Stony Brook University
The Graduate School

Doctoral Defense Announcement

Abstract

Salivary Cortisol and Depression Risk: Relations with Child Temperament, Maternal History of Depression, Parenting and Life Stress

By

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Dysregulation of the HPA system has been consistently associated with the affective disorders. The current study aims to examine whether low positive emotionality (PE), a possible temperamental precursor to depression, is associated with HPA axis functioning in children prior to the onset of a depressive disorder. This study also aims to examine the unique and joint effects of some of the complex factors related to children's early HPA axis functioning and depression risk, including maternal history of depression, parenting and life stress, alongside early temperament. These factors were examined in relation to children's salivary cortisol levels in response to a laboratory stressor and to children's home basal cortisol levels. One hundred and sixty-six preschool-aged children and their biological parents were recruited from a larger study examining temperamental risk for mood disorders: 166 children completed all four salivary cortisol samples during the laboratory visit; 94 children provided a home morning salivary cortisol sample; and 93 children provided a home evening salivary cortisol sample. Child temperament and parenting were assessed using laboratory observational measures, and maternal depression and child life stressors were assessed with semi-structured interviews.

First, mixed effects modeling was used to examine predictors of children's laboratory cortisol reactivity. We found that temperamental negative emotionality (NE), behavioral inhibition (BI), parental hostility and life stress were significantly associated with components of the trajectory of cortisol during the laboratory visit. Furthermore, several significant interactions emerged that were associated with greater laboratory cortisol reactivity, including low PE X maternal melancholic depression, child BI X maternal melancholic depression, child low PE X parental hostility, and maternal depression X parental hostility. In addition, child BI interacted with parental support to predict lower laboratory baseline cortisol levels. Next, multiple regression was used to examine predictors of children's home basal cortisol levels. We found that both child low PE and maternal melancholic depression were significantly associated with higher morning cortisol levels, suggesting potential depression endophenotypes. The study's findings are summarized in terms of risk, resilience, and potentiation as they relate to the development of neuronendocrine dysfunction in young children, and suggest potential avenues for future research.

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