



Research paper

Depressive symptom composites associated with cortisol stress reactivity in adolescents

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ABSTRACT

Background: Altered hypothalamic-pituitary-adrenal (HPA) function is common in youth with major depressive disorder (MDD) but variability in the strength and direction of HPA alterations has prompted a search for symptom-based subtypes with unique neuroendocrine signatures. This study investigated the extent to which depressive symptom composites were differentially associated with cortisol responses to psychosocial stress.

Methods: This study examined salivary cortisol responses to the Trier Social Stress Test (TSST) in 145 adolescents who varied in their risk for MDD: 38 had current MDD; 35 were healthy but at high risk for MDD based on having one or both parents with unipolar MDD; and 72 were healthy youth with no personal or family history of a psychiatric disorder. Multilevel models examined within-person change in cortisol levels during a 2-h resting phase prior to the TSST and both linear and quadratic changes in cortisol levels following the TSST.

Results: Anticipatory cortisol reactivity was lower in MDD youth compared to low-risk youth, and in youth with higher compared to lower depressive symptom severity. Whereas affective symptoms were associated with increased anticipatory cortisol reactivity and more rapid recovery to the TSST, neurovegetative symptoms were associated with decreased anticipatory cortisol reactivity and slower recovery.

Limitations: The cross-sectional design does not permit inferences regarding temporal relations between cortisol responses and depressive symptom composites.

Conclusions: The present findings suggest that heterogeneity among studies examining HPA reactivity in depressed youth may be driven, in part, by differences in depressive symptom composites across samples.

1. Introduction

Hypothalamic-pituitary-adrenocortical (HPA) alterations in youth with major depressive disorder (MDD) are not universal (for reviews, see Lopez-Duran et al. (2009) and Stetler and Miller (2011)). Heterogeneity in stress responses in healthy individuals has spurred the development of criteria for distinguishing cortisol “responders” from “nonresponders” (Miller et al., 2013). These individual differences are especially pronounced in depressed youth (for a review, see Lopez-Duran et al. (2009)) and have likely contributed to inconsistent findings on the relation between depression and HPA reactivity in youth. Some efforts to explain why only 40–60% of depressed individuals exhibit HPA alterations (for a review, see Parker et al. (2003)) have proposed depressive subtypes with unique neuroendocrine signatures and distinct etiologies (for a review,

see Antonijevic (2006)). These efforts, driven in part by growing dissatisfaction with descriptive phenomenological approaches to psychiatric diagnosis, examined whether neuroendocrine abnormalities might adhere more closely to specific depressive symptom clusters than to depressive diagnoses (Halbreich, 2006). The present study adopted a symptom-based approach to investigate the determinants of cortisol stress reactivity in adolescents who varied in their depressive symptom presentation.

1.1. Psychosocial stressors and cortisol reactivity in youth

Adolescence is a developmental period characterized by elevated stress levels, due to changes in academic environment, social activities, and physical development (Ge et al., 2003; Wigfield et al., 1991).

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Further, youth who experienced stressful life events were more likely to develop MDD (Grant et al., 2004). Interpersonal stressors predicted first onsets and recurrences of MDD (Sheets and Craighead, 2014), and the HPA axis appeared to play a role in this increased vulnerability (Colich et al., 2015; Rao et al., 2008). For example, cortisol reactivity to psychosocial stressors predicted depressive symptom trajectories in formerly depressed emerging adults (Morris et al., 2012) and development of MDD in girls (Colich et al., 2015). Therefore, cortisol reactivity may be especially useful as a risk marker for depression. Further supporting this view, Stroud et al. (2009) proposed that changes in the stress response over the adolescent transition may account, in part, for the growing risk for MDD during adolescence (Hankin et al., 1998).

Most research to date examining relations between depressive symptoms and HPA activity has focused on diurnal cortisol secretion (Rao et al., 1996) and responses to a pharmacologic challenge (for reviews, see O'Keane et al. (2012) and Stetler and Miller (2011)). Psychosocial stress paradigms complement these approaches by probing suprahypothalamic circuits involved in the HPA response to stress. Studies of cortisol reactivity to psychosocial stress in child and adolescent samples, however, have yielded inconsistent findings. Greater cortisol responses have been reported for depressed compared to non-depressed adolescents (Rao et al., 2008), dysphoric post-pubertal youth compared to non-dysphoric youth (Hankin et al., 2010), and adolescents with more recent onset of depressive symptoms (Booij et al., 2013). In contrast, blunted cortisol responses have been reported for dysphoric pre-pubertal youth compared to non-dysphoric youth (Hankin et al., 2010), adolescents with more severe depression severity (Harkness et al., 2011), adolescents with internalizing symptoms (Spies et al., 2011), children with higher self-reported depressive symptoms (Dieleman et al., 2010), children with current, past or sub-syndromal MDD (Suzuki et al., 2013), and adolescents with more chronic depressive symptoms (Booij et al., 2013). Taken together, these findings suggest that alterations in cortisol reactivity are not limited to youth with depressive diagnoses and may be determined by a variety of factors including depression severity and developmental timing. Recent evidence suggests that prepubertal girls with blunted cortisol responses and postpubertal girls with elevated cortisol responses were at greatest risk for MDD onset (Colich et al., 2015). The present study considered associations between pubertal stage and cortisol response parameters.

1.2. Depressive symptom composites

MDD is a syndrome characterized by heterogeneity in symptom presentation: there are 227 possible symptom combinations that satisfy the diagnostic criteria (van Loo et al., 2012). Although youth with MDD must present with at least one core affective symptom (depressed or irritable mood and/or anhedonia), they may exhibit different combinations of neurovegetative symptoms (i.e., sleep or appetite disturbances, fatigue, psychomotor retardation) or cognitive symptoms (i.e., impaired concentration, worthlessness, guilt, suicidal ideation). Heterogeneity in the presentation of depressive symptoms may help to explain why youth exhibit increased or decreased cortisol reactivity to psychosocial stressors. Research in depressed preschoolers suggested that those with anhedonia have higher cortisol reactivity than those without anhedonia (Luby et al., 2004) but did not differ from hedonically depressed or non-depressed preschoolers in cortisol levels at the end of a stress task (Luby et al., 2003). Research on neurovegetative symptoms indicated that higher fatigue was associated with lower cortisol reactivity to a laboratory stressor in non-depressed adults (Lennartsson et al., 2015), and relations between sleep disturbances and cortisol reactivity to laboratory stressors differed for boys and girls (Pesonen et al., 2012). To our knowledge, no studies have examined relations between cognitive depressive symptoms and cortisol reactivity in depressed youth. However, self-esteem has been implicated as a vulnerability factor for depression and is closely tied to

one of the cognitive depressive symptoms – feelings of worthlessness (Beck, 1991; Roberts and Monroe, 1999). Research in healthy adults indicated that higher self-esteem was associated with lower anticipatory cortisol reactivity to a stress task (Turan, 2015) and lower cortisol reactivity to interpersonal rejection (Ford and Collins, 2010).

1.3. Maltreatment history

Exposure to childhood maltreatment (including physical, sexual or emotional abuse, physical or emotional neglect, and exposure to domestic violence) can produce enduring changes in HPA function and can increase vulnerability to MDD (for a review see Heim et al. (2008)). Neurodevelopmental traumatology models posit that the long-term negative sequelae of childhood maltreatment are caused, in part, by the adverse effects of stress response dysregulation on brain development during critical vulnerability periods (for a review, see De Bellis et al. (2011)). Previous studies indicated that adolescents with a history of maltreatment exhibit elevated cortisol reactivity and delayed cortisol recovery to psychosocial stress tasks (MacMillan et al., 2009), except in cases of more severe depressive symptoms (Harkness et al., 2011). The importance of accounting for childhood maltreatment in studies examining associations between HPA reactivity and MDD is underscored by researchers who posit that MDD in the context of maltreatment may represent a distinct subtype (for a review, see Heim et al. (2004) and Rao et al. (2008)). Accordingly, the present study accounted for the effects of maltreatment history while examining the association between depressive symptoms and cortisol response parameters.

1.4. The present study

The present study sought to examine the extent to which specific depressive symptoms (affective, cognitive, neurovegetative) predict cortisol reactivity to a psychosocial stressor, thereby accounting for the mixed findings regarding depression and cortisol reactivity. As a first step, our aim was to replicate prior research on the associations between MDD status (currently depressed, at high risk for depression, or low risk for depression) and cortisol reactivity in youth. We expected that our largely post-pubertal sample of adolescents with MDD would exhibit greater cortisol responses than adolescents at high- or low-risk for depression. Second, based on evidence that alterations in cortisol reactivity were present in both syndromal and sub-syndromal depressed youth (Suzuki et al., 2013), we examined the association of depression severity and cortisol responses. We hypothesized that higher overall depression severity, regardless of diagnostic status, would be associated with more blunted anticipatory cortisol reactivity and recovery in adolescents (Harkness et al., 2011).

Third, our primary study aim was to examine affective, neurovegetative and cognitive depressive symptom composites as predictors of cortisol stress responses. This method of categorizing depressive symptoms conceptually has proven useful in prospective studies (Kouros et al., 2016) and limits the number of predictors tested per model. Based on prior work in depressed adolescents (Hankin et al., 2010; Rao et al., 2008), we hypothesized that affective symptoms would be associated with a robust cortisol response pattern in adolescents marked by increased anticipatory reactivity and more rapid cortisol recovery. Although no studies, to our knowledge, have examined associations between the neurovegetative symptom cluster and cortisol reactivity in youth, we nonetheless hypothesized that neurovegetative symptoms would be associated with a pattern of blunted anticipatory cortisol reactivity and recovery in adolescents based on research examining fatigue in adults (Lennartsson et al., 2015). Given the lack of research on cognitive symptoms and cortisol reactivity in youth, no specific hypotheses were made. However, based on studies in adults (Ford and Collins, 2010; Turan, 2015), we expected that higher levels of cognitive symptoms would be associated with blunted anticipatory

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