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Depression history as a moderator of relations between cortisol and shame responses to social-evaluative threat in young adults

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Abstract

Changes in cortisol and shame are commonly elicited by psychosocial stressors involving social-evaluative threat. According to social self preservation theory, this coordinated psychobiological response is adaptive. Individuals with a history of depression, however, may exhibit diminished cortisol reactivity to acute stressors, which could interfere with coordinated cortisol and shame responses. The present study examined temporal relations between cortisol and shame responses to a psychosocial stress task in young adults who varied in their history of depression (56 remitted-depressed, 46 never-depressed). Lagged effects multilevel models revealed that depression history moderated relations between cortisol levels and shame ratings 25 to 55 minutes later. The pattern of these interactions was similar: whereas higher cortisol levels predicted increases in shame in never-depressed individuals, cortisol levels were unrelated to shame responses in remitted-depressed individuals. Findings suggest a dissociation between cortisol and shame responses to stress in individuals with a history of depression.

Keywords

depression; cortisol; shame; stress; social-evaluative threat

When an individual perceives an event as threatening, a series of emotional, behavioral, physiological, and endocrine responses are elicited that together comprise the stress response. Coordination of these stress response systems is considered to be important for successful adaptation to environmental challenges (Lazarus, 1991; Levenson, 2014). Less clear, however, is how these systems are coordinated during the stress response (Andrews, Ali, & Pruessner, 2013; Campbell & Ehlers, 2012), and whether problems with coordination are associated with risk for stress-related psychopathology. Whereas subjective emotional

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experiences and sympathetic nervous system activity are triggered within milliseconds of stress exposure (Ulrich-Lai & Herman, 2009), the hypothalamic-pituitary-adrenal (HPA) axis reacts more slowly, with cortisol responses peaking between 20 and 40 minutes after stressor onset (Dickerson & Kemeny, 2004). Hence, capturing moment-to-moment interactions among these stress response systems has been a challenge.

Psychosocial stress tasks that incorporate uncontrollable social-evaluative threat reliably trigger cortisol responses and psychological distress in healthy individuals. A meta-analysis of this literature, however, did not detect an association between cortisol levels and psychological distress (Dickerson & Kemeny, 2004). According to the integrated specificity model (Kemeny, 2003), the nature of the threat posed by a stressor determines the particular psychobiological responses elicited. Moreover, emotions orchestrate coordinated responses to challenge (e.g., Ekman, 1999) and are thought to be linked to unique neurobiological correlates (Damasio et al., 2000; Ekman, Levenson, & Friesen, 1983; Herman, Ostrander, Mueller, & Figueiredo, 2005). A more consistent pattern of findings emerges when global constructs such as psychological distress are defined in specific terms (Campbell & Ehlert, 2012). The present study investigated relations between cortisol responses and an emotion commonly elicited by social-evaluative threat – shame (Kemeny, Gruenewald, & Dickerson, 2004).

The social self preservation model posits a coordinated psychobiological response to social threat that involves increases in both shame and cortisol (Kemeny et al., 2004). Increases in shame are triggered by negative interpersonal appraisals (Tangney, Miller, Flicker, & Barlow, 1996) and are associated with withdrawal and disengagement behaviors that could serve to deescalate social conflict (Gilbert, 2000). Increases in cortisol levels have been proposed as a mechanism for mobilizing resources necessary to cope with the challenge of social-evaluative threat (Dickerson et al., 2004). Although acute shame and cortisol responses may be adaptive, chronic hyper- or hypo-activation of cortisol and shame responses are linked to increased risk for negative health outcomes (Raison & Miller, 2003). Cortisol responses to social evaluation were found to be positively correlated with shame in healthy young adults following a stress task (Denson, Creswell, & Granville-Smith, 2012). In healthy children, higher shame responses to task failure were associated with higher cortisol reactivity and delayed cortisol recovery (Mills, Imm, Walling, & Weiler, 2008). Greater cortisol responses to stress also have been observed in healthy individuals who experienced greater pre- to post-stress task increases in shame (Gruenewald, Kemeny, Aziz, Fahey, 2004). Taken together, these studies demonstrate a positive *association* between shame and cortisol responses to social evaluation in healthy individuals; less is known, however, regarding the temporal relation between cortisol and shame responses to stress. In particular, do cortisol responses to a social-evaluative threat *predict* subsequent shame responses?

In a recent review examining correspondence between emotional and physiological responses to stress, Campbell and Ehlert (2012) concluded that inconsistencies in this literature may be explained by methodological factors including a reliance on single or pre-post assessments of emotional states during stress tasks. Repeated affective and salivary cortisol assessments rarely have been combined to capture the dynamic interplay between

these stress response systems (Hellhammer & Schubert, 2013). One exception is a study by Schlotz and colleagues (Schlotz, Kumsta, Layes, Entringer, Jones, & Wüst, 2008) in which psychological and HPA axis responses to both pharmacological and psychosocial challenges were repeatedly and simultaneously assessed. Lagged effects models revealed significant covariation between psychological and HPA axis measures across laboratory challenges, such that higher lagged cortisol levels predicted lower subsequent levels of self-reported state anxiety and arousal (Schlotz et al., 2008). The present study extends this literature by using lagged effects models to test whether within-individual changes in cortisol levels during a psychosocial stressor predict changes in subsequent shame.

Stress, Cortisol, and Depression History

Altered relations between HPA axis and psychological stress responses may be a marker of risk for stress-related psychiatric conditions such as major depressive disorder (MDD). Higher diurnal cortisol secretion, impaired HPA axis negative feedback, and persistent negative mood are associated with MDD and contribute to risk for recurrence (Holsboer, 2000). When confronted with psychosocial stressors, depressed and non-depressed individuals exhibit different patterns of cortisol secretion. Compared to non-depressed youth, currently depressed adolescents typically show enhanced cortisol reactivity and delayed cortisol recovery to psychosocial stressors (Rao, Hammen, Ortiz, Chen, & Poland, 2008; Stewart, Mazurka, Bond, Wynne-Edwards, & Harkness, 2013). In contrast, currently depressed adults tend to show elevated pre-stress cortisol levels, diminished cortisol reactivity, and delayed cortisol recovery compared to non-depressed adults (Burke, Davis, Otte, & Mohr, 2005).

Remitted-depression designs are well-suited to identifying depression vulnerability factors because they allow researchers to rule out the confounding effects of the depressive episode (*state markers*). These vulnerability factors may be present before first onset of an MDE (*trait markers*) or could emerge as a consequence of an MDE (*scar markers*) (Adam, Sutton, Doane & Mineka, 2008). Remitted-depressed adults generally show diminished cortisol reactivity to laboratory stressors (Ahrens, Deuschle, Krumm, van der Pompe, den Boer, & Lederbogen, 2008; Brown, 2001; Trestman, Coccaro, Bernstein, & Lawrence, 1991; see also Bagley, Weaver, & Buchanan, 2011; Lange, Zschucke, Ising, Uhr, Birmphohl, & Adli, 2013) and higher pre- to post-stressor negative affect (Bagley et al., 2011) compared to never-depressed adults. Biological challenge studies revealed that HPA responses to the dexamethasone/corticotropin-releasing hormone test during remission predicted depression recurrence (Appelhof et al., 2006; Aubry et al., 2007; Hatzinger, Hemmeter, Baumann, Brand, & Holsboer-Trachsler, 2002; Zobel, Yassouridis, Frieboes, & Holsboer, 1999), suggesting that cortisol responses to psychosocial stressors during remission could represent trait or scar markers and increase vulnerability to recurrent episodes.

One possible extension of social self-preservation theory (Kemeny et al., 2004) is that diminished cortisol responses to acute stressors in remitted-depressed individuals could interfere with the adaptive coordination of cortisol and shame responses to social-evaluative threat. To our knowledge, the time course of cortisol effects on shame has not been examined in remitted-depressed individuals using lagged effects analysis, nor has depression

history been tested as a moderator of these relations. Therefore, the primary goal of the current study was to determine whether depression history moderated within-individual relations between cortisol levels and shame during a psychosocial stress task. Based on the social self-preservation model (Kemeny et al., 2004) and evidence of diminished cortisol reactivity in remitted-depressed individuals (e.g., Ahrens et al., 2008; Brown, 2001; Trestman et al., 1991), we hypothesized the following interaction pattern: first, higher cortisol responses would be associated with higher shame over time in never-depressed individuals, whereas cortisol responses would not be significantly associated with shame responses in remitted-depressed individuals. Second, based on the work of Schlotz and colleagues (2008) showing time lagged cross-correlations between salivary cortisol and subsequent psychological measures during a psychosocial stress test, we anticipated that differences in the strength of cortisol-shame relations between remitted- and never-depressed individuals would become more pronounced with greater lag intervals between cortisol and subsequent shame.

Method

Participants

Participants were 102 individuals (56 remitted-depressed and 46 never-depressed), ages 18 to 31 years (mean age = 22.97, $SD = 3.87$). Inclusion in the remitted-depressed group required a past diagnosis of MDD as determined by the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1997). Full remission was defined as an absence of significant symptoms of depression for at least two months (Frank et al., 1991). The never-depressed group had no lifetime history of a depressive disorder.

Participants were screened and excluded for current or past bipolar disorder or posttraumatic stress disorder (PTSD), health conditions known to influence HPA axis function (e.g., Cushing's disease, Addison's disease, diabetes) or use of prescription or non-prescription drugs (e.g., benzodiazepines) that might affect the HPA system. One individual was excluded due to pregnancy and another for use of mirtazapine. Participants using antidepressant medication ($n = 13$ SSRIs; $n = 1$ SNRI; $n = 1$ tricyclic) or birth control ($n = 40$) were not excluded². Participants were recruited from undergraduate and graduate programs at a mid-size university in the southeastern United States and through a medical center listserv email; individuals received course credit or \$30 for participation in the study. All procedures were approved by the University Institutional Review Board for the Protection of Human Subjects.

Measures

Depression—The SCID-I (First et al., 1997) was used to assess current and lifetime diagnoses of MDD, bipolar disorder, and PTSD. Other current or lifetime anxiety disorders were not assessed. Depression history was operationalized as a dichotomous variable (i.e., yes or no). All interviews were audio-taped and a random 20% were re-rated for reliability

²Results of multilevel analyses did not differ when antidepressant medication use and birth control use were included as covariates.

by an independent evaluator who was unaware of the ratings of the primary interviewer. Inter-rater reliability for history of depression yielded a kappa of 1.00.

The *Beck Depression Inventory-II* (BDI-II; Beck, Steer, & Brown, 1996) was used to assess participants' current level of depressive symptoms. The BDI-II is a 21-item, widely used, self-report measure with good reliability and validity (Beck, Steer, Ball, & Ranieri, 1996). In this sample, coefficient alpha for the BDI-II was .85.

The *Shame* subscale of the *State Shame and Guilt Scale* (SSGS; Marschall, Sanftner, & Tangney, 1994) consists of 5 items assessing shame in the moment (e.g., "I feel humiliated, disgraced;" "I want to sink into the floor and disappear."). Respondents rated each item on a 5-point scale (1 = *not feeling this way at all*; 3 = *feeling this way somewhat*; 5 = *feeling this way strongly*); a mean score for *shame* was computed. The SSGS was administered while participants provided each saliva sample (6 times). In this sample, coefficient alphas ranged from .80 to .88. The 5 guilt items and the 5 pride items of the SSGS were not included in the present study.

Psychosocial Stressor—The Trier Social Stress Test (TSST) consisted of a 5-minute free-speech task followed by a 5-minute mental arithmetic task. In the current study, participants were randomly assigned to either a social evaluation (stress) condition or to a no social evaluation (control) condition. Similar to procedures outlined by Gruenewald and colleagues (2004), participants in the stress condition were told that the examiner would be in the room during their speech, their performance would be audio- and videotaped, and then a panel of judges would evaluate their speech in comparison to others. Participants randomized to the control condition were informed that they would perform the tasks alone in the room and they would not be observed, recorded, or evaluated.

Cortisol—Salivary cortisol samples were collected using a saliva collection device (Salivette; Sarstedt Inc., Newton, NC). Cortisol levels were determined in duplicate using a commercially available enzyme immunoassay kit (Enzyme-Linked ImmunoSorbent Assay, ALPCO diagnostics, Salem, NH). The lower detection limit, or sensitivity, of this assay is 1.0ng/ml. Inter- and intra-assay coefficients of variation were 7.0% and 4.4%, respectively.

Procedures

Individuals meeting study criteria based on the telephone screen were invited to participate. Participants were instructed not to drink alcohol, smoke, use nonprescription drugs, engage in strenuous exercise, or visit the dentist within the 24 hours prior to their appointment, and to refrain from drinking (except water), eating, or brushing their teeth 1 hour before the session. Participants were screened for these behaviors at the beginning of the laboratory assessment; none required rescheduling. To control for diurnal variations in cortisol, all laboratory sessions were conducted between 2:00 and 7:00 p.m.

After providing written informed consent, participants were administered the Mood Disorders and PTSD sections of the SCID-I by a trained Masters level graduate student (MCM) supervised by an expert clinician (JG). Participants then completed the BDI-II and sat quietly for 10 minutes. Following this rest period, participants provided their first saliva

sample and shame rating (T0: baseline). Then they were informed about the laboratory task to which they were randomly assigned and provided their second saliva sample and shame rating at the end of the 10-minute preparation period (T1: anticipatory stress, 10 minutes after T0). Participants then were escorted to another room where they performed the tasks. For all participants, the speech task preceded the mental arithmetic task. Participants provided their third (T2: mid-task, 17 minutes after T0) saliva sample and shame rating between the speech and arithmetic tasks and their fourth (T3: post-task, 25 minutes after T0) saliva sample and shame rating immediately following the arithmetic task. They next completed a demographics questionnaire, rested for 10 minutes, provided their fifth (T4: recovery 1, 40 minutes after T0) saliva sample and shame rating, rested another 10 minutes, and then provided their sixth (T5: recovery 2, 55 minutes after T0) and final saliva sample and shame rating. At the end of the study, participants were fully debriefed regarding the nature of the experimental manipulation.

Data Analytic Plan

Cortisol data were log-transformed to reduce skewness. To address the hypotheses regarding within- and between-individual change simultaneously, we specified a series of multilevel models (MLM) using hierarchical linear models (HLM 6) (Raudenbush et al., 2004) consisting of a within-person (i.e., level-1) sub-model describing how each individual's shame ratings changed over time, and a between-person (i.e., level-2) sub-model describing how these changes varied across individuals (Bryk and Raudenbush, 1992; Singer and Willett, 2003). For MLM, person means for the time-varying predictor (i.e., cortisol levels) were included in level-2 models to remove between-person variance from within-person variables and prevent predictors from correlating with individual intercepts or between-person factors (Hoffman and Stawski, 2009).

To determine the extent to which cortisol levels predicted subsequent shame, we ran a series of lagged effects models varying the lag interval (n minutes), such that shame at time t for individual i was predicted by cortisol levels at time $t - n$. Depression history was included in all models as a moderator of relations between lagged cortisol levels and subsequent shame. The impact of social-evaluative threat on pre-stress levels of shame and changes in shame over time was tested in all models by including stressor condition as a predictor of shame intercept and by including a stressor condition $\times$ time interaction. Sex was included as a predictor of shame intercept and a sex $\times$ time interaction was included in all models based on evidence of sex differences in cortisol responses to the TSST (Kudielka & Kirschbaum, 2005).

Results

Preliminary Analyses

In the overall sample, cortisol levels increased more rapidly in the stress as compared to the control condition ($b = .001$, $SE = .0004$, $p = .007$) (Figure 1), increased more rapidly for males as compared to females ($b = -.001$, $SE = .0004$, $p = .008$), but were not significantly associated with age ($b = .0001$, $SE = .0001$, $p = .252$) or depressive symptoms in the past two weeks ($b = -.0003$, $SE = .0002$, $p = .206$). Changes in shame ratings in the overall

sample were negatively associated with depressive symptoms in the past two weeks ($b = -.003$, $SE = .001$, $p < .001$) but were not associated with stressor condition ($b = -.002$, $SE = .001$, $p = .173$), sex ($b = -.001$, $SE = .001$, $p = .511$), or age ($b = -.0001$, $SE = .0002$, $p = .602$). Remitted- and never-depressed individuals did not differ in their baseline (T0) cortisol levels (remitted-depressed mean = 11.06, $SD = 4.05$; never-depressed mean = 12.44, $SD = 6.85$; $t = 1.25$, $p = .21$). In subsequent lagged effects analyses, the stressor condition $\times$ time and sex $\times$ time interactions were included as predictors of shame, along with the main effects of stressor condition, sex, and depressive symptoms.

Does Depression History Moderate Relations between Lagged Cortisol and Shame?

Associations between cortisol and shame responses were examined using lagged effects models. Cortisol was included as a predictor of concurrent shame (lag $n = 0$ minutes) and shame 10 minutes, 17 minutes, 25 minutes, 40 minutes, and 55 minutes later.

The Level 1 model was as follows:

$$\text{Shame}_{ti} = \pi_0 + \pi_1 \text{Time}_{ti} + \pi_2 \text{Cortisol}_{(t-n)i} + e$$

The Level 2 model was as follows:

$$\pi_0 = \beta_{00} + \beta_{01} \text{Cortisol Mean} + \beta_{02} \text{Stress} + \beta_{03} \text{Sex} + \beta_{04} \text{BDI} + \beta_{05} \text{MDD} + r_0$$

$$\pi_1 = \beta_{10} + \beta_{11} \text{Stress} + \beta_{12} \text{Sex} + r_1$$

$$\pi_2 = \beta_{20} + \beta_{21} \text{MDD}$$

In this equation, Time denotes the number of minutes elapsed since baseline; Cortisol denotes the individual's level of cortisol (log-transformed); Cortisol Mean denotes the individual's person mean for Cortisol (grand-mean centered), Stress denotes stressor condition (0 = control; 1 = stress); Sex denotes male (0) or female (1); BDI denotes depressive symptoms in the past two weeks; and MDD denotes history of depression (0 = never depressed; 1 = remitted depressed). Shame_{ti} indicates the shame rating at time t for person i . The subscript $(t - n)$ for cortisol indicates cortisol level n minutes prior to Shame_{ti}. Of primary interest was the interaction between lagged cortisol levels and depression history (β_{21}).

Results of multilevel models predicting changes in shame are presented in Table 1 (for lag intervals associated with significant MDD $\times$ cortisol interactions: 25, 40, and 55 minutes). The interaction of cortisol and depression history predicted changes in shame 25 minutes later ($b = -.488$, $SE = .170$, $p = .005$). Simple slope analysis revealed that whereas higher cortisol levels predicted higher shame 25 minutes later in never-depressed individuals ($b = .243$, $SE = .117$, $p = .039$), lagged cortisol levels did not significantly predict shame in remitted-depressed individuals ($b = -.246$, $SE = .129$, $p = .056$) (Figure 2). Similar interaction patterns were observed for cortisol levels predicting shame 40 minutes later ($b = -.646$, $SE = .181$, $p = .001$) and 55 minutes later ($b = -.536$, $SE = .201$, $p = .008$). The interaction of cortisol and depression history did not predict changes in shame concurrently ($b = .381$, $SE = .233$, $p = .102$), 10 minutes later ($b = -.280$, $SE = .180$, $p = .120$), or 17 minutes later ($b = -.217$, $SE = .168$, $p = .196$).

Discussion

Depression history moderated relations between cortisol and shame responses to acute social-evaluative threat. Whereas higher cortisol predicted increases in shame in never-depressed individuals, the association between cortisol and shame responses was not significant in remitted-depressed individuals. Results extend prior research in healthy individuals (Gruenewald et al., 2004; Matheson & Anisman, 2009; Mills et al., 2008) by revealing the time course of cortisol effects on shame: cortisol levels predicted higher shame ratings 25 to 55 minutes later but were not significantly associated with shame ratings 0 to 17 minutes later.

Shame is triggered by the belief that one is responsible for the occurrence of a negative event, and is associated with feelings of worthlessness, shrinking and smallness, and a concern that the self will be exposed as defective (Tangney & Fischer, 1995; Tangney, Wagner, & Gramzow, 1992). Nonverbal behaviors linked to shame, such as lowered gaze, slumped posture, and avoiding eye contact, are positively correlated with cortisol responses in children (Lewis & Ramsay, 2002). Similar behaviors indicative of social defeat or subordination have been found to be associated with elevated cortisol levels in animals (Sapolsky, 1993). Although trait measures of shame have not been found to be associated with diurnal cortisol secretion in young women (Rohleder, Chen, Wolf, & Miller, 2008), the present study showed that acute cortisol responses to social-evaluative threat predict subsequent increases in state shame ratings in healthy young men and women. Shame-related submissive behaviors may serve an adaptive function by reducing interpersonal aggression and eliciting cooperation (Keltner, Young, & Buswell, 1997). To the extent that cortisol elevations mobilize resources to engage in submissive displays, they also may be considered adaptive. Consistent with this perspective, higher cortisol reactivity has been found to be associated with increased social avoidance behavior in individuals with social anxiety disorder (Roelofs, van Peer, Berretty, de Jong, Spinhoven, & Elzinga, 2009).

Cortisol responses did not significantly predict subsequent shame in remitted-depressed individuals. A similar decoupling of cortisol and affect was observed in a study of individuals with chronic stress who were administered a mental arithmetic stress task (Pike et al., 1997). Why might there be less concordance between cortisol and shame responses to acute social-evaluative threat in individuals with a history of depression or elevated chronic stress levels? One possibility is that depression and chronic stress dampen cortisol reactivity to acute stressors. Decreased cortisol responses to laboratory stressors generally have been reported in studies of remitted-depressed individuals (Ahrens et al., 2008; Brown, 2001; Trestman et al., 1991) and also have been linked to higher chronic stress levels (Bellingrath & Kudielka, 2008).

“Blunted” cortisol reactivity may be affected by cognitive factors such as trait rumination. For example, higher trait rumination was associated with reduced cortisol reactivity to social-evaluative threat in healthy adults (Zoccola, Dickerson, & Zaldivar, 2008). In contrast to trait rumination, higher state rumination immediately following the TSST is associated with *higher* acute cortisol responses and with non-habituation of cortisol responses to subsequent stressors (e.g., Gianferante et al., 2014). Taken together, these findings suggest

that a *relatively stable* tendency to ruminate about negative emotions and their causes and consequences without engaging in problem solving (Nolen-Hoeksema, 2012), could contribute to lower cortisol responses in remitted-depressed individuals. This diminished cortisol reactivity, in turn, may disrupt covariation of cortisol and shame responses.

Salivary cortisol levels increased more rapidly in response to the TSST for males than females, which is generally consistent with prior research (Kirschbaum, Pirke, & Hellhammer, 1995; Lopez-Duran, Mayer, & Abelson, 2014; Schoofs & Wolf, 2011; see also Rohleder, Schommer, Hellhammer, Engel, & Kirschbaum, 2001). Sex differences in cortisol reactivity may be affected by a variety of biological and psychosocial factors. Some studies have found that sex differences disappear when women are administered laboratory stress tasks during their luteal phase (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999). A limitation of the current study is that menstrual cycle phase was not assessed. Prior research also suggests that women using oral contraception may exhibit diminished cortisol responses to psychosocial stress (Kirschbaum et al., 1995); however, the present findings were not affected when oral contraceptive use was included as a predictor of intercepts or changes in cortisol levels throughout the TSST. Finally, given that women report greater use of rumination when distressed than men (Nolen-Hoeksema, 2012) and the aforementioned link between rumination and blunted cortisol reactivity in healthy individuals (Zoccola et al., 2008), a ruminative response style may partially account for the observed sex differences in cortisol responses to psychosocial stress, and therefore should be assessed in future studies of responses to stress.

The present study addressed some shortcomings of prior research on the covariation of psychobiological stress responses (Campbell & Ehlert, 2012), including over-reliance on pre-post measures of affect. Nevertheless, limitations remain that provide directions for future research. The cross-sectional design cannot distinguish whether the lack of covariation between cortisol and shame responses to stress in remitted-depressed individuals represents a pre-existing vulnerability factor or a ‘scar’ left behind by prior depressive episodes. Inclusion of currently-depressed individuals in the present study would have further clarified whether the observed lack of covariation between cortisol and shame responses is present during an MDE. In addition, the sample primarily consisted of undergraduate and graduate students; replication in broader normative as well as clinical samples is needed. Finally, remitted- and never-depressed individuals were not excluded based on current or lifetime anxiety disorders; hence, we cannot rule out the possibility that undiagnosed anxiety disorders in remitted- and/or never-depressed individuals might have altered relations between cortisol and shame responses in the present study. Prior research has shown both lower (e.g., Klumbies, Braeuer, Hoyer, & Kirschbaum, 2014) and higher (e.g., Roelofs et al., 2009) cortisol reactivity in individuals with social phobia compared to healthy controls. Given the high comorbidity between MDD and social phobia (Kessler, Stang, Wittchen, Stein, & Walters, 1999) and evidence that cortisol responses to psychosocial stress predict avoidance behavior in individuals with social phobia (Roelofs et al., 2009), future studies should examine whether social anxiety impacts relations between cortisol and shame.

In conclusion, the present findings in never-depressed individuals are consistent with social self preservation theory (Kemeny et al., 2004) by demonstrating time-sensitive covariation of cortisol and shame responses to social-evaluative threat. In contrast, individuals with a history of depression exhibited little-to-no covariation between cortisol and shame. Whether this decoupling interferes with shame-related submissive behaviors and increases the likelihood of escalating interpersonal conflict (Gilbert, 2000; Keltner et al., 1997) remains to be studied. These findings suggest a novel psychobiological pathway through which formerly depressed individuals may contribute to the occurrence of interpersonal stressors and, ultimately, increase risk of recurrence.

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Figure 1. Mean cortisol levels ($\pm$ standard error of the mean) to stress and control conditions of a standardized laboratory stressor (TSST).

Figure 2.

Within-individual change: history of MDD by lagged cortisol predicting shame responses to the Trier Social Stress Test (TSST) [cortisol lagged 25 minutes prior to shame]. $*p < .05$.

Table 1

Multilevel Models Predicting Shame Responses to a Psychosocial Stress Test.

Cortisol Lag Interval	Predictor	<i>b</i> (<i>SE</i>)
25 minutes	Intercept	0.94 (.23) ^{***}
	Cortisol mean	0.09 (.21)
	Stress condition	0.12 (.08)
	Sex	0.003 (.08)
	BDI	0.22 (.04) ^{***}
	MDD	0.37 (.19) [*]
	Time	−0.001 (.001)
	Stress condition × time	−0.002 (.001)
	Sex × time	−0.002 (.001)
	Cortisol	0.24 (.12) [*]
	MDD × cortisol	−0.49 (.17) ^{**}
40 minutes	Intercept	0.96 (.23) ^{***}
	Cortisol mean	0.16 (.20)
	Stress condition	0.10 (.08)
	Sex	−0.01 (.08)
	BDI	0.22 (.04) ^{***}
	MDD	0.53 (.20) ^{**}
	Time	−0.002 (.001)
	Stress condition × time	−0.002 (.001)
	Sex × time	−0.001 (.001)
	Cortisol	0.17 (.11)
	MDD × cortisol	−0.65 (.18) ^{**}
55 minutes	Intercept	0.96 (.23) ^{***}
	Cortisol mean	0.07 (.20)
	Stress condition	0.13 (.08)
	Sex	−0.003 (.08)
	BDI	0.22 (.04) ^{***}
	MDD	0.42 (.22)
	Time	−0.001 (.001)
	Stress condition × time	−0.002 (.001)
	Sex × time	−0.001 (.001)
	Cortisol	0.25 (.12) [*]
	MDD × cortisol	−0.54 (.20) ^{**}

* $p < .05$;** $p < .01$;*** $p < .001$

Note Stressor condition (0 = control; 1 = stress); sex (0 = male; 1 = female); BDI = depressive symptoms in the past two weeks; MDD (0 = never-depressed; 1 = remitted-depressed).

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