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Neurocognitive function and state cognitive stress appraisal predict cortisol reactivity to an acute psychosocial stressor in adolescents

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Summary Stress and associated alterations in hypothalamic—pituitary—adrenal (HPA) function have deleterious influence on the development of multiple mental and physical health problems. Prior research has aimed to identify individuals most at risk for the development of these stress-related maladies by examining factors that may contribute to inter-individual differences in HPA responses to acute stress. The objectives of this study were to investigate, in adolescents, (1) whether differences in neurocognitive abilities influenced cortisol reactivity to an acute stressor, (2) whether internalizing psychiatric disorders influenced this relationship, and (3) whether acute cognitive stress-appraisal mechanisms mediated an association between neurocognitive function and cortisol reactivity. Subjects were 70 adolescents from a community sample who underwent standardized neurocognitive assessments of IQ, achievement, and declarative memory measures at mean age 14 and whose physiological and behavioral responses to a standardized psychosocial stress paradigm (Trier Social Stress Test, TSST) were assessed at mean age 18. Results showed that, among all adolescents, lower nonverbal memory performance predicted lower cortisol reactivity. In addition, internalizing disorders interacted with verbal memory such that the association with cortisol reactivity was strongest for adolescents with internalizing disorders. Finally, lower secondary cognitive appraisal of coping in anticipation of the TSST independently predicted lower cortisol reactivity but did not mediate the neurocognitive—cortisol relationship. Findings suggest that declarative memory may contribute to inter-individual differences in acute cortisol reactivity in adolescents, internalizing disorders may influence this relationship, and cognitive stress appraisal also predicts cortisol reactivity. Developmental, research, and clinical implications are discussed.
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1. Introduction

Extensive research has focused on the deleterious impact of stress on the development of multiple mental and physical health problems including depression, anxiety, cardiovascular disease, and cancer, and the association of these disorders with functional alterations in the hypothalamic–pituitary–adrenal (HPA) axis, the primary physiological mediator of the stress response (Tsigos and Chrousos, 1994; Heim et al., 2000). Much less is known about which individuals are at increased risk for the development of HPA dysregulation and associated stress-related disorders. Thus, while many studies differentiate patterns of HPA dysregulation between groups (e.g., depressed vs. healthy adults), a growing number of investigations have begun to focus on inter-individual differences to identify mechanisms of increased risk for adverse outcomes. Studies to date have largely concentrated on demographic and health-behavior variables (e.g., age, gender, contraceptive use, caffeine, and alcohol), as well as personality features, using laboratory psychosocial stress challenges such as the Trier Social Stress Test (TSST Kirschbaum et al., 1993) within healthy subjects (for reviews see Foley and Kirschbaum, 2010; Kudielka and Wust, 2010).

Neurocognitive factors may contribute to differences in the physiological transduction of psychosocial stress given their significant influence on skills essential for adaptation to the stress and demands of daily life (Fiocco et al., 2007; Stawski et al., 2011). Accordingly, neurocognitive weaknesses may increase subjective and physiological experiences of stress, including activation of the HPA axis (Power et al., 2008; Franz et al., 2011). Existing studies have varied in assessing the temporal relationship of neurocognitive and HPA function, and whether neurocognitive and HPA function is assessed at baseline or under stress. Most investigations have examined the impact of reactive cortisol levels on measures of neurocognitive function, with most, but not all (Domes et al., 2002; Nater et al., 2007), suggesting that increased cortisol levels predict impaired cognition, particularly in both verbal and visual memory (Kirschbaum et al., 1996; Lee et al., 2007; Quesada et al., 2012). Several longitudinal community studies, however, suggest a bidirectional effect of neurocognitive and HPA function; that is, lower baseline neurocognitive performance predicts changes in HPA axis diurnal function characterized by lower morning and higher afternoon/evening cortisol levels and a flatter diurnal cortisol slope presumably due to chronic stress (Lupien et al., 2005; Power et al., 2008; Franz et al., 2011; Stawski et al., 2011). Some, but not all of these investigations, also controlled for anxiety and depressive symptoms (Lupien et al., 2005; Franz et al., 2011).

Little is known about whether and how baseline neurocognitive abilities predict adolescent HPA reactivity to acute stress. Ginty et al. (2012) assessed the relationship of baseline general intelligence (IQ) and verbal memory with acute cortisol and cardiovascular (CV) responses following a series of laboratory stress tasks in a large community sample of adults and found that lower cortisol and CV responses to the stressors were associated with lower verbal memory ability, but not IQ. A study of children 5–18 years of age similarly found that higher HPA reactivity to a speech task was associated with higher global academic achievement scores post-task (Mathewson et al., 2012). Using education level as a proxy for academic abilities, Fiocco et al. (2007) found that adults with lower educational levels had greater cortisol responses to the TSST. Secondary analyses found that those with lower levels of education also performed more poorly on tests of verbal fluency, but groups did not differ on measures of digit span, self-esteem, or appraisal of the TSST as stressful. Although limited in scope and number, these investigations suggest that specific domains of neurocognitive function and acute cortisol reactivity to stress may be related and influenced by developmental factors.

Very little is also understood about whether the bidirectional associations of neurocognitive and HPA function are influenced by the presence of psychiatric conditions such as anxiety and depression despite evidence of the (1) high prevalence of these disorders among adults and children (Kessler et al., 2005; Merikangas et al., 2010), (2) frequent co-occurrence of neurocognitive deficits and learning disorders with depression and anxiety (Wenz-Gross and Siperstein, 1998; Feurer and Andrews, 2009), and (3) association of depressive and anxiety disorders with stress and HPA dysfunction (Tafet and Bernardini, 2003; Von Werne Baes et al., 2012). Existing studies of post-traumatic stress disorder, depression, and anxiety suggest a differential effect among clinical subjects compared to controls, with higher levels of cortisol generally associated with greater deficits in neurocognitive function in those with internalizing disorders (Gomez et al., 2009; Hinkelmann et al., 2009; Lagarde et al., 2010; Lenze et al., 2012; Wingenfeld et al., 2012).

A separate growing body of cognition-focused studies have examined the proximal impact of cognitive stress appraisal on cortisol reactivity. A transactional model of stress posits that perceptions of increased threat and decreased ability to cope are associated with greater experience of subjective stress to specific stressors (Folkman et al., 1986). Support for this model is derived from a recent meta-analysis of laboratory acute stress challenges that found social evaluative threat and lack of control as central to inducing cortisol reactivity (Dickerson and Kemeny, 2004). Contemporary measures of acute cognitive stress processes distinguish between Primary Appraisal (threat and personal relevance of a situation) and Secondary Appraisal (self-assessment of ability to cope with the stressor) (Gaab et al., 2005). Although limited, findings support an association of subjective reports of increased acute cognitive stress appraisal with increased cortisol reactivity (Wirtz et al., 2007; Juster et al., 2012), with one investigation suggesting an effect of increased primary (threat) but not secondary (coping ability) stress appraisal (Gaab et al., 2005).

The current study aims to investigate the relationship of neurocognitive function and cortisol reactivity in a longitudinal community sample of adolescents using verbal and nonverbal subtests of commonly used measures of IQ, academic achievement, and declarative memory; and whether these relationships are influenced by the presence of psychiatric internalizing disorders. The study further examines the influence of acute cognitive appraisal of stress in this relationship. We hypothesize that (1) youth with lower cognitive abilities in both verbal and nonverbal domains will have decreased cortisol reactivity, (2) that this finding will be more robust in those with internalizing disorders due to the compounding, or interactive, effect of having anxiety and/or depression, and (3) that higher levels of acute cognitive

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