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Maternal Disrupted Communication During Face-to-Face Interaction at 4 months: Relation to Maternal and Infant Cortisol Among at-Risk Families

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Abstract

The study evaluated the association between maternal disrupted communication and the reactivity and regulation of the psychobiology of the stress response in infancy. Mothers and infants were recruited via the National Health Service from the 20% most economically impoverished data zones in a suburban region of Scotland. Mothers (*N* = 63; *M* age = 25.9) and their 4-month-old infants (35 boys, 28 girls) were videotaped interacting for 8 min, including a still-face procedure as a stress inducer and a 5-min coded recovery period. Saliva samples were collected from the dyads prior to, during, and after the still-face procedure and later assayed for cortisol. Level of disruption in maternal communication with the infant was coded from the 5-min videotaped interaction during the recovery period which followed the still-face procedure. Severely disrupted maternal communication was associated with lower levels of maternal cortisol and a greater divergence between mothers' and infants' cortisol levels. Results point to low maternal cortisol as a possible mechanism contributing to the mother's difficulty in sensitively attuning to her infant's cues, which in turn has implications for the infant's reactivity to and recovery from a mild stressor in early infancy.

Controlled animal models have established that caregiver responsiveness is one factor that externally regulates the psychobiology of the stress response in infants (Barr et al., 2004; Coplan et al., 1996; Francis, Diorio, Liu, & Meaney, 1999). The effects of caregiver responsiveness during this sensitive period in infancy have also been shown to be long

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lasting, affecting the set points of the activity and reactivity of the hypothalamic-pituitary-adrenal (HPA) axis into adulthood (Francis et al., 1999). The long-term effects demonstrated in these studies underscore the importance of understanding how social features of the early caregiving environment shape the developing stress response system among human infants. In this study, we assess quality of maternal communication with 4-month-old infants following a stressful still-face procedure as one potentially critical regulator of infant stress. We examine how maternal communication relates to maternal and infant cortisol reactivity, as well as to the covariation of mother and infant cortisol.

INFANT CORTISOL AND MATERNAL RESPONSIVE CAREGIVING

Several studies confirm that, compared with securely attached infants, infants with insecure or disorganized attachment relationships with their caregivers at 12–18 months have higher cortisol levels following exposure to a stressor such as the strange situation procedure (Gunnar & Donzella, 2002; Hertsgaard, Gunnar, Erickson, & Nachmias, 1995; Spangler & Grossman, 1993) or an approaching novel stimulus (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). Disorganized attachment behavior by the infant has also been repeatedly linked to maternal frightened, frightening, or atypical behavior in interaction with the infant (Madigan et al., 2006). Thus, we would expect that the sensitivity and responsiveness of the caregiver would also impact the psychobiology of the stress response in infants (Gunnar, 1992; Gunnar & Donzella, 2002). In support of this hypothesis, in a sample of 9-month-old infants, a sensitive and responsive substitute caregiver prevented elevations in cortisol during maternal separation (Gunnar, Larson, Hertsgaard, Harris, & Brodersen, 1992). Further, maternal sensitivity during a mild everyday stressor was associated with faster recovery in infants' cortisol levels following that stressor (Albers, Riksen-Walraven, Sweep, & Weerth, 2008; Blair et al., 2008). In contrast, when a caregiver does not recognize an infant's cues or when a caregiver responds to an infant's cues in a way that is inappropriate or noxious, we would expect less optimal infant physiological regulation. For example, Bugenthal, Martorell, and Barraza (2003) have shown that maternal withdrawal is associated with higher baseline cortisol in the child. However, developmental science is still far from characterizing with any precision how much disruption in maternal responsiveness is required before there are adverse effects on the reactivity and regulation of the infant's HPA axis (Blair, Raver, Granger, Mills-Koonce, & Hibel, 2011).

It is also important to note that under extreme conditions of neglectful or abusive care, some studies find that children show reduced rather than increased cortisol responses to stressors (for a review see Gunnar & Vazquez, 2001). For example, in a study of preschool children who had experienced abuse and neglect, maltreated children had lower cortisol reactivity to the level of conflict in the classroom compared with children not maltreated (Hart, Gunnar, & Cicchetti, 1995). In addition, girls who had experienced chronic and severe abuse in childhood displayed blunted physiological reactivity to social evaluative threat during the transition to adolescence (Gordis, Granger, Susman, & Trickett, 2006). Finally, lower *waking* cortisol levels, as well as flattened diurnal cortisol slopes, have been repeatedly associated with severe neglect in early childhood (Bruce, Fisher, Pears, & Levine, 2009; Dozier et al., 2006). Lower baseline cortisol levels have also been associated with exposure to combat among army veterans (see Weiner, 1992; for review). In short, both lower baseline values and reduced response to threat are thought to develop after extended periods of elevated cortisol levels associated with sustained stressors. These reduced baseline values, as well as blunting of cortisol reactivity, are thought to be adaptive responses to avoid the negative physiological effects associated with long-term exposure to high levels of glucocorticoids (e.g., cortisol). Thus, there is consensus across studies that the relationship between adverse experiences and physiological responses to stress may not be linear (Weiner, 1992); instead, lower cortisol responses to stress may result either from responsive or from exceptionally unresponsive caregiving. However, very little is known about the types or degree of disturbance in caregiving that would be associated with higher vs. lower cortisol responses to a stressor in the child.

One well-validated coding instrument for assessing frightened, frightening, or atypical maternal behavior in interaction with the infant is the Atypical Maternal Behavior Instrument for Assessment and Classification (AMBIANCE), which yields an overall rating for the level of disrupted maternal communication with the infant. An infant exposed to high levels of maternal disrupted communication at 12–18 months is almost four times more likely to exhibit disorganized attachment behavior than a child who has not experienced such interaction (odds ratio 3.7; meta-analysis Madigan et al., 2006). In addition, infants of mothers with high levels of disrupted communication are also more likely to exhibit behavior problems and psychiatric symptoms from toddlerhood to age 20 (Najmi, Bureau, Chen, & Lyons-Ruth, 2009; Dutra, Bureau, Holmes, Lyubchik, & Lyons-Ruth, 2009; Lyons-Ruth, Bureau, Holmes, Easterbrooks, & Brooks, in press; Madigan, Moran, Schuengel, Pederson, & Otten, 2007; Shi, Bureau, Easterbrooks, Zhao, & Lyons-Ruth, 2012). *Mothers* who display higher levels of disrupted communication with the infant are more likely to have higher levels of psychosocial risk, including depressive symptoms, psychiatric disturbance, or prior maltreatment of a child (Lyons-Ruth, Bronfman, & Parsons, 1999), and disrupted communication has been shown to mediate the effects of these maternal risk factors on disturbed infant attachment behavior (Lyons-Ruth, Bureau, Riley, & Atlas-Corbett, 2009). Disrupted maternal communication with the infant, then, may be one mechanism through which environmental risk is translated into individual differences in infant physiological dysregulation.

MATERNAL CORTISOL AND MATERNAL RESPONSIVE CAREGIVING

Consistent with a biosocial model of the family, which highlights the interdependence of biological processes within families (Booth, Carver, & Granger, 2000), we propose that interacting with a distressed infant may also impact the stress physiology of the mother. Although fewer studies have examined *maternal* physiology when a child is faced with a stressor (Out, Pieper, Bakermans-Kranenburg, & Van Ijzendoorn, 2010), sensitive and responsive mothers may have particularly pronounced cortisol responses when their infants are distressed. For example, the sound of a crying infant is enough to increase the cortisol levels of mothers of newborns, but only for mothers who score higher on sympathy toward their infants (Stallings, Fleming, Corter, Worthman, & Steiner, 2001). Further, increase in mothers' degree of behavioral synchrony with the infant (e.g., mimicking infants vocalizations), following a stressful event for the infant, is associated with higher maternal cortisol levels (i.e., greater reactivity; Thompson & Trevathan, 2008).

There are good theoretical and empirical grounds for predicting that mothers who display *less* attunement and *more* disrupted communication with their infants might also be more likely to experience *attenuated* cortisol responses when their child exhibits distress (Hibel, Granger, Blair, & Cox, 2009). The experience of trauma early in life is associated with later attenuated diurnal cortisol patterns among adults, marked by lower cortisol levels on rising as well as slower rates of decline over the day (Heim, Ehlert, Hanker, & Hellhammer, 1998; King, Mandansky, King, Fletcher, & Brewer, 2001; Yehuda, Halligan, & Grossman, 2001; Yehuda et al., 2000). In addition, among mothers exposed to interpersonal violence, high levels of maternal posttraumatic stress symptoms were associated with exceptionally low cortisol reactivity when mothers were interacting with their preschool children after a period of separation stress (Schechter et al., 2004). These higher levels of maternal posttraumatic stress symptoms were also significantly associated with higher levels of disrupted maternal communication, and disrupted communication, in turn, was marginally associated with lowered maternal cortisol (Schechter et al., 2004). However, to date only the Schechter et al. (2004) study has directly assessed cortisol levels and their relation to disrupted communication among at-risk mothers.

MUTUAL INFLUENCE BETWEEN MOTHER'S AND INFANT'S CORTISOL LEVELS?

Consistent with the interdependencies found between infant behavior and maternal behavior (e.g., Beebe et al., 2010), mothers' and infants' cortisol levels may also be related to one another. For example, in a study of infants in the early months of life, infant cortisol levels were significantly associated with maternal cortisol levels (Spangler & Grossman, 1993). Thus, the degree of physiological synchrony in cortisol activity may be associated with maternal empathy and behavioral mutuality. In a study in which a child between the ages of 2 and 4 years was exposed to a novel and potentially challenging task (i.e., walking on a balance beam for the first time), the degree of physiological attunement between mother's and child's cortisol during the task (i.e., correlation between infant and maternal cortisol) was associated with the mother's sensitive responsiveness toward her child during the task (Hofstad, Stansbury, & Rice, 2001). It seems that just as sensitive mothers mirror their child's behavioral and emotional cues, they may also mirror their physiological reactions to events. However, in other studies, while mothers and infants may start off with similar cortisol levels, they sometimes diverge over the course of a task, depending on whether or not the mother and infant stay in close proximity or on whether they experience the event similarly (Middlemiss, Granger, Goldberg, & Nathans, 2012; Thompson & Trevathan, 2008). Thus, the magnitude of the association between maternal and infant cortisol may be dependent on how attuned a mother is to her infant's cues.

CURRENT STUDY

Most studies linking the quality of mother–infant interaction to either maternal or infant cortisol have been conducted on infants older than 7 months of age (e.g., Blair et al., 2008). Little is known about the relation between insensitive maternal responses and cortisol activity during earlier infancy when the child is most dependent on parental regulation. In addition, most studies to date have been carried out in normative middle-income samples.

However, middle-income samples are unlikely to capture the full range of stressful circumstances associated both with lowered maternal cortisol and with more severely disrupted levels of mother–infant interaction. Therefore, the current work studied mothers and their 4-month-old infants from an economically impoverished geographic suburban area of Scotland. To the best of our knowledge, this is the first study of its type with infants this young and from an economically disadvantaged context.

Our hypotheses were as follows: First, we expected that mothers' cortisol responses would be related to the level of disrupted communication with their infants. As noted, Schechter et al. (2004) reported that mothers' high levels of disrupted communication were associated with low levels of maternal cortisol, both at baseline and during a stressor procedure. Thus, we predicted that mothers with extremely disrupted behavior would be more likely to have blunted HPA axis activity (Hypothesis 1).

Second, based on the studies reviewed above, we expected that infants exposed to disrupted maternal communication, which is likely to constitute less effective regulation of stressful arousal for the infant, would show heightened cortisol responses to a stressor. However, the literature suggests that the relation between stress exposure and cortisol baseline levels and reactivity to a stressor may be nonlinear, with those exposed to moderate stress expected to show higher cortisol responses but those exposed to very high levels of stress expected to show *lower* cortisol reactivity. Thus, given the lack of work in at-risk samples, an additional exploratory hypothesis was that infants exposed to the highest levels of disrupted maternal communication might show blunted, rather than elevated, cortisol responses to a stressor (Hypothesis 2).

Third, consistent with past work on concurrent synchrony and maternal sensitivity (e.g., Hibel et al., 2009; Hofstad et al., 2001), we expected that mothers who displayed appropriate, nondisrupted interactions with their 4-month-old infants would exhibit concurrent cortisol levels that were more closely related to those of their infants, while mothers who displayed disrupted communication with their infants would show greater discrepancies between their own cortisol levels and those of their infants. To evaluate this question, we assessed two separable components of concurrent synchrony: a) degree of covariation between maternal and infant cortisol values, which can be high even if mother and infant cortisol are at very different absolute levels and b) degree of similarity in the actual values of maternal and infant cortisol scores (Hypothesis 3a,b).

METHOD

Participants

Sixty-three mothers and their four-month-old infants (35 boys and 28 girls) participated. Over a predetermined period of 4 months, every mother of a newborn infant living with the 20% most income-deprived data zones of a fixed geographic suburban area in Scotland was approached. Mothers were told of the study during a regular health visit as part of their routine care within the Scottish National Health Service (NHS). Eighty-five percent of mothers approached agreed to participate in the study. Mothers were on average 25.9 years old (*SD* = 5.6), and 33% of the mothers reported an annual household income of less than

£10,000 GBP (32% reported between £10,000 to £20,000 GBP, 18% reported £20,000 to £30,000 GBP, and 13% reported higher than £30,000 GBP). In contrast, the average annual household income in the UK for 2007/2008 was approximately £30,000 GBP; only the bottom 20% of households had an average income of about £15,000 GBP (National Office for Statistics, 2009). Five percent of mothers had only completed some basic elementary education, 41% had completed “standards” (the equivalent of completing middle school), 42% has completed “highers” (the equivalent of completing high school), and 7% had a university degree. Fifty-five percent of the mothers were unemployed. Twenty-one percent of mothers were married, 58% were cohabitating but not married, 10% had a partner but were not cohabitating, and 9% were single.

Procedures

Two research-trained Scottish National Health Service (NHS) health visitors collected all data in the homes of the participants. All mothers gave informed consent for their infants and their own participation, and the research was approved and monitored by a Scottish National Health Service Research Ethics Board. After informed consent was elicited and equipment was setup, an initial cortisol sample was taken from both mother and infant (this first saliva sample was taken approximately 20 min after the health visitor arrived). Next, mothers and infants engaged in a filmed standard still-face interaction procedure followed by a 5-min recovery period. The still-face procedure creates a mild-to-moderate stressor for the infant, allowing the researcher to then observe how a mother is able to restore her child’s prior emotional state after an upset (for review, see Muir & Lee, 2003). The infant was placed in a baby chair while the mother sat opposite the infant. A mirror was placed adjacent to the baby, and the camera was placed behind the mother’s shoulder so that the video frame contained the infant’s full body and face, the mother’s full-face mirror reflection, and the mother’s profile. Recording the interaction in this manner allowed for eye contact to be easily determined (for similar procedures at home, see Cohn & Tronick, 1989; Cohn, Tronick, Matias, & Lyons-Ruth, 1986). The interaction followed a standard still-face procedure (Tronick, Als, Adamson, Wise, & Brazelton, 1978) in which the mother was instructed to play with her infant for 1 min, then to freeze her face of all emotion and not speak or interact with the infant for 2 min, followed by a period of normal face-to-face interaction with the infant for 5 min without the use of toys. This final 5 min of normal interaction was later coded for disrupted maternal affective communication (see Methods below). Review of the 2-min still-face portion of the videotape confirmed that all mothers followed instructions to freeze their faces and not respond to the infants’ initiatives.

The researcher left the room to give the mother and infant privacy, cracking the door in case she was needed. When the interaction procedure was completed, demographic data from mothers was collected, including recent experiences of anxiety and depression as well as variables to be controlled for in the cortisol analysis. Finally, additional cortisol samples were collected 18 min after the end of the 2-min still-face period and again 38 min after the end of the 2-min still-face period.

Determination of salivary biomarkers

Saliva samples were collected from both the mothers and the infants at three different time points during the home visit: approximately 20 min after arriving for the appointment (i.e., baseline), 18 min after the end of the still-face procedure (i.e., stress response), and 38 min after the end still-face procedure (i.e., recovery). Mothers' saliva was collected by asking them to gently mouth a 30-mm × 10-mm swab for 45 sec. Infants' saliva was collected using a hydrocellulose microsponge. The sponge was placed under the infants tongue for 15–30 sec at a time, for a minimum of 60–90 sec total. Following Granger et al. (2007), participants were asked to not eat, drink, or brush their teeth for an hour prior to their appointment. Further, all visits were made after noon to account for cortisol's diurnal pattern (and time of day samples were collected was controlled for in the analysis). The mean collection time for the baseline sample (sample 1) was 1:25 pm (*SD* 1 h, 15 min). The mean collection time for the stress response sample (sample 2) was 1:58 pm (*SD* 1 h, 18 min). The mean collection time for the recovery sample (sample 3) was 2:20 pm (*SD* 1 h, 22 min). After collection, all samples were stored frozen at –20°C until assay.

Cortisol levels were determined employing a competitive solid-phase time-resolved fluorescence immunoassay with flouromeric end point detection (DELFIA). The sample test volume was 50 μL, and the assays sensitivity was .17 nmol/L. All samples were assayed in duplicate, and the average of the duplicates was used in the analyses. Average intra- and interassay coefficients of variation were less than 6.7% and 9.0%, respectively.

Level of maternal disrupted communication with the infant

Maternal disrupted communication was coded from the videotaped 5-min period of normal mother–infant face-to-face interaction following the 2-min still-face period, using the four-month version of the AMBIANCE (Atypical Maternal Behavior Instrument for Assessment and Classification: Bronfman, Parsons, & Lyons-Ruth, 1993–2009) as adapted by Kelly (2004; Kelly, Ueng-McHale, Grienenberger, & Slade, 2003). The coding system targets atypical aspects of maternal responsiveness not adequately described in generalized coding systems for warmth or sensitivity, by focusing upon frightened, frightening, withdrawing, disoriented, or other odd or contradictory behaviors in interaction with the infant. In a recent meta-analysis, disrupted communication as coded by the AMBIANCE demonstrated substantial stability over time (stability coefficient .56**, time periods from 6 months to 5 years, $N = 203$) and demonstrated good concurrent and predictive validity in relation to infant disorganized attachment behavior ($r = .35$**, $N = 384$) and to maternal unresolved loss or trauma on the Adult Attachment Interview ($r = .20$**, $N = 311$; Madigan et al., 2006). Maternal disrupted communication in infancy has also been shown to predict child behavior problems from toddlerhood to age 20, including antisocial personality disorder, dissociation, and suicidality (Dutra et al., 2009; Lyons-Ruth et al., in press; Madigan et al., 2007; Pechtel, Woodman, & Lyons-Ruth, 2012; Shi et al., 2012).

There are two stages to the rating procedure. First, the coder notes all instances of disrupted maternal affective communication using itemized examples for five dimensions of disrupted communication described in the coding manual, as follows:

1. *Affective communication errors*, coded when the parent gives contradictory affective signals to the infant (e.g., using a sweet voice with a derogatory message or with a threatening posture) or makes inadequate or inappropriate responses to the infant's signals (e.g., fails to comfort a distressed infant).
2. *Role confusion*, coded when the mother calls the infant's attention to herself in ways that override or ignore the infant's cues (e.g., asking the infant for a kiss when the infant is distressed).
3. *Frightened/disoriented behavior*, as shown in fearful, hesitant, or deferential behavior toward the infant (e.g., asking permission of the infant) or as expressed in disoriented behavior, including loss of affect and movement (e.g., "freezing"), frenetic and uncoordinated overtures toward the infant, or sudden and unusual shifts in voice tone.
4. *Negative-intrusive behavior*, either in physical interaction (e.g., pulling the infant by the wrist) or in verbal communication (e.g., mocking or teasing the infant; attributing negative feelings or motivation to the infant, as in "He/she hates me").
5. *Withdrawing behavior*, as shown by creating physical distance from the infant (e.g., sitting back in the chair rather than leaning forward toward the infant to interact) or creating verbal distance (e.g., interacting silently).

Second, based on both the frequency and seriousness of these observed forms of disrupted communication, as well as the relative absence of ameliorating interactions, a rating is given on a 7-point scale for overall level of disrupted affective communication. Parents rated at 5 or above on the scale are considered as disrupted in parent–infant communication. The manual gives explicit guidelines for judging each level, including specific descriptions of behavior that might satisfy criteria for the level. For example, partial criteria for a score of 5 are as follows: "While the parent appears to be trying to interact appropriately with the infant, the parent cannot divert from own needs or cannot seem to understand infant's signals....The parent displays persistent mixed affective signals or persistent errors in responding to infant needs...Interactions appear arrhythmic, with delayed, inappropriate, or absence of response to infant signals....The parent may display elevated withdrawal, mildly fearful behavior, confusion or disorientation with the infant.... Even when little or no overt hostility is displayed, there may be a sense of not enough availability of parent to the infant or little assistance to the infant in coping with a difficult situation" (Bronfman et al., 1993–2009; p. 13).

A maximum score of 7 is given when "disrupted communication predominates with almost no ameliorating behaviors. The parent is highly unresponsive, ineffective, or inappropriate in relation to the needs of the infant. Not only do the parent's needs take priority, but the infant's needs are not attended to in any significant manner. There is persistent evidence of mixed affective signals, intrusive behavior, withdrawal, hostility, lack of boundaries, role reversal, and/or disorientation, with little ameliorating contact." The intraclass reliability coefficient for the rating between two coders on a randomly selected sample of 10% of the tapes was ICC = .91 (McGraw & Wong, 1996).

Given that we might expect a nonlinear relation between the degree of maternal disrupted communication and infant cortisol response, it was important to distinguish between moderately disrupted levels of mother–infant interaction and severely disrupted levels of interaction. Because lowered cortisol reactivity has primarily been associated with extreme adverse experiences (i.e., violence, abuse, or neglect), we made a conservative decision to include only those at the highest level of disturbance on the AMBIANCE in the severely disrupted group (rating of 7 on the 7-point scale). Mothers with AMBIANCE scores of 5–6 were assigned to the moderately disrupted group, and, consistent with the manual's instructions, mothers with scores of 1–4 were placed in the not disrupted group.

Analytic strategy

Cortisol values were first examined for outliers and for skew. The one cortisol value more than three standard deviations from the mean was deleted from the data set (n = 1), yielding an N of 62 for all analyses. Due to positive skew, cortisol values were then subjected to a log transformation, which was successful in normalizing the distributions for both maternal and infant cortisol (skewness =−.51 to .98). Although we measured cortisol in nmol/L, nontransformed μg/dL units are reported in the text and tables to ease interpretation.

In this design, multiple cortisol assessments were nested within individual, and individuals were nested within the mother–infant dyad. To account for the dependency in the data, we used a mixed model ANOVA that includes both between-subjects and within-subjects measures in SPSS (similar to MLM; Raudenbush & Bryk, 2002). These analyses simultaneously models the error involved with sampling observations at multiple levels, making it a preferred strategy to ordinary least squares regression (Kenny, Kashy, & Cook, 2006). However, it is important to note that for many of the analyses, dependency at the dyad level was not relevant. For example, if psychological variables were only collected from the mother, they could be used to predict both mother and infant cortisol outcomes in separate analyses without violating assumptions of data independence.

To address Hypotheses 1 and 2, we evaluated relations between severity of maternal disrupted communication and levels of maternal and infant cortisol. To examine concurrent synchrony between maternal and infant cortisol (Hypothesis 3a,b), we evaluated the relations between the level of maternal disruption and a) the covariation between maternal and infant cortisol and b) the absolute value of the differences between maternal and infant cortisol levels.

RESULTS

Maternal and infant cortisol responses to the still-face assessment: preliminary and descriptive analyses

Descriptive characteristics and correlations between mothers' and infants' cortisol at all three measurement occasions are displayed in Table 1. Forty-four mothers in the sample (69.8%) were rated as not disrupted (AMBIANCE ratings 1–4), 18 mothers (28.6%) were rated as moderately disrupted (ratings of 5–6), and three mothers (4.8%) were rated as severely disrupted (rating of 7).

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Infants displayed a typical stress response to the still-face procedure in that infants' cortisol levels increased from time 1 (M = .16 μg/dL) to time 2 (M = .19 μg/dL; $t(48,2) = 2.66$, p = .01) and then decreased during recovery ($M_{Time\ 3}$ = .16 μg/dL; $t(52,1) = 5.5$, $p < .01$). In contrast, mothers exhibited a significant decrease in cortisol throughout the study session (M_{Time1} = .13 μg/dL, M_{Time2} = .11 μg/dL, M_{Time3} = .10 μg/dL; $F(39,2)= 4.51$, $p = .02$). Level of mothers' cortisol was correlated across all measurement occasions (p's < .05). Level of infants' cortisol at Time 2 was correlated with level of infants' cortisol at Time 1 and Time 3 (p's < .05); however, infants' cortisol level at Time 1 was not significantly associated with infants' cortisol level at Time 3 ($p = .14$).

Several additional variables were examined for their relation to maternal and infant cortisol levels to evaluate whether they should be included as covariates in subsequent analyses, including maternal age, maternal education, and household income. Results indicated that none of these variables were associated with maternal or infant cortisol levels (*p's* > .2). However, variables with established associations with maternal or infant cortisol responses (i.e., food and dairy consumption within the last hour, depression, anxiety, and time of day cortisol was assessed) were included in the relevant analyses as specified below.

In the subsequent sections, we first examine predictors of maternal and infant cortisol separately. We then examine two types of synchrony or mutual influence between maternal cortisol and infant cortisol. First, do maternal cortisol and infant cortisol vary together and is any obtained covariation associated with timing of assessment or severity of disrupted communication? Second, are the absolute levels of maternal and infant cortisol more similar among nondisrupted dyads than among more severely disrupted dyads?

Maternal cortisol and maternal disrupted communication

To assess the relation between maternal disrupted communication and mothers' cortisol responses (Hypothesis 1), a mixed-design ANOVA was estimated with mothers' cortisol as the outcome variable and severity of maternal disrupted communication (1–3), timing of cortisol assessment (baseline, reactivity, recovery; 0–2), and the interaction of maternal disrupted communication and timing of cortisol assessment as the predictor variables. Variables known to affect maternal cortisol responses (i.e., food and dairy consumption within the last hour, depression, anxiety, and time of day cortisol was assessed) were included as control variables. Results of this analysis revealed that severity of maternal disrupted communication was significantly associated with lower maternal cortisol [$F(52.27) = 3.32$, $p = .04$]. There was a significant effect of timing [$F(89.98) = 4.67$, $p = .03$, as described above], but no interaction between timing of assessment and severity of maternal disrupted communication on maternal cortisol [$F(89.14) = 1.52$, $p = .22$; see Figure 1].

Follow-up analyses were run to assess which contrasts among the three categories of maternal disrupted communication were associated with lower maternal cortisol. Using the same mixed-design ANOVA as above, two orthogonal models were examined, one comparing nondisrupted to moderately disrupted mothers, and one comparing severely disrupted mothers to both other groups combined. Only the contrast between mothers with severely disrupted maternal communication and all other mothers reached significance.

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Mothers who displayed severely disrupted maternal communication with their infants had significantly lower mean levels of cortisol than did other mothers [$b = .28$, $SE = .11$, $t(52.83) = 2.59$, $p = .01$, 95% CI (0.06, 0.50)]. The effect size was large, $d = .86$ (effect sizes above $d = .80$ are considered large effects, Morris & DeShon, 2002).

Infant cortisol and maternal disrupted communication

To test the effect of maternal disrupted communication on infants' cortisol responses (Hypothesis 2), a mixed-design ANOVA was estimated with infants' cortisol as the outcome variable and severity of maternal disrupted communication (1–3), timing of cortisol assessment (baseline, reactivity, recovery; 0–2), and the interaction of maternal disrupted communication and timing as the predictor variables. Variables known to affect infant cortisol responses (i.e., food and dairy consumption within the last hour as well as time of day cortisol was collected) were included as covariates/control variables. Severity of maternal disrupted communication was not significantly associated with the infants' mean cortisol levels ($p > .20$); nor was time linearly related to infant cortisol ($p = .17$). There was, however, a marginally significant interaction between maternal disrupted communication and timing of infant cortisol assessment [$F(93.19) = 2.43$, $p = .09$]. Typically, a marginally significant interaction term would not be followed up statistically due to concerns about replicability. However, the underlying effect size driving this interaction was very large, and so, we mention it here to stimulate attempts to replicate this in larger samples. Applying the same two orthogonal contrasts as above revealed that infants of mothers who displayed severely disrupted communication had significantly higher rates of increase in cortisol over the course of the still-face procedure (steeper slopes) than did infants of mothers who did not display severely disrupted behavior [$b = -.22$, $SE = .11$, $t(92.18) = -2.04$, $p = .04$, 95% CI (−.43, −0.01)]. This effect can be seen in Figure 1. The effect size was large, $d = 2.07$. Other contrasts were not significant. Given that the initial interaction term did not reach significance, however, this finding must be viewed with caution.

Evaluating concurrent relations between maternal and infant cortisol: covariation and similarity in cortisol levels

To assess the degree of concurrent covariation between infants' and mothers' cortisol (i.e., associations within measurement occasion), a model was run that included maternal cortisol, maternal disrupted communication (1–3), timing of assessment (0–2), all two-way and three-way interactions, and relevant control variables including infant dairy intake, infant food intake, mother's dairy intake, mother's food intake, maternal depression, maternal anxiety, and sample time. Mother's cortisol was not significantly correlated with infants' concurrent cortisol ($p > .20$), nor did the lack of association between mothers' and infants' concurrent cortisol levels change by timing of assessment ($p > .20$), or by severity of maternal disrupted communication ($p > .20$) or their interactions (see Table 1 for raw correlations).

The second question of interest concerned whether mothers and infants exhibited similar levels of cortisol overall, as well as whether mothers' and infants' cortisol values *converged or diverged systematically* as a function of timing of assessment (baseline, reactivity, recovery) or severity of disruption in maternal communication. To capture similarity and divergence between maternal and infant cortisol levels, following Griffin, Murray, and

Gonzalez (1999), we computed the dependent variable as the absolute value of the difference between mothers' and infants' cortisol levels, and also entered mothers' and infants' cortisol values into the model as control variables. Standard cortisol control variables were also added to the model (i.e., food and dairy consumption within the last hour, and time of day cortisol was assessed). Finally, timing of assessment (0–2) and severity of disruption (1–3) were entered as the predictor variables. The interaction term was also assessed but was not significant [$F(98.59) = .38$, $p = .68$] and was trimmed from the final model. Results indicated that the severity of maternal disrupted communication was significantly associated with the magnitude of difference between maternal and infant cortisol levels [$F(46.11) = 3.77$, $p = .03$]. Timing of assessment was not associated with the degree of difference between maternal and infant cortisol values. Computing the same two orthogonal follow-up models as above, significantly greater differences between mothers' and infants' cortisol values were found in the severely disrupted group compared with the less disrupted groups, $b = -.28$, $SE = .10$, $t(45.80) = -2.66$, $p = .01$, 95% CI (−0.48, −0.07). The effect size again was large, $d = 1.30$.

This finding indicated that cortisol levels at any given assessment point were more similar between mothers and infants who were *not* severely disrupted than among those who *were* severely disrupted. As can be seen in Figure 1, this divergence in cortisol levels between mothers and infants in the severely disrupted group was due to severely disrupted mothers maintaining unusually low cortisol levels overall assessment points, while their infants' cortisol levels rose dramatically over the still-face and recovery periods.

DISCUSSION

Results extend our understanding of the relations between the quality of mother–infant interaction and maternal and infant cortisol responses to a stressor. The first finding of the study, and the one with the most far-reaching implications, was that *lower* maternal cortisol was associated with the severity of maternal disrupted communication with the infant. Importantly, this association between disrupted mother–infant interaction and low maternal cortisol was consistent across all three assessment points including the baseline period, which occurred prior to both the stress of the still-face interaction and to the assessment of disrupted communication. The consistency of this relation, then, suggests that low maternal cortisol may be a general risk factor for impaired parent–infant relationships. This association between mothers' lowered cortisol levels and her difficulty interacting with her infant in an empathic and nondisrupted way echos the similar finding of Schechter et al. (2004) among mothers of preschool-aged children and thus has potentially important implications for understanding parenting behavior, which will be discussed in more detail below.

Analysis of the relations *between* maternal and infant cortisol levels further illuminated these findings. There was no overall correlation between maternal and infant cortisol levels at particular assessment points. However, correlations do not take into account of the absolute levels of cortisol, only the extent of parent–infant covariation. When the absolute values of the differences between maternal and infant cortisol levels were assessed, these differences were significantly predictable from the severity of maternal disrupted communication. As maternal affective communication with the infant became more disrupted, the difference between maternal and infant cortisol levels became significantly greater. This dyadic divergence in cortisol levels appeared to be a joint effect of the sustained low cortisol levels of severely disrupted mothers combined with the rising slope of the cortisol levels of their stressed infants across the still-face procedure. Thus, the divergence between maternal and infant physiological stress responses appears to mirror the mismatch between the infant's signals and the parent's severely disrupted responses to those signals coded at the behavioral level.

It is also notable that these cortisol findings pertain to mothers of very young infants. Prior studies of both maternal and child cortisol have primarily studied children aged 1 year and older. These findings extend into the early months of life the prior work relating impaired parenting to atypical child cortisol responses and suggest that studies of maternal and infant cortisol now need to be extended into the prenatal and early postnatal periods.

Why might low maternal cortisol be related to mothers' severe disruption in attuned, empathic responding to their infants? To account for this link, we propose a model in which normative maternal cortisol levels serve as an indicator of mother's flexible access to her own emotional states. Such flexible access in the face of affective arousal allows the parent to use her own affective responses to attune to, and regulate, her infant's level of arousal. In this model, downregulation of the mother's HPA axis response carries with it a blunting of access to her own affective experience, which then impairs the mother's ability to empathically attune to her infant's experience.

Consistent with this model, mothers who were not disrupted in communication with their infants were neither elevated nor blunted in their cortisol responses over the course of the still-face procedure. Instead, their mean cortisol levels were similar in value to those of their infants, but appeared to vary independently around that maternal mean in response to their assessments of the environment. We would speculate that these maternal and infant cortisol values represent "normative" midrange levels that allow each partner to respond flexibly and independently to the challenges posed by the presence of the researcher in the home and the need to maintain the face-to-face interaction. As described in microanalytic videotape analyses, parents follow the general valence and direction of the infants' affective arousal, while also modulating the mutual affective state by downregulating or upregulating the affect and intensity as appropriate to maintain the infants' positive affective state (Beebe et al., 2010; Cohn & Tronick, 1989). The observed midrange cortisol values seem to be consistent with the mother's sensitivity and flexibility in responding to the infant's cues and working to maintain the infant's positive state, while their infants are able to show the expected reactivity to the challenge posed by the stressor but also recover to baseline in response to the mother's nondisrupted regulation in face-to-face interaction subsequent to the stressor.

In contrast, among severely disrupted dyads, very low maternal cortisol levels were associated with a striking lack of sensitivity to infant cues, as well as with infant cortisol levels that increased sharply across the stressor and recovery periods. A visual inspection of the infants' cortisol patterns over time is particularly revealing (see Figure 1). Even as young as 4 months, infants' of mothers who displayed severely disrupted communication had very low mean cortisol levels, similar to their mothers. In contrast to their *mothers*, however, *infants'* of severely disrupted mothers exhibited cortisol responses that were very reactive to the still-face procedure and did not decrease during the recovery period.

The validity of this model will need to be explored in future work. We cannot determine from the present analyses whether the mother's sustained low cortisol is a response to some aspect of the infant's responses or whether the infant's responses are conditional on aspects of the mother's functioning. It will be important for future work in larger samples to look at direction of influence between mother and infant over time.

Limitations and future directions

The present findings must be considered in light of the study's strengths and limitations. Most importantly, the small sample size, particularly in the severely disrupted group, makes these findings preliminary and in need of replication. Although the effect sizes were large and significant, we cannot be confident that sampling issues do not play a role in the findings. Thus, replication studies are important. However, the maternal results do converge with previous work by Schechter et al. (2004) indicating that particularly high-risk mothers, as indexed by posttraumatic stress symptoms, had significantly lower cortisol levels than other mothers and also had significantly higher levels of disrupted communication in interaction with their preschool-aged children. This convergence with previous work in an independent laboratory, in addition to the large effect sizes obtained in the present work, suggest that these findings may be pointing to the existence of an important subgroup of mothers and infants who need further consideration in subsequent work.

A second limitation is that the subcategorization of the group of disrupted mothers into moderately disrupted and severely disrupted categories has not been validated beyond the data presented here. In previous work with the AMBIANCE scoring system, both the ordinal (1–7) rating scale and the categorical distinction between nondisrupted and disrupted mothers *have been* well validated and have also shown substantial stability over time periods from 6 months to 5 years (r = .56; N = 203; see meta-analysis Madigan et al., 2006). No external validity data are available on the further subcategorization of the disrupted group used here. However, in all previous work, a linear relation has been expected between degree of maternal disruption and the outcome variable of interest (e.g., infant disorganized attachment; child behavior problems). Cortisol measures have been distinct in the curvilinear relation that has emerged in relation to severity of risk factors, with moderate risks associated with *elevated* cortisol response to stressors and severe risks associated with *blunted* cortisol response to stressors. Thus, use of the disrupted communication measure as a continuous measure of increasing severity was not appropriate for testing the possibility of such a curvilinear relation to cortisol responses of mothers and infants in the present study, leading to the further subcategorization of disrupted communication into moderate and severe categories. In addition, due to the likely rarity of blunted cortisol responses in the general population, we assigned severe status only the highest scale point (7) on the level of disruption scale. Further research is needed to assess whether this cut-point for distinguishing moderate and severe groups continues to show validity beyond the findings of the present study.

Third, we do not have data on the behavior of the infants in this study. Such data would be important to gather in future work to assess whether normative, high, and blunted cortisol levels are associated with particular infant behavioral presentations, for example mild distress, extreme distress, or apathy.

Perhaps the most notable strength of the current study was the use of health visitors within a community sample. Because all mothers within a defined geographic area were approached for participation, the community sampling procedure should yield a study sample that reflects the distribution of disrupted relationships among economically stressed families. In the current cohort, 4.8% of mothers were assessed as severely disrupted in interaction with their infants. In addition, because health visitors had preexisting relationships with these families, and were able to visit in participants' homes, it was likely that the mothers and infants were more comfortable than they would have been had the study taken place in a laboratory requiring travel to an unfamiliar place with an infant. This naturalistic setting was likely to have increased the rate of participation, and hence the representative nature of study findings, and decreased sources of error in both mothers' and infants' physiological reactivity to the study procedures, enabling more accurate baseline measures of maternal and infant cortisol.

The findings of the current study also raise a number of avenues for future research. The patterning of infant cortisol in the current work suggests that even as young as 4 months, infants are starting to show signs of lower baseline cortisol, greater reactivity to a stressor, and slower recovery to baseline. Given the association between slow cortisol recovery and the eventual deregulation of cortisol (e.g., Ryff & Singer, 2001; Seeman & McEwen, 1996; Wimbush & Nelson, 2000), longitudinal assessment of cortisol trajectories for infants as a function of degree of maternal disrupted communication is needed to assess whether these early signs might be indicative of the eventual development of lower cortisol baselines, flattened diurnal slopes, and lessened reactivity to stressful events.

Further work should also explore whether low maternal cortisol may be one mediating mechanism through which maternal stressful life experience impacts maternal communication with the infant. Mothers who display severely disrupted communication with their infants are more likely to have experienced childhood abuse (Lyons-Ruth & Block, 1996), to endorse high levels of PTSD symptoms (Schechter et al., 2004), and to exhibit unresolved states of mind regarding past trauma (Madigan et al., 2006). The experience of trauma is also associated with later lower cortisol levels (Heim et al., 1998; King et al., 2001; Yehuda et al., 2000, 2001). Thus, mothers' attenuated levels of cortisol may be one important mechanism through which experiences of extreme stress modify a mother's ability to respond emotionally to her infant. Further work is needed relating maternal cortisol levels to maternal life experiences.

Finally, future studies should evaluate whether low maternal cortisol levels precede and thus might be a causal contributor to the infant's difficulties in regulating cortisol responses to a mildly stressful situation. Understanding direction of effect between low maternal cortisol and infant cortisol dysregulation will be critical to the design of interventions to help mother–infant relationships at risk.

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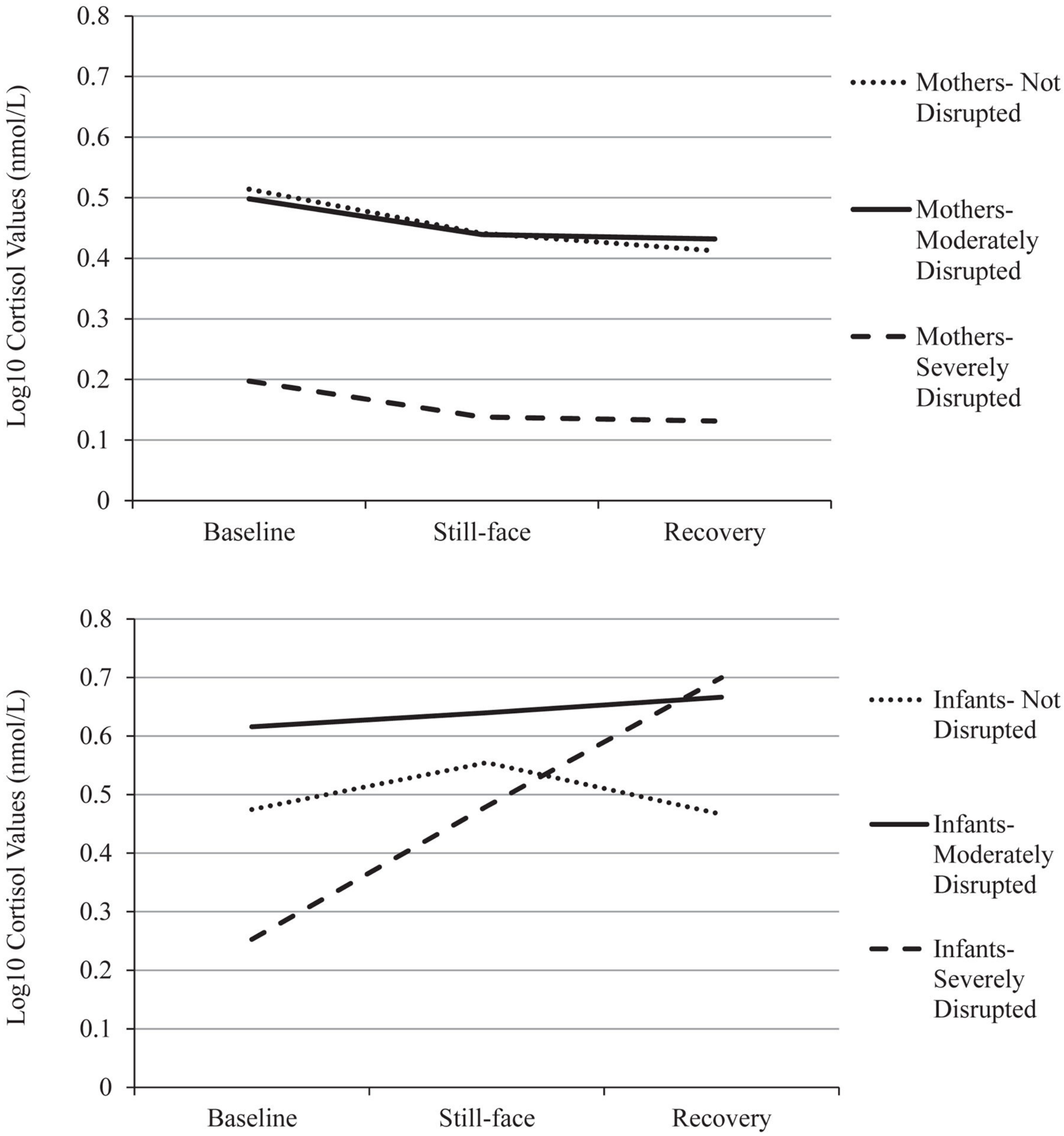

Figure 1.
Maternal and infant cortisol over time by level of maternal disrupted communication.

TABLE 1

Correlations Among Cortisol Levels at the Three Assessment Points ($N = 62$)

Variables	1	2	3	4	5	6
1. Maternal cortisol (1)	–					
2. Maternal cortisol (2)	.88**	–				
3. Maternal cortisol (3)	.86**	.87**	–			
4. Infant cortisol (1)	.16	.13	.08	–		
5. Infant cortisol (2)	−.09	−.09	−.24+	.38**	–	
6. Infant cortisol (3)	−.04	.03	−.07	.20	.61**	–
M	.13	.11	.10	.16	.19	.16
SD	.06	.04	.04	.14	.14	.13
Range	.02–.27	.02–.21	.02–.22	.04–.83	.03–.67	.04–.83

Note. Cortisol assessed at baseline (1); 18 min after end of still-face procedure (2); and 38 min after end of still-face procedure (3). Cortisol values reported are raw values in μg/dL (these values were then transformed to correct for skewness).

+ $p < .10$;

** $p < .01$