ABSTRACT: Electroencephalogram (EEG) patterns may reflect a vulnerability to depression. In an effort to understand their earliest origin, we examined their stability and consistency and their associations with perinatal depressive symptoms. Depressive symptoms were measured prospectively throughout the perinatal period in 83 women with histories of depression and/or anxiety. Infant’s EEG was recorded during baseline, feeding, and play at 3 and 6 months of age. Prenatal and postpartum depressive symptoms interacted significantly to predict 3- and 6-month-olds’ EEG asymmetry scores. Asymmetry scores were consistent across contexts, except from baseline to feeding and play at 6 months, and stable across ages, except during feeding. Changes in depressive symptoms across ages were not associated with changes in infant EEG. Findings highlight the importance of considering both prenatal and postpartum depressive symptoms in the prediction of infant EEG, as well as the need to consider context to understand stability of infant EEG patterns.

INTRODUCTION

Electroencephalogram (EEG) patterns of right frontal activity being higher than left frontal activity have been found to be associated with emotion in general and depression more specifically. The same pattern of greater relative right frontal EEG activation, operationally defined as greater relative right frontal EEG asymmetry, that has been found in adults with depression (Henriques & Davidson, 1990, 1991) has also been found in infants of prenatally (Field, Diego, Hernandez-Reif, Schanberg, & Kuhn, 2002; Field et al., 2004) and postnatally (Dawson et al., 2001; Field, Fox, Pickens, & Nawrocki, 1995) depressed mothers, although we found no published reports of possible additive or interactive associations across pre- and post-natal depression. Moreover, these asymmetry scores in infants predict levels of inhibition at age 4 (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001). EEG asymmetry scores in infants have been found to be independent of current affective state (Jones, Field, Fox, Lundy, & Davalos, 1997) and consistent across contexts, but it is unclear when this consistency first emerges in development (Dawson et al., 2001; Dawson, Frey, Panagiotides, & Osterling, 1997). Also, infant asymmetry scores from baseline EEG recordings have been found to be stable over time, albeit with small effect sizes (Diego, Field, Jones, & Hernandez-Reif, 2006; Jones, Field, Davalos, & Pickens, 1997), and we found no reports on the stability of asymmetry scores in contexts involving maternal interaction, such as play or feeding. This is important, as infant EEG patterns recorded during interactions with their mothers may reflect the quality of the interaction at the moment as well as the infant’s history of interaction with a depressed mother (Jones, Field, Fox, et al., 1997). More broadly, the ability to determine the earliest time when infants demonstrate stable right frontal asymmetry may have clinical implications that inform the development of screening guidelines for identifying vulnerable individuals who might most benefit...
from preventive intervention. Alternatively, knowing the predictors of naturally occurring change in infant EEG asymmetry may reveal exacerabring or corrective factors, which could be targets of intervention.

Although more information is needed, these findings suggest that frontal EEG asymmetry may reflect, even in infancy, a trait marker of vulnerability to depression; however, the associations of infant EEG asymmetry scores over time being small, for the most part, suggests that infants vary from each other in the stability of these scores. Thus, it is critical to determine the predictors of EEG asymmetry scores at distinct times and well-characterized contexts in infancy as well as the predictors of stability or variability. The aim of the current study was to explore the relation between maternal prenatal and postpartum depressive symptoms and infant EEG patterns over the first several months of life. In particular, this study adds to the literature in several key ways: by examining how maternal prenatal and postpartum depressive symptoms might have an additive or interactive effect on infant EEG asymmetry scores at 3 and 6 months of age, as well as exploring the stability of infant EEG asymmetry scores over this time and the consistency across contexts. A long-term goal of this line of research is to distinguish among four subsets of infants: those who start out early on negative trajectories of development and continue on this pathway relative to those who may “recover,” and still others in whom a negative trajectory emerges later in infancy relative to “resilient” infants who never evidence the asymmetry pattern of concern.

EEG and Emotion

EEG patterns within the frontal lobes have been theorized to be indicative of emotion regulation at the physiological level, based on their association with two distinct behavioral systems. Fox et al. (1991) proposed a model that includes separate behavioral approach and withdrawal systems that are present at birth, with approach including joy, interest, and anger, and withdrawal including distress, disgust, and fear. As these alternative behavioral systems are thought to be associated with different frontal lobes, measures of EEG have been used to examine the association between these behavioral systems and brain activation. Findings revealed that greater relative left frontal activation was associated with approach behavior and positive affect, whereas greater relative right frontal activation was associated with withdrawal behavior and negative affect. Thus, EEG patterns appear to reflect individual differences in emotion regulation (Fox, 1991, 1994).

EEG as a Biomarker of Vulnerability to Depression

In addition, greater relative right frontal EEG activation has been suggested to reflect a vulnerability to depression. In several studies of adults, greater relative right frontal EEG asymmetry has been found in those diagnosed as depressed or those with higher levels of depressive symptoms compared to nondepressed individuals, who displayed greater relative left frontal EEG activation (Diego, Field, & Hernandez-Reif, 2001; Field et al., 1995; Henrichs & Davidson, 1991; Jones, Field, Fox, et al., 1997). This association between depression and right frontal asymmetry remains even beyond a depressive episode and is unrelated to self-reported emotional state (Gotlib, Ranganath, & Rosenfield, 1998; Henriques & Davidson, 1990), suggesting that EEG patterns may reflect a trait-like marker of depression in adults. Similar to these findings in adults, a few studies have reported evidence for the state independence of EEG patterns in 1-month-old and 13- to 15-month-old infants of depressed and nondepressed mothers (Dawson et al., 1997, 2001; Jones, Field, Fox, et al., 1997). Taken together, these findings yield support for EEG patterns reflecting a trait-like marker independent of affective state, suggesting a vulnerability marker in infants for the later development of depression.

Consistent with the notion of EEG patterns as a vulnerability marker for the later development of depression, Fox et al. (2001) found that infants with greater relative right frontal EEG asymmetry at both 9 and 14 months of age displayed stable behavioral inhibition over the first 4 years of life. This is in contrast to infants who showed less relative right frontal EEG asymmetry at 9 months of age and greater relative left frontal EEG asymmetry at 14 months of age, as inhibition scores for these infants declined over the first 4 years of life (Fox et al., 2001). These findings suggest an association between infant EEG patterns and later behavioral inhibition, which is an early temperament marker of vulnerability for depression and other internalizing problems (Biederman et al., 2001; Muris, Merckelbach, Wessel, & van de Ven, 1999). Thus, individual differences in EEG asymmetry appearing as early as 9 months of age may reflect a vulnerability to the later development of depression.

Infant EEG and Maternal Depression

Given the support for the predictive importance of EEG asymmetry scores as early as infancy, it is essential to identify predictors of these scores in infants. One such predictor that has been examined is maternal depression, as depression in mothers is known to confer
risk for the development of psychopathology on offspring (Goodman et al., 2011). Consistent with the Goodman and Gotlib model (1999), abnormal EEG patterns may describe one developmental pathway through which this risk is transmitted.

The association between maternal depressive symptoms or diagnosed depression in the postpartum period and infant EEG patterns has been particularly well documented. In one study, significantly more (10 of 17) 3- to 6-month-old infants of mothers with concurrently high levels of depressive symptoms showed greater relative right frontal EEG asymmetry during a neutral condition compared to infants of mothers with low levels of depressive symptoms (3 of 15; Field et al., 1995). This finding has been replicated in infants as early as 1 week of age, and extends to 1 month olds, 3–6 months olds, and 13–15 months olds, with infants of mothers currently diagnosed with depression or high on depressive symptoms showing greater relative right frontal EEG asymmetry compared to infants of nondepressed mothers or those low on depressive symptoms (Dawson et al., 2001; Diego et al., 2006; Jones, Field, Davalos, et al., 1997; Jones, Field, Fox, et al., 1997). Yet these studies are limited in two ways. First, these studies solely relied on concurrent measures of maternal depressive symptoms, rather than prospective measures of depressive symptoms, which would capture the infants’ longitudinal exposure since birth. Second, the majority of these findings were from studies that sampled predominately ethnic minority mothers of low to middle socioeconomic status (SES), and some samples included high percentages of teenage mothers. Thus, the findings may at least partly reflect infants’ exposure to general stressors associated with low SES or teenage parenting, making the contribution of maternal depression less clear, an idea which, to our knowledge, has not been tested. Therefore, it is important to examine the relation between infant EEG and prospectively studied maternal depression in more broadly middle SES samples.

In addition to the findings of associations between infant EEG asymmetry scores and concurrent (postpartum) maternal depression, maternal prenatal depression is also suggested to influence offspring EEG (Goodman & Gotlib, 1999). Maternal prenatal depression may impact infants’ psychophysiology through variations in the intrauterine environment (Dawson et al., 2001). A few studies have now shown an association between prenatal depressive symptom severity and single measures of newborn or childhood EEG. In two studies of newborns, those with greater relative right frontal EEG asymmetry had mothers with higher prenatal depressive symptoms compared to those with greater relative left frontal EEG asymmetry (Field et al., 2002, 2004). At 14 months of age, children of mothers who were depressed prenatally exhibited greater relative right frontal EEG asymmetry, while at 3½ years of age, these children showed reduced EEG activity across all scalp regions (Dawson et al., 2003). It is important to note, however, that maternal prenatal depression was measured retrospectively in the Dawson study; retrospective measures of depression are known to be unreliable (Newport et al., 2008). Also, we found no studies other than Dawson et al. that reported having examined associations between maternal prenatal depression and infant EEG at any points later than the newborn time period. Thus, very little is known about associations between prenatal depression and infant EEG beyond the newborn time period. In this study, we test the theory that depression during pregnancy would influence infants’ psychophysiology (Dawson et al., 2001; Goodman & Gotlib, 1999) by examining the extent of prenatal depression’s prospective influence over a longer span of infant development, the first 6 months of life.

Moreover, despite the support for both prenatal depression and postpartum depression predicting infant EEG, no studies were found to have examined the possible additive or interactive effects of pre- and postnatal depression on infant EEG asymmetry scores. Doing so is critical for several reasons. First, prenatal depression is the strongest predictor of postpartum depression (O’Hara & Swain, 1996) and thus many infants are dually exposed. Studies limited to only prenatal or only postpartum depression on infant EEG miss the potential contribution of dual exposure. Second, despite prenatal depression being the strongest predictor of postpartum depression, some women have depression that remits early in the postpartum period (Campbell, Cohn, & Meyers, 1995; Field, 1992). Thus some but not all infants of prenatally depressed mothers will also be exposed in the postpartum. Consistent with the Goodman and Gotlib (1999) model, the effects of maternal prenatal and postpartum depression on infant EEG asymmetry scores could be additive, such that higher levels of depressive symptoms at both time points predict greater relative right frontal EEG asymmetry in infants. Alternatively, they could be interactive in that, for example, maternal prenatal depression is only associated with greater relative right frontal EEG asymmetry in infants when the mother is also depressed during the postpartum period. That is, consistent with theories on biological sensitivity to the environment (Belsky, Bakermans-Kranenburg, & van Ijzendoorn, 2007), maternal elevated postpartum depression may only be associated with infant EEG asymmetry scores in the context of having been exposed to higher prenatal depression. Thus knowledge of infants’ exposures both pre- and
postnatally might enhance the prediction of infant EEG asymmetry scores.

**Consistency of Infant EEG Patterns**

Further support for EEG patterns as an index of vulnerability to the later development of depression would come from evidence of these patterns being consistent across contexts. The vast majority of the literature reports on infant EEG patterns during baseline conditions alone. In the two studies found to have included other contexts, consistency of infant EEG patterns was supported. Thirteen- to 15-month-old infants of depressed mothers had greater relative right frontal EEG asymmetry compared to infants of nondepressed mothers not only during a baseline condition, but also during two social interaction contexts (one with the mother and one with the experimenter; Dawson et al., 1997, 2001). These results support the idea that infant EEG asymmetry scores are consistent across contexts. However, we found no studies examining consistency in the EEG scores of younger infants. Such findings would yield support for the earlier emergence of a trait-like vulnerability.

**Stability of Infant EEG Patterns**

At least as important as cross-situational consistency, it is important to understand whether infant EEG patterns are stable and the possible role of the ongoing course of maternal depression in predicting stability. Evidence is beginning to emerge on the predictive value of stable infant EEG patterns, yielding further support for infant EEG patterns representing a vulnerability to the development of depression. Specifically, infants who showed right frontal asymmetry stably from 10 to 24 months of age were rated by their mothers as higher in internalizing behavior problems at 30 months of age; in contrast, those who showed stable left frontal asymmetry were rated by their mothers as higher in externalizing behavior problems (Smith & Bell, 2010). Whereas earlier studies had found support for the predictive value of EEG scores at specific time points, these findings expand on this idea by demonstrating the predictive value of the stability of these scores across ages.

Thus, it is important to know the stability of infant EEG asymmetry over time. Researchers have found evidence for the stability of EEG scores over time in both infants of mothers with high and low depressive symptoms. Findings revealed moderate to large correlations between baseline frontal EEG asymmetry scores recorded within a few days after birth and again at 3–6 months of age (Diego et al., 2006) and between 1 and 3 months of age (Jones, Field, Fox, et al., 1997). In addition, nearly all infants (7 out of 8) who showed right frontal asymmetry at 3–6 months showed the same patterns at 3 years of age (Jones, Field, Davalos, et al., 1997). However, the previous studies only examined stability of baseline EEG scores; the stability of asymmetry scores across early infancy in maternal-interaction contexts remains unexplored. Demonstrating stability of asymmetry scores in maternal-interaction contexts would lend further support for infant EEG scores being a trait marker of vulnerability.

Despite accumulating evidence that infant EEG scores are stable over time, there is evidence for individual differences in stability of baseline EEG scores. Aside from one finding of a large positive association between baseline frontal EEG asymmetry scores across ages (Diego et al., 2006), other findings suggest only a moderate association, lending support to this idea of individual differences in stability. In Smith and Bell (2010), for example, over half of the infants demonstrated a change in their EEG patterns from 10 to 24 months of age. Diego et al. (2006) found that infants of mothers with high depressive symptoms shifted in baseline EEG asymmetry scores from the neonatal period to 3–6 months of age, with the direction of the shift differing based on maternal self-reported behavioral approach or withdrawal (Diego et al., 2006). However, maternal depressive symptoms levels were only measured once, within a few days after birth. Thus the question of whether change in maternal depression in the postpartum might have been related to the changes in infant baseline EEG patterns remains unanswered. In the current study, we build on these efforts to understand why some infants show stability of EEG scores over time and others do not by examining changes in maternal depression symptoms as a predictor of stability in baseline EEG patterns across early infancy.

**Current Study**

The current study aimed to address unanswered questions about the relation between maternal perinatal depressive symptoms and infant EEG asymmetry scores in a prospective study. Depressive symptoms were measured in pregnancy and through 6 months postpartum and EEG was measured at both 3 and 6 months of age in baseline and in two maternal interaction contexts (play and feeding). These ages were chosen given support for long-term EEG stability from as early as 3 months of age to early childhood (albeit restricted to baseline EEG; Jones, Field, Davalos, et al., 1997) at the same time that EEG shifts from 3 to 6 months have been demonstrated (Diego et al., 2006), suggesting
that some, but not all infants of perinatally depressed mothers may be on a negative trajectory from these early ages. However, factors associated with such shifts are not well understood. We hypothesized the following: (1) maternal postpartum depressive symptoms would be associated with infants’ greater relative right frontal EEG asymmetry during baseline at both 3 and 6 months of age; (2) maternal prenatal and postpartum depressive symptoms would either additively predict infant baseline frontal EEG asymmetry scores at 3 and 6 months of age, such that higher levels of both would predict greater relative right frontal EEG asymmetry in infants, or interactively, such that greater prenatal exposure would predict greater relative right frontal EEG asymmetry specifically among infants whose mothers had more (or increasing) depressive symptoms postnatally; (3) infant frontal EEG asymmetry scores would be consistent across contexts (baseline, feeding, and play) at both 3 and 6 months of age; (4) infant frontal EEG asymmetry scores would be stable from 3 to 6 months of age not only in baseline but also (5) in maternal-interaction contexts (feeding and play); and (6) the relation between infant baseline frontal EEG asymmetry scores at 3 and 6 months of age would be moderated by changes in maternal depressive symptom levels between those two time points. We restricted our hypothesized role of changes in maternal depressive symptoms in predicting changes in EEG asymmetry to baseline EEG, rather than EEG during maternal interactive contexts, given the primary focus on baseline in studies of associations between maternal depressive symptoms and infant EEG asymmetry as well as in studies of the stability of infant EEG. Conversely, less is known about EEG during maternal interactive contexts and thus tests of this hypothesis in those contexts were not justified. We tested these hypotheses in women who had been depressed and/or anxious prior to their pregnancy in order to enhance the likelihood that infants would be exposed to maternal depressive symptoms either during the prenatal period, during the postpartum period, or both.

METHODS

Participants

Data were collected as part of the longitudinal study Perinatal Stress and Gene Influences: Pathways to Infant Vulnerability. Women were recruited during pregnancy through a women’s mental health program in a psychiatry department in a large, southeastern United States city. Referrals came from women in the community, their doctors, other clinics, and other research studies. Participants all met DSM-IV criteria for a previous Major Depressive Episode (MDE), Obsessive Compulsive Disorder (OCD), Generalized Anxiety Disorder (GAD), or Post-Traumatic Stress Disorder (PTSD). Further inclusion criteria were as follows: being <16 weeks pregnant measured from last menstrual period and being between ages 18 and 45. Exclusion criteria included: active suicidality or homicidality, having psychotic symptoms, meeting DSM-IV criteria for bipolar disorder, schizophrenia, or currently active eating disorder, having an active substance use disorder within 6 months prior to last menstrual period or positive urine drug screen, illness requiring treatment that can influence infant outcomes, such as epilepsy, asthma, autoimmune disorders, and having abnormal thyroid stimulating hormone or anemia.

For this report, we focused on the first enrolled 83 women and their infants (41% female and 59% male) and their data from pregnancy and 3 and 6 months postpartum. This sample size is larger than similar studies. In addition, power analyses were conducted using G’Power (Faul, Erdfelder, Lang, & Buchner, 2007) in order to determine an appropriate sample size to achieve good statistical power. We computed the minimal sample size needed in order for correlational tests (alpha level of .05) to yield a medium effect size. Results suggested that for our analyses, a sample size of 82 would be large enough to achieve a statistical power level of .803.

Of the women, 67 (81%) met DSM-IV criteria for a previous MDE, 10 (12%) met for OCD, 24 (29%) for GAD, and 11 (13%) for PTSD (sum is >83 since some women had more than one diagnosis). Infants’ estimated gestational age at delivery ranged from 29 to 41 weeks ($M = 38.14 \text{ weeks}$, $SD = 2.22$) and their birth weight ranged from 1.28 to 4.54 kg ($M = 3.20, SD = .66$). For infants who were born premature (gestational age of <37 weeks), laboratory visits were scheduled based on the infant’s full term due date in order to correct for prematurity.

A total of 76 mother–infant dyads participated at infant age 3 months and an additional 7 at infant age 6 months only. In all, 65 mother–infant dyads had complete data at both time points. Women ranged from 23.4 to 44.5 years of age at delivery ($M = 33.80 \text{ years}$, $SD = 4.35$). Most (88%) were married. On average, women had completed 16.71 years ($SD = 2.09$) of education. Nearly half (43.40%) were primiparous. Most were white (84%), with the remaining 11% being African American, 2% Native American, and 2% Asian. Most of the women (82%) were taking antidepressant medications during pregnancy as were 73% during the first 6 months postpartum. Data on antidepressant exposure during the postpartum were missing for 10 participants.

Procedure

Data were collected from the women at multiple time points throughout pregnancy and the first 6 months postpartum. During pregnancy, women completed an average of 5.50 Beck Depression Inventory scales (BDI; A. T. Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), with a range from 1 to 10 times ($SD = 1.79$). At infant ages 3 and 6 months, women also completed the Beck Depression Inventory-Second Edition (BDI-II; A. T. Beck, Steer, & Brown, 1997) to assess depressive symptoms concurrent with the EEG measurement.
At 3 and 6 months of infant age, mothers and their infants were video-recorded and infants’ EEG was recorded during a 3-min baseline, 5-min feeding, and 5-min play segment, following Dawson et al. (1997). Prior to the baseline segment, an EEG cap was secured to the infant’s head while a research assistant manipulated toys in order to distract the infant. The baseline segment was designed to keep the infant quiet and alert and minimize eye movements and gross motor movements. Infants sat on their mothers’ laps and a research assistant blew bubbles for the infants to watch (Dawson et al., 2000), moving the toy to direct the infant’s attention. The baseline segment was designed to keep the infant quiet and alert and minimize eye movements and gross motor movements. Infants sat on their mothers’ laps and a research assistant blew bubbles for the infants to watch (Dawson et al., 1997). Mothers were instructed not to talk to their infant during this segment of the EEG recording. Following Lehtonen, Könnönen, Purhonen, Partanen, and Saarikoski (2002), during the feeding segment, the mother breast- or bottle-fed her infant. Finally, consistent with Morasch and Bell (2011), during the play segment, the mother was provided with toys and instructed to play with her child in any way she would like.

**Measures**

**EEG Measures.** The baseline EEG recordings were made from 16 left and right scalp sites: frontal pole (FP1, FP2), medial frontal (F3, F4), lateral frontal (F7, F8), central (C3, C4), anterior temporal (T3, T4), posterior temporal (T7, T8), parietal (P3, P4), and occipital (O1, O2), referenced to Cz. EEG was recorded using a stretch cap (Electro-Cap, Inc., Eaton, OH) with electrodes in the 10/20 system pattern and recommended procedures regarding EEG data collection with infants and young children were followed (Pivik et al., 1993). After the cap was placed on the infant’s head, a small amount of abrasive gel was placed into each recording site and the scalp gently rubbed. Following this, conductive gel was placed in each site. Electrode impedances were measured and accepted if they were below 5 K ohms. The electrical activity from each lead was amplified using separate SA Instrumentation Bioamps (San Diego, CA) and band passed from 1 to 100 Hz. Activity for each lead was displayed on the monitor of the acquisition computer. The EEG signal was digitized on-line at 512 samples per second for each channel so that the data were not affected by aliasing. The acquisition software was Snapshot-Snapstream (HEM Data Corp., Southfield, MI) and the raw data were stored for later analysis.

Infant EEG data were examined and analyzed using EEG Analysis System software developed by James Long Company (Caroga Lake, NY). First, the data were re-referenced via software to an average reference configuration. Average referencing, in effect, weighted all the electrode sites equally and eliminated the need for a noncephalic reference. Active (F3, F4, etc.) to reference (Cz) electrode distances vary across the scalp. Without the re-referencing, power values at each active site may reflect interelectrode distance as much as they reflect electrical potential. The average reference configuration requires that a sufficient number of electrodes be sampled and that these electrodes be evenly distributed across the scalp. Currently, there is no agreement concerning the appropriate number of electrodes (Davidson, Jackson, & Larson, 2000; Hagemann, Naumann, & Thayer, 2001; Luck, 2005), although the 10/20 configuration that we used does satisfy the requirement of even scalp distribution.

The average reference EEG data were artifact scored for eye blinks using Fp1 and Fp2 (Myslobodsky et al., 1989), with a peak-to-peak criterion of 100 μV or greater. Artifact associated with gross motor movements over 200 μV peak-to-peak was also scored. These artifact-scored epochs were eliminated from all subsequent analyses. The data then were analyzed with a discrete Fourier transform (DFT) using a Hanning window of 1-s width and 50% overlap. Power was computed for the 6–9 Hz frequency band. Infants and young children have a dominant frequency between 6 and 9 Hz (Bell & Fox, 1994; Marshall, Bar-Haim, & Fox, 2002), and this particular frequency band has been correlated with patterns of emotion reactivity and emotion regulation during infancy (Bell & Fox, 1994; Buss et al., 2003; Dawson, 1994) and early childhood (Fox et al., 2001). The power was expressed as mean square microvolts and the data transformed using the natural log (ln) to normalize the distribution.

Frontal EEG asymmetry values were computed by subtracting ln power at left frontal (F3) from ln power at right frontal (F4). In infants and young children, power in the 6–9 Hz band has been shown to be inversely related to cortical activation during emotion reactivity and regulation (Bell & Fox, 1994). Thus, a negative asymmetry score reflects greater right frontal activation, whereas a positive asymmetry score reflects greater left frontal activation. Descriptive statistics on the EEG asymmetry scores are in Table 1.

**Depression Measures.** Depression symptom levels were measured with both the BDI and BDI-II. Data were collected as part of a larger, long-standing clinical research protocol that relied on the BDI. When the infants were in the laboratory for the EEG data collection, there was the opportunity to add measures and the BDI-II was selected as the measure of concurrent depressive symptoms, for its advantages of being more parallel to the DSM-IV relative to its predecessor.

**Beck Depression Inventory** (A. T. Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The BDI is a 21-item self-report measure of depression symptom severity in the past week, with each item rated on a 4-point scale, ranging from 0 to 3. The score is a sum across items, with higher scores indicating greater severity of depressive symptoms. Scores of 0–9 indicate no depression, 10–18 indicates mild-moderate depression, 19–29 indicates moderate-severe depression, and 30–63 indicates severe depression (A. T. Beck et al., 1961). The BDI has been found to be both a valid and reliable measure of depression severity, with an especially high degree of content validity and internal consistency reliability (A. T. 1961; Ji et al., 2011). For pregnancy, we calculated area under the curve (AUC) scores for each woman to represent the overall depression severity level across the pregnancy, standardized to a 40-week pregnancy. For the postpartum, separate AUC scores were calculated for each woman to represent the overall depression severity level during the first 3 months postpartum and during the first 6 months postpartum. Descriptive statistics for these AUC scores are in Table 1.
Table 1. Descriptive Statistics of Maternal Depression Symptoms and Infant EEG Variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>M</th>
<th>SD</th>
<th>Minimum</th>
<th>Maximum</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prenatal depression AUC</td>
<td>362.97</td>
<td>270.14</td>
<td>35.64</td>
<td>1207.82</td>
<td>77</td>
</tr>
<tr>
<td>Postpartum depression AUC through 3 months</td>
<td>115.06</td>
<td>97.40</td>
<td>0</td>
<td>519.00</td>
<td>79</td>
</tr>
<tr>
<td>Postpartum depression AUC through 6 months</td>
<td>223.80</td>
<td>173.92</td>
<td>0</td>
<td>926.80</td>
<td>79</td>
</tr>
<tr>
<td>Concurrent depression at 3 months</td>
<td>9.40</td>
<td>8.63</td>
<td>0</td>
<td>44.00</td>
<td>72</td>
</tr>
<tr>
<td>Concurrent depression at 6 months</td>
<td>8.64</td>
<td>8.33</td>
<td>0</td>
<td>43.00</td>
<td>72</td>
</tr>
<tr>
<td>Baseline EEG 3 months</td>
<td>.01</td>
<td>.25</td>
<td>−.89</td>
<td>1.02</td>
<td>75</td>
</tr>
<tr>
<td>Feeding EEG 3 months</td>
<td>−.04</td>
<td>.17</td>
<td>−.46</td>
<td>.39</td>
<td>73</td>
</tr>
<tr>
<td>Play EEG 3 months</td>
<td>.03</td>
<td>.25</td>
<td>−.58</td>
<td>.78</td>
<td>71</td>
</tr>
<tr>
<td>Baseline EEG 6 months</td>
<td>.10</td>
<td>.28</td>
<td>−.40</td>
<td>1.15</td>
<td>71</td>
</tr>
<tr>
<td>Feeding EEG 6 months</td>
<td>.06</td>
<td>.21</td>
<td>−.45</td>
<td>.59</td>
<td>71</td>
</tr>
<tr>
<td>Play EEG 6 months</td>
<td>.13</td>
<td>.35</td>
<td>−.96</td>
<td>1.86</td>
<td>71</td>
</tr>
</tbody>
</table>

Note: AUC, Area Under the Curve. Depression symptoms AUC were measured with the Beck Depression Inventory (BDI) except for the concurrent measures, which were measured with the BDI-II.

Beck Depression Inventory-Second Edition (A. T. Beck, Steer, & Brown, 1997). The BDI-II is a 21-item self-report measure of depressive symptom severity. As answers are based on the past 2 weeks, the BDI-II parallels the DSM-IV duration criterion for a MDE. Items are rated on a 4-point scale, ranging from 0 to 3 and summed, with higher total scores indicating greater severity of depressive symptoms. Depression scores ranging from 0 to 13 indicate no or minimal depression; 14–19 indicates mild depression; 20–28 indicates moderate depression; 29–63 suggests a severely depressed individual. Based on these empirically established cut scores, a score of 14 or higher is considered to indicate depression (A. T. Beck, Steer, & Brown, 1997). The BDI-II is a valid and reliable measure of depression severity, with an especially high degree of content validity, construct validity, and internal consistency (A. T. Beck et al., 1997). The BDI-II has good concurrent validity with measures of both antenatal and postpartum depression (Boyd, Le, & Somberg, 2005; Steer, Scholl, & Beck, 1990). Table 1 provides descriptive statistics for the BDI-II scores at 3 and 6 months postpartum.

Demographics, infant gestational age, and antidepressant usage. Data on women’s socio-demographics were obtained from interview. Gestational age was obtained from hospital birth records. Antidepressant usage was determined by clinician interview, prospectively tracking medication across pregnancy and the first 6 months postpartum.

Data Analytic Strategy

We tested separately the degree of association between maternal age, Hollingshead score, baby gestational age at birth, antidepressant usage, infant gender, and infant EEG asymmetry scores in order to determine whether to control for these variables. To test the first hypothesis, that higher maternal postpartum depressive symptoms would be associated with 3- and 6-month-old infants’ greater relative right frontal baseline EEG asymmetry scores, both a concurrent measure of maternal depressive symptoms and a cumulative, AUC, measure of depressive symptom levels over the first 3 or 6 months postpartum were used in Pearson product moment correlations with infant EEG asymmetry scores at both ages.

In order to test our second hypothesis that maternal prenatal and postpartum depressive symptoms would either additively predict infant baseline EEG asymmetry scores at 3 and 6 months of age, such that higher levels of both would predict greater relative right frontal EEG asymmetry in infants, or that they would interact, such that greater prenatal exposure would predict greater relative right frontal EEG asymmetry specifically among infants whose mothers had more (or increasing) depressive symptoms postnatally, we conducted a multiple regression analysis. The predictor variables were the AUC BDI scores during the prenatal period and over the first 3 or 6 months postpartum, as well as the interaction term of these two variables. For both regression equations, the predictor variables were centered and a statistical interaction term was created following the widely accepted approach to testing for interaction effects using multiple regression (Cohen, Cohen, West, & Aiken, 2003).

The third through fifth hypotheses, that infant EEG asymmetry scores would be consistent across contexts at both ages and stable across ages in all contexts, was tested with Pearson product moment correlations. Finally, in order to examine the sixth hypothesis, that the relationship between infant baseline EEG asymmetry scores at 3 and 6 months of age would be moderated by changes in maternal depressive symptoms between those two time points, we ran two separate analyses. For the first, we calculated a difference score for infant EEG asymmetry scores, subtracting each infant’s asymmetry score at 3 months of age from their asymmetry score at 6 months of age. A similar difference score was created for maternal concurrent depressive symptoms, by subtracting maternal BDI-II scores at infant age 3 months from maternal BDI-II scores at infant age 6 months. We then ran a Pearson product moment correlation between these two values—the infant EEG asymmetry difference score and the maternal concurrent depressive symptoms difference score. For the second analysis, we conducted a multiple regression analysis. Infant
3-month baseline EEG asymmetry scores and maternal postpartum depressive symptoms through infant age 3 months were entered in the first step, followed by maternal concurrent depressive symptoms at 6 months in the second step, in order to predict infant 6-month EEG asymmetry scores.

RESULTS

Preliminary Analyses

Fifty-one women (66%) exceeded the BDI cutoff indicating clinically significant levels of depression at some point during the prenatal period and 25 women (30%) exceeded the BDI-II cutoff at 3 or 6 months postpartum. Of the 76 infants who visited the laboratory at 3 months of age, usable EEG data were obtained from 75 (99%) infants at baseline, 72 (95%) during feeding, and 71 (93%) during play. Of these, baseline asymmetry scores for two infants were excluded as outliers (±3 standard deviations from the mean) and baseline data for two infants were excluded for an insufficient quantity of data (<10 s of good data), yielding a sample size of 71 infants with good baseline data at 3 months of age. No infants were excluded in the feeding context at 3 months of age. In the play context, 1 infant was excluded as an outlier and 10 infants were excluded for an insufficient quantity of data, yielding a sample size of 60 infants with good play data at 3 months of age. Of the 72 infants who visited the laboratory at 6 months of age, 71 (99%) had usable baseline data, 71 had usable feeding data, and 71 had usable play data. Of these, baseline data for one infant were excluded as an outlier and baseline data for one other infant were excluded for insufficient quantity of data, yielding a sample of 69 participants with good baseline data at 6 months of age. In the feeding context, data for one infant were excluded for insufficient quantity of data, yielding a sample of 70 infants with good feeding data at 6 months of age. Finally, data were excluded as an outlier for two infants in the play context at 6 months of age, and data were excluded for three other infants due to insufficient quantity of data, yielding a sample of 66 infants with good data during play at 6 months of age. Infants who did not contribute usable data for the various contexts were retained in the final dataset but were excluded from analyses using an appropriate data filter.

We tested for possible confounding variables that might have been associated with infant EEG asymmetry scores at 3 and 6 months of age. There were no significant associations between EEG asymmetry scores in any of the contexts at either age and maternal age at delivery, baby’s gestational age, or baby gender. Mother’s education level was only significantly associated with EEG asymmetry scores during feeding at 6 months of age, \( r(68) = -.30, p < .05 \). The number of prenatal weeks that infants were exposed to maternal antidepressant use was not significantly associated with EEG asymmetry scores in any context at either age. Given the likelihood that the one significant association may be due to chance, none of these variables were controlled for in the analyses.

In addition, mothers differed in whether they breast- or bottle-fed their infants during the feeding context: 29 women (41%) breast fed at 3 months as did 25 women (36%) at 6 months. There were no significant group differences in infant EEG asymmetry scores during feeding based on whether the mother breast or bottle fed at 3 months, \( t(69) = .41, p = .68 \), or at 6 months, \( t(67) = 1.18, p = .24 \).

Hypothesis Testing

Postpartum Depressive Symptoms and EEG. The first hypothesis was that higher maternal postpartum depressive symptom levels would be associated with 3- and 6-month-old infants’ greater relative right frontal baseline EEG asymmetry. At infant age 3 months, Pearson product moment correlations revealed that there was not a significant association between infant baseline frontal EEG asymmetry scores and either maternal concurrent depressive symptoms (BDI-II), \( r(65) = -.06, p = .63 \), or maternal postpartum depressive symptoms from birth through infant age 3 months (BDI AUC postpartum), \( r(65) = .05, p = .71 \). That is, contrary to prediction, postpartum depressive symptoms were neither concurrently, nor over the course of the first 3 months postpartum, significantly associated with infant baseline EEG asymmetry scores at 3 months of age.

At infant age 6 months, there was also not a significant association between infant baseline frontal EEG asymmetry scores and either maternal concurrent depressive symptoms (BDI-II), \( r(64) = -.16, p = .21 \), or maternal postpartum depressive symptoms from birth through infant age 6 months (BDI AUC postpartum), \( r(66) = .01, p = .95 \). Again, contrary to prediction, postpartum depressive symptoms were neither concurrently, nor over the course of the first 6 months postpartum, significantly associated with infant baseline EEG asymmetry scores at 6 months of age.

Pre- and Post-Natal Depression Symptoms and EEG: Additive or Interactive. We hypothesized that maternal prenatal and postpartum depressive symptoms would either additively predict infant baseline EEG asymmetry scores at 3 and 6 months of age, such that higher levels of both would predict greater relative right frontal EEG asymmetry in infants, or that they would interact, such that greater prenatal exposure would predict greater relative right frontal EEG asymmetry.
specifically among infants whose mothers had more (or increasing) depressive symptoms postnatally. Regression analyses revealed that maternal prenatal depressive symptoms did not predict infant baseline frontal EEG asymmetry scores at 3 or 6 months of age. Also, the additive effect of maternal prenatal and postpartum depressive symptoms did not explain a significant amount of additional variance in infant baseline EEG asymmetry scores at either 3 or 6 months of age. Rather, at both ages, maternal prenatal and postpartum (through infant age 3 or 6 months, respectively) depressive symptoms interacted significantly to predict infant baseline frontal EEG asymmetry scores (see Tab. 2). Consistent with the interaction hypothesis, at 3 months of age, prenatal depressive symptoms and infant EEG asymmetry scores were not significantly associated among women with low postpartum depressive symptom levels, $r(31) = .18, p = .16$. In contrast, prenatal depressive symptoms and infant EEG asymmetry scores were significantly associated among women with high postpartum depressive symptoms, $r(26) = -.44, p = .01$, a moderate effect size (Cohen, 1992; see Fig. 1). Findings at infant age 6 months were somewhat consistent with those at 3 months, as predicted by the interaction hypothesis: prenatal depressive symptoms and infant EEG asymmetry scores were not significantly associated among women with low postpartum depressive symptom levels, $r(31) = .04, p = .41$. In contrast, there was a trend for the association between prenatal depressive symptoms and infant EEG asymmetry scores among women with high postpartum depressive symptoms, $r(30) = -.26, p = .08$, a small effect size (see Fig. 2).

**Consistency of EEG.** Our third hypothesis was that infant EEG asymmetry scores would be consistent across contexts, from baseline to the two maternal interaction contexts (feeding and play) at both 3 and 6 months of age. As hypothesized, results of Pearson product-moment correlations revealed significant positive associations between contexts at both ages (see Tab. 3). That is, greater relative right frontal EEG asymmetry during baseline was associated with greater relative right frontal EEG asymmetry during play with a moderate effect size at infant age 3 months, and with a small, trend level association at infant age 6 months. Greater relative right frontal EEG asymmetry during feeding was significantly associated with greater relative right frontal EEG asymmetry during play with moderate (at 3 months) and large (at 6 months) effect sizes. Greater relative right frontal EEG asymmetry during baseline was also significantly associated with greater relative right frontal EEG asymmetry during 3-month olds’ feeding with a moderate effect size; this association was not significant at infant age 6 months.

**Stability of EEG.** Our fourth hypothesis was that infant baseline EEG asymmetry scores would be stable from 3 to 6 months of age. Pearson correlations revealed a significant association between baseline frontal EEG asymmetry scores at 3 and 6 months of age, $r(57) = .27, p < .05$. Greater relative right frontal baseline EEG asymmetry at 3 months of age was associated with greater relative right frontal EEG asymmetry at 6 months of age with a moderate effect size.

Our fifth hypothesis was that infant EEG asymmetry scores in maternal-interaction contexts (feeding and play) would be stable from 3 to 6 months of age. This hypothesis was partially supported. Infant EEG asymmetry scores in the play condition were significantly associated across age, $r(43) = .38, p < .05$ with a moderate effect size, such that greater relative right

<table>
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<td></td>
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<td>6 Months</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$\Delta R^2$</td>
<td>$\beta$</td>
<td>$\Delta R^2$</td>
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<td>-.408**</td>
<td>.089*</td>
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<td>Interation term</td>
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<tr>
<td>Total $R^2$</td>
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* $p < .05$ level.
** $p < .01$ level.

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**Table 2. Summary of Hierarchical Regression Analyses for Maternal Depression Variables Predicting Infant EEG Asymmetry Scores**
Frontal EEG asymmetry at 3 months of age was associated with greater relative right frontal EEG asymmetry at 6 months of age. There was no significant association between EEG asymmetry scores during feeding from 3 to 6 months of age, $r(57) = .12, p = .36$.

Role of Maternal Depressive Symptoms in the Prediction of Stability of Infant EEG. Finally, we hypothesized that the relation between infant baseline EEG asymmetry scores at 3 and 6 months of age would be moderated by changes in maternal depressive symptoms between those two time points. In order to further understand EEG stability at baseline, we examined stability descriptively. We found that only 37 infants (59%) demonstrated a stable pattern of either left or right frontal EEG asymmetry from 3 to 6 months of age, whereas the others (41%; $n = 26$) showed a change from either left to right or right to left frontal asymmetry over this time period.

We found no significant association between changes in maternal concurrent depressive symptoms from infant ages 3 to 6 months and changes in infant baseline frontal EEG asymmetry scores from 3 to 6 months, $r(54) = -.21, p = .12$, though the association was in the predicted direction, with increasing maternal depressive symptoms associated with decreasing infant EEG asymmetry scores. Results of the regression analyses revealed that only 3-month infant EEG asymmetry scores significantly predicted infant 6-month EEG asymmetry scores. Maternal postpartum depressive symptoms did not account for significant additional variance in infant 6-month EEG asymmetry scores (see Tab. 4).

DISCUSSION

With a prospective longitudinal design beginning in pregnancy, we investigated the relation between maternal prenatal and postpartum depressive symptoms and infant EEG asymmetry scores measured at 3 and 6 months of age in baseline and two maternal
interaction contexts (feeding and play). Our findings suggest that maternal prenatal and postpartum depressive symptoms interact to predict infant EEG baseline asymmetry scores at both ages. In addition, we found consistency of EEG asymmetry scores across contexts as early as 3 months of age and stability across infancy of EEG asymmetry scores in the baseline and play contexts.

Findings from our first hypothesis were not consistent with previous findings that maternal depressive symptoms in the first year postpartum were significantly negatively correlated with infants’ frontal EEG asymmetry scores (Dawson et al., 2001; Diego et al., 2006; Jones, Field, Davalos, et al., 1997; Jones, Field, Fox, et al., 1997). Instead, our findings on EEG in infants at 3- and 6-months of age revealed that not only were maternal concurrent depressive symptom levels not significantly associated with infant EEG asymmetry scores, but neither was a more comprehensive measure capturing infants’ lifetime of exposure to maternal postpartum depressive symptoms (collected during the period from the infant’s birth through the times of the EEG recordings).

There are several possible explanations for our failure to support the expected associations between infant EEG and postpartum depression. First, the Field studies (Diego et al., 2006; Field et al., 1995; Jones, Field,

Table 3. Intercorrelations Among Infant EEG Asymmetry Scores in All Three Contexts at Both Ages

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<td>Play 6 months</td>
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*p < .05 level (two-tailed).

**p < .01 level (two-tailed).
Davalos, et al., 1997; Jones, Field, Fox, et al., 1997) sampled predominantly low to middle SES, ethnic minority women. Within our sample of broadly middle and upper SES women, 16% of whom were from ethnic minorities, we found no significant associations between maternal age and infant EEG asymmetry scores, and maternal education level was only significantly associated with EEG during one of six observations: feeding at infant age 6 months. Nonetheless, these differences in sample characteristics across studies could have played a role in the different findings. In particular, it is possible that the stresses associated with poverty and being of minority ethnicity may have partially explained the associations between depressive symptoms and infant EEG asymmetry scores in the previous studies. A second possible explanation for the discrepant findings is that previous studies included community samples regardless of depression history, whereas the current study relied on a clinical sample of mothers with lifetime histories of depressive or anxiety disorders. Infants of women with histories of depression are important to study given these women’s significantly greater risk of perinatal depression relative to general population samples (O’Hara & Swain, 1996). Thus, the common history of depression in the women in the current sample may better explain the infant EEG asymmetry scores, whereas the postpartum depressive symptoms alone may not add significant variance. If replicated with samples at similar risk for depression, albeit not at psychosocial risk, our findings suggest that postpartum depression symptom levels alone, at least among women with histories of depression, do not play a significant role in infant EEG patterns.

The second aim of the current study was to examine the potential additive or interactive associations between both maternal prenatal and postpartum depressive symptoms, prospectively measured, in predicting infant EEG asymmetry scores at 3 and 6 months of age. This was, to our knowledge, the first attempt to examine both maternal prenatal and postpartum depressive symptoms together as a predictor of infant EEG asymmetry scores. Previous studies have focused solely on postpartum depression or the relation between maternal prenatal depressive symptoms and newborns’ EEG asymmetry scores (Field et al., 2002), or maternal prenatal depression measured retrospectively and older infants’ or children’s EEG asymmetry scores (Dawson et al., 2003).

In corroboration with Belsky’s (2007) theory, we found strong support for the interactive model: maternal prenatal and postpartum depressive symptoms interacted significantly to predict infant EEG asymmetry scores with a moderate effect size at both ages. In particular, we found that higher levels of prenatal depressive symptoms were associated with greater relative right frontal EEG asymmetry specifically among infants whose mothers had higher levels of depressive symptoms postnatally, albeit at the trend level at 6 months of age. In contrast, the relation between maternal prenatal depressive symptoms and infant EEG asymmetry scores was not significant in the context of low postpartum maternal depressive symptoms. Such findings point to the importance of taking both prenatal and postnatal maternal depressive symptoms into account as predictors of infant EEG asymmetry scores and support a moderating role of postpartum depressive symptom levels in the association between prenatal depressive symptom levels and infant baseline EEG asymmetry scores at 3 and 6 months of age. These findings are consistent with the notion of stress sensitization as one possible pathway to offspring vulnerability, with sensitization to stress-related disorders being associated with prenatal exposure (Goodman & Gotlib, 1999).

The third aim was to extend the findings on the consistency of infant EEG asymmetry scores across contexts down to younger infants. Consistent with

<table>
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<th>Predictor</th>
<th>ΔR²</th>
<th>β</th>
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<td>3-Month infant baseline EEG asymmetry scores</td>
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<td>.296*</td>
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<td>Concurrent depression at 6 months</td>
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*p < .05 level.

### Table 4. Summary of Hierarchical Regression Analyses for Maternal Depression Variables and 3-Month Infant EEG Asymmetry Scores Predicting 6-Month Infant EEG Asymmetry Scores

Developmental Psychobiology
Dawson’s findings in older infants (Dawson et al., 1997, 2001), the current findings supported our hypothesis that infant EEG asymmetry scores would be consistent across contexts, particularly at 3 months of age, with moderate to large effect sizes. At 6 months of age, although EEG asymmetry scores during the two maternal interaction contexts were highly correlated, association between baseline and play were only associated at the trend level and associations between baseline and feeding were not significant. These findings suggest that consistency of infant EEG patterns across contexts, from baseline to interactions with the mother, emerges even earlier than had previously been shown. By 6 months, context seems to matter more, such that EEG patterns during baseline may need to be distinguished from EEG obtained during interactions.

The fourth aim was to examine stability over time. We attempted to replicate the findings that infant baseline EEG asymmetry scores were stable across early infancy (Diego et al., 2006; Jones, Field, Davalos, et al., 1997; Jones, Field, Fox, et al., 1997) as well as extend these findings to an examination of the stability of EEG asymmetry scores obtained in maternal interaction contexts. We found strong support for our hypothesis that infant EEG asymmetry scores during baseline would be stable; specifically, greater infant EEG asymmetry scores at 3 months were significantly associated with greater infant EEG asymmetry scores at 6 months with a moderate effect size in both the baseline and play contexts. In contrast, infant EEG asymmetry scores during feeding were not stable across ages, which may reflect important differences in the feeding context relative to baseline and even as compared to the other maternal interactive context of play.

Further, during baseline, somewhat similar to Smith and Bell’s (2010) finding, only slightly more than half (59%) of infants maintained a stable pattern of either left or right frontal EEG asymmetry from 3 to 6 months of age, whereas the others showed a change from either left to right or right to left frontal asymmetry over this time period. This finding places even further importance on exploring the potential moderating role of ongoing maternal depressive symptoms in predicting stability. Contrary to our prediction, the relation between infant baseline EEG asymmetry scores at 3 and 6 months of age was not moderated by changes in maternal depressive symptoms between those two time points, although the association was in the predicted negative direction. The direction of the association suggests that as maternal depressive symptoms increase from infant age 3 to 6 months, infant EEG asymmetry scores become more negative, reflecting a shift toward greater relative right frontal EEG asymmetry. An alternative approach to this question indicated similar findings, such that only 3-month infant EEG asymmetry scores significantly predicted 6-month infant EEG asymmetry scores, whereas maternal postpartum depressive symptoms did not. The lack of significance may be accounted for by the fact that the full trajectory of maternal depressive symptoms from infant age 3 to 6 months was not examined. Therefore, a change in maternal concurrent depressive symptoms, captured by the 2-week “snapshot” based on the symptom measures from 3 to 6 months, may not accurately reflect the exposure that infants received in the months between these assessments. More frequent sampling of mothers’ depressive symptom levels may be important to help explain the observed changes in infant EEG asymmetry scores across ages. Also, there may be other variables that contribute to this association and would help to explain which infants demonstrate stable EEG patterns.

Two additional variables that may help to explain associations between changes in maternal depressive symptoms and change or stability of infant EEG asymmetry scores are infants’ concurrent affect and mothers’ parenting quality. Although findings of concurrent affect show that 1-month-old and 13- to 15-month-old infants of depressed mothers show different infant EEG patterns despite not differing in concurrent affect (Dawson et al., 1997, 2001; Jones, Field, Fox, et al., 1997), such measures of concurrent affect have not been examined in 3- and 6-month-olds. Additionally, parenting quality may moderate or mediate associations between maternal depressive symptoms and infant EEG asymmetry scores.

Overall, the findings of the current study demonstrate support for infant EEG as a trait marker of vulnerability, with findings suggesting that infant EEG patterns are consistent across contexts even as early as 3 months of age and moderately stable from 3 to 6 months of age in both baseline and play contexts. However, by 6 months of age, consistency begins to diverge, and EEG in the feeding context was not stable across ages. Thus, despite support for consistency and stability, the pattern of findings and the effect sizes implicate individual differences in infant EEG patterns across contexts and over time, and highlight the importance of identifying variables that may influence these patterns.

The current findings also indicate that, at least in predicting infant EEG asymmetry scores at 3 and 6 months of age, maternal prenatal and postpartum depressive symptoms must both be considered. Specifically, the association between maternal prenatal depressive symptoms and infant EEG asymmetry scores is only significant in the context of high levels of postpartum
depressive symptoms, albeit at a trend level at 6 months. Further research is needed in order to investigate the mechanisms by which perinatal depressive symptoms are associated with infant EEG asymmetry scores, as well as to more closely examine the impact of changes in maternal depressive symptoms on changes in infant EEG patterns.

NOTES

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