ARTICLE

PREDECTING MATERNAL SENSITIVITY: THE ROLES OF POSTNATAL DEPRESSIVE SYMPTOMS AND PARASYMPATHETIC DYSREGULATION

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ABSTRACT: Previous research has established that maternal depression is a risk factor for a variety of negative developmental outcomes among infants and children. Although low levels of maternal sensitivity have been hypothesized to explain this risk, the biological mechanisms underlying the association between postnatal depressive symptoms and low levels of maternal sensitivity have been largely underexplored. This study examined the roles of postnatal depressive symptoms and parasympathetic nervous system functioning as predictors of low levels of maternal sensitivity, during a stressful mother–infant interaction—the reunion phase of the Still-Face Paradigm. Depressive symptoms and traitlike predispositions toward parasympathetic dysregulation, as indexed by low resting levels of respiratory sinus arrhythmia, were associated independently with less sensitive parenting. Discussion considers that during stressful mother–infant interactions, both mothers with depressive symptoms and mothers predisposed to parasympathetic dysregulation may have fewer emotional, physiological, and psychological resources with which to respond sensitively to their infants’ cues.

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Perinatal depression is an issue of considerable importance. Epidemiological studies have reported that depression during the first 3 to 5 months of the postnatal period affects roughly 10 to 15% of all new mothers (Cooper & Murray, 2007; Cox, Murray, & Chapman, 1993). In addition to its prevalence, there are clear risks associated with maternal depressive symptoms for very young children (Campbell, Cohn, & Meyers, 1995; Campbell, Matestic, von Stauffenberg, Mohan, & Kirchner, 2007; Cicchetti, Rogosch, & Toth, 1998; Cummings & Davies, 1994; Downey & Coyne, 1990; Field, 1992; Goodman & Gotlib, 1999, 2002). In particular, mothers with depressive symptoms tend to be less emotionally available during interactions with their infants, and this emotional unavailability is correlated with difficulties in interpreting and responding appropriately to their infants’ behavioral and emotional cues (Cicchetti, Ganiban, & Barnett, 1991; Field, 1992; Field, Sandberg, Vega-Lahr, Goldstein, & Guy, 1985). As such, the children of mothers with depressive symptoms are often deprived of critical developmental support (Campbell et al., 2007; Cohn, Campbell, Matias, & Hopkins, 1990; Cohn & Tronick, 1987; Jameson, Gelfand, Kulcsrar, & Teti, 1997).

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Maternal sensitivity, or the attuned, timely, and ultimately effective response to an infant’s needs (Lohaus, Keller, Ball, Elben, & Voelker, 2001), also has been associated with child cognitive and socioemotional development and attachment security (Bowlby, 1969; Campbell et al., 2007; Carlson & Sroufe, 1995; DeWolff & van IJzendoorn, 1997). Specifically, mothers who are more sensitive to their infants’ cues are more likely to have children who are securely attached. This finding has been supported repeatedly in the literature, as demonstrated in the meta-analysis by DeWolff and van IJzendoorn (1997). In this analysis, the composite index of the association between maternal sensitivity and child attachment security was in the moderate range, \( r(1,097) = 0.25 \), according to Cohen’s (1992) conventional criteria.

Goodman and Gotlib (1999, 2002) proposed that a lack of maternal sensitivity likely mediates the association between maternal depressive symptoms and cognitive and socioemotional impairments in children. However, few studies have directly tested this mediation-model (Burrous, Crockenberg, & Leerkes, 2009; Shaw, Connell, Dishion, Wilson, & Gardner, 2009). In addition, evidence has accumulated suggesting that perhaps depressive symptoms are better conceptualized as a moderator of the association between maternal sensitivity and child outcomes (Campbell et al., 2007; Campbell et al., 2004; Murray, Fiori-Cowley, Hooper, & Cooper, 1996; Murray, Stanley, Hooper,
A third possibility also exists, which suggests that maternal depressive symptoms, difficulties with sensitivity, and other maternal characteristics might be critical, yet independent, sources of risk for child impairments (Maughan, Cicchetti, Toth, & Rogosch, 2007).

The specific nature of the statistical relationship (i.e., mediation, moderation, or independent/proxy risk factors) underlying the connection among postnatal depressive symptoms, maternal sensitivity, and child outcomes remains unclear. Nonetheless, more recent attempts to specify the basis for these associations have proposed the role of biological processes common to both problems with depressive symptoms and maternal sensitivity (Mills-Koonce et al., 2007; Rottenberg, Salomon, Gross, & Gotlib, 2005). In light of the role that difficulties with emotion regulation play in both affective disturbances (Beauchaine, 2001; Beauchaine, Gatze-Kopp, & Mead, 2007) and impaired parenting behaviors (Moore et al., 2009), physiological processes associated with the parasympathetic nervous system (PNS) have become targets of interest.

Depressive symptoms and the disorder of depression are widely viewed as problems of emotion dysregulation at both behavioral and psychophysiological levels (Beauchaine, 2001; Rottenberg, Wilhelm, Gross, Biuckians, & Gotlib, 2001). One psychophysiological index of PNS activity associated with emotion regulation is respiratory sinus arrhythmia (RSA). RSA is a computed index reflecting the increase in heart rate during inhalation and decrease in heart rate during exhalation, as a result of modulation by vagus nerve activity at the cardiac sinoatrial node (Beauchaine, 2001; Porges, Dousnard-Roosevelt, & Maiti, 1994). Control of the heart by the vagus nerve has been shown to be a valid characteristic of PNS functioning, and vagal control of the heart also is associated with homeostatic functions as well as regulation of the flight-or-fight response (Berntson et al., 2002).

Low basal levels of RSA have been associated with emotion regulation difficulties in both children and adults (Beauchaine, 2001; Beauchaine et al., 2007). Despite the emotion regulation difficulties characterizing individuals with depressive symptoms, studies examining basal levels of RSA among individuals with depressive symptoms have produced mixed results. Several researchers have shown that individuals with clinically significant depressive symptoms have lower basal levels of RSA than do individuals without depressive symptoms (Carney et al., 1995; Dalack et al., 1990; Licht et al., 2008; Rechlin, Weis, Spiter, & Kaschka, 1994; Rottenberg, 2007; Rottenberg et al., 2001; Rottenberg, Wilhelm, Gross, & Gotlib, 2002). However, other studies have found no differences between participants with depressive symptoms and healthy controls with respect to basal levels of RSA (Lehofer et al., 1997; Moser et al., 1998). A likely reason for these inconsistent results involves the heterogeneity of depressive symptoms experienced by the participants in these studies. For example, Rottenberg et al. (2002) found that low baseline levels of RSA were positively associated with cognitive depressive symptoms such as thoughts of sadness and feelings of guilt, but negatively associated with suicidal ideation. As such, it may be that specific depressive symptoms are associated with specific patterns of parasympathetic dysregulation. This study examines new mothers’ basal levels of RSA in relation to cognitive depressive symptoms, as reported on the Edinburgh Postnatal Depression Scale (EPDS; Cox, Holden, & Sagovsky, 1987). This measure was chosen because women in the postnatal period typically experience many physical depressive symptoms such as changes in sleep patterns, appetite, and weight as a consequence of parturition and/or postnatal fatigue. Furthermore, recent research examining postpartum fatigue has reported that it is a distinct entity from postpartum depressive symptoms (Bozoky & Corwin, 2002; Groer et al., 2005). Thus, for the purposes of this study, the cognitive features of depressive symptoms were examined.

In addition to the research that has linked depressive symptoms and basal RSA levels, mounting evidence has implicated the role of parasympathetic functioning in social functioning, generally, and in parenting behavior, more specifically (Beauchaine, 2001; Beauchaine et al., 2007; Moore et al., 2009). Previous work has associated high basal levels of RSA with social competence and resilience in the face of stress (Eisenberg et al., 1995; Fabes & Eisenberg, 1997). Other studies have associated low basal RSA levels with a lack of emotional understanding, inability to regulate emotions, and lack of social competence (Neumann, Sollers, Thayer, & Waldstein, 2004). In addition, it has been hypothesized that the association between basal RSA levels and social competence extends to interactions between mothers and their infants, such that mothers with high basal levels of RSA tend to be more sensitively attuned to their infants’ behavioral and emotional cues while mothers with low basal levels of RSA tend to be less responsive to their infants’ cues (Moore et al., 2009). This hypothesis is based on previous research showing that sensitive mothers typically display autonomic responses consistent with approach tendencies to care for their infants (Ham & Tronick, 2006; Tronick, 1989). However, some individuals show atypical patterns of autonomic reactivity when confronted with infant cues, even prior to having children of their own (Bugental, Lewis, Lin, Lyon, & Kopeikin, 1999). Specifically, Bugental, Lewis, Lin, Lyon, and Kopeikin (1999) found that women at risk for low maternal sensitivity responded to an unfamiliar infant’s emotional cues with a pattern of autonomic reactivity consistent with a mobilization to flee or avoid interactions (i.e., increases in heart rate).

Previous research has shown that maternal sensitivity is associated with maternal adult attachment styles, such that mothers with secure adult attachment styles are more likely to engage in sensitive maternal behaviors with their infants (Raval et al., 2001; van IJzendoorn, 1995; Ward & Carlson, 1995). Recently, Laurent and Ablow (2012) found that secure-autonomous expectant mothers responded to listening to an unfamiliar infant cry stimulus with reductions in RSA. However, insecure-dismissing women responded to an unfamiliar infant’s emotional cues with a pattern of autonomic reactivity consistent with a mobilization to flee or avoid interactions (i.e., increases in heart rate).
Furthermore, maternal autonomic functioning, sensitive maternal behavior, and infant attachment security have been associated with the entrainment of the autonomic nervous system of the infant, further highlighting the importance of examining physiological predictors of maternal behavior (see Conradt & Ablow, 2010; Schore, 2001, 2010).

While previous studies linking maternal behavior to autonomic functioning have examined psychophysiology reactivity, it may be that baseline or traitlike features of autonomic functioning may be more strongly associated with maternal behavioral responses to infant cues, as many of the maternal characteristics associated with sensitivity also are traitlike, long-standing predispositions (i.e., attachment; Laurant & Ablow, 2012). In addition, the rapidly developing area of study examining allostatic load has focused on how environmental factors and physiological reactivity in the face of stress directly influence basal levels of autonomic functioning over time as well as how these basal levels subsequently affect behavior (McEwen & Stellar, 1993). Furthermore, according to the “law of initial values,” baseline levels of physiological functioning may directly affect the direction and magnitude of physiological reactivity and serve to predispose mothers toward specific patterns of autonomic and behavioral responses (Porges et al., 1994). Thus, an examination of baseline levels of RSA may provide additional insight into the understanding of mothers’ traitlike autonomic dysregulation as it relates to predispositions toward sensitive maternal behavior.

**CURRENT STUDY**

The research reviewed has suggested that maternal depressive symptoms are a risk factor for problems with maternal sensitivity. Furthermore, adults with depressive symptoms have been shown to have low baseline levels of RSA. Thus, it is possible that mothers with depressive symptoms have difficulty regulating their physiological responses to stress, given traitlike difficulties with emotion regulation, as evidenced by low baseline RSA. As such, mothers with depressive symptoms may have fewer emotional, physiological, and psychological resources with which to respond sensitively to their infants’ cues.

In an attempt to clarify possible associations among mothers’ depressive symptoms, baseline RSA levels, and behavioral sensitivity, three quantitative models were tested in the present study. Specifically, resting RSA levels were tested as a mediator (Model 1) of the association between depressive symptoms and low maternal sensitivity. In a second model, depressive symptoms were tested as a possible moderator of the association between baseline RSA and maternal sensitivity (Model 2). Finally, resting RSA levels and depressive symptoms were examined as independent risk factors for low maternal sensitivity (Model 3). To better test the effects of long-standing depressive versus postpartum depressive symptoms, the data for this study were collected at 5 months postpartum, as it has been shown that postpartum depressive symptoms resolve typically by the fifth month of the postnatal period (Cooper & Murray, 2007; Cox et al., 1993).

It was hypothesized that mothers with depressive symptoms would be less sensitive while interacting with their infants than would mothers with few or no depressive symptoms. It also was hypothesized that mothers with depressive symptoms would have correspondingly lower resting levels of RSA. In addition, we initially hypothesized that low resting levels of RSA would mediate the relationship between postnatal depressive symptoms and low maternal sensitivity (i.e., support for Model 1). However, as with all meditational models, there are several important caveats which must be considered.

Kraemer, Stice, Kazdlin, Offord, and Kupfer (2001) suggested that mediation models must meet several criteria. The first criterion, temporal precedence, states that the mediated factor must occur prior to the mediating factor in time. The second criterion, correlation, states that the two risk factors must be correlated with one another. The final criterion, domination, states that for full mediation to be present, the association must be accounted for by the mediated factor. Temporal precedence of the predictors being tested in this study could not be established with complete certainty. However, in keeping with previous work that has examined the temporal relationship between depressive symptoms and parasympathetic regulation, it was assumed that maternal depressive symptoms precede decreases in baseline RSA levels (see the meta-analysis by Rottenberg, Clift, Bolden, & Salomon, 2007, which has suggested that depressive symptoms precede abnormal cardiac vagal control). As such, these assumptions enabled a test of statistical mediation rather than a test of true mediation. If supported, this will be a useful starting point for replication in longitudinal studies where the temporal sequencing of baseline RSA levels and the onset of depressive symptoms may be determined.

Alternatively, depressive symptoms could operate as a moderator of the link between baseline RSA levels and maternal sensitivity. As noted by Kraemer et al. (2001), moderation occurs when two risk factors are uncorrelated with one another. Kraemer et al. also argued that the moderator typically precedes the moderated construct and is more readily conceptualized as a dichotomous factor (e.g., depressed vs. nondepressed). Findings in the depression literature suggestive of a lack of association between depressive symptoms and baseline RSA levels have been supportive of moderation. As such, if low baseline RSA is indeed a proxy for traitlike levels of dysregulation, high versus low levels of depressive symptoms may help to specify for which mothers problems with emotion regulation act as a risk factor for insensitive parenting. In our test of moderation (Model 2), it was hypothesized that higher numbers of depressive symptoms would augment the link between low baseline RSA and lower maternal sensitivity while lower levels of depressive symptoms would buffer women with low baseline RSA from problems with maternal sensitivity.

Finally, it may be that baseline RSA levels serve as neither a mediator nor moderator of the association between maternal postnatal depressive symptoms and low sensitivity. Again, findings in the literature showing a lack of association between depressive symptoms and baseline RSA levels, but an association between each of these factors and low sensitivity, have been supportive of
such a model. In such a case, it may be that both maternal postnatal depressive symptoms and low baseline RSA levels function as independent risk factors for maternal insensitivity. This model (Model 3) also was examined.

Note that the majority of studies examining the association between maternal postnatal depressive symptoms and behavioral sensitivity have done so in the context of positive face-to-face or free-play-style interactions (Murray, Fiori-Cowley, & Cooper 1996). However, it may be that mothers with depressive symptoms find responding sensitively during times of stress more challenging (Laurent, Ablow, & Measelle, 2011). Thus, an examination of maternal behavior and maternal postnatal depressive symptoms in the context of a stressful mother–infant interaction may provide a clearer view of conditions associated with low maternal sensitivity. Tronick, Als, Adamson, Wise, and Brazelton (1978) provide a clearer view of conditions associated with low maternal sensitivity. Tronick, Als, Adamson, Wise, and Brazelton (1978) and Tronick and Cohn (1989) developed the Still-Face Paradigm (SFP) to explore the dynamics of mother–infant interactions under conditions of stress. The current study examined and coded maternal behaviors associated with low sensitivity during the reunion phase of the SFP to gain an understanding of the associations between postnatal depressive symptoms and low sensitivity during times of parenting stress.

METHOD

Sample

Participants were recruited from local childbirth education classes, hospitals, and public assistance programs during their third trimester of pregnancy (mean duration of gestation at the time of recruitment = 33.1 weeks, SD = 1.4). At the time of prenatal recruitment, 100 participants enrolled in the study. To sample for women at relative risk for postnatal depressive symptoms, mothers’ depressive symptoms were assessed prenatally, at the time of recruitment, using the Beck Depression Inventory-II (BDI-II; Beck, 1996). The BDI-II is a 21-question, self-report inventory of maternal depressive symptom severity (Beck, 1996). At the time of enrollment, 30 mothers (30%) met the clinical criteria for depression (a score of >19), based on BDI-II assessment scores. In accordance with requirements of the local Institutional Review Board, all women endorsing suicidal ideation or items related to feelings of hopelessness or helplessness were given referrals to local mental health agencies. Furthermore, all women enrolled in the study were given information on local parenting classes and public assistance programs.

Between the prenatal assessment and the 5-month postnatal assessment, 5 participants (3 meeting criteria for depression and 2 controls) chose not to take part in the longitudinal portion of the study. Thus, participants were 95 first-time mothers, seen in the laboratory when their infants were 5 months of age. Participants ranged in age from 18 to 38 years (M = 24.5, SD = 4.68). The majority of the sample identified as White/not of Hispanic origin (77.8%), and 40.5% of the sample had annual household incomes below $20,000 (M = $9,630, SD = $1,130). The sample was predominantly unmarried or never married (56.2%).

Procedure

Upon arrival at the laboratory, women were seated, and written informed consent was obtained. Questionnaire booklets, including the EPDS, basic demographic information, and a number of mental health and parenting measures, were completed by participants in their homes 1 week prior to the 5-months’ postpartum laboratory session. Questionnaires were returned at the time of this lab session. At the beginning of this session, participants and their infants were positioned approximately 2.5 ft from a 13.5-in. monitor and silver–silver chloride electrodes were attached as described later. Resting baseline RSA levels were recorded while participants viewed a 2-min, neutral Baby Einstein (Clark & Dunham, 2002) film clip, which included music and moving shapes. A film-clip baseline was selected based on prior research indicating that minimally demanding tasks such as watching an emotionally neutral film clip result in more stable estimates of physiological functioning than do no-task baselines. These neutral baselines also account for the physiological response of orienting and attending (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992). Participants then completed the SFP, described next.

SFP

The SFP is a 6-min laboratory procedure used to explore the dynamic aspects of caregiver–infant interactions (Tronick et al., 1978). The experimental procedure consists of three episodes: a 2-min face-to-face play interaction; a 2-min period in which the mother holds a still, flat-affect face; and a 2-min interaction in which the mother attempts to reengage her infant. The still-face portion of the procedure is believed to mimic a depressive episode, and it has been shown to be distressing to the infant. The reengagement episode was coded for maternal sensitivity, as it is believed that this episode would require the greatest level of sensitive responding to the infants’ behavioral and emotional cues due to increased infant distress associated with the still face. All interactions were videotaped and coded, as described next.

Maternal Sensitivity Coding

Videotapes were coded using the Global Coding Scheme of Mother–Infant Interactions, a well-validated coding measure of maternal sensitive behavior that has been shown to be predictive of numerous child cognitive and socioemotional outcomes (Murray & Cooper, 1996). This measure was chosen because it primarily assesses characteristics of maternal responsiveness to infant communicative cues, which is in line with our hypothesis that low baseline RSA levels may be associated with the availability of fewer emotional, physiological, and psychological resources with which to respond sensitively to infants’ cues.

Videos were coded by a trained researcher who was unaware of the mothers’ numbers of depressive symptoms. Reengagement episodes were coded on three dimensions of maternal warmth,
Responsiveness, and sensitivity using a series of scales with ratings of 1 (cold, unresponsive, or insensitive) to 5 (warm, responsive, or sensitive). Composite scores of overall maternal sensitivity were calculated by summing the participants’ scores across each of the domains for the entire 2-min episode, as proposed by Murray and Cooper (1996).

To establish reliability, 30 cases, including participants both with and without depressive symptoms, were rated independently by a second blinded researcher. Intraclass correlations for each of the global rating domains were high (maternal warmth: 0.89, maternal responsiveness: 0.93, and maternal sensitivity: 0.92).

EPDS

The EPDS (Cox et al., 1987) is a 10-item, self-report scale used to measure depressive symptoms during the postnatal period. Research has demonstrated that there are limitations associated with the use of several well-established measures of depressive symptoms during the perinatal period. Specifically, Cox et al. (1987) suggested that both the Anxiety and Depression Scale (SAD) (Bedford & Foulds 1978) and the Center for Epidemiological Studies Depression Scales (CES-D) (Radloff 1977) emphasize somatic depressive symptoms, which may be associated with both the pregnancy period and the transition to motherhood rather than depression specifically. The EPDS overcomes these limitations by focusing on cognitive depressive symptoms such as thoughts of sadness, worthlessness, and inappropriate guilt, which have been shown to be associated uniquely with depression in the perinatal period (Cox et al., 1987).

The EPDS uses a scale from 0 (no, not at all) to 3 (yes, quite a lot). The items are summed to create a total score ranging from 0 to 30. Clinical cutoff scores range between 9 and 13; however, for the purpose of this study, a continuous scoring system of depressive symptoms was used. The EPDS has been shown to have good user acceptability when administered as a questionnaire (92% response rate). The EPDS also has been shown to have acceptable sensitivity (79%) and specificity (85%) (Cox, Chapman, Murray, & Jones, 1996).

Physiological Data Collection

Physiological responses during resting baseline were collected using a nine-channel bioamplifier (Model JCA-09). During the baseline, physiological channels were sampled continuously with low-pass filtering at 1000 Hz. High-pass filtering was recorded at 0.03 Hz. Silver–silver chloride film electrodes were used and placed axially on the left and right rib at the elevation of the heart. A ground electrode was place on the left collarbone to monitor for physiological channels were sampled continuously with baseline, physiological channels were sampled continuously with low-pass filtering at 1000 Hz. High-pass filtering was recorded at 0.03 Hz. Silver–silver chloride film electrodes were used and placed axially on the left and right rib at the elevation of the heart. A ground electrode was place on the left collarbone to monitor for A ground electrode was place on the left collarbone to monitor for R–R wave artifacts related to movement.

To obtain heart rate data, the interbeat interval (IBI) was measured as the interval between successive R-waves in the electrocardiogram (ECG). IBI data were manually edited for R–R wave interval outliers associated with movement artifacts and/or ectopic myocardial activity. Heart periods (HP) were edited offline with IBIEDIT software (James Long Company, 1999). The edited R-wave series was converted to a prorated HP series with an equal time interval of 125 ms between each prorated HP. Mean HP was calculated from the prorated HP series.

RSA was computed using respiration rates, edited IBI, and HP data, as outlined by Grossman, van Beek, and Wielen (1990), and the difference between the minimum IBI during inhalation and the maximum IBI during exhalation was used to calculate RSA using James Long Company software (1999).

Data Reduction of Physiological Data

Each participant’s RSA data were inspected both graphically and computationally for outliers. Six participants were excluded from the full analyses: Two participants had equipment problems resulting in a loss of ECG/RSA acquisition, and 4 participants had baseline RSA data lying further than 3 SD from the mean. Therefore, a total of 89 participants were used in the full data analysis.

To reduce the RSA data, the mean of each 5-s epoch was calculated across the 2 min of the baseline episode. Correlations among of the 5-s epochs were high (r=0.74), indicating little variability in RSA across the baseline period. Therefore, the mean baseline level of RSA was computed for each participant across the full 2-min period, and these values were used in all subsequent analyses.

RESULTS

Descriptive and Preliminary Analyses

Descriptive statistics for the primary variables of interest are presented in Table 1. On average, mothers’ levels of depressive symptoms on the EPDS were in the nonclinical range, although 31.5% of the sample (n = 30 women) scored above the clinical cutoff of 9. The mean baseline RSA value for the sample was 6.80 (SD = 4.81) (see Table 1). Although difficult to characterize this level of RSA, it is comparable to the means reported for other samples comprised of females with and without depressive symptoms (e.g., Rottenberg et al., 2002). In terms of maternal sensitivity, the mothers in this sample generally scored toward the middle (M = 9.15, SD = 2.67) on a scale ranging from 5 to 15 (5 is highly cold, unresponsive or insensitive and 15 is highly warm, responsive, and sensitive). Qualitatively, this coincided with low to moderate sensitivity and was similar to the levels of maternal sensitivity reported by Murray, Fiori-Cowley et al. (1997) in a sample of both.
Predicting Maternal Sensitivity From Postnatal Depressive Symptoms and Baseline RSA

Regression analyses, both univariate and multivariate, were used to examine the main and interactive effects of depressive symptoms and baseline RSA levels on maternal sensitivity. Two initial models were tested (Table 2a) to examine the univariate contributions of depressive symptoms and baseline RSA levels to individual differences in maternal sensitivity. As shown in Table 2a, both depressive symptoms and baseline RSA accounted for small, but significant, amounts of variance in maternal sensitivity in their own models, $R^2 = 0.074$, $p < .001$, and $R^2 = 0.042$, $p < .05$, respectively. Specifically, higher levels of depressive symptoms were associated with less maternal sensitivity whereas higher baseline RSA levels were associated with greater maternal sensitivity.

Model 3 tested the unique, nonoverlapping contributions of both predictors. Overall, the model accounted for a significant amount of the variance in maternal sensitivity, $R^2 = 0.106$, $p < .01$. However, as shown in Table 2b, only maternal depressive symptoms, $\beta = −0.254$, $p < .01$, provided unique prediction whereas baseline RSA levels provided trend-level prediction, $\beta = 0.177$, $p = .08$.

A final regression model tested the potential moderating effect of maternal depressive symptoms on the association between baseline RSA and maternal sensitivity scores (Model 2). In this model, maternal sensitivity was regressed on baseline RSA levels, maternal depressive symptoms, and the mean-centered interaction of maternal depressive symptoms and baseline RSA levels. As shown in Table 2c, although the overall model was significant, $R^2 = 0.109$, $p < .05$, the interaction did not account for a significant portion of the variance in maternal sensitivity, $\beta = −0.118$, $p = .78$; thus indicating that mothers’ postnatal depressive symptoms did not moderate the association between baseline RSA levels and maternal sensitivity.

### DISCUSSION

The results of this study are consistent with prior work showing that higher numbers of postnatal depressive symptoms and lower baseline RSA levels are both significant predictors of lower maternal sensitivity. Although numerous studies have found this association in the context of maternal behavior during free-play-style tasks (Campbell et al., 2004; Murray, Fiori-Cowley et al., 1996; Murray, Hipwell, & Hooper, 1996; Pederson et al., 1990), few studies, if any, have sought to determine whether this association exists with depressed and nondepressed mothers. In terms of the distribution, 46.3% of the women ($n = 44$) fell in the 5 to 8 or lower sensitivity range, 26.3% ($n = 25$) fell in the 9 to 11 or moderate sensitivity range, and 27.4% ($n = 26$) fell in the 12 to 15 or high maternal sensitivity range.

Prior to running our central analyses, maternal age, ethnicity, income, educational level, marital status, infant gender, infant distress during the Still-Face episode, and maternal resting HR were tested as predictors of maternal sensitivity. None of these variables was a significant predictor of maternal sensitivity; thus, all were removed from further analysis to reduce the potential for model oversaturation and collinearity.

Next, the correlation between mothers’ depressive symptom scores on the EPDS and baseline RSA levels was examined to determine whether the prerequisites for mediation versus moderation were indicated. Contrary to prediction, mothers’ depressive symptoms and baseline RSA levels in these data, and depressive symptoms whereas other studies have reported no statistically significant associations between baseline RSA levels produced mixed results; some studies have reported small, but significant, associations between baseline RSA levels and depressive symptoms whereas other studies have reported no associations at all. Given the lack of association between mothers’ depressive symptoms and baseline RSA levels in these data, this precluded tests of mediation (Model 1) while providing the necessary preconditions for testing moderation (Model 2).

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**TABLE 2. Hierarchical Regression Predicting Maternal Sensitivity From Postnatal Depressive Symptoms and Baseline RSA Levels**

<table>
<thead>
<tr>
<th>Criterion/Predictors (step)</th>
<th>$\beta$</th>
<th>B</th>
<th>$R^2$</th>
<th>$R^2$</th>
<th>† 95%</th>
<th>CI of $B_t$</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Maternal Sensitivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Depressive symptoms</td>
<td>−0.273</td>
<td>−0.313</td>
<td>0.074</td>
<td>–</td>
<td>(−0.548, −0.078)</td>
<td>−2.645**</td>
</tr>
<tr>
<td>2. Baseline RSA level</td>
<td>0.211</td>
<td>11.293</td>
<td>0.042</td>
<td>–</td>
<td>(−0.010, 22.597)</td>
<td>1.989*</td>
</tr>
<tr>
<td>(b) Maternal Sensitivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Depressive symptoms</td>
<td>−0.254</td>
<td>−0.291</td>
<td>0.074</td>
<td>–</td>
<td>(−2.471, 0.015)</td>
<td>−2.471**</td>
</tr>
<tr>
<td>2. Baseline RSA level</td>
<td>0.177</td>
<td>9.724</td>
<td>0.106</td>
<td>0.032</td>
<td>(−1.459, 20.907)</td>
<td>1.729</td>
</tr>
<tr>
<td>(c) Maternal Sensitivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Depressive symptoms</td>
<td>−0.203</td>
<td>−0.232</td>
<td>0.074</td>
<td>–</td>
<td>(−0.705, 0.241)</td>
<td>−0.977</td>
</tr>
<tr>
<td>2. Baseline RSA level</td>
<td>0.285</td>
<td>15.627</td>
<td>0.106</td>
<td>0.032</td>
<td>(−27.458, 58.712)</td>
<td>0.721</td>
</tr>
<tr>
<td>3. Depressive Symptoms × Baseline RSA Level</td>
<td>−0.118</td>
<td>−0.879</td>
<td>0.107</td>
<td>0.001</td>
<td>(−7.074, 5.316)</td>
<td>−0.282</td>
</tr>
</tbody>
</table>

RSA = respiratory sinus arrhythmia mean across a 2-min baseline.

* $p < .05$, two-tailed. ** $p < .01$, two-tailed.

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behavior observed during a stressful mother–infant interaction. Individuals with depressive symptoms often experience difficulties coping and regulating their emotions during times of stress (Laurent, Ablow, & Measelle, 2011). However, women with depressive symptoms can look reasonably effective as parents during interactions that pose little challenge (Laurent et al., 2011). Thus, it may be that mothers with elevated depressive symptoms are less able to regulate their emotional responses when caring for a distressed infant or when they are distressed themselves. As such, these mothers may be less sensitively attuned to their infants’ distress cues and may have fewer cognitive, emotional, and physical resources to devote to soothing their infants, perhaps because they are focused on soothing themselves (Laurent & Ablow, 2012).

On the whole, the women in this study did not experience clinical levels of depressive symptoms, with over two thirds of the sample falling in the low to moderate range of severity. However, even with mild to moderate depressive symptoms, nearly half of all mothers scored in the lowest third on our composite measure of maternal sensitivity. Thus, although our sample was only experiencing mild to moderate postnatal depressive symptoms, the association between women’s depressive symptoms and less sensitive maternal behavior persisted. Subclinical levels of depression have been implicated in impaired parenting (Goodman & Gotlib, 2002), and there also is evidence that sustained exposure to chronic low levels of maternal depressive symptoms my negatively impact infants’ stress biology (Laurent et al., 2011). As such, a longitudinal design would best test the effects of milder maternal depression and depressive symptoms which are nevertheless associated with low maternal sensitivity on childhood outcomes.

Baseline RSA levels also were a significant predictor of maternal sensitivity. Consonant with theory and prediction, higher levels of baseline RSA were associated with greater maternal sensitivity. Higher basal RSA levels are thought to support the capacity for active engagement with and attention to the demands of the environment (Beauchaine, 2001). Because individuals with high baseline RSA levels are predisposed in general toward more effective regulatory capacities, it may be that these same individuals also are better able to sustain attention and engage with environmental demands during times of stress. Individuals with low baseline RSA levels, by contrast, may be more likely to respond to stressors with a flight-or-fight response, given their predisposition toward less regulated PNS activity while at rest. As maternal sensitivity includes orientation toward and engagement with infants’ communicative cues, it follows that mothers with high levels of baseline RSA would be better able to respond to their infants’ cues and emotional bids. Conversely, mothers with low baseline RSA levels might be more likely to respond to their infants’ distress by missing or avoiding their emotional bids or by responding with hostility and aggression, as is the case with flight-or-fight responding.

Although predictive in the respective models, baseline RSA was not a significant, unique predictor of maternal sensitivity when modeled simultaneously with maternal depressive symptoms. One potential explanation for this finding is that very little research has examined the nature of baseline RSA levels specifically during the postnatal period. Thus, it may be that other factors such as weight gain, changes in sleep patterns, and changes in hormone levels may influence baseline RSA levels, resulting in a weaker association between baseline RSA levels and sensitivity during the postnatal period. In future studies, it will be necessary to examine changes in baseline RSA levels across the perinatal period as well as how these changes are associated with both maternal sensitivity and depressive symptoms.

The lack of association between mothers’ postnatal depressive symptoms and baseline RSA levels precluded a formal test of mediation, which had been our initial prediction. However, this finding is not entirely inconsistent with the literature: Several studies have reported no association between basal levels of RSA and depressive symptoms (Carney et al., 1995; Lehrer et al., 1997; Moser et al., 1998). This lack of association may be explained partially by the cognitive nature of the symptoms assessed by the EPDS. Other studies examining the association between baseline RSA levels and depressive symptoms have included both cognitive and physical symptoms (Carney et al., 1995; Dalack et al., 1990; Licht et al., 2008; Rechlin et al., 1994; Rottenberg, Clift, Bolden, & Salomon 2007; Rottenberg et al., 2001). Thus, it could be that physical depressive symptoms are more strongly associated with baseline indices of autonomic functioning, which itself is largely influenced by fairly stable characteristics such as weight, cardiovascular health, and diabetes (Berntson, Normen, Hawkley, & Cacioppo, 2008).

The heterogeneity of mothers’ cognitive depressive symptoms might be a different, but related, explanation. As noted earlier, Rottenberg et al. (2002) found that RSA was positively associated with cognitive depressive symptoms, but negatively associated with suicidal ideation. It may be that individuals who tend toward withdrawn forms of depressive symptoms have higher baseline RSA levels whereas individuals with emotional instability and impulsivity have lower baseline RSA levels. In addition, it may be that more chronic or severe depressive symptoms correlate more strongly with baseline RSA. Women in the present sample generally reported subclinical levels of postpartum depressive symptoms. Thus, it is possible that the milder form of depression report by mothers in the present study precluded associations with a more stable measure of autonomic functioning. As such, in future studies, it will be important to more broadly examine the features of depressive symptoms to test possible associations with baseline RSA and to more fully examine the proposed mediation model.

The results of this study also were not supportive of the hypothesis that depressive symptoms would moderate the effect of baseline RSA on maternal sensitivity. That is, mothers’ postpartum depressive symptoms did not help to determine for whom baseline RSA served as a risk factor for insensitive parenting. One plausible explanation is statistical; namely, the size of our sample and corresponding power limitations, which make the detection of significant interactions difficult in linear regression models (Cohen, 1992). In all likelihood, however, the small effect size of the interaction term points to other possibilities. Although we are making a case for the importance of studying these associations
while mothers and infants are engaged in stressful interaction, it could be that individual differences in depressive symptoms moderate the association between baseline RSA and maternal behavior during nonstressful interactions. Laurent et al. (2011) found that the resting-state cortisol levels of depressed and nondepressed mothers were related to their prestress maternal behavior, yet peak cortisol levels predicted behavioral difficulties during and following a stressful separation from their infant only for depressed women. In our own attempts to further this research, we will include baseline RSA, as well as RSA reactivity, during and following stressful mother–infant interactions to determine if and how depressive symptoms moderate vagal associations with maternal behavior.

It is important to consider several limitations when interpreting the findings of this study. First, a larger sample with greater racial, ethnic, and socioeconomic diversity is desirable to generalize the findings and to examine subgroups of women with varying types of depressive symptoms and a greater range of maternal behaviors. The sample also was limited with respect to the number of women with clinically significant depressive symptoms. The EPDS does not yield a formal diagnosis of postnatal depression; yet, the sample as a whole scored below the suggested clinical range. The EPDS also does not allow for the examination of depression subtypes, and as such, this study can generalize only to new mothers experiencing postnatal depressive symptoms rather than to clinical populations.

An additional limitation of this research is that baseline RSA was the only measure of autonomic functioning tested. Thus, inferences about PNS functioning must be made without knowledge of associated sympathetic nervous system (SNS) functioning as well as PNS reactivity and recovery. In future studies, it will be important to examine multiple features of PNS and SNS functioning, as prior research has shown that cardiac autonomic balance and cardiac autonomic regulatory capacity may provide additional information related to health and behavioral outcomes (Berntson et al., 2008).

Despite these limitations, this study revealed that both maternal depressive symptoms and physiological predispositions toward emotion dysregulation serve to decrease maternal sensitive behavior during stressful mother–infant interactions. These findings are important on several levels. Specifically, the association between maternal depressive symptoms and impaired maternal sensitivity persisted even when the depressive symptoms were few or mild. In addition, while other studies have shown that autonomic reactivity is associated with parenting behavior, this study revealed that traitlike predispositions toward emotion dysregulation, indexed by homeostatic autonomic nervous system functioning, also are predictive of sensitive parenting. These results provide further support that mothers with depressive symptoms and mothers with emotion dysregulation attend to their own regulatory needs rather than to those of their infants, which may result in negative consequences for infant socioemotional development. Continuation of this work may help to elucidate the etiology of maladaptive mother–infant interactions as well as add specificity to the development of interventions designed to enhance sensitive parenting behavior.

REFERENCES


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