Maternal sensitivity and infant and mother adrenocortical function across challenges

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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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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 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
mothers and more modest increases in the context of the TFP. (That being said, we must also recognize that infant laboratory stressors only unreliably cause cortisol increases and in many cases cortisol levels decrease from baseline (Jansen et al., 2010), with high baseline levels attributed to “arrival effects” or anticipatory anxiety (Ruttle et al., 2011)).

2. Methods and materials

2.1. Participants

Following approval from Research Ethics Boards at the Centre for Addiction and Mental Health and Ryerson University, we recruited families via postings in community centers and in-person visits to mother—infant activity centers and consumer baby shows across a large urban and suburban area. Infants were full term and healthy. Mothers were 18 years or older at childbirth, with no known hormonal or psychiatric disorder and with sufficient English to complete questionnaires. Infant—mother dyads were assessed at mean infant ages 15.94 (SD = 1.18) and 16.95 (SD = 1.71) months. The sample includes 297 mother—infant dyads. Table 1 shows sample demographics. The sample is low risk across maternal age, education, marital status, and family size.

With respect to missingness, between 11.1% (16-month baseline) and 23.23% (17 month baseline) datapoints were missing for infants due to some reluctance to engage in saliva sampling. Mothers were missing between 4.40% and 17.51% of any given data point; the latter figure involves the +40 SSP sample and is primarily due to early termination of some lab visits due to infant fatigue. Missing data were not related to any sociodemographic factors. Growth models for the cortisol concentrations were fitted using maximum likelihood estimation, which accounts for missing data (described below).

2.2. Procedures

Two research assistants visited the dyads’ homes, observed mothers and infants as they interacted, and coded maternal sensitivity over a two-hour period. The research assistants also observed the infants in a standardized, 6-min toy frustration procedure, and collected saliva samples during that same visit. One month later, the dyads visited the lab, where researchers observed the infants in a maternal separation procedure (SSP) and collected saliva samples. Maternal sensitivity was not coded during this second visit due to our emphasis on ecologically valid observation in the home and because we wished to minimize participant time in the lab. All appointments started between 0900 h and 1000 h. Morning visits are typical practice with infants. It has been argued that during infancy, effects of daytime stressors, meals, and naps are highly variable from child to child and from day to day. Morning collection is advised so that perturbations of routine do not confound cortisol levels (Gunnar and White, 2001; Goldberg et al., 2003; van Bakel and Riksen-Walraven, 2004). However, afternoon visits are preferable for adults because morning cortisol levels are relatively high, making it difficult to detect reactivity (Dickerson and Kemeny, 2004). The greater sensitivity to infant compared to maternal cortisol reactivity was a necessary trade-off in study design.

2.3. Challenges

At 16 months, infants participated in a TFP (Braungart-Rieker and Stifter, 1996), consisting of 4 90-second episodes, wherein mother alternately engaged baby with an attractive toy provided by experimenter and deprived baby of the toy by placing it in a transparent container. If the infant cried continuously for 20 s, the episode was terminated.

At 17 months, we observed infant and mother in the SSP (Ainsworth et al., 1978), consisting of 8 three-min episodes wherein the toddler is with mother, with mother and female stranger, with stranger, and alone in an unfamiliar but child-friendly room. The procedure involves two separations from mother, terminated if the child cries hard for 20 s. Mothers observed their toddlers throughout. The SSP was used only as a challenge and was not coded for attachment classification. On completion of the SSP, the dyad was shown to a second room, not associated with the separations, where post-challenge cortisol samples were collected.

2.4. Saliva collection/assay

Mothers were asked, with reference to both themselves and their infants, to refrain from brushing their teeth, eating, and drinking 60 min before procedures to avoid contamination (Kirschbaum and Hellhammer, 1994). Given the timing of the visits, it is not surprising that many mothers did not comply with this request; according to maternal report, 79 mothers and 78 infants ate/drank within an hour of the TFP, and 34 mothers and 42 infants ate/drank within an hour of

Table 1 Sample characteristics.

<table>
<thead>
<tr>
<th>Variable</th>
<th>147 (53%) female</th>
</tr>
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<tbody>
<tr>
<td>Infant sex</td>
<td>147 (53%) female</td>
</tr>
<tr>
<td>Maternal age at child’s birth</td>
<td>31.54 (4.35; 20.00–45.08)</td>
</tr>
<tr>
<td>Marital status</td>
<td>82.8%</td>
</tr>
<tr>
<td>Married</td>
<td>82.8%</td>
</tr>
<tr>
<td>Common-law</td>
<td>11.4%</td>
</tr>
<tr>
<td>Single/separated</td>
<td>5.5%</td>
</tr>
<tr>
<td>Highest level of maternal education</td>
<td>47.4%</td>
</tr>
<tr>
<td>Primary school</td>
<td>1.0%</td>
</tr>
<tr>
<td>Secondary school</td>
<td>22.7%</td>
</tr>
<tr>
<td>Community college</td>
<td>7.8%</td>
</tr>
<tr>
<td>Undergraduate university</td>
<td>21.1%</td>
</tr>
<tr>
<td>Post-graduate university</td>
<td>47.4%</td>
</tr>
<tr>
<td>Maternal behavior</td>
<td>0.49 (0.33; –.69 to .90)</td>
</tr>
<tr>
<td>Q-set M (SD; range)</td>
<td>0.49 (0.33; –.69 to .90)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Number of siblings at Visit 1</th>
<th>76.1%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>17.1%</td>
</tr>
<tr>
<td>2</td>
<td>5.6%</td>
</tr>
<tr>
<td>&gt;3</td>
<td>1.2%</td>
</tr>
</tbody>
</table>
the SSP. Saliva was collected 5 min pre-challenge (baseline) and 20 and 40 min after each challenge was completed; the challenges were designed to be increasingly stressful and the cortisol samples were expected to encompass the effects of this escalating stress, rather than the stress of a single episode (see Bernard and Dozier, 2010). Saliva was collected with Sorbettes (Salimetrics, State College PA) placed in the mouth for 60 s. Once saturated, Sorbettes were deposited in a 2-mL cryovial, sealed, and stored at −70 °C.

For assay, salivettes were thawed and centrifuged for 10 min at 3000 rpm at 4 °C. Samples were assayed using a high sensitivity enzyme immunoassay kit (Salimetrics, State College, PA). Samples from each dyad across both visits were assayed in the same batch. All samples were assayed in duplicate and averaged. The interassay variability was 10.6%; the intra-assay variation was 8.3%, for samples with low values, 6.9% for samples with high values.

2.5. Maternal sensitivity

A strength of this literature involves observational assessments of maternal sensitivity. Typically, these observations are based on instrumentation derived from attachment research (Ainsworth et al., 1978) and consist of Likert scales with anchored descriptions of key sensitivity domains. Meta-analytic review shows that such measures are consistent predictors of infant attachment security (r = .21; Atkinson et al., 2000), even where observations are brief, as in the literature reviewed above (range = 7–20 min, median = 10 min).

However, Pederson et al. (1990) amalgamated and systematized such sensitivity observations in the Maternal Behavior Q-sort (MBQS). For the MBQS, observers sort 90 cards, each describing a particular maternal behavior, into 9 piles of 10, with piles denoting “most like” to “least like” mother. A single score is derived from the MBQS, ranging from −1.00 to +1.00, depending on how the sort for a particular mother correlates with a prototypically ideal sort (the lower the score, the less sensitive the mother; Pederson et al., 1990). MBQS observations are associated with infant attachment security at r = .48, significantly larger than the aforementioned effect size linking traditional observations to infant attachment security (r = .21; Atkinson et al., 2000). The MBQS has not been used in the cortisol literature to date but its power may render it an important addition. In the current study, two female observers, blind to other measures, made a two-hour visit to the home for MBQS (Version 3.1) observation. They attained inter-observer agreement (intraclass correlation) of .88, p < .0005. We used the mean score across both observers in the following analyses.

2.6. Statistical analysis

The data were analyzed using multilevel modeling (MLM; Kashy and Donnellan, 2008) with restricted maximum likelihood to estimate dyadic growth models in which participant role (infant versus mother) and maternal sensitivity score predicted changes in cortisol over time. MLM is optimal for cortisol analysis because it permits comparison of mother and infant cortisol concentrations and rates of change (trajectories or slopes). Trajectories are important to assessing attunement across time within challenge. Indeed, some investigators incorporate synchronous trajectories into their definition of attunement (Hibel et al., 2009; Laurent et al., 2011, 2012). Thus, Laurent et al. (2012) argued that “stress trajectories may be characterized as synchronous when they unfold in a parallel fashion (but not necessarily at the same level) over time or asynchronous when they unfold either independently of one another or inversely (one rising as the other falls)” (p. 36). This contrasts with operationalizing attunement as the correlation between single point estimates. Additional advantages of MLM are that it does not require complete data for every participant, nor that observations are evenly spaced in time (Hruschka et al., 2005; Hibel et al., 2009). The MLM analysis used measurements of both participants’ natural logarithm of cortisol across three samples (0, +20, +40) from two visits (TFP, SSP). These data formed a multilevel structure in which infants and mothers were nested within families but visit and sampling occasion were crossed with families (i.e., infants and mothers both participated in each visit and were each sampled on the same three occasions).

To account for statistical interdependence within dyads, data analysis was informed by the actor–partner interdependence model (APIM; Kashy and Kenny, 2000). Bright et al. (2012) successfully adopted the APIM in assessing cortisol attunement in mother–infant dyads. The APIM assumes that when individuals are involved in an interdependent relationship, their outcome depends not only on their own characteristics and inputs (actor effect) but their partners’ as well (partner effect). In these analyses, role was effects (dummy) coded such that mother = 1 and infant = −1. Visit was also effects coded such that TFP = 1 and SSP = −1, and maternal sensitivity score was grand mean centered.

Growth curve models estimated the baseline levels of cortisol (intercepts) and the trajectories of cortisol levels across the samples over the two visits. After controlling for wake time, moderated growth models of cortisol were estimated with linear effect of sample. The model included fixed effects for role (infant versus mother), visit (TFP and SSP), the linear term for sample, and the hypothesized moderator (maternal sensitivity). This resulted in a possible four-way interaction between role, visit, sample, and maternal sensitivity. Although dimensional sensitivity data were entered into the model, significant interactions were graphed and interpreted (Fig. 1) using 1 SD above and below the grand mean as high and low values for continuous predictors (Aiken and West, 1991).

In addition to the fixed effects, the random effects part of the model included random intercepts, slopes for sample and residual variances for mothers and their infants. Finally, covariances between mother and infant intercepts and slopes were estimated (to assess attunement) following procedures outlined by Kenny et al. (2006); this involves dividing the covariance between mothers’ and infants’ (e.g.) slopes and dividing it by the square root of the product of maternal variance and infant variance), as was the covariance between the two residuals (to assess whether factors beyond those studied here influenced degree of attunement).
3. Results

3.1. Preliminary analysis

Demographics. Neither infant nor mother cortisol concentrations (at 0, +20, +40) were significantly related to child sex, maternal education, maternal smoking status, family income, or date of last menstruation (assessed for luteal stage).

Wake and feeding times. At TFP, child wake time correlated with child baseline ($\rho = .24, p < .001$) and +20 ($\rho = .16, p < .05$) concentrations. Child feeding times did not correlate significantly with cortisol concentrations. Maternal wake time and time of last meal/drink did not correlate with maternal cortisol concentrations. At SSP, neither babies’ nor mothers’ wake or feeding times correlated with cortisol indices. For the purposes of our main analyses, we covaried child wake time at TFP (wake times for the two procedures correlated at $r = .47, p < .0005$).

3.2. Main analyses

The findings are depicted in Fig. 1. Table 2 presents means and standard deviations for cortisol values and MBQs ratings. Table 3 presents regression coefficients and standard errors for the fixed parameters from the multilevel dyadic growth curve model with log cortisol as the dependent variable.

There was a significant main effect for role; mothers’ cortisol levels were higher than infants’ (mother: $b = .81, SE = .01$; infant: $b = .66, SE = .02$). There was also a significant main effect for sample (indicating that sample concentrations vary over time — baseline, +20, +40). There were two significant two-way interactions involving role: one with challenge (indicating that the difference between infant and mother cortisol levels depends on the challenge; see Fig. 1), the other with sample (indicating that sample values at any given point (baseline, +20, +40) depend on whether the sample is maternal or infant); as well as a significant two-way interaction between challenge and sample (sample concentrations at any given time point depend on the challenge). There was a significant three-way interaction involving role, challenge, and sample and a three-way interaction approached significance for challenge, sample and maternal sensitivity. However, all of these effects occurred within a marginally significant four-way interaction with role, challenge, sample, and maternal sensitivity ($b = -.003, t(665) = 1.90, p = .058$).

Above, we forwarded hypotheses regarding the relations between maternal sensitivity and (a) infant cortisol levels, (b) mother cortisol levels, and (c) between-challenge variability among infants; this interaction shows the complexity of these issues and may go some way to explaining the inconsistency of results in the literature. All other interactions were nonsignificant (Table 3).

To break down the four-way interaction, we computed the three-way interaction between challenge, sample, and maternal sensitivity separately for mothers and infants. For mothers, the three-way interaction was not statistically significant ($b = .0004, t(749) = .183$); regardless of their sensitivity, mothers show a comparable decrease in cortisol across both visits. For infants, the interaction was significant ($b = .007, t(645) = 2.28, p < .01$). For infants, the interaction between challenge and sample differed between high and low levels of maternal sensitivity. We estimated the effect for sample by challenge interaction separately for high and low maternal sensitivity. For the TFP, infants of both low and high sensitive mothers exhibit a decrease in cortisol across samples, although this decrease is more pronounced for infants of high sensitive mothers than for infants of low sensitive mothers (low sensitive: $b = -.004, t(286) = 2.71, p < .01$;
Table 3  Fixed effects estimates from dyadic growth curve model of cortisol levels across time.

<table>
<thead>
<tr>
<th></th>
<th>b (SE)</th>
<th>t</th>
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<tbody>
<tr>
<td>Intercept</td>
<td>0.732 (0.074)</td>
<td>52.56***</td>
</tr>
<tr>
<td>Role</td>
<td>0.074(0.009)</td>
<td>8.67***</td>
</tr>
<tr>
<td>Challengea</td>
<td>0.018(0.011)</td>
<td>1.56</td>
</tr>
<tr>
<td>Sampleb</td>
<td>-0.005 (0.0007)</td>
<td>-7.40***</td>
</tr>
<tr>
<td>Maternal sensitivity</td>
<td>0.047 (0.042)</td>
<td>1.12</td>
</tr>
<tr>
<td>Role by challenge</td>
<td>0.021 (0.007)</td>
<td>3.03**</td>
</tr>
<tr>
<td>Role by sample</td>
<td>-0.004 (0.0006)</td>
<td>-6.49***</td>
</tr>
<tr>
<td>Challenge by sample</td>
<td>0.002 (0.007)</td>
<td>3.84**</td>
</tr>
<tr>
<td>Role by challenge by sample</td>
<td>-0.003 (0.0006)</td>
<td>-5.23***</td>
</tr>
<tr>
<td>Challenge by maternal sensitivity</td>
<td>0.004 (0.002)</td>
<td>1.816</td>
</tr>
<tr>
<td>Role by challenge by sample by maternal sensitivity</td>
<td>-0.003 (0.002)</td>
<td>-1.09</td>
</tr>
</tbody>
</table>

Note. The analysis used the natural logarithm of cortisol. For role, 1 = mother, and -1 = infant. SE: standard error. 
a Visit (toy frustration and strange situation). 
b Measured in 20-min increments. 
* p = .058. 
** p < .01. 
*** p < .001

high sensitive: b = -.009, t(283) = 5.56, p < .001). By contrast, in the SSP, infants of highly sensitive mothers show an increase in cortisol (b = .007, t(280) = 3.21, p < .002); infants of low sensitive mothers show no such increase across samples (b = .002, t(290) = 1.03, ns). To reiterate, these results show that infant cortisol levels depend on both challenge and maternal sensitivity, with infants of more highly sensitive mothers showing greater decreases in response to the frustration challenge than infants of less sensitive mothers and greater increases in the separation paradigm, i.e., infants of more sensitive mothers show greater between-challenge cortisol variability than infants of less sensitive mothers.

There were also several significant random effects for the model. First, there was significant variability in the intercepts for mothers (variance = 0.05, Wald Z = 10.29, p < .0001; the Wald test is essentially a z test, with degrees of freedom calculated in the context of multilevel regression) and infants (variance = 0.07, Wald Z = 9.60, p < .0001). This indicates that there were individual differences in mothers’ and infants’ baseline cortisol levels. Second, there was significant variability in the slopes for mothers (variance = 0.029, Wald Z = 10.02, p < .0001) and infants (variance = 0.04, Wald Z = 9.365, p < .0001). This indicates the existence of individual differences in the trajectories of cortisol for each member of the dyad. Third, the intercepts for mothers and infants were significantly correlated (rho = .53, Wald Z = 6.09, p < .0001). The slopes for mothers and infants were also significantly correlated (rho = .60, Wald Z = 7.27, p < .0001). These results indicate mother–infant attunement in cortisol secretion. Finally, there was a significant positive correlation of the residuals (rho = .18, Wald Z = 4.52, p < .001). This correlation suggests that after controlling for average cortisol levels and the change in cortisol over challenges, samples, and maternal sensitivity, there is still considerable similarity in cortisol levels between mothers and infants.

To assess for differences in degree of attunement between dyads with more sensitive mothering and dyads with less sensitive mothering, we split the sample at the sensitivity median and re-ran the analyses. Attunement was significant for baseline cortisol levels (low sensitive dyads: rho = .46, Wald Z = 4.31, p < .0001; high sensitive dyads: rho = .59, Wald Z = 5.30, p < .0001). The difference between these coefficients, however, was nonsignificant (z = 1.58, p = .11). Attunement was also significant for adrenocortical slopes (low sensitive dyads: rho = .42, Wald Z = 3.58, p < .0001); high sensitive dyads: (rho = .69, Wald Z = 5.96, p < .0001). The difference between these coefficients was significant (z = 3.51, p < .0004), indicating that for this index, high sensitivity dyads show greater adrenocortical attunement than low sensitivity dyads.

4. Discussion

In this study, we addressed relations between maternal sensitivity and: infant cortisol secretion; maternal cortisol secretion; and mother–infant cortisol attunement. Previous findings regarding these relations have been inconsistent. In addition, we addressed the issue of intra-individual, between-challenge variability in the context of maternal sensitivity. This issue has not been studied before but it is important because flexible reactivity is a central indicator of healthy adrenocortical function. We addressed these issues with a combination of features designed to minimize confounding and maximize statistical power; these features include large sample size, repeated and differentially stressful challenges, robust assessment of maternal behavior, and powerful statistical approach.

The analyses revealed a four-way interaction incorporating role (mother versus infant) × sample (baseline, +20, +40) × challenge (TFP, SSP) × maternal sensitivity. Mothers had higher overall cortisol concentrations than infants. However, this was not true for every sample and even in those samples where it was true, the difference between maternal and infant cortisol concentrations varied. Thus, in the TFP, mother and infant cortisol concentrations moved toward convergence over time as both partners downregulated. In the SSP, maternal baseline was higher than infant baseline, but by +40 this situation reversed because mothers downregulated while infants did not. Finally, all these factors (role, challenge, and sample) interacted with maternal sensitivity. In particular, the infants of highly sensitive mothers showed a strong cortisol decrease in response to the TFP and a strong increase in response to the SSP. By contrast, infants of less sensitive mothers showed relatively weak decreases and increases, respectively. This four-way interaction goes some way to explaining inconsistencies in the existing literature. No studies of maternal sensitivity and infant cortisol simultaneously account for all factors represented here, although all are apparently important to understanding infant HPA function.

This interaction must be understood in the context of the high baseline findings. Maternal cortisol concentrations decreased in the context of the TFP and SSP and infant levels decreased in the context of the TFP (Fig. 1). Ruttle et al.
Indeed, responses but also differentiation between mother and infant intercepts suggests that the high baseline concentrations among infants may reflect attunement with mothers.

Hitherto, frustration and separation paradigms have not been administered in a single sample in the context of maternal sensitivity, precluding within-subject, between-challenge comparisons. The current findings indicate that maternal sensitivity is related to infant cortisol reactivity, but this relation depends on challenge. The significant interaction shows that infants in high sensitivity dyads show greater intra-individual, between-challenge variability across the SSP and TFP than do infants in low sensitivity dyads. Infants with more sensitive mothers evince robust elevations in response to the SSP and robust declines in response to the TFP. In contrast, infants of less sensitive mothers show negligible increases in the context of the SSP and blunted decreases in the context of the TFP.

This intra-individual, between-challenge variability is a key finding when one considers the purpose of stress neurobiology — to maintain viability through change via short-term adaptive actions (allostasis); “turning on and turning off responses efficiently is vital” (McEwen and Seeman, 1999, p. 33). A strong HPA response permits vigorous activation of neurobiological systems under conditions of acute challenge. However, chronically high cortisol secretion is neurobiologically damaging, so its marked decrease as the challenge dissipates is also crucial (Gunnar and Quevedo, 2007). Indeed, both negative parenting and atypical cortisol secretion have been linked to psychopathology (Gunnar and Quevedo, 2007). In this regard, it is possible that flexible HPA function mediates, at least partially, the relation between parenting and psychopathology.

Not only were infants of more and less sensitive mothers differentially responsive across challenges, sensitive mother–infant dyads also showed greater cortisol attunement than did less sensitive mother–infant dyads. Overall, maternal and infant baselines and slopes correlated at rho = .53 and .60, respectively. However, mother–infant baseline cortisol levels correlated .59 and .46 for high sensitivity and low sensitivity dyads, respectively, and slopes correlated .69 and .42. The difference between the slope coefficients was significant, indicating that for this index, high sensitivity dyads share greater adrenocortical attunement than low sensitivity dyads. The finding that baseline cortisol levels were not significantly different when high sensitivity and low sensitivity dyads were compared, but that the trajectories were, may lend support to Laurent et al.’s (2011, 2012) contention that trajectory is a particularly sensitive measure of attunement.

Prior studies have been inconsistent with respect to mother–child cortisol attunement, with correlations between mother and child cortisol concentrations typically varying between .01 (Hibel et al., 2009) and .31 (Sethre-Hofstad et al., 2002; van Bakel and Riksen-Walraven, 2008). More recently, however, Middlemiss et al. (2011) studied dyads at a hospital-based infant sleep program and found correlations of .75 and .78 at initiation of infant bedtime routine and following the infants’ transition to sleep, respectively. Although sample sizes were small (N = 16 and 12, respectively), the present findings also suggest that dyadic cortisol attunement may be stronger than earlier estimates suggest.

The present study also addressed the link between maternal sensitivity and maternal cortisol secretion, important because previous investigations show conflicting findings (c.f., van Bakel and Riksen-Walraven, 2008; Feldman et al., 2010). We found no significant relation between maternal sensitivity and maternal cortisol concentrations. Keeping in mind the fact that the TFP and SSP challenges were designed for infants, not adults, one possible explanation for the discrepant findings across studies involves the timing of maternal sensitivity and infant distress measures. Neither we nor van Bakel and Riksen-Walraven (2008) assessed maternal sensitivity as part of the infant challenge. In contrast, Feldman et al. (2010) incorporated maternal sensitivity into the context of challenging the infant, charging the mother with minimizing the infant’s discomfort. This methodology may ingeniously challenge infant and mother simultaneously, fostering a direct link between maternal behavior and maternal HPA function. The issue requires further research.

It is also important to note that we found a significant, positive correlation between residuals, indicating that even after accounting for mean cortisol concentrations, change in cortisol over samples and across visits, and maternal sensitivity, there was still similarity in cortisol levels between mothers and infants. This indicates that maternal sensitivity does not account for all the variance in mother–infant attunement. One obvious additional factor may involve genetics. Human studies suggest a heritable component in cortisol reactivity (Kirschbaum et al., 1992), cortisol levels after awakening (Wüst et al., 2000), and circadian rhythm (Bartels et al., 2003). There is a need to study of gene–environment interactions as they influence infant HPA function.

There are limitations to this study. First, we interpreted all fixed effects in the context of a near-significant four-way interaction (p = .058). However, it appeared more misleading to disregard this finding because it missed traditional significance levels by .8% than to interpret lower-order effects without the qualifications set by all factors. This is particularly so given that higher-order interactions are difficult to demonstrate in field studies, inflating Type II error rates (McClelland & Judd, 1993). Second, we measured change in cortisol secretion over time against a backdrop of high morning cortisol levels and diurnal decrease. This may have undermined the detection of stressor impact (Dickerson and Kemeny, 2004). Third, HPA activity only partially characterizes the stress response; no definitive conclusions regarding the stress-inducing nature of toy frustration and strange situation procedures are possible until sympathetic nervous system activity is assessed (Laurent et al., 2011, 2012). Fourth, neither challenge was designed to stress mothers, so the relation between maternal sensitivity and maternal cortisol remains moot. Such limitations should be addressed in future.
To summarize, the present findings indicate that maternal sensitivity is linked to infant adrenocortical activity, but this link is dependent on challenge and sample. More sensitive mothers have infants who show greater between-challenge variability, with robust cortisol elevations in response to perceived threat and strong decreases where the challenge proves nonthreatening. Less sensitive mothers have infants who show lower between-challenge variability, with relatively shallow increases and decreases in response to challenge. The results also suggest that mother–infant adrenocortical activity is strongly attuned, albeit more so among high sensitivity dyads than low sensitivity dyads. Nevertheless, maternal sensitivity does not explain all the attunement in mother–infant adrenocortical activity. Overall, the results are congruent with the hypothesis that maternal behavior serves as an external organizer of biological regulation in infancy (Spangler et al., 1994). This attunement appears exquisitely dimensional and sculpted to even ordinary variations in normative care (Atkinson, 2012; Feldman, 2012; Hane and Philbrook, 2012).

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Conflict of interest

The authors have no conflict of interest respecting this study, financial, personal, or otherwise.

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