Biological Psychology 109 (2015) 159–165

Contents lists available at ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

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

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ARTICLE INFO

Article history:
Received 20 September 2014
Received in revised form 27 May 2015
Accepted 31 May 2015
Available online 3 June 2015

Keywords:
Depression
Cortisol
Shame
Stress
Social-evaluative threat

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 25–55 min 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.

1. Introduction

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 & Ehlert, 2012), and whether problems with coordination are associated with risk for stress-related psychopathology. Whereas subjective emotional 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 min 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, & Fiqueiredo, 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,

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http://dx.doi.org/10.1016/j.biopsycho.2015.05.007
0301-0511/

Flicker, & Barlow, 1996) and are associated with withdrawal and disengagement behaviors that could serve to de-escalate 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, Gruenewald, & Kemeny, 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 (Gruenwald, 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 et al. (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.

2. 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 et al., 2008; Brown, 2001; Trestman et al., 1991; see also Bagley, Weaver, & Buchanan, 2011; Lange, Zschucke, Ising, Uhr, & Bermpohl, 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 et al. (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.

3. Method

3.1. Participants

Participants were 102 individuals (56 remitted-depressed and 46 never-depressed), ages 18–31 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 excluded[1]. Participants were recruited from

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