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Adolescent adrenocortical activity and adiposity: Differences by sex and exposure to early maternal depression

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Summary Prior research has linked either basal cortisol levels or stress-induced cortisol responses to adiposity; however, it remains to be determined whether these distinct cortisol measures exert joint or independent effects. Further, it is unclear how they interact with individual and environmental characteristics to predict adiposity. The present study aims to address whether morning cortisol levels and cortisol responses to a psychosocial stressor independently and/or interactively influence body mass index (BMI) in 218 adolescents (117 female) participating in a longitudinal community study, and whether associations are moderated by sex and exposure to early maternal depression. Reports of maternal depressive symptoms were obtained in infancy and preschool. Salivary cortisol measures included a longitudinal morning cortisol measure comprising sampling points across ages 11, 13, 15, and 18 and measures of stress-induced cortisol responses assessed via the Trier Social Stress Test (TSST) at age 18. Lower morning cortisol and higher TSST cortisol reactivity independently predicted higher age 18 BMI. Morning cortisol also interacted with sex and exposure to early maternal depression to predict BMI. Specifically, girls exposed to lower levels of early maternal depression displayed a strong negative morning cortisol—BMI association, and girls exposed to higher levels of maternal depression demonstrated a weaker negative association. Among boys, those exposed to lower levels of maternal depression displayed no association, while those exposed to higher levels of maternal depression displayed a negative morning cortisol—BMI association. Results point to the

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independent, additive effects of morning and reactive cortisol in the prediction of BMI and suggest that exposure to early maternal depression may exert sexually dimorphic effects on normative cortisol—BMI associations.

1. Introduction

Obesity is a global health concern with many negative short- and long-term consequences for well-being (Kraak and Story, 2010). Altered hypothalamic—pituitary—adrenal (HPA) axis activity, indexed by glucocorticoids such as cortisol, has been linked to higher adiposity through the interaction of the HPA axis with multiple neurophysiological systems, including activation of brain regions controlling food intake and hormones implicated in metabolism and appetite regulation (for a review, see Spencer and Tilbrook, 2011). Although basal cortisol levels and stress-induced cortisol responses have been separately linked to adiposity, research has yet to examine how these distinct cortisol measures work together. Furthermore, separate lines of research suggest that cortisol—adiposity associations may be influenced by the sex of the individual (Walker et al., 2000; Larsson et al., 2009) as well as prior stress exposure (Donoho et al., 2011). Understanding these complex associations may have important implications for improved identification of who is at risk of obesity as well as potential targets for intervention.

Studies of cortisol and adiposity have examined both diurnal measures and stress-induced responses of the HPA axis (Chrousos, 2000). Daily fluctuations in level of cortisol output typically follow a diurnal rhythm characterized by a surge upon awakening followed by a decline across the day. In response to a stressor, cortisol levels generally begin to increase upon encountering the stressor, peak approximately 10—30 min after the stressor's onset (Foley and Kirschbaum, 2010), and then decline depending on the nature of the challenge and the individual's resources. In general, studies of adiposity and HPA-axis functioning, both diurnal and stress-induced, have yielded mixed results (e.g., Wallerius et al., 2003; Brydon, 2011; Phillips et al., 2012). Nevertheless, many studies have linked greater adiposity to either lower morning levels of cortisol (Björntorp and Rosmond, 2000; Walker et al., 2000; Ranjit et al., 2005; Larsson et al., 2009; Kumari et al., 2010; Champaneri et al., 2013) or to higher cortisol responses to an experimental psychosocial stressor, with the latter indexed by a variety of measures including change from baseline to peak (Benson et al., 2009), total cortisol output (Epel et al., 2000), and cortisol output relative to baseline (Benson et al., 2009; Dockray et al., 2009). In our previous work examining associations of adolescent adiposity with diurnal cortisol, including morning, afternoon, and evening levels and decline over the day, we found that lower morning levels were most closely and consistently associated with higher BMI (both concurrently and longitudinally) at multiple time points across adolescence; moreover, a longitudinal measure of morning cortisol across ages 11, 13, and 15 was a stronger predictor of BMI than morning cortisol at any single age (Ruttle et al., 2013a). However, to our knowledge, no studies have considered both morning levels and stress-induced cortisol responses, leaving unexamined whether they jointly or independently predict adiposity.

The mixed findings regarding adiposity and HPA-axis functioning suggest that important individual characteristics or contextual factors may moderate those associations. In particular, there is some evidence of the moderating effects of an individual's sex and history of stress exposure. Although the majority of cortisol—adiposity studies solely examine one sex (e.g., Björntorp and Rosmond, 2000; Epel et al., 2000; Benson et al., 2009; Brydon, 2011; Hillman et al., 2012), a few studies have considered sex differences, including two that found lower morning cortisol levels to be associated with higher adiposity in women but not men (Walker et al., 2000; Larsson et al., 2009). Further, one study of adolescent females found that a higher cortisol awakening response was more strongly associated with higher adiposity among those exposed to recent chronic school-related stress (Donoho et al., 2011).

Previous studies of the cortisol—adiposity association have not considered the potential moderating influence of early chronic stress exposure. Among the more common early life stressors, exposure to maternal depression may be particularly salient due to associated deficits in mother—child interactions and insecure attachments that can alter HPA-axis activity (Ashman et al., 2002; Brennan et al., 2008; Dougherty et al., 2011) and influence feeding behaviors (Ertel et al., 2010). We previously demonstrated that early exposure to higher maternal depressive symptoms had a particularly potent effect on later HPA-axis activity both in childhood and adolescence (Essex et al., 2002b, 2011; Ruttle et al., 2013b). There is also some evidence that exposure to early, chronic maternal depression is associated with increased likelihood of later overweight or obesity; however, research into adolescence is sparse and authors suggest examining moderating factors to clarify associations (Lampard et al., 2014).

The current study builds on prior research, including our own, first by investigating the independent and joint associations of morning cortisol levels and cortisol responses to a psychosocial stressor with adiposity (i.e., body mass index; BMI) in a longitudinal community sample of 18-year-olds, and second by considering whether these cortisol—BMI associations are moderated by the individual's sex and exposure to maternal depression across the infancy and preschool periods. We hypothesized that lower morning cortisol levels would be associated with higher BMI. We also anticipated that greater cortisol responses to the psychosocial stress paradigm, the Trier Social Stress Test (TSST), would also be associated with higher BMI; however, due to inconsistencies in the measures used across previous studies, we took an exploratory approach by examining three indices of stress-induced cortisol responses. We also anticipated moderating effects of sex and exposure to early maternal depression on