Psychoneuroendocrinology (2013) 38, 2943—2951

Available online at www.sciencedirect.com

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Maternal sensitivity and infant and mother adrenocortical function across challenges

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Received 19 May 2012; received in revised form 25 June 2013; accepted 2 August 2013

KEYWORDS
Cortisol;
Maternal sensitivity;
Dyadic attunement;
Mother;
Infant;
Strange situation

Summary Findings regarding associations between maternal sensitivity and infant and mother adrenocortical function have been inconsistent. Nor have studies addressed the issue of intra-individual, between-challenge cortisol variability in the context of maternal sensitivity. In this study, we combine several design features aimed at sensitizing analyses to these issues. Cortisol secretion of 297 infants and their mothers was assessed in response to different challenges at 16 and 17 months. Extensive, structured observations of maternal sensitivity were conducted at infant age 16 months. Data were analyzed with multilevel modeling using an actor—partner interdependence model. We found that maternal sensitivity was related to infant, but not maternal, cortisol levels and also to infant—mother cortisol attunement. Infants of more sensitive mothers, as compared to infants of less sensitive mothers, showed greater cortisol variability across challenges, with relatively steep cortisol decreases and increases, depending on challenge. Mother and infant cortisol levels were highly correlated and this attunement was higher among dyads with more sensitive mothers than among dyads with less sensitive mothers. The results show nuanced attunement in a low-risk sample, with the infants of higher sensitivity mothers showing greater intra-individual variability across challenges. High cortisol response variability across challenges may simultaneously permit adaptation to threat and protect the infant from overexposure to corticosteroids.

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0306-4530/$ — see front matter
http://dx.doi.org/10.1016/j.psyneuen.2013.08.001

1. Introduction

The impact of normal variation in home environments, particularly with respect to maternal sensitivity toward the child, is emerging as a vital aspect of early hypothalamic–pituitary–adrenal (HPA) function. However, findings are ambiguous and questions remain unaddressed. This study evaluates the associations between maternal sensitivity and: infant cortisol secretion; maternal cortisol secretion; mother–infant cortisol attunement; and intra-individual variability in infant cortisol secretion across challenges (between-challenge variability). To enhance power and reliability of findings, the current study combines several methodological features, including large sample size, repeated and varied infant challenges, robust assessment of maternal sensitivity, and a powerful data analytic strategy.

Maternal sensitivity involves behaviors that are contingent and appropriate to infant signals. The construct is central to child psychology, particularly it predicts quality of infant–caregiver attachment (Ainsworth et al., 1978) and has been studied in the context of infant HPA function (Spangler et al., 1994; Nachmias et al., 1996; Blair et al., 2005, 2008; Albers et al., 2008). However, results are not entirely consistent. For example, some studies show that maternal sensitivity is related to infant baseline cortisol (Blair et al., 2005, 2008), reactivity (change in cortisol concentrations from baseline to a post-stressor value roughly timed to capture peak response) (Spangler et al., 1994; Blair et al., 2005, 2008), and recovery (return of cortisol levels toward baseline) (Blair et al., 2005, 2008; Albers et al., 2008); other studies show no such associations (Haley and Stansbury, 2003; Thompson and Trevathan, 2009; Jansen et al., 2010) or only some of them (Albers et al., 2008). With respect to cortisol reactivity, in particular, maternal sensitivity has been associated with decreased infant reactivity (i.e., infants of higher-sensitivity mothers show lower increases in cortisol concentrations than infants of lower-sensitivity mothers) (Blair et al., 2008; Feldman et al., 2010), increased infant reactivity (Blair et al., 2005, 2008; van Bakel and Riksen-Walraven, 2008), and no significant change in infant cortisol concentrations (Blair et al., 2008). Blair et al. (2005, 2008) showed almost all aforementioned variants in a single longitudinal study, suggesting the need for careful examination of important moderators.

Furthermore, while several studies address the link between maternal sensitivity and infant HPA function, few address the relation between maternal sensitivity and maternal cortisol. Feldman et al. (2010) found that mothers who provided stimulatory touch while their infants signaled disengagement secreted more cortisol at baseline, in reactivity, and at recovery than mothers who provided less missynchronous touch. However, van Bakel and Riksen-Walraven (2008) found no such association. Further research is needed.

Related research involves attunement (Field, 1994), defined in the psychological literature as behavioral coordination between mother and infant (Feldman, 2007). Research has also addressed adrenocortical attunement, the synchrony of HPA response across mother and infant dyad partners. Adrenocortical attunement is sometimes defined as involving causal/lagged associations (Laurent et al., 2011, 2012; Middlemiss et al., 2011) and sometimes in terms of non-causal covariation. Different studies define/assess attunement using varied cortisol indices (e.g., baseline, reactivity, response trajectory). Some investigators report significant dyadic attunement (Middlemiss et al., 2011; Neu et al., 2009; Bright et al., 2012; Ruttle et al., 2011; Laurent et al., 2012), others do not (Sethre-Hofstad et al., 2002; van Bakel and Riksen-Walraven, 2008; Poggi Davis and Granger, 2009). Hibel et al. (2009) found that maternal and infant baseline cortisol concentrations were significantly correlated but not their slopes. Furthermore, some studies show significantly higher adrenocortical attunement in dyads with more sensitive mothers, as compared to dyads with less sensitive mothers (Sethre-Hofstad et al., 2002; van Bakel and Riksen-Walraven, 2008), but this finding is not entirely consistent (Hibel et al., 2009; Ruttle et al., 2011).

To date, the literature neglects the issue of intra-individual, between-challenge variability (i.e., the individual's differential cortisol secretion in response to differentially challenging circumstances) as this relates to maternal sensitivity. We cannot ethically stress infants beyond what they might experience in a typical day (Gunnar et al., 2009). Nevertheless, there are differences in the degree to which standard infant challenges promote adrenocortical activity (Gunnar et al., 2009; Jansen et al., 2010; Laurent et al., 2012). This variability is important to the challenges used in the present study, specifically the Toy Frustration Procedure (TFP; Braungart-Rieker and Stifter, 1996) and Strange Situation Paradigm (SSP; Ainsworth et al., 1978). Meta-analytic data (Jansen et al., 2010) indicate that challenges inducing anger precipitate cortisol increases corresponding to a *d* (standardized difference between pre-stressor and post-stressor cortisol concentrations) of .13; by contrast, the SSP induces cortisol reactivity corresponding to $d = .34$. While the number of studies in this meta-analysis undermines formal comparison of effect sizes, the discrepancy does suggest that the SSP may be the more potent stressor. Furthermore, Laurent et al. (2012) found that cortisol trajectories were higher in both mothers and infants during the SSP (which Laurent et al. considered a "threat"), compared to a clean-up task (which Laurent et al. considered a "challenge") and that dyadic cortisol attunement was stronger during the SSP than during the clean-up task.

Such findings indicate that the differential impact of two challenges may provide a means of assessing *between-challenge variability* in infant HPA function. Healthy function involves flexible response, i.e., a robust cortisol increase in response to acute stressors and lower increase, or no increase at all, under more quotidian circumstances. In contrast, one might expect muted increases and decreases where the HPA axis is less elastic, as under conditions of chronic stress (Miller et al., 2007).

We advance several hypotheses. (1) Maternal sensitivity is positively related to infant cortisol intercept (baseline) and trajectory. (2) Mothers and infants show attunement across intercept and trajectory. (3) This attunement is stronger among more sensitive mothers and their infants than among less sensitive mothers and their infants. (4) Compared to infants with less sensitive mothers, infants with more sensitive mothers show greater variability in HPA response across challenges, i.e., *infants with more highly sensitive mothers show greater cortisol increases (as manifested in their trajectories) to the SSP than do children of less sensitive*