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Early calibration of the HPA axis by maternal psychopathology☆

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ABSTRACT

Given the central role of stress-responsive neurophysiology in mental and physical health, it is important to understand how particular patterns of stress responsivity may become entrained by the early caregiving environment. In this study we investigated links between maternal depression and anxiety symptom profiles and within-infant development of hypothalamic-pituitary-adrenal (HPA) axis responses from 6 to 18 months of life. Associations with infant cognitive and social-emotional development were also tested to gauge the adjustment implications of HPA response trajectories. Mothers from a low-income community sample reported their symptoms at 3, 6, 12, and 18 months postnatal, and infants engaged in interpersonal stress tasks at 6, 12, and 18 months. Four saliva samples were taken at each time to assess cortisol responses, and a developmental screener at 18 months provided an index of infant adjustment. Multilevel modeling results revealed an association between maternal symptoms and infant HPA axis sensitization—i.e., a higher cortisol reactivity slope that increased over time. In particular, early (3-month) depression symptoms among mothers who had crossed a diagnostic threshold for major depressive disorder predicted this pattern of response, which in turn related to poorer infant developmental outcomes. Results are considered in terms of adaptive calibration of stress response systems, which may come at a cost to individual psychosocial functioning.

1. Introduction

The functioning of stress-responsive neurophysiology is central to health (McEwen, 2013), making it important to understand its origins—how a system such as the hypothalamic-pituitary-adrenal (HPA) axis learns to respond in ways that increase susceptibility to disorder. The early caregiving environment is thought to shape child stress systems to adapt to prevailing risk conditions. To identify and intervene with high-risk families, it is important to understand which parental characteristics calibrate child stress responsiveness, and how this maps onto child adjustment. The current study adds to this understanding by investigating infants' HPA response patterns from 6 to 18 months in relation to both maternal psychopathology and infant development.

Parental psychopathology increases offspring risk for disorder, with particularly strong evidence for maternal depression effects on child internalizing and externalizing (Goodman et al., 2011). One likely risk pathway is functioning of the HPA axis, the neuroendocrine system culminating in cortisol release. Individuals at risk for and suffering from depression show altered patterns of cortisol response to stress (e.g., Bouma et al., 2011; Powers et al., 2016). Whereas *level* of response has varied, there is more consistent evidence that *dynamics* of response are altered, with depression-prone individuals showing extended reactivity/impaired recovery (Burke et al., 2005). In early development, maternal depression symptoms were shown to predict infants' cortisol response to interpersonal stress; different symptom courses predicted higher vs. lower levels, but consistently delayed recovery (Laurent et al., 2011). Thus, exposure to a depressed mother may tune the infant's developing HPA axis toward a profile enhancing risk for psychopathology. Given evidence that low socioeconomic status increases risk of depression (Musliner et al., 2016) and that maternal negativity acts as a mechanism connecting low income to child HPA dysregulation (Zalewski et al., 2012), it may be particularly important to examine maternal symptom-related effects in low-income samples.

A useful theoretical model for understanding how and why the HPA axis is tuned in a particular way is the Adaptive Cal-

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ibration Model (ACM; Del Giudice et al., 2011). Grounded in evolutionary theory, the ACM proposes that stress response systems are calibrated by early environmental conditions to increase the organism's fitness in that environment, even if this comes with a cost to well-being. Environmental threat and unpredictability should entrain greater stress responsiveness, fostering a "vigilant" phenotype characterized by HPA hyperresponsivity and both internalizing and externalizing behaviors. Research in humans and rodents has demonstrated sensitization of the HPA axis—increased responsivity to subsequent stress—following exposure to emotional stressors (Belda et al., 2015), providing a model for how the vigilant phenotype could develop in higher-stress environments.

Because caregivers are thought to transmit information about conditions in the broader environment (de Baca et al., 2016), parental stress and symptomatology should influence infant HPA calibration. That is, while a variety of parent characteristics contribute to the child's development, depression—which has been consistently tied to life stress (Hammen, 2005; Monroe et al., 2009)—may offer particularly salient information about environmental risk vs. safety. Indeed, research involving diurnal cortisol suggests parental depression symptoms may impact child cortisol levels and stability over time, which in turn relate to internalizing and externalizing problems (Laurent et al., 2014). Other research highlights HPA sensitization—increasing cortisol levels across childhood, which predicted cortisol non-recovery from acute stress and internalizing symptoms by late childhood—in a poor caregiving context (Laurent et al., 2015). Together, these studies support ACM predictions that exposure to a symptomatic parent could sensitize the HPA axis, leading to a pattern of responding that, even if "fit" for a higher-risk environment, limits psychosocial functioning. However, there is little within-individual research addressing such calibration, particularly during early development.

Besides establishing whether stress calibration is evident, research must clarify the importance of different types of exposures. While the bulk of research has focused on maternal depression, there is evidence that anxiety—with which it is often comorbid—also relates to child outcomes (e.g., Feldman et al., 2009; O'Connor et al., 2002), and the degree to which these syndromes exert distinct or similar effects requires further study. The general distress component underlying both depression and anxiety could be the active ingredient shaping child stress sensitivity, or there may be effects attributable to unique components of these syndromes. A mix of studies involving categorical diagnoses or continuous symptoms leaves questions about whether symptom effects vary depending on mothers' history of disorder; current symptom elevations may carry greater weight for individuals who have crossed the threshold from subclinical distress to clinically diagnosable conditions, intensifying the signal received by the child. Finally, there are variable indications that exposure to consistently elevated maternal depression, symptoms during early development, or increasing maternal symptoms is the most potent influence on child HPA function (Apter-Levi et al., 2016; Essex et al., 2002; Laurent et al., 2011, 2012). Each of these possibilities must be interrogated to understand the conditions for risky stress calibration.

The present study aimed to shed light on early HPA axis calibration by testing associations between mothers' internalizing symptoms and their infant's cortisol response to developmentally appropriate interpersonal stressors at 6, 12, and 18 months. Infants' HPA responses during dyadic stress do not occur in a vacuum, but rather are part of a dyadic response that may itself be influenced by maternal depression (Laurent et al., 2011); thus, cortisol was assessed in both mother and infant and modeled together. To discern parental symptom effects against a backdrop of moderately stressful environmental conditions that themselves foster psychopathology, families comprised a low-income community sample. A developmental screener was used to gauge adjustment implications of infant cortisol response trajectories. Guided by the ACM, it was hypothesized that exposure to maternal depression or anxiety symptoms would predict infant HPA sensitization, characterized by cortisol non-recovery during stress that becomes more marked from 6 to 18 months. These effects were expected to be exacerbated in cases where mothers had crossed diagnostic threshold for a lifetime depressive or anxiety disorder, and for earlier and/or increasing maternal symptom exposure. Finally, it was predicted that HPA sensitization would relate to poorer infant cognitive and social-emotional development.

2. Material and methods

2.1. Participants

Mothers were recruited from the Women Infants Children (WIC) program and other community agencies serving low-income families in a mid-sized city in the northwest. To be eligible, mothers had to speak English, have a <12-week-old infant, and anticipate remaining in the area until this target child was 18 months old. Table 1 gives demographic information about the sample at the first assessment.

Of the 91 mothers who completed the time 1 study assessment, 54 completed the final (time 4) assessment. Compared to non-completers, study completers tended to be older ($M = 28.32$ vs. 25.18, $t[89] = 2.84$, $p = 0.006$), in a longer-term romantic relationship (median length = 2-5 yrs vs. 1–2 yrs, $\chi^2[4] = 10.00$, $p = 0.041$), report higher household income (median = \$20,000-\$29,999 vs. \$10,000-\$19,999, $\chi^2[7] = 14.39$, $p = 0.045$), and have more biological children ($M = 2.94$ vs. 2.55, $t[89] = 2.01$, $p = 0.047$). There were no differences in racial/ethnic group identification, likelihood of being in a relationship with the target child's biological father or degree of contact with the father, education, or employment status. Of the mental health-related variables reported at time 1, the only difference that emerged was for current (3-month) depressive symptoms: $M = 7.75$ for completers vs. 12.36 for non-completers, $t(85) = 2.58$, $p = 0.011$. Analyses of maternal symptom effects are based on the 73 cases with complete time 1 questionnaire data who also participated in the time 2 stress session. Analyses of infant adjustment effects are based on the 53 cases with complete time 4 questionnaire data (in addition to stress session data).

2.2. Procedure

Prior to study participation, mothers gave written informed consent to all study procedures, which had been approved by the University of Oregon Institutional Review Board. Mothers completed study assessments at four times: time 1 at 3 mos postnatal, time 2 at 6 mos, time 3 at 12 mos, and time 4 at 18 mos. At each time, mothers responded to questionnaires via Qualtrics. At time 1, they also completed a home visit that involved a diagnostic interview, and at times 2–4 they completed laboratory sessions with their infant. Each laboratory session included a developmentally appropriate interpersonal stressor involving maternal unavailability and/or confrontation with a strange adult. For each of these tasks, if the infant showed signs of extreme distress (i.e., crying continuously for more than 30s), the mother was allowed to re-engage and comfort him/her. Each session began with mothers answering questions about conditions that would preclude participation at that time (see below). Mother-infant dyads were then led through the stress task/s, followed by a period of 45–50 mins during which the mother completed questionnaires with her infant in the room.