Self-esteem, cortisol reactivity, and depressed mood mediated by perceptions of control

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Abstract

This study investigated cortisol reactivity (CR) as a moderator and perceptions of control as a mediator between low self-esteem (SE) and depressed mood. Fifty-four participants completed SE and mood inventories before an uncontrollable laboratory stressor. Salivary cortisol was determined before and after the stressor. Analyses indicated significance for SE (β = −0.30), CR (β = −0.92) and their interaction (β = −0.90) in predicting depressed mood (P = 0.03 for all). Low SE and decreased CR predicted the highest levels of depressed mood. The interaction indicated that depressed mood was predicted by increases in cortisol in individuals with higher SE, but by decreases in cortisol in individuals with lower SE. These relationships were statistically explained by low perceptions of control at baseline. Findings support biopsychological explanations for depression, with SE, CR, and uncontrollability as putative markers of depressed mood that may be even more pronounced in depressive disorders. © 2002 Elsevier Science B.V. All rights reserved.

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1. Introduction

Evidence consistently supports the relationship between low self-esteem (SE) and depression. For example, low SE has been found to predict the onset of depressive...
symptoms, the course of depressive disorders, and the ability to recover (Brown et al., 1990a,b; Roberts and Monroe, 1992). Another factor thought to influence depression and negative mood is exposure to uncontrollable stressful events (Alloy and Clements, 1992; Breier et al., 1987; Lloyd, 1980). It is possible that the established relationship between SE and depression is influenced by individual differences in response to uncontrollable stress. Such individual differences can be reflected in cortisol reactivity (CR), which is a physiological response of the hypothalamic pituitary adrenal (HPA) axis that is sensitive to real or perceived uncontrollability (Biondi and Picardi, 1999; Mason, 1968; Stansbury and Gunnar, 1994).

In the only known study to address the relationship between SE, CR, and uncontrollability, a combination of low SE with low internal locus of control indeed was associated with increased CR specifically in response to a task designed to induce failure (Pruessner et al., 1999a). On the other hand, in a subgroup of teachers with both high burnout and perceived stress, low SE and low internal locus of control have been associated with low basal levels of cortisol secretion in the hour after awakening (Pruessner et al., 1999b). These same teachers, however, also showed stronger increases in cortisol after dexamethasone suppression, suggesting possible HPA hyperactivity in some circumstances.

Lastly, dysregulation of the HPA axis has been associated with depressive disorder, especially endogenous depression, and can be briefly summarized as showing basal hypercortisolemia, blunted ACTH and increased cortisol in response to injected CRH, and relative resistance to cortisol suppression by oral dexamethasone (see Plotsky et al., 1998; Stokes, 1995 for reviews). More recently, studies are beginning to indicate a relationship between depressive symptoms or negative affect and increased CR to stress in particular (Scarpa et al., 1997; Susman et al., 1997). These studies are consistent with the notion of cortisol hyper-responsivity to both internal and external stressors in depression, possibly as a result of lowered glucocorticoid feedback sensitivity in the HPA axis.

Other types of cortisol responses, however, have also been noted in relationship to depression and to stress-related disorders. Cortisol response to the opioid antagonist naloxone, for example, was found to be reduced in depressed subjects relative to healthy controls (Burnett et al., 1999). Reduced cortisol to a naloxone challenge was interpreted as reflecting a failure of the typical inhibitory effect of opioids on the HPA axis, which the authors suggested could explain the HPA overactivity that is usually seen in depressed patients. On the other hand, hyporenin rather than hyper-cortisolism has also been reported in the development of stress-related disorders such as post-traumatic stress disorder, chronic fatigue syndrome, and other somatoform disorders (see Heim et al., 2000 for a review). These authors suggest that conditions of chronic stress may lead to a deficiency in cortisol, including reduced adrenocortical secretion and reactivity, or increased negative feedback inhibition of the HPA axis. If depression is viewed as a stress-related disorder, therefore, some forms may be related to a similar reduction in cortisol.
The above findings suggest that the relationship of depressed mood to HPA activity is indeed complex. Overall, low SE and changes in CR (as a physiological measure of responsivity to uncontrollable stress and/or possible HPA axis dysregulation) have been implicated as risk factors for depressed mood. To the authors’ knowledge, however, no studies have investigated the role of CR and perceived uncontrollability in the SE/depressed mood relationship. This study assessed the influence of CR as a moderator and perceptions of control as a mediator of this relationship. Based upon the predominant literature on SE, HPA hyperactivity, and uncontrollable stress in relation to depression, it was hypothesized that individuals with low SE and increased CR would display the highest levels of depressed mood, and that this effect would be statistically accounted for by self-reports of low control. As such, these factors would reflect a biopsychological marker for depressed mood that may be even more pronounced in the presence of a depressive disorder.

2. Method

2.1. Participants

Fifty-four undergraduate psychology students (17 males, 37 females; aged 18–45, \(M = 22.2\) years) completed questionnaires for depressed mood and SE before a laboratory stressor and provided saliva samples for cortisol before and after the stressor. Perceptions of controllability and helplessness were also obtained before and after the stressor.

2.2. Laboratory stressor

Based on Breier et al. (1987), the uncontrollable stressor consisted of 60 white noises (100 dB, duration 2–5 s, intertrial interval 10–40 s). Four buttons were located above the dominant hand of the participant who was told that he/she could stop the noise if a correct button sequence was mastered by trial and error. In actuality, no correct sequence existed, thus giving no opportunity to eliminate the noise. The task lasted about 30 min. This task was designed to assess individual differences in the cortisol response to an uncontrollable stressor, rather than attempting to elicit a cortisol response in all participants.

2.3. Self-report measures

SE was assessed with the Coopersmith self-esteem inventory (SEI, adult version; Coopersmith, 1981), consisting of 25 items rated as 0 (unlike me) or 1 (like me). Items were summed for a total score (\(M = 17.11\), Range = 1–25), with higher scores reflecting higher SE. For some analyses, participants were divided into high (\(n = 30; M = 21.43\)) and low (\(n = 24; M = 11.71\)) SE groups based upon a median split (Md = 19).
Depressed mood was assessed with the depression–dejection subscale of the profile of mood states (POMS; McNair et al., 1971), consisting of 15 depressive adjectives rated from 0 (not at all) to 4 (extremely). Items were summed for a total score ($M = 5.24$, Range $= 0–32$).

As a task manipulation check and to assess perceptions of controllability, participants were asked to rate their sense of helplessness and control both before and after the stressor on a scale of 0 (not at all) to 100 (extremely). The means for helplessness reported before and after the stressor were 18.27 and 68.27, respectively. The means for control reported before and after the stressor were 59.37 and 10.04, respectively.

2.4. Assessment of cortisol reactivity

Saliva was collected using a commercial kit from Sarstedt, Inc. (Arlington Heights, IL). Participants soaked gauze in the mouth for 60 s, which was then put in a tube, centrifuged, and stored at $-80 \, ^\circ C$ until assayed. Cortisol concentrations in $\mu g/dl$ were determined using a solid-phase radioimmunoassay procedure outlined by Diagnostic Products Corporation (Los Angeles, CA). Each sample was processed in duplicate, and the mean intra-assay coefficient of variation was 4.22%.

Three saliva samples were obtained—two before the stressor (averaged to form a baseline) and one immediately after the stressor. Saliva was always sampled in the afternoon, between 1 p.m. and 5 p.m. Though cortisol can peak in response to a meal such as lunch, afternoon sampling was chosen because this is a time when endogenous cortisol levels tend to drop and stabilize, thus maximizing the likelihood that observed cortisol increases are task dependent. Moreover, there was no reason to suspect systematic meal differences in relation to SE or controllability in our sample. Lastly, CR was assessed with a percent change score, calculated by the following formula: $[(\text{post-stressor cortisol level} - \text{baseline cortisol level})/\text{baseline cortisol level}] \times 100$. This formula provides a continuous measure indicating the percent cortisol change from baseline, with positive scores reflecting an increase and negative scores a decrease from pre- to post-stressor. A percent change score was chosen because it assesses the change in cortisol levels that occurred specifically in reaction to the task, regardless of initial absolute values of cortisol. Thus, CR was standardized across individuals. For some analyses, participants were classified as Reactors if their reactivity score was positive ($n = 17$; $M = 28.93\%$) and Nonreactors if their reactivity score was negative ($n = 37$; $M = -38.60\%$). In all cases, change scores were at least 5%, which is the lowest change possible to be detected by the assay procedure used.

2.5. Procedures

The participant completed the SEI and POMS, rinsed his/her mouth with water, and was seated at a computer for 5 min to habituate. Two saliva samples were then collected, separated by 10 min, after which participants rated their sense of helplessness and control. Headphones were placed, instructions given, and the
stressor began. Immediately after, headphones were removed and the third saliva sample collected. Participants then rated their sense of helplessness and control felt during the task and were debriefed.

3. Results

3.1. Stressor manipulation check

Paired t-tests were conducted to compare baseline and post-stressor ratings of helplessness and control. Results indicated a significant increase in helplessness, $t(51) = 10.81$, $P = 0.00$; $M = 18.27$ versus $68.27$, and a significant decrease in control, $t(51) = 12.26$, $P = 0.00$; $M = 59.37$ versus $10.04$, suggesting that the task did produce the desired effect of perceived uncontrollability overall.

3.2. Effects of demographics

Independent t-tests and correlations were conducted to determine if gender or age affected SE, CR, or depressed mood. No significant effects were found for gender. Age was significantly correlated with CR, $r(52) = 0.28$, $P = 0.04$, but not with SE or depressed mood.

3.3. Self-esteem and depression with cortisol reactivity as a moderator

A stepwise regression analysis was conducted with SE and CR entered in Step 1, and their interaction term (i.e. SE × CR) entered in Step 2, as predictors of depressed mood. The model was significant at Step 1, $F(2, 51) = 5.98$, $P = 0.01$, accounting for 19% of the variance, with a significant change at Step 2, $F(1, 50) = 4.86$, $P = 0.03$, accounting for an additional 7% of the variance. Thus, the overall model was significant, $F(3, 50) = 5.91$, $P = 0.00$, and accounted for 26% of the variance, with a significant main effect for SE, $\beta = -0.30$, $t = 2.23$, $P = 0.03$, a significant main effect for CR, $\beta = -0.92$, $t = 2.31$, $P = 0.03$, and a significant interaction, $\beta = -0.90$, $t = 2.21$, $P = 0.03$. The main effects indicated that low SE and decreases in post-task cortisol each predicted higher levels of depressed mood. The interaction was further examined by regressing depressed mood onto CR separately for groups with high or low SE (See Fig. 1 for a graph of the results). Results indicated that decreased CR predicted depressed mood for the low SE group at the level of a trend, $\beta = -0.35$, $t = 1.76$, $P = 0.09$, but increased CR significantly predicted depressed mood for the high SE group, $\beta = 0.38$, $t = 2.16$, $P = 0.04$. Overall, the highest mean levels of depressed mood were found in individuals with lower levels of SE and decreased CR over the stressor task.

For purposes of clarification, Table 1 presents the means for absolute cortisol levels both before and after the stressor task for the SE groups with high and low levels of depressed mood (as determined by a median split). As can be seen, cortisol levels declined for all groups except for those with both higher levels of SE and
3.4. Mediating role of perceptions of control

Since a significant interaction indicated that SE was qualified by CR, it was most appropriate to test the mediating role of controllability on the interaction rather than on SE alone. Mediation was tested using criteria set forth by Baron and Kenny (1986). Specifically, if B is a mediator between A and C, then (1) A must relate to C, (2) A must relate to B, (3) B must relate to C, and (4) B must statistically account for the relationship between A and C. In this case, (1) the interaction of SE and CR must relate to depressed mood, (2) the interaction must relate to perceived controllability, (3) perceived controllability must relate to depressed mood, and (4) perceived controllability must statistically account for the interaction effect on depressed mood.

Table 1

<table>
<thead>
<tr>
<th>Self-esteem</th>
<th>Depressed mood</th>
<th>Pre-stressor cortisol</th>
<th>Post-stressor cortisol</th>
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</thead>
<tbody>
<tr>
<td>Low</td>
<td>Low</td>
<td>0.27</td>
<td>0.21</td>
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<tr>
<td></td>
<td>High</td>
<td>0.44</td>
<td>0.42</td>
</tr>
<tr>
<td>High</td>
<td>Low</td>
<td>0.79</td>
<td>0.53</td>
</tr>
<tr>
<td></td>
<td>High</td>
<td>0.39</td>
<td>0.50</td>
</tr>
</tbody>
</table>
Fig. 2. Mean control ratings at baseline and post-stressor for SE and CR groups.

Criterion 1 was already established in the above regression analyses indicating a significant interaction between SE and CR in predicting depressed mood. Criterion 2 was tested in two ways. First, a two-way (high/low SE \times cortisol Reactor/Nonreactor) repeated measures ANOVA was conducted, with the repeated measure being ratings of control at baseline and post-stressor task (See Fig. 2 for a graph of the results). This analysis was conducted to determine if perceptions of control were related to SE and CR both before and after the stressor, or if one time point was
more influential. Results indicated a significant Time × SE × CR group interaction, \( F(1, 48) = 4.53, P = 0.04 \), whereby Nonreactors reported reduced control in the low SE group, but Reactors reported reduced control in the high SE group; and this difference was most pronounced at baseline. In other words, Nonreactors with low SE reported the lowest levels of baseline control and Reactors with high SE reported the second lowest levels of baseline control. Thus, Criterion 2 was supported. Because differences in perceived control were greatest at baseline, only this time point was used in further analyses of the mediational effect.

The second test of Criterion 2 was conducted using a stepwise regression, with SE and CR entered in Step 1 and their interaction term entered in Step 2, predicting baseline levels of control. The model was a nonsignificant trend at Step 1, \( F(2, 49) = 3.09, P = 0.06 \), accounting for 11% of the variance, with a significant change at Step 2, \( F(1, 48) = 11.23, P = 0.00 \), accounting for an additional 1% of the variance. Thus, the overall model was significant, \( F(3, 48) = 6.23, P = 0.00 \), and accounted for 28% of the variance, with a significant main effect for CR, \( \beta = 1.33, t = 3.32, P = 0.00 \), and a significant interaction, \( \beta = -1.37, t = 3.35, P = 0.00 \). The main effect indicated that increases in post-task cortisol predicted higher levels of baseline control. The interaction was further examined by regressing baseline control onto CR separately for groups with high or low SE. Results indicated that increased CR predicted low perceptions of baseline control for the high SE group at the level of a trend, \( \beta = -0.32, t = 1.76, P = 0.09 \), but decreased CR significantly predicted low perceptions of baseline control for the low SE group, \( \beta = 0.57, t = 3.06, P = 0.01 \). Thus, consistent with the ANOVA results, reduced perceptions of control at baseline were related to CR differentially, depending upon level of SE. Based upon the ANOVA and regression analyses, Criterion 2 was supported.

Criteria 3 and 4 were tested using a stepwise regression analysis to predict depressed mood, where ratings of baseline control were entered in Step 1, SE and CR in Step 2, and the interaction term for SE × CR in Step 3. The model was significant at Step 1, \( F(1, 50) = 6.23, P = 0.02 \), accounting for 11% of the variance, with a significant change at Step 2, \( F(2, 48) = 3.73, P = 0.03 \), accounting for an additional 12% of the variance. There was no significant change at Step 3, \( F(1, 47) = 2.45, P = 0.12 \), which accounted for an additional 4% of the variance. Specifically, at Step 1, baseline reports of control were significantly negative related to depressed mood, \( \beta = -0.33, t = 2.50, P = 0.02 \), thus supporting Criterion 3. The model was also significant at Step 2, \( F(3, 48) = 4.79, P = 0.01 \), with a main effect for low SE predicting depressed mood, \( \beta = -0.37, t = 2.73, P = 0.01 \). Lastly, the overall model at Step 3 was significant, \( F(4, 47) = 4.31, P = 0.01 \). In this step, once levels of controllability were included in the model, SE remained significant, \( \beta = -0.30, t = 2.11, P = 0.04 \), but CR and the interaction between SE and CR became nonsignificant, \( P = 0.12 \) for both. Thus, the inclusion of perceptions of control statistically accounted for the SE × CR interaction on depressed mood, providing support for Criterion 4.
4. Discussion

The results are consistent with the literature showing depression to be associated negatively with SE (Roberts and Monroe, 1992; Brown et al., 1990a,b) and positively with CR (Scarpa et al., 1997; Susman et al., 1997), although the latter occurred only for individuals with higher levels of SE. Contrary to this literature and the study by Pruessner et al. (1999a), depressed mood was negatively associated with CR, particularly for individuals with lower levels of SE. Indeed, the highest levels of depressed mood were reported in individuals with low SE who also showed decreases in cortisol after the stressor task. These individuals also reported the lowest perceptions of controllability before the task, which statistically mediated the SE × CR interaction effect. These latter findings are more consistent with the notion of hypocortisolism, particularly in reaction to stress, as posited by Heim et al. (2000). Overall, the findings are supportive of biopsychological theories of emotions (e.g. Lang, 1984) and, in particular, suggest that SE, HPA reactivity to uncontrollable stress, and perceptions of control dynamically interact in the presence of depressed mood.

One implication is that such interactions may also occur and be even more pronounced in manifest depressive disorders. Recent perspectives in the field of developmental psychopathology suggest that depression is the result of many interactive factors (including biological features, family experiences, cognitive representations, social competencies, and life stress) occurring within a developmental context (Hammen and Rudolph, 1996). Findings from this study, which show an interaction between cognitive appraisals of SE and biological reactivity to uncontrollable stress, are consistent with such a multifaceted and biopsychological framework. The finding that perceptions of controllability statistically explain these effects provides yet further evidence for the dynamic interplay of multiple factors. Thus, a combination of SE, CR, and perceptions of control may act as a biopsychological marker of clinical depression.

It is intriguing to note that CR was positively related to depressed mood only for individuals reporting relatively higher levels of SE, which is typically viewed as a protective factor from depression. One interpretation of this result might be that heightened CR predicts depressed mood only in the absence of psychological risk factors. This would be consistent with the literature on hypercortisolism, reduced glucocorticoid feedback sensitivity, and other HPA disturbances in individuals with endogenous, but not exogenous, depression (see reviews by Amsterdam et al., 1989; Carroll, 1982). Still, the highest levels of depressed mood were reported by individuals with low levels of SE, and these individuals showed reduced cortisol responsivity. Perhaps, similar to the disorders described by Heim et al. (2000), these individuals reflect a subgroup affected by conditions of chronic stress, thus leading to problems with SE, hypocortisolism, and reactive depression.

An additional interpretation of the current findings is that the high and low SE groups were experiencing the test situation differently. This interpretation is supported by the finding that low perceptions of control were related to increased CR for participants with relatively higher levels of SE, but to decreased CR for those
with lower levels of SE. Psychological influences have long been described in relation to cortisol responsivity. In a seminal review, for example, Mason (1968) noted that, in addition to qualities of the stimulus itself influencing adrenocortical activation, there were marked individual differences in the psychoendocrine response to the same stimulus. Thus, he concluded that individual differences in psychological variables needed to be addressed. The psychological variables noted to relate to adrenocortical activation included (1) active coping to master a difficult task or illness and (2) observing that previously effective behavior no longer accomplishes mastery of a task. Low adrenocortical activity, on the other hand, was associated with passive coping or extensive denial as a defense mechanism. Applied to the current findings, it could imply that the increased CR in participants with higher levels of SE reflected an active (though failed) attempt to overcome initial feelings of low controllability and to master the task at hand. Reduced CR in participants with lower levels of SE, on the other hand, could imply a tendency to use passive coping strategies or denial in reaction to a difficult task.

Though intriguing, the results of this study are qualified by several limitations, including the use of a nonclinical sample with low reports of depressed mood, which limits interpretations regarding clinical levels of depression. Additionally, the correlational and cross-sectional design does not allow for inferences of causality, or developmental vulnerability and progression. While this study requires replication with an expanded design to address these limitations, the results are encouraging and are presented here to prompt continued research on the role of CR in biopsychological studies of depression and depressed mood.

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