $\alpha_{T1} = .77$, $\alpha_{T2} = .82$) and negative mood (sad, irritable, hopeless, low, downcast, distressed; $\alpha_{T1} = .87$, $\alpha_{T2} = .88$) at each time point.

Self-blame attributions. Self-blame attributions were coded from a free response question included in the final questionnaire. Participants were asked "Why do you think the other participant did not continue with the study?" Responses were coded by two raters who were unaware of participants' experimental condition or level of self-esteem. Responses were coded as 0 if the participant made no self-blame attributions and as 1 if the participant made any self-blame attributions. Some examples of self-blame attributions were "The other participant disliked my statement/picture," and "I think she thought I was probably weird due to my personal description I wrote and photo." Interrater agreement was 97%, and disagreements were settled by Máire B. Ford. Self-blame attributions were spontaneously made by 17% of the total sample.

Manipulation check. At the end of the final questionnaire, participants rated the degree to which they felt rejected by the other participant on a scale from 1 (*not at all*) to 7 (*extremely*).

Results

Preliminary analyses.

Premanipulation motivation and expectations. Before testing our primary hypotheses, we wanted to determine whether HSE and LSE individuals differed in their premanipulation motivations and expectations. We found that there was no significant correlation between self-esteem and ratings of partner attractiveness ($r = .11$, $p = .35$) or importance of the interaction ($r = .13$, $p = .27$). Thus, HSE and LSE individuals were equally attracted to their partner

and equally invested in the upcoming interaction. However, individuals with HSE had more positive expectations about how well the interaction would actually go ($r = .37$, $p < .01$).

Manipulation check. To determine whether our rejection manipulation was effective, we conducted a regression analysis predicting feelings of rejection from experimental condition (0 = control, 1 = rejection) and self-esteem (continuous) on Step 1 and the Condition \times Self-Esteem (SE) interaction on Step 2. Results revealed a significant main effect of experimental condition ($\beta = .40$, $p < .001$), with participants in the rejection condition feeling more rejected than those in the control condition ($M_{\text{rejection}} = 4.14$, $M_{\text{control}} = 2.61$). There was also a significant main effect of SE ($\beta = -.40$, $p < .001$), with LSE participants feeling more rejected on average. Finally, there was no SE \times Condition interaction ($\beta = -.01$, $p = .93$), indicating that the manipulation was equally effective for LSE and HSE individuals. An examination of the predicted means based on the two additive main effects shows that LSE and HSE participants in the rejection condition felt more rejected ($M_{\text{LSE}} = 4.9$, $M_{\text{HSE}} = 3.3$) than did those in the control condition ($M_{\text{LSE}} = 3.4$, $M_{\text{HSE}} = 1.9$), but that LSE individuals in the rejection condition felt the most rejected overall.

Psychological responses to rejection.

Changes in self-appraisals. A hierarchical regression analysis predicting postmanipulation self-appraisals controlling for premanipulation self-appraisals revealed no main effect of condition ($\beta = -.07$, $p = .20$) or SE ($\beta = .07$, $p = .25$) but did reveal a significant interaction ($\beta = .18$, $p = .04$). As shown in Figure 2, an analysis of simple slopes (Aiken & West, 1991) revealed no effect of SE in the control condition (simple $\beta_{\text{control}} = -.05$, $p = .60$) but did reveal a significant effect in the rejection condition (simple $\beta_{\text{rejection}} = .20$, $p = .02$), with LSE individuals reporting a significant decrease in social self-appraisals following rejection.

Self-blame attributions. Because self-blame attributions was a dichotomous outcome (0 = no self-blame, 1 = self-blame), we used logistic regression for this dependent variable. Our initial analysis would not converge to an adequate solution due to low frequencies in the control condition; only 2 of 36 participants (5.5%) made self-blame attributions in this condition, compared with 11 of 42 participants (26%) in the rejection condition, $\chi^2(1$,

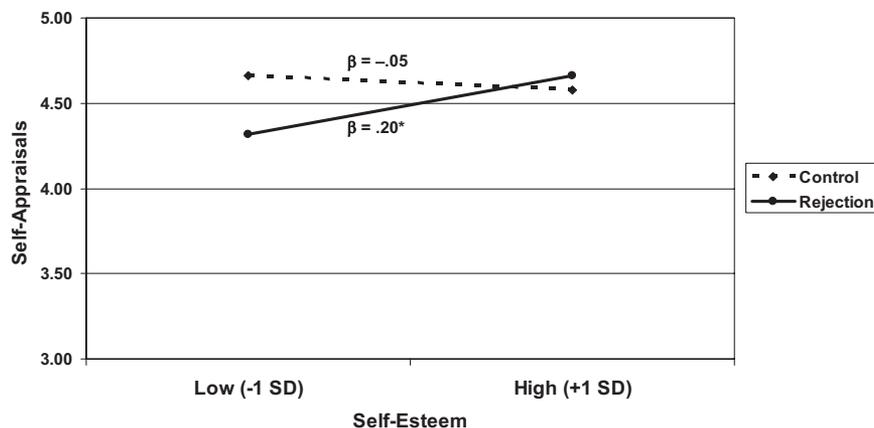


Figure 2. Social self-appraisals by self-esteem and condition. * $p < .05$.

$N = 78$) = 5.94, $p = .02$. (This pattern is not surprising, given that these participants were given a clear external explanation for their partner's decision to discontinue with the study.) Therefore, we conducted a more focused analysis predicting self-blame attributions from SE within the rejection condition only. Results revealed a significant effect of SE ($b = -1.02$), Wald $\chi^2(1, N = 42) = 5.26$, $p < .01$; individuals with LSE were much more likely than those with HSE to blame themselves for the rejection (42% vs. 9%).³

Derogation of the interaction partner. A regression analysis predicting postmanipulation partner evaluations controlling for premanipulation evaluations revealed a significant effect of condition ($\beta = .25$, $p < .01$), no effect of SE ($\beta = -.09$, $p = .24$), and a significant interaction ($\beta = -.24$, $p = .03$). As shown in Figure 3, there was no association between SE and partner derogation in the control condition (simple $\beta_{\text{control}} = .11$, $p = .32$), but there was a significant negative association in the rejection condition (simple $\beta_{\text{rejection}} = -.21$, $p = .03$), with LSE individuals derogating their partner more in response to rejection.

Mood. A regression analysis predicting postmanipulation positive mood controlling for premanipulation positive mood revealed no significant effect of condition ($\beta = .08$, $p = .30$) or SE ($\beta = .06$, $p = .52$) and no interaction ($\beta = -.07$, $p = .56$). Likewise, results for negative mood revealed no effect of condition ($\beta = -.07$, $p = .22$) or SE ($\beta = -.09$, $p = .11$) and no interaction ($\beta = .06$, $p = .51$). Thus, participants reported no changes in mood following the rejection manipulation, and there was no link between SE and changes in mood.

Cortisol reactivity. Because cortisol scores were slightly positively skewed we square-root transformed them for all analyses. Next, we computed a single baseline value by taking the mean of the first two samples, both of which reflected HPA activity prior to the manipulation. For descriptive purposes, Figure 4 shows mean levels of pre- and postmanipulation cortisol for all groups.⁴

Before testing our hypotheses, we explored whether there were any individual differences in baseline cortisol. Regression analyses revealed no effect of SE ($\beta = .15$, $p = .19$) and no SE \times Condition interaction ($\beta = -.14$, $p = .40$). However, we did find an unexpected effect of experimental condition ($\beta = -.32$, $p < .01$). Participants in the control group had slightly higher baseline cortisol levels ($M = .28$, $SD = .12$) than did those in the rejection condition ($M = .20$, $SD = .11$).⁵ To control for this difference, and to explore within-person changes in cortisol levels, we entered baseline as a predictor in all analyses.⁶

Stress phase cortisol. The analysis of stress phase cortisol revealed no effect of condition ($\beta = -.02$, $p = .80$), a significant effect of SE ($\beta = -.22$, $p < .01$), and a significant interaction ($\beta = -.24$, $p = .05$). As shown in Figure 5, there was no association between SE and cortisol reactivity in the control condition (simple $\beta_{\text{control}} = -.04$, $p = .78$), but there was a significant negative association in the rejection condition (simple $\beta_{\text{rejection}} = -.35$, $p < .01$), with LSE individuals exhibiting higher levels of stress phase cortisol following rejection.

Early recovery phase cortisol. Regression analysis revealed no main effect of condition ($\beta = -.03$, $p = .74$), a marginal effect of SE ($\beta = -.17$, $p = .06$), and a significant interaction ($\beta = -.29$, $p = .04$). Once again, there was no association between SE and cortisol in the control condition (simple $\beta_{\text{control}} = .05$, $p = .71$), but there was a significant negative association in the rejection

condition (simple $\beta_{\text{rejection}} = -.33$, $p < .01$), with LSE individuals exhibiting higher levels of early recovery cortisol following rejection.

Late recovery phase cortisol. Regression analysis revealed no effect of condition ($\beta = -.07$, $p = .50$) or SE ($\beta = .00$, $p = .97$) but did reveal a significant interaction ($\beta = -.33$, $p = .04$). This interaction, however, was driven by differences in the control condition, not the rejection condition. By the late recovery phase, HSE and LSE individuals in the rejection condition no longer differed significantly in their cortisol levels (simple $\beta = -.18$, $p = .19$). However, in the control condition, individuals with HSE exhibited marginally higher levels of cortisol (simple $\beta = .26$, $p = .10$), suggesting that they were approaching their resting baseline somewhat more slowly than were individuals with LSE. Nevertheless, as illustrated in Figure 4, all participants in the control condition exhibited a steady decline in cortisol levels across the 2 hour study period.

Area under the curve. The analyses thus far indicate that participants with LSE had higher levels of stress phase and early recovery cortisol following an ambiguous interpersonal rejection. Moreover, Figure 4 shows that the only participants who experienced an increase in HPA activity following the manipulation were those with LSE in the rejection condition. But, did LSE individuals experience significant increases in cortisol levels over baseline, or did they simply fail to decline from the baseline measure as rapidly as those with HSE? To address this question, we examined the stress phase and early

³ Because self-esteem is a continuous variable, these values are predicted proportions computed at 1 SD above and below the mean on self-esteem.

⁴ The values shown in this graph for the HSE and LSE groups are the predicted values (plotted at 1 SD above and below the mean on SE) based on moderated regression analyses. Also, 1 participant in the control condition had an extremely high (greater than 3 SDs above the mean) baseline level of cortisol and was, therefore, excluded from any analyses involving cortisol.

⁵ We carefully examined the data to determine whether the unexpected baseline difference was driven by an outlier, but there was no evidence that this was the case. We also compared the control and experimental groups on a long list of variables that were assessed in our background questionnaire to determine whether the groups differed on factors that might be relevant to cortisol reactivity or to the psychological processes investigated in this study. We found no group differences on personality factors (e.g., self-esteem, neuroticism, anxious attachment, avoidant attachment), emotional well-being (e.g., depression, anxiety, somatic symptoms), or demographic factors (e.g., gender, age, weight). We also compared the groups on all premanipulation measures (self-evaluations, partner evaluations, and mood) and found no differences. Thus, the initial difference in baseline cortisol was not systematically related to major factors known to affect cortisol levels or to the psychological processes being investigated in this study.

⁶ Although baseline levels of cortisol will be controlled in all analyses, it is also important to note that the primary goal in our study is to examine self-esteem as a moderator of cortisol reactivity in response to rejection. It is important to note there were no self-esteem differences in baseline levels of cortisol and no Self-Esteem \times Condition interactions at baseline. Thus, the unexpected elevation in control group cortisol at baseline is unlikely to interfere with our ability to draw clear inferences about the effects of self-esteem on stress phase cortisol reactivity in response to rejection.

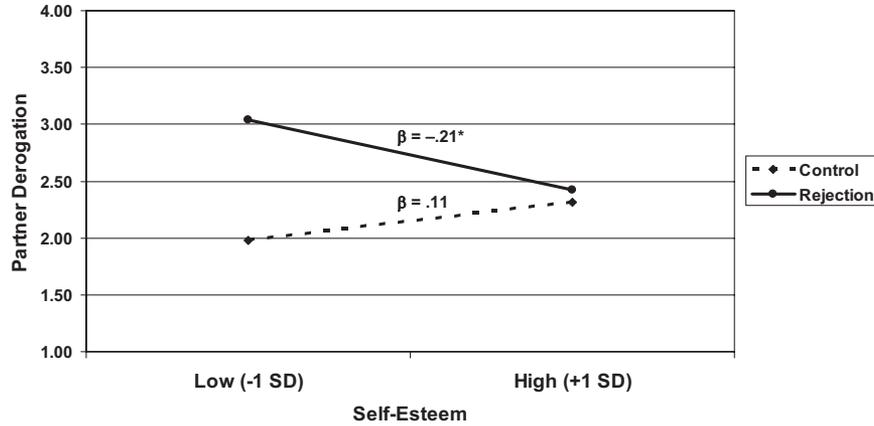


Figure 3. Partner derogation by self-esteem and condition. * $p < .05$.

recovery cortisol scores more closely by computing an index of area under the curve with respect to increases over baseline (AUC_1), using the trapezoid formula (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). The AUC_1 index provides a single score that quantifies each participant's total cortisol concentration relative to baseline across repeated measurements. Individuals who showed no total increase over baseline were assigned a score of zero. Thus, this index provides an unambiguous measure of total increases over baseline during the stress and early recovery phase.

A regression analysis predicting AUC_1 revealed no significant effect of condition ($\beta = .17, p = .14$), a significant effect of SE ($\beta = -.29, p = .01$), and a significant interaction ($\beta = -.37, p = .03$). As shown in Figure 6, simple slopes analysis revealed a strong and significant association between SE and AUC_1 in the rejection condition (simple $\beta_{\text{rejection}} = -.48, p < .01$) but no association in the control condition (simple $\beta_{\text{control}} =$

.00, $p = .98$). Thus, individuals with LSE showed significantly greater HPA activity in response to rejection, as evidenced by increases in cortisol reactivity during the stress phase and early recovery phase.

Gender. We conducted a final series of regression analyses to investigate whether any of our cortisol effects were moderated by gender. We found no significant interactions with gender.

Testing the mediation model. In our final analysis, we used structural equation modeling to test the proposed theoretical model shown in Figure 1. This model hypothesized that the link between SE and cortisol reactivity (HPA activation) in response to rejection would be mediated by perceived threat to the self and that cortisol reactivity would, in turn, predict self-protective action. Because these processes were hypothesized to operate only in the presence of rejection—and, as expected, there were no SE effects in the control condition—we tested this model only within the rejection condition.

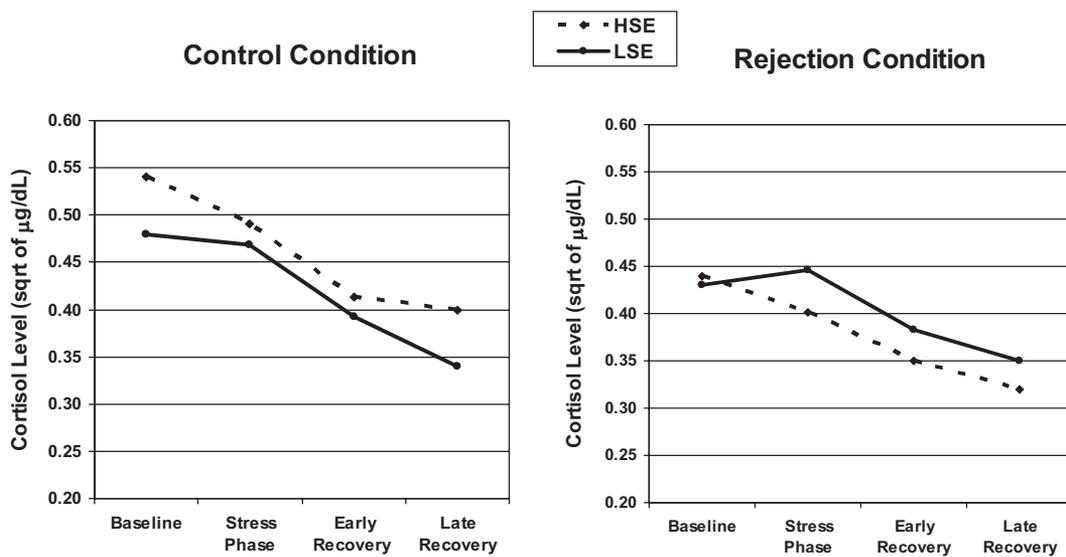


Figure 4. Mean cortisol levels at each time point by self-esteem and condition. HSE = high self-esteem; LSE = low self-esteem; sqrt = square root.

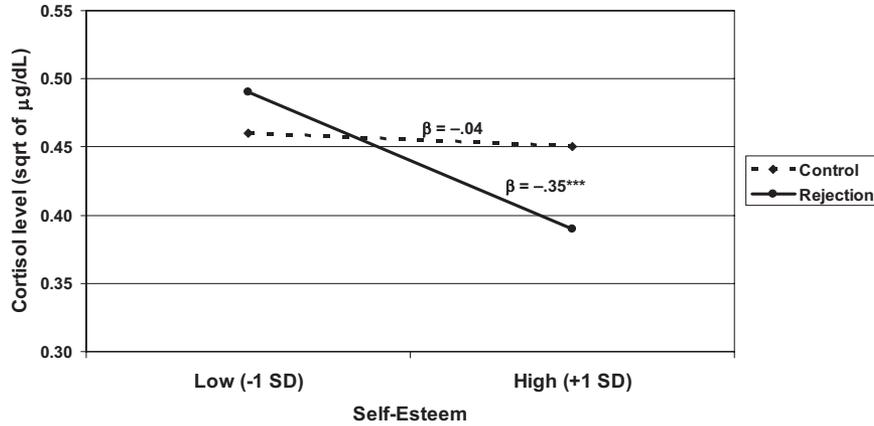


Figure 5. Stress phase cortisol levels by self-esteem and condition. Sqrt = square root. *** $p < .001$.

Model specification. We specified a path model in which perceived self-threat was indexed by two observed variables, self-blame attributions and negative social self-appraisals. (Mood was excluded from the model because it was unrelated to SE and rejection.) Next, HPA activation was indexed by our stress phase cortisol variable, which was most temporally proximal to the stressor and thus provided the most powerful index of HPA activation. Finally, social distancing was indexed by our partner derogation variable. For variables that were measured before and after the manipulation (self-evaluations and partner derogation), we used residualized change scores to determine whether changes in appraisals from pre- to postmanipulation predicted cortisol reactivity. In addition, stress phase cortisol was a residualized change score controlling for baseline cortisol.

Model estimation. To test the model, we used AMOS software (Arbuckle, 2003) and maximum likelihood estimation. Model fit was assessed with a joint consideration of the chi-square statistic, the chi-square-to-degrees-of-freedom ratio (χ^2/df), the standardized root-mean residual (SRMR, the average absolute correlation residual), the comparative fit index (CFI), and the root-mean-square error of approximation (RMSEA). Desirable model fit is evidenced by a nonsignificant χ^2 , a χ^2/df ratio of 3 or

lower, a SRMR of .10 or less, a CFI of .95 or greater, and a RMSEA no greater than .08 and preferably less than .05 (Kline, 2005). In addition, because of our relatively small sample, and because one of our mediators (self-blame attributions) was a dichotomous variable, we verified all of our results by running additional analyses with bootstrapped parameter estimates and standard errors.

The estimated model with standardized path coefficients is shown in Figure 7. The goodness-of-fit statistics indicated that the model fit the data very well, $\chi^2(5, N = 42) = 5.42, p = .37$; $\chi^2/df = 1.083$; SRMR = .08, CFI = .985, RMSEA = .045. As shown in Figure 7, LSE individuals were more likely to make self-blaming attributions ($\beta = -.41, p < .01$) and to experience negative social self-appraisals ($\beta = -.37, p = .01$) following rejection. Self-blame attributions were, in turn, significantly associated with increased stress phase cortisol ($\beta = .47, p < .01$). Negative self-appraisals were also positively associated with increased cortisol, but this association was not significant ($\beta = .17, p = .21$). (It should be noted that this was not simply due to the presence of self-blame in the model, as negative self-appraisals did not serve as a significant mediator even when self-blame was removed.) Finally, individuals who experienced higher stress

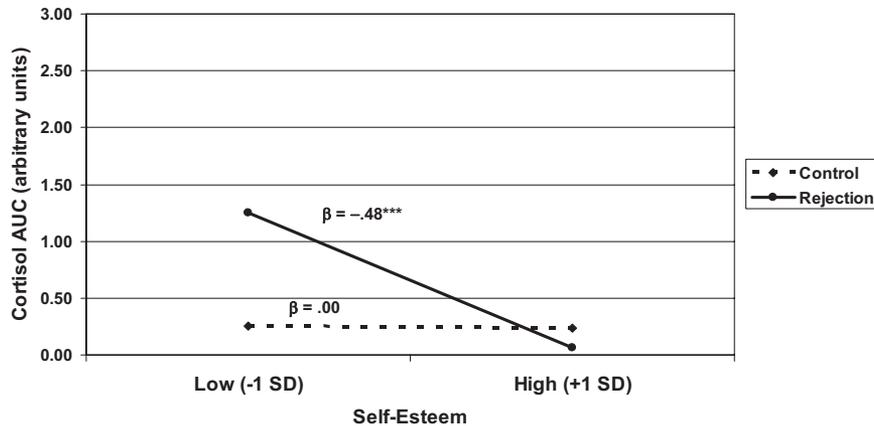


Figure 6. Cortisol AUC by self-esteem and condition. AUC = area under the curve. *** $p < .001$.

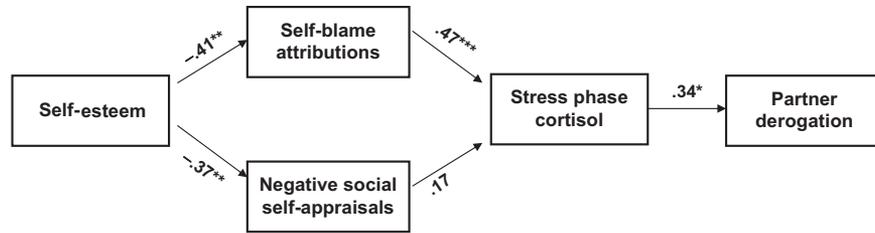


Figure 7. Path analysis testing the mediation model within the rejection condition. $\chi^2(5, N = 42) = 5.42, p = .37$; $\chi^2/df = 1.083$; standardized root-mean residual = .08; comparative fit index = .985; root-mean-square error of approximation = .045. * $p < .05$. ** $p < .01$. *** $p < .001$.

phase cortisol were more likely to derogate their partner ($\beta = .34, p = .02$). We ran an additional model in AMOS in which we estimated the path coefficients (and corresponding standard errors) with bootstrapping. This analysis resulted in path coefficients and significance tests (based on confidence intervals) that were highly similar to those shown in Figure 7.

The above model assumes full mediation of the SE effects, and although this model provided a good fit to the observed data, it is possible that the SE effects are only partially mediated. Hence, we tested two nested models in which we added direct effects between SE and cortisol and between SE and partner derogation, one at a time, and computed the change in the chi-square statistic ($\Delta\chi^2$). A significant $\Delta\chi^2$ indicates that the addition of the direct effect significantly improved model fit. In our first model, we added a direct path from SE to stress phase cortisol. This path did not improve model fit, $\Delta\chi^2(1) = 2.51, p = .11$. In addition, the direct path from self-esteem to cortisol was not significant ($\beta = -.24, p = .11$), but the path from self-blame attributions to cortisol remained significant ($\beta = .39, p < .01$). In our second model, we added a path from SE to partner derogation. This path did not improve model fit, $\Delta\chi^2(1) = 1.06, p > .50$. In addition, the path from SE to partner derogation was not significant ($\beta = -.17, p = .27$), but the path from stress phase cortisol to partner derogation was marginally significant ($\beta = .27, p = .08$). Thus we can conclude that the initial, fully mediated model provided the best fit to the data. However, because our sample size is relatively small and the remaining direct effects were greater than zero, it is more prudent to conclude that we have evidence of partial mediation.

Lastly, to rule out the possibility that negative appraisals (rather than HPA activation) are the more proximal predictor of derogation, we tested an additional alternative model. In this model we reordered variables so that SE predicted cortisol level, which then predicted perceived threat to the self (self-blame attributions and negative evaluations), which in turn predicted derogation. Path coefficients for this model indicated that self-esteem significantly predicted stress phase cortisol ($\beta = -.43, p < .01$), stress phase cortisol significantly predicted self-blame attributions ($\beta = .50, p < .001$), and self-blame attributions predicted partner derogation ($\beta = .32, p = .03$). However, stress phase cortisol did not significantly predict negative social self-appraisals ($\beta = .25, p = .10$), and negative social self-appraisals did not predict derogation ($\beta = -.06, p = .69$). Because this alternative model and our original model were not nested, we were unable to compute a chi-square difference test between them. However, the fit indices for this alternative model revealed a relatively poor fit to the data, $\chi^2(5,$

$N = 42) = 9.994, p = .08, SRMR = .11, CFI = .82, RMSEA = .155$, and a comparison of these fit indices with those obtained for the original model (see above) shows that the original model provided a much better fit than did the alternative model.

Taken together, these results are consistent with our proposed theoretical model. They suggest that individuals with LSE experienced significant increases in HPA reactivity in large part because they blamed the rejection on something negative or unworthy about the self. Increases in HPA reactivity then partially explained why LSE individuals were more likely to socially distance themselves from their partner by derogating them as a potential source of social connection.

Discussion

The overarching goal of this investigation was to gain a deeper understanding of the role of self-esteem as part of a broader psychobiological response system for regulating and coping with threats to social acceptance. Overall, results showed dramatic differences between HSE and LSE individuals in their psychological and neuroendocrine responses to ambiguous interpersonal rejection. As predicted, compared to those with HSE, individuals with LSE reported significant declines in social self-evaluations, were more likely to blame themselves for the rejection, and were more likely to derogate their interaction partner's interpersonal traits and personal qualities. LSE individuals also exhibited a greater physiological stress response as indicated by increases in cortisol levels immediately following the rejection and during the early recovery phase.

These findings provide clear evidence that individuals with LSE experienced even a mild interpersonal rejection as a meaningful threat to their social worth. In contrast, HSE individuals experienced the very same social cues as much less threatening; although they reported feeling rejected, they were buffered from further harm to the self. They showed no declines in social self-appraisals, made external rather than internal attributions for the rejection, and showed no evidence of partner derogation, suggesting little need for self-protection. Most importantly, they showed no increase in cortisol levels. Thus, although they acknowledged that their interaction partner may have lacked interest in them, individuals with HSE stopped short of internalizing this information and allowing it to pose a threat to their overall sense of worthiness. It is important to note that differences between HSE and LSE individuals in the rejection condition cannot be attributed to a general pattern of negative affectivity or social anxiety associated with LSE because

we found no self-esteem differences in psychological or physiological responses in the control condition—in which participants were given a clear external attribution for their partner's decision to end their interaction.

Results also provided support for our proposed mediation model in which we tested several key hypotheses concerning the links between psychological and physiological responses to rejection. Path analysis revealed that within the rejection condition, individuals with LSE experienced a greater degree of social self-threat, which in turn predicted higher stress phase cortisol levels. This greater sense of physiological alarm then predicted greater partner derogation. Thus, as predicted, the association between self-esteem and cortisol reactivity was mediated by self-threat (primarily self-blame attributions), and the association between self-esteem and partner derogation was mediated by cortisol reactivity (and indirectly mediated by self-blame attributions).

One feature of the path analysis deserves additional comment. Although both self-blame attributions and negative self-evaluations were associated with LSE and increased cortisol reactivity, self-blame clearly played a stronger mediating role. One reason for this finding may be that our measure of self-blame was better at differentiating those who experienced a potent sense of social devaluation from those who were only mildly offended—individuals who made self-blaming attributions clearly located the rejection in a flawed self. In contrast, our measure of negative social self-evaluations may have captured a lowered sense of social acceptance without necessarily implying that it was rooted in something inherently unworthy about the self. In fact, there was only a weak correlation between declines in social self-appraisals and self-blame attributions ($r = .17$). Although this distinction may seem subtle, we believe it may have important theoretical implications in terms of identifying the precise cognitions that trigger a meaningful threat to social self-preservation. It is also noteworthy that self-blame attributions were correlated with partner derogation ($r = .31, p = .06$), but changes in self-evaluations were not ($r = .01$), which again suggests that self-blame attributions did a better job at identifying those who felt more threatened and, hence, more motivated to engage in self-protective action.

We had predicted that self-esteem differences in injury to the self would also be revealed in feelings of emotional distress following rejection. However, the rejection manipulation had no discernable effects on mood. Instead the results revealed that LSE individuals reported more negative affect regardless of condition and that they did not display significantly more negative affect than HSE individuals following rejection. Although these results may seem surprising, our findings parallel a number of other studies that have failed to show differences in self-reported affective responses to rejection (e.g., Leary, Haupt, Strausser, & Chokel, 1998) and that show that broad affective measures, such as the one used in this study, may be insensitive to brief rejection manipulations within the laboratory (Gardner, Pickett, & Brewer, 2000; Nezlek et al., 1997). One reason may be that the effects of rejection on affective states, and subsequent links to HPA activation, are limited to only certain emotional states. For instance, recent work suggested that rejection and other forms of social evaluative threat may elicit the shame family of emotions (shame, embarrassment, humiliation), which appears to play a key role in HPA activation (Dickerson, Gruenewald, & Kemeny, 2004; Gruenewald, Dickerson, & Kemeny, 2007). Rejection may also trig-

ger hurt feelings rather than generalized distress (MacDonald & Leary, 2005). We were not aware of these findings when we were designing our study, so we did not measure shame or hurt feelings. In future studies, it will be important to measure these additional emotional states as well as to include implicit measures of emotion whenever possible.

Theoretical implications. This investigation has a number of implications for psychobiological models of social stress and for interpersonal models of self-esteem. First, this study adds to a small but growing literature on the physiological and neural processing dynamics of social rejection. Consistent with social self-preservation theory (Dickerson, Gruenewald, & Kemeny, 2004) and social pain theory (Eisenberger & Lieberman, 2004; MacDonald & Leary, 2005), our findings demonstrate that threats to social acceptance—even relatively mild threats—are capable of activating the HPA system. Our results extend this work by highlighting the importance of individual differences in threat sensitivity and by demonstrating the mediating role of appraisals of social devaluation. Our study is the first to show that HPA activation in response to interpersonal rejection is moderated by self-esteem and mediated by self-blame attributions. These findings provide empirical support for two key, but previously untested, theoretical assumptions of social self-preservation theory, namely that HPA activation will be modulated by protective or vulnerability factors within the individual and that HPA activation will be directly linked to subjective appraisals of social devaluation.

Additionally, our findings contribute to the literature on risk regulation in close relationships (Murray, Holmes, & Collins, 2006) and to the broader literature on social responses to rejection (e.g., Bourgeois & Leary, 2001; Sommer, 2001; Twenge, Baumeister, Tice, & Stucke, 2001; Warburton, Williams, & Cairns, 2006). Our study replicates the previously established link between LSE and defensive social responses to rejection and sheds new light on the potential mechanisms through which these behaviors may be triggered. Our findings show that for people with LSE, even mild cues to interpersonal rejection are sufficiently powerful to trigger a biological stress response and that this stress response may play a role in shaping defensive social behaviors that may further weaken their social connections. In doing so, our study goes beyond prior studies in specifying both psychological and biological mediators that may play a role in motivating defensive responses to rejection, and it underscores the importance of investigating biological mediators of social behavior.

Our findings suggest that individual differences in cortisol reactivity may help explain individual differences in partner derogation, but the relationship between stress hormones and social behavior is not well understood. From a theoretical perspective, there are at least two ways in which we can conceptualize the role of cortisol as a mediator of partner derogation. First, cortisol may be conceptualized as an implicit, biological marker of psychological threat. In this view, cortisol levels are associated with partner derogation because higher levels of cortisol reflect an increased state of psychosocial threat. Second, cortisol may be more directly involved in shaping behavior by mobilizing or potentiating self-protective action. Of course, any pathways relating cortisol to social behavior are likely to be extremely complex, and we do not suggest that cortisol directly triggers specific social responses. Cortisol is involved in a wide range of metabolic functions and has

diverse effects on physiology and the brain (see Erickson, Drevets, & Schulkin, 2003, for a review). Nevertheless, there are good reasons to speculate that cortisol may be implicated in shaping social behavior. A small number of experimental studies have shown that administration of glucocorticoids can affect the interpretation of emotional stimuli and subjective feelings of arousal in humans (e.g., Schmidt, Fox, Goldberg, Smith, & Schulkin, 1999; Soravia et al., 2006) and the expression of aggressive behavior in nonhuman species (Haller, Halász, Mikics, & Kruk, 2004). Cortisol also increases activation in some areas of the brain, including the amygdala, which is implicated in the perception, memory, and experience of emotional events (Erickson, Drevets, & Schulkin, 2003, for a review). The wide ranging effects of cortisol on metabolic and cognitive functioning makes it reasonable to speculate that it may influence the production of social behaviors. In a recent review, Erickson et al. (2003) concluded that cortisol may affect behavior by increasing the readiness to behave—the likelihood of performing certain behaviors in suitable environments. Thus, although the current study cannot explain the specific mechanism linking cortisol reactivity to partner derogation, we believe our findings point to a potential role for cortisol in shaping both adaptive and maladaptive responses to interpersonal rejection and other social stressors. We view the current finding as preliminary and worthy of additional research attention.

Results from this study also advance our understanding of psychosocial variables that contribute to stress reactivity and to health. Stress researchers have suggested that personality may play an important role in driving perceptions of external stressors, which can impact HPA activation and ultimately health (Chida & Hamer, 2008; McEwen, 2002; Sapolsky, 1990, 1999). The current study suggests that self-esteem may be one important individual difference variable that shapes stress appraisals and has implications for long-term health and well-being. Specifically, the current study shows that people with LSE have an HPA system that is easily triggered, even in response to mild or ambiguous rejection cues. Because daily social life is complex, and social threats such as rejection are often subtle and ambiguous, people with LSE are at risk for over-perceiving threat. This tendency, coupled with the tendency to overreact to rejection cues, can lead to a maladaptive accumulation of stress hormones that ultimately leads to poor health outcomes. There is substantial evidence that long-term exposure to stress hormones can have deleterious effects on health (McEwen, 1998; Sapolsky, 1998), can damage the nervous system, and can accelerate brain aging, which can have harmful effects on learning and memory (Goossens & Sapolsky, 2007). Stress-related neuronal loss (especially in the hippocampus) has also been implicated in the etiology of major depression (Lee, Ogle, & Sapolsky, 2002).

Thus, for individuals with LSE, who may experience increased exposure to interpersonal stressors (Stintson et al., 2008) and heightened reactivity to those stressors (as suggested by our findings), cumulative exposure to cortisol may have considerable impact on their health and emotional well-being. Indeed, a growing number of studies have demonstrated an association between LSE and poor health outcomes (Antonucci, Peggs, & Marquez, 1989; Brown & McGill, 1989; Shimizu & Pelham, 2004; Stintson et al., 2008). However, little is known about the nature of this mind-body connection. One recent longitudinal study of college students revealed that the association between LSE and poor health

was mediated by interpersonal stressors (Stintson et al., 2008), but it is still unclear how interpersonal stress is converted into poor health outcomes. The current investigation helps to fill this gap by suggesting that maladaptive patterns of HPA activation (in response to social stress) may be one important mechanism linking interpersonal experiences to long-term health. In addition, if we assume that self-esteem acts as a sociometer (Leary & Baumeister, 2000) then it is likely to modulate responses to a variety of stressors (e.g., performance failure) that either directly or indirectly threaten social inclusion. In future studies, researchers should investigate the role of self-esteem in moderating HPA activation in response to other categories of stressors that may impact health.

Although the current study focused on self-esteem, other individual difference variables are also likely to play a role in modulating physiological responses to social stress. Results from prior studies on individual difference variables that are associated with LSE (namely rejection sensitivity and attachment-related anxiety) also point to the important role that personality may play in moderating physiological reactivity to interpersonal stressors. For example, research has demonstrated a link between attachment style and HPA responses to conflict discussion (Laurent, & Powers, 2007; Powers, Pietromonaco, Gunlicks, & Sayer, 2006) and to travel-related separations (Diamond, Hicks, & Otter-Henderson, 2008). Researchers have also shown that people who are high in rejection sensitivity respond to rejection with an increased sensitivity of the eye blink response, suggesting increased activation of a defensive motivational system (Downey, Mougios, Ayduk, London, & Shoda, 2004; Gyurak & Ayduk, 2007) and that individuals who are quicker to respond to rejection (based on a response latency task) show heightened cortisol reactivity to a social evaluation task (a difficult mental arithmetic task with social criticism; Dandeneau et al., 2007). Although these studies did not focus specifically on HPA responses to interpersonal rejection, they demonstrate the important role that individual differences can play in modulating physiological responses to social stressors. Findings from the current study extend this area of research by investigating self-esteem as an important component of a larger psychobiological system that drives psychological, biological, and social responses to rejection.

Finally, this study sheds light on an important debate in the literature concerning the benefits and the costs of HSE (Baumeister, Campbell, Krueger, & Vohs, 2003; Leary, 2004). If the benefits of HSE are not as great as theory suggests then we might have expected a discrepancy between self-report measures of social threat (e.g., self-evaluations, self-blame), which are consciously controlled and subject to self-presentation biases, and physiological measures of threat, which are automatic and not easily controlled. We observed no such discrepancies in our data. Thus, we found no evidence that HSE individuals were simply presenting a veneer of resilience. Nevertheless, our findings do not rule out the possibility that there may be variability among those with HSE. For example, it may be important to differentiate individuals who have stable versus labile HSE (Kernis & Paradise, 2002; Seery, Blascovich, Weisbuch, & Vick, 2004) and those with defensive versus nondefensive HSE (Jordan, Spencer, Zanna, Hoshino-Browne, & Correll, 2003).

Limitations and future directions. Some limitations of this study must be noted. First, because self-esteem was not manipu-

lated, we cannot draw unqualified causal inferences or rule out the possibility that unmeasured third variables may explain the self-esteem effects reported here. Likewise, although results from our path analysis were consistent with the proposed causal model, these findings are correlational and other plausible models may fit the data equally well. Second, we cannot assume a one-to-one correspondence between HPA activity and psychological threat. Cortisol is an end product of a complex system that is influenced by numerous biological factors, making it a very blunt measure that lacks psychological specificity. In future studies, it would be useful to investigate other physiological parameters that may offer a more fine-grained analysis of the links between physiology and specific psychological appraisals. For example, impedance cardiography would be useful for differentiating challenge and threat states (Blascovich & Mendes, 2000), which would allow us to identify those who feel confident in their ability to cope with social threat (a challenge state) and those who feel they lack the necessary coping resources (a threat state). Third, in the current study, we investigated processes related to romantic relationships by investigating rejection by a potential romantic relationship partner. Although we have no reason to believe that these same processes would not operate in long-term relationships, in the future, researchers should investigate these processes within the context of well-established relationships.

Although HSE individuals in the current study were clearly buffered from the threat of mild rejection cues, we believe it would be inaccurate to conclude that they will, or should, always be immune to the pain of social rejection. Indeed, individuals with HSE are motivated to seek social inclusion and are likely to experience declines in well-being—including increased HPA activity—in response to more potent rejection experiences, especially from valued relationship partners. Moreover, because rejection violates their expectations, it is possible that they will experience marked levels of psychological and physiological disequilibrium in response to explicit rejection. Nevertheless, even in such circumstances, we suspect that HSE individuals will cope better by repairing their damaged ego, mitigating long-term harm to their broader self-concept, and showing more rapid physiological recovery (quicker return to equilibrium). Thus, an important goal for future research will be to examine how HSE and LSE individuals differ in response to more potent interpersonal rejection experiences.

Another avenue for future research is to explore additional mechanisms by which HSE buffers individuals from the negative implications of interpersonal rejection. In the current study, individuals with HSE experienced the social situation as nonthreatening in part because they made external attributions for the rejection. Individuals with HSE may also engage in other coping strategies that protect their overall sense of worth in the face of rejection or social exclusion (Sommer, 2001). For example, they may be better able to protect their self-image by spontaneously affirming their worth in other valued domains of personal identity or by reminding themselves of others who love and value them (Creswell et al., 2005). Overall, it will be useful in future research to explore the strengths associated with HSE and not simply the vulnerabilities associated with LSE.

This study provides preliminary evidence for an association between HPA activation and one specific social response, partner derogation. In future studies, it will be useful to investigate other

social responses to rejection and the potential role of the HPA system in understanding these responses. Of course, as noted above, links between the HPA system and social behavior are likely to be extremely complex, and we do not expect a simple one-to-one association between HPA activation and specific social behaviors. It is likely that HPA activation mobilizes self-protective action, broadly defined, which can include prosocial as well as antisocial actions that serve the underlying goal of self-protection in the face of potential or actual rejection. Indeed, although social withdrawal and aggression are common responses to social rejection, individuals may also cope by reaching out to close others for social support or by seeking alternative relationship partners (Maner, DeWall, Baumeister, & Schaller, 2007). It is possible that HPA activation may serve to potentiate behavior in the direction afforded by the situation (e.g., is the situation controllable, are there other sources of social connection currently available?) and by individual differences in specific action tendencies.

Lastly, research is needed on the role of other individual difference factors that may modulate physiological responses to rejection; these include rejection sensitivity and insecure attachment style. We believe these factors, along with self-esteem, are part of a broader self-system that helps regulate the social self-preservation system. We focused on self-esteem because there are well-documented findings relating LSE to negative psychological responses to rejection and because there is substantial evidence that LSE also poses a risk factor for relationship difficulties (Murray, Holmes, & Collins, 2006). There is also evidence that self-esteem and other self-resources (e.g., optimism, positive illusions) are related to long-term health outcomes (e.g., Stinson et al., 2008; Taylor, Lerner, Sherman, Sage, & McDowell, 2003). Of course, there are differences between self-esteem and these other constructs, and it will be important to investigate their shared and unique roles in regulating social construal and physiology in response to social threat.

Conclusion. A great deal of research has focused on individual differences in psychological responses to rejection, but scientists are only just beginning to understand the neural processing and physiological consequences of social rejection. The current study contributes to this effort by serving as a bridge between research on physiological responses to rejection and research on self-esteem as an important regulator of perceived social threat. This article provides support for an integrated perspective that illuminates the process through which self-esteem may shape cognitive, physiological, and social responses to rejection in close relationships. We hope the current investigation encourages other researchers to explore the complex links among social situations, biological processes, and personality vulnerability and resilience.

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