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Parent-child adrenocortical concordance in early childhood: The moderating role of parental depression and child temperament

Stephanie M. Merwin, Victoria C. Smith, Marissa Kushner, Edward P. Lemay Jr., Lea R. Dougherty*

University of Maryland College Park, USA

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ABSTRACT

This study examined biological concordance between parent and child morning cortisol and whether parent and child-level risk factors for depression moderated this association. Participants included 136 parents and their preschool-aged children. Parents and children obtained salivary cortisol samples at waking, and 30 and 45 min post-waking across two days to assess the cortisol awakening response. Parental lifetime depression was assessed using a clinical interview and child temperamental negative emotionality (NE) and positive emotionality (PE) were assessed using an observational laboratory-based assessment. Results indicated significant parent-child concordance between both average cortisol levels and cortisol fluctuations across waking. Greater concordance was observed for dyads with parents with a lifetime history of depression and with children high in NE and PE. These parent- and child-level moderators were associated with different indices of concordance. Findings highlight the need to examine the role of parent and child risk factors for depression on parent-child adrenocortical concordance.

1. Introduction

The parent-child dyad is a dynamic system that involves bidirectional and transactional processes that begin in infancy and evolve over time. The profound effects of the parent-child relationship on children's cognitive, social, emotional, and neurobiological development has been well-documented (Belsky & de Haan, 2011). Moreover, emerging research supports the significance of parent-child synchrony, or the matching of parent and child behavior and/or biological rhythms within the parent-child dyad (Feldman, 2007). Evidence suggests that behavioral synchrony, or the temporal matching of discrete events (e.g., eye-gaze, vocalizations, touch, affective states), within the parent-child dyad predicts children's positive outcomes including increased self-regulation, attachment security, stress management, and fewer behavior problems (Feldman, 2007, 2012a, 2012b). However, very little is known about biological synchrony, which is characterized by the matching of biological rhythms or physiological processes between parent and child. Importantly, biological synchrony may serve as a potential mechanism underlying individual differences in the parent-child relationship and in children's developmental pathways and outcomes (Feldman, 2012a, 2012b).

Recent research has investigated parent-child concordance of the hypothalamic-pituitary-adrenal (HPA) axis, one of the body's primary stress response systems, as an aspect of biological synchrony, which reflects the attunement or association of the stress hormone cortisol between the parent and child. The HPA axis holds particular importance as it has been consistently linked to social and emotional functioning, life stress, and numerous physical and psychological health outcomes (Chida & Steptoe, 2009). Findings demonstrate significant parent-child adrenocortical concordance both in their cortisol response to stressors (Atkinson et al., 2013; Hibel, Blair, Granger, & Cox, 2009; Middlemiss, Granger, Goldberg, Nathans, 2012; Ruttle, Serbin, Stack, Schwartzman, & Shirtcliff, 2011; Saxbe et al., 2014; Sethre-Hofstad, Stansbury, & Rice, 2002; van Bakel & Riksen-Walraven, 2008) and their diurnal cortisol patterns (Bright, Granger, & Frick, 2012; Hibel, Trumbell, & Mercado, 2014; LeMoult, Chen, Foland-Ross, Burley, & Gotlib, 2015; Papp, Pendry, & Adam, 2009; Williams et al., 2013). Greater parent-child adrenocortical concordance in response to an acute stressor task has been associated with both maternal sensitivity (Sethre-Hofstad et al., 2002; van Bakel & Riksen-Walraven, 2008) and harsh parenting styles (e.g., Hibel et al., 2009). Although these findings

* Corresponding author at: Lea Rose Dougherty, Department of Psychology, University of Maryland, College Park, MD 20742, USA.
E-mail address: ldougher@umd.edu (L.R. Dougherty).

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support the link between parent-child adrenocortical concordance and behavior, the findings are mixed as both positive and negative parenting behaviors have been linked to greater adrenocortical concordance. It is probable that concordance is heterogeneous, and research is needed to identify specific characteristics within the parent-child relationship that impact adrenocortical concordance.

One critical aspect of adrenocortical functioning that has received little scientific investigation in the study of concordance is the cortisol awakening response (CAR) (Hibel et al., 2014; LeMoult et al., 2015; Williams et al., 2013). The CAR captures the morning rise in cortisol across the waking period. The CAR is a unique aspect of the HPA axis, such that the awakening response is superimposed on the diurnal response. It is moderately heritable, but also sensitive to environmental factors including day-to-day demands (Fries, Dettenborn, & Kirschbaum, 2009). Abnormalities in the CAR and morning cortisol levels have been linked to a number of adverse physical and mental health outcomes (Chida & Steptoe, 2009). Both high and low CAR and morning cortisol levels have been linked to life stress, depression, and other stress-related disorders (Adam et al., 2010; Bhagwagar, Hafizi, & Cowen, 2003; Chida & Steptoe, 2009; Stetler & Miller, 2005), highlighting the complexity of the CAR and morning cortisol levels as well as the conflicting evidence in the literature. In recent work, parent CAR and child CAR, quantified as slope of change scores between cortisol levels at waking and 30 min post-waking, have been found to be positively correlated in preschool-aged (2–5 years; Hibel et al., 2014) and school-aged children (7–15 years; LeMoult et al., 2015; Williams et al., 2013) and their parents, with small to large effect sizes. These studies provide preliminary evidence that parent-child concordance of the CAR emerges early in development and is present across childhood. However, no study, to our knowledge, has examined parent-child adrenocortical concordance of average and person-centered cortisol levels across the waking period. Concordance of average cortisol reflects similarity in total cortisol levels across morning samplings, whereas concordance of person-centered cortisol levels reflects similar fluctuations in cortisol across the morning. Examining specific aspects of the morning cortisol response may shed light on the inconsistencies in the literature on adrenocortical concordance and whether parents and children demonstrate concordance of specific cortisol indices across the morning.

Moreover, a critical next step is to investigate individual differences in the adrenocortical parent-child concordance of morning cortisol levels in order to elucidate how this biological process may serve as a mechanism of risk for adverse child outcomes. Thus, the goal of the current study was to examine adrenocortical concordance of parent and child average and person-centered cortisol levels across waking in a sample of 136 parents and their preschool-aged children and to investigate how parent and child risk factors for depression moderate this association. Consistent with previous evidence (e.g., Feldman, 2012a, 2012b; Hibel et al., 2009; Papp et al., 2009; Sethre-Hofstad et al., 2002; Woody, Feurer, Sosoo, Hastings, & Gibb, 2016), the strength of physiological concordance is likely related to parent and child factors within the dyad. For the current study, we chose to explore the role of two well-established parent- and child-level risk factors for depression as moderators of parent-child adrenocortical concordance: parental history of depression and child temperament. We focused specifically on risk factors for depression given that abnormalities in HPA-axis functioning, particularly morning cortisol levels, are hypothesized to play a critical role in the pathophysiology of depression and may be one mechanism involved in the intergenerational transmission of risk for depression (Heim, Ehlert, & Hellhammer, 2000). Moreover, both parental depression (Feldman et al., 2012) and child temperament (Feldman, 2003) have been found to disrupt behavioral synchrony and these disruptions may extend to the level of physiological concordance as well. Below, we detail the evidence supporting the possible moderating role of these risk markers for depression on parent-child adrenocortical concordance.

First, parental depression has widespread effects on the parent and child, including disruptions in parenting and adverse child outcomes, including increased risk for psychopathology, suicidality, physical illnesses, earlier mortality, and psychosocial impairment (Hammen, 2009; Weissman et al., 2006). Moreover, abnormalities in the CAR and morning cortisol levels have been observed in currently depressed patients, recovered patients, and the high-risk offspring of depressed parents (Bhagwagar & Cowen, 2008; Vreeburg et al., 2010), and the CAR has also been found to be a unique predictor of depression onset in adolescence (Adam et al., 2010). These findings suggest that the CAR and morning cortisol levels may play a role in the pathophysiology of depression. Previous findings suggest that characteristics associated with parental depression, such as negative parenting behaviors and parent negative affect, are associated with greater concordance between parent and child diurnal cortisol levels (Papp et al., 2009) and cortisol reactivity (Hibel et al., 2009). In contrast, LeMoult et al. (2015) did not find evidence that maternal depression moderated parent-child concordance of the slope between cortisol levels at waking and 30 min post-waking in a sample of 112 mothers and their daughters ages 9–15 years-old; nevertheless, this study focused exclusively on older female offspring and their mothers and the examination of parent-child concordance of the change in cortisol from waking to 30 min post-waking. Examining the role of parental depression on parent-child concordance in younger children and in both sexes is critical because parental depression has a more profound effect on the parent-child relationship during early childhood (Goodman et al., 2011), a time when young children rely heavily on their parents for most of their basic physical and social needs, and as sex differences in adrenocortical functioning have been observed (Wüst, Federenko et al., 2000). Moreover, given the importance of the cortisol response across waking, examining concordance of parent-child average cortisol levels across waking and fluctuations in cortisol levels across waking (i.e., person-centered cortisol levels) informs us whether specific indices of concordance may be differentially related to parent and child risk factors for depression. Further examination of the moderating role of parental depression on adrenocortical concordance may also shed light on the origins of neuroendocrine dysfunction observed in high-risk offspring and the transmission of risk from depressed parents to their offspring.

Second, child temperamental emotionality has been found to have a profound impact on social relationships with parents (e.g., Belsky, 1984; Coplan, Bowker, & Cooper, 2003; Deater-Deckard, 2004), peers (e.g., Coplan & Bullock, 2012; Gunnar, Sebanc, Tout, Donzella, & van Dulmen, 2003), and teachers (e.g., Rudasill & Rimm-Kaufman, 2009) from an early age. Temperament refers to behavioral and emotional reactivity and regulatory processes that are relatively stable and are rooted in early developing biological systems (Rothbart, 2007; Shiner et al., 2012). Two higher-order domains in major models of child temperament are negative emotionality (NE) and positive emotionality (PE), which have been identified as important risk factors for depression (Shankman & Klein, 2003). NE is characterized by the tendency to experience negative emotions such as anger, sadness, anxiety, and irritability, whereas PE is characterized by positive affect such as joy, excitement, interest, and sociability; empirical models of child temperament document temperamental child NE and PE as distinct and orthogonal constructs (e,g, Measelle, John, Ablow, Cowan, & Cowan, 2005; Rothbart, Ahadi, Hershey, & Fisher, 2001). High levels of temperamental NE in children are related to increased difficulties in parent-child and peer relationships, harsher parenting styles, and increased risk for psychopathology, including depression (Bates, Schermerhorn & Petersen, 2012; Coplan & Bullock, 2012; Martorell & Bugental, 2006; Rothbart et al., 1994; Tackett, 2006). Conversely,