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Electroencephalogram Patterns in Infants of Depressed Mothers

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Electroencephalogram Patterns in Infants of Depressed Mothers

By

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B.A., Emory University, 2006

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An abstract of
A thesis submitted to the Faculty of the
James T. Laney School of Graduate Studies of Emory University
in partial fulfillment of the requirements for the degree of
Master of Arts
in Psychology
2011

Abstract

Electroencephalogram Patterns in Infants of Depressed Mothers
By Cara M. Lusby

The goal of the current study was to examine the association between maternal prenatal and postpartum depressive symptoms and infant electroencephalogram (EEG) asymmetry scores. Participants were 66 women with a history of depression and/or anxiety and their infants. Women were recruited during pregnancy and their depressive symptoms were measured at multiple time points throughout pregnancy and the postpartum. Infant EEG asymmetry scores were collected at 3 and 6 months of age, and mothers' concurrent depressive symptoms were also measured. Maternal prenatal and postpartum depressive symptoms separately did not predict infant EEG asymmetry scores at either age, nor did the additive effect of the two variables. The interaction of maternal prenatal and postpartum depressive symptoms did significantly predict infant EEG asymmetry scores at 6 months of age, with a trend in the association at 3 months of age. Specifically, at infant age 6 months, prenatal depressive symptoms and infant EEG asymmetry scores were significantly associated among women with high postpartum depressive symptoms only. Infant EEG asymmetry scores were consistent across contexts at both ages and were stable across ages in the baseline and free play segments, but not feeding. Findings highlight the importance of considering both prenatal and postpartum maternal depressive symptoms in the prediction of infant EEG asymmetry scores, as well as pointing to the need to further understand the differences between infants who display changes in patterns versus those whose patterns are stable across ages.

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Electroencephalogram Patterns in Infants of Depressed Mothers

Electroencephalogram (EEG) patterns have been found to be associated with emotion in general and depression more specifically. The same pattern of greater relative right frontal EEG asymmetry scores that has been found in adults with depression has also been found in infants of antenatally (Field, Diego, Hernandez-Reif, Schanberg, & Kuhn, 2002; Field et al., 2004) and postnatally (Dawson et al., 2001; Field, Fox, Pickens, & Nawrocki, 1995) depressed mothers. Moreover, these scores in infants predict later levels of inhibition (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 this consistency has only been examined in older infants (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 (Diego, Field, Jones, & Hernandez-Reif, 2006; Jones, Field, Davalos, & Pickens, 1997), although 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 interaction at the moment as well as an infant's history of interaction with a depressed mother (Jones, Field, Fox, et al., 1997).

Although more information is needed, these findings suggest that EEG asymmetry scores may reflect, even in infancy, a trait marker of vulnerability to depression; however, the associations are not strong and suggest that there is variability in the stability and consistency of these patterns. Thus, it is critical to determine what the predictors of this stability or variability may be. In particular, this study adds to the

literature on associations between maternal prenatal and postpartum depression and infant EEG by examining how maternal prenatal and postpartum depressive symptoms might have an additive or interactive effect on EEG asymmetry scores at 3 and 6 months of age as well as on the stability of these scores over time. The aim of the current study was to explore the relationship between maternal prenatal and postpartum depressive symptoms and infant EEG patterns over the first several months of life. A long term goal of the current research is to distinguish between subsets of infants who start out early on negative trajectories of development and continue on this pathway from others who may "recover," and still others in whom a negative trajectory emerges later in infancy.

EEG and Emotion

EEG patterns have been theorized to be indicative of emotional dysregulation at the physiological level, based on their association with two distinct behavioral systems. Fox and colleagues (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 behavioral systems are thought to be associated with different frontal hemispheres of the brain, 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 Vulnerability to Depression

Further, researchers have suggested that greater relative right frontal EEG asymmetry scores reflect a vulnerability to depression. In several studies of adults, patterns of greater relative right frontal EEG activation have been found in those diagnosed as depressed or those with high 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; Henriques & Davidson, 1991; Jones, Field, Davalos, et al., 1997; 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, & Rosenfeld, 1998; Henriques & Davidson, 1990), suggesting that EEG patterns may reflect a trait-like vulnerability to depression in adults.

Given this association between EEG patterns and depression in adults, it is critical to be able to identify the earliest markers of this vulnerability. Fox and colleagues (2001) found that infants who had greater relative right frontal EEG asymmetry at nine and fourteen months of age displayed stable inhibition over the first four years of life. This is in contrast to infants who showed less relative right frontal EEG asymmetry at nine months of age and greater relative left frontal EEG asymmetry at 14 months of age. For the latter two groups, their inhibition scores declined over the first four years of life (Fox et al., 2001). These findings suggest an association between infant EEG patterns and later inhibition, which is an early temperamental vulnerability for depression and other internalizing problems (Biederman et al., 2001; Muris, Merckelbach, Wessel, & van de Ven, 1999). Thus individual differences in EEG patterns may be a vulnerability that

reflects an individual's affective style and can place the individual at risk for the development of depression (Davidson, 1998).

Infant EEG and Maternal Depression

As the previous findings support 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. EEG patterns in offspring of depressed mothers may be a potential vulnerability marker of the risk for the later development of psychopathology, as depression in mothers is known to confer this risk 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. In particular, the association between maternal postpartum depressive symptoms or diagnosed depression and infant EEG patterns has been 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 relative right frontal activation during a neutral condition compared to infants of mothers with low levels of depressive symptoms (3 of 15) (Field et al., 1995). This difference between infants of concurrently depressed and nondepressed mothers has been found in infants as early as one week of age, and extends to one month olds, 3 to 6 month olds, and 13 to 15 month olds, with infants of mothers diagnosed as depressed 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). Further,

infants of mothers with diagnosed major depression displayed greater relative right frontal asymmetry scores than infants of mothers with sub-threshold depression, who in turn displayed greater relative right frontal asymmetry scores than infants of nondepressed mothers (Dawson et al., 2001; Dawson et al., 1997). Despite the consistency of these findings across studies, it is important to note that the majority of these findings were from samples composed of predominately ethnic minority mothers of low to middle socioeconomic status and thus may reflect general stressors associated with low SES as well as the infant's exposure to maternal depression. Therefore it is important to test the relationship between maternal depression and infant EEG in more diverse samples.

Despite the limitation of restricted sample characteristics, several studies now have found differences in infant EEG patterns in relation to concurrent (postpartum) maternal depression. In addition, maternal prenatal depression is also suggested to influence offspring EEG (Goodman & Gotlib, 1999). In particular, it has been theorized that maternal prenatal depression impacts infants' psychophysiology through variations in the intrauterine environment (Dawson et al., 2001). A few studies have now shown the association between prenatal depressive symptom levels in mothers and single measures of newborn or childhood EEG. Newborns with greater relative right frontal EEG activation had mothers with higher prenatal depressive symptoms compared to those with greater relative left frontal EEG activation (Field et al., 2002). Similarly, maternal depressive symptoms during pregnancy were found to be negatively correlated with newborns' EEG patterns, such that higher levels of maternal depressive symptoms were associated with newborns' greater relative right frontal EEG (Field et al., 2004). At 14

months of age, children of mothers who were depressed prenatally exhibited greater relative right frontal EEG asymmetry, while at 3 ½ 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. Also, we found no studies that reported having examined associations between maternal prenatal depression and offspring EEG at earlier points in infancy.

Given the support for both prenatal depression and postpartum depression independently predicting offspring EEG, examining the possible additive or interactive effects of depression occurring at these two time points and the impact on infant EEG asymmetry scores is critical for several reasons. For one, prenatal depression is the strongest predictor of postpartum depression (O'Hara & Swain, 1996) and thus many infants are dually exposed. Also, research suggests that women with postnatal depression might recover in early infancy (Goodman & Brand, 2009). Thus if a mother was depressed in pregnancy and only briefly (or not at all) in the postpartum, the question remains as to whether infants might recover from the maladaptive EEG patterns with which they may have been born. 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 scores in infants. Alternatively, they could be interactive. For example, it is possible that maternal prenatal depression is only associated with greater relative right frontal EEG patterns in infants when the mother is also depressed during the postpartum period. That is, infants may 'recover' from effects of prenatal exposures if mothers are low in depression in the

postpartum. In contrast, 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. Therefore, knowledge of infants' exposures both pre- and postnatally might enhance the prediction of infant EEG asymmetry scores.

Given the impact of maternal depression on infant EEG asymmetry scores as well as the association between infant EEG patterns and early temperamental vulnerabilities for internalizing disorders, it is important to understand whether these patterns are stable and persistent. This is particularly crucial as these patterns may confer vulnerability for the later development of depression. In order to address this question, researchers have examined whether infant EEG patterns are independent of current affective state, consistent across contexts, and stable over time. These findings are reviewed in the following sections.

State Independence of Infant EEG Patterns

Similar to findings in adults, which show that relative right frontal EEG patterns remain beyond a depressive episode, several studies have found evidence for state independence of infant EEG patterns. For example, the difference in EEG patterns between infants of depressed and nondepressed mothers is not explained by differences in infants' concurrent affect or behavior (Dawson et al., 2001; Dawson et al., 1997). Thirteen to fifteen month old infants of depressed mothers versus infants of nondepressed mothers had greater relative right frontal EEG asymmetry despite the fact that these two groups of infants did not differ in their affective behavior coded from infants' facial

expression and vocal quality during the baseline condition. Jones et al. (1997) also found that although 1-month-old infants of depressed and nondepressed mothers differed in their EEG patterns, they did not significantly differ in their concurrent affect (Jones, Field, Fox, et al., 1997). These findings yield support for EEG patterns reflecting trait-like vulnerability in infants.

Consistency of Infant EEG Patterns

The consistency of infant EEG patterns across contexts has also been documented, suggesting that EEG patterns do not change with changes in context. Thirteen to fifteen month old infants of depressed mothers had greater relative right frontal EEG activation 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., 2001; Dawson et al., 1997). These results support the consistency of EEG patterns across contexts. However, we found no studies reporting consistency in the EEG patterns of younger infants. Such findings would yield support for the earlier emergence of a trait-like vulnerability.

Stability of Infant EEG Patterns

Finally, evidence is beginning to emerge on the predictive value of the stability of infant EEG patterns, yielding further support for infant EEG patterns representing a vulnerability to the development of depression. Smith and Bell (2010) examined the association between stability of infant EEG patterns from 10 to 24 months of age on infant internalizing and externalizing behavior problems at 30 months of age. Infants were divided into groups based on their EEG patterns at both 10 and 24 months of age: whether they were stable left, stable right, or demonstrated a change over that time in

development. Those infants who showed stable right asymmetry displayed more maternal-rated internalizing behavior problems at 30 months of age compared to those who showed stable left asymmetry. In contrast, infants who showed stable left asymmetry displayed more maternal-rated externalizing behavior problems compared to those infants who showed stable right asymmetry or displayed a change (Smith & Bell, 2010). Whereas previous research has suggested the predictive value of EEG patterns at specific time points, these findings expand on this idea by demonstrating the predictive value of the stability of these patterns across ages.

Researchers have found evidence for the stability of infant EEG patterns over time in both infants of mothers with high depressive symptoms as well as infants of mothers with low depressive symptoms. Findings reveal moderate to large correlations between baseline frontal EEG asymmetry scores recorded within a few days after birth and again at 3 to 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 to 6 months showed the same patterns at 3 years of age (Jones, Field, Davalos, et al., 1997). As previously noted, however, these samples included predominately ethnic minority mothers of low to middle socioeconomic status, so the stability of infant baseline frontal EEG asymmetry scores across ages in a more diverse sample remains unexplored.

Also, the previous studies only examined stability of baseline EEG. One theory linking maternal postpartum depression and infant EEG patterns suggests that exposure to maternal depression in the postpartum impacts infants' emotion regulation abilities through the lower quality of maternal interactions with her infant, which is known to be

associated with maternal depression (Diego et al., 2006; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Therefore, infant EEG patterns recorded during interactions with their mothers may reflect the quality of interaction at the moment as well as an infant's history of interaction with a depressed mother (Jones, Field, Fox, et al., 1997). In particular, researchers have examined infant EEG asymmetry scores in the context of play with the mother (Dawson et al., 2001; Dawson et al., 1997) and feeding (Jones, McFall, & Diego, 2004). Thus it is important to extend studies of stability of baseline EEG patterns to also examine the stability of infant EEG patterns in contexts involving maternal interactions to address the questions of the extent to which changes in maternal depression over time may influence stability or change in infant EEG asymmetry scores.

Despite accumulating evidence that infant EEG patterns reflect state-independent vulnerabilities that are consistent across contexts and stable over time, there is evidence for variability of the stability of these baseline EEG patterns. Despite one finding of a large positive association between baseline frontal EEG asymmetry scores across ages, other findings suggest only a moderate association, lending support to this idea of variability. Also, in the Smith and Bell (2010) sample, over half of the infants demonstrated a change in their EEG patterns from 10 to 24 months of age. Further evidence of instability of infant EEG patterns across age comes from findings that infants of mothers with high depressive symptoms showed a shift in baseline EEG asymmetry scores from the neonatal period to 3 to 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 within a few days after birth and not at a later time point, thus leaving unanswered the question of

whether change in maternal depression in the postpartum might have been related to the changes in infant baseline EEG patterns. Therefore it is important to identify the factors that predict stability in order to understand why some infants show stability of EEG patterns and others do not.

Current Study

The current study aimed to address unanswered questions about the relationship between maternal depressive symptoms and infant EEG asymmetry scores, with a focus on EEG measured longitudinally at 3 and 6 months of age in three contexts (baseline, play with mother, and feeding). In particular, the study addressed six specific aims: 1) to attempt to extend the findings of the association between maternal postpartum depressive symptoms and infant baseline EEG asymmetry scores at both 3 and 6 months of age in a more broadly middle class sample relative to previous research involving low to middle socioeconomic status, predominately ethnic minority samples; 2) to examine the extent to which maternal prenatal and postpartum depressive symptoms may additively or interactively predict infant EEG asymmetry scores at both ages; 3) to downward extend the findings of the consistency of infant EEG asymmetry scores across contexts to younger infants; 4) to test for the replication of findings of the stability of infant baseline EEG asymmetry scores from 3 and 6 months of age in a more broadly middle class sample relative to previous research involving a low to middle socioeconomic status, predominately ethnic minority sample; 5) to extend the findings of the stability of infant baseline EEG asymmetry scores across ages to the stability of EEG asymmetry scores across ages in maternal-interaction contexts; and 6) to examine the association between

changes in maternal postpartum depressive symptoms and the stability of infant baseline EEG asymmetry scores across ages.

In order to accomplish these aims, we examined infant EEG asymmetry scores at 3 and 6 months of age given previous findings yielding strong support for the stability of infant EEG asymmetry scores beginning as early as 3 months of age. We hypothesized the following: 1) maternal postpartum depressive symptoms would be associated with infants' greater relative right frontal baseline EEG asymmetry scores at both ages; 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 scores specifically among infants whose mothers had more (or increasing) depressive symptoms postnatally; 3) infant frontal EEG asymmetry scores would be consistent across contexts at both 3 and 6 months of age; 4) infant baseline frontal EEG asymmetry scores would be stable from 3 to 6 months of age; 5) infant frontal EEG asymmetry scores in maternal-interaction contexts (feeding and free play) would be stable from 3 to 6 months of age; and 6) the relationship between infant baseline frontal EEG asymmetry scores at 3 and 6 months of age would be moderated by changes in maternal depressive symptoms symptom levels between those two time points.

Method

Participants

The data for this study were collected as part of the longitudinal study *Perinatal Stress and Gene Influences: Pathways to Infant Vulnerability*. Women were recruited

during pregnancy through several sources. The majority were referred by their doctors to the Women's Mental Health Program (WMHP) of the Department of Psychiatry and Behavioral Sciences at Emory University. Women who underwent clinical evaluation at the WMHP and Grady Satellite Clinic were recruited as well. In addition, women screened for research participation at the Emory Mood and Anxiety Disorders Program who were excluded for a positive pregnancy test were referred to the study. Various additional recruitment strategies were employed: annual mailing, flyers at WMHP, flyers at local obstetrics practices in the Atlanta area, periodical education of staff at the WMHP and other clinical programs at Emory about ongoing research studies, and the annual referral dinner for community clinicians, hosted by WMHP.

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 less than 16 weeks pregnant measured from last menstrual period, being between ages 18 and 45, fluency in both written and verbal English, being able to give informed consent and abide by study procedures, being able to identify the biological father of the infant, and completing at least one prenatal assessment, including the Beck Depression Inventory (BDI; A. T. Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Exclusion criteria included: active suicidality or homicidality, having psychotic symptoms, meeting DSM-IV criteria for bipolar disorder, schizophrenia, and/or currently active eating disorder, having an active substance use disorder within six months prior to last menstrual period and/or positive urine drug screen, illness requiring treatment that can

influence outcomes such as epilepsy, asthma, autoimmune disorders, and having abnormal thyroid stimulating hormone or anemia.

For this report, data were collected on 66 women and their infants. Participants ranged from 25.1 to 44.5 years of age at delivery (M = 34.3 years, SD = 3.9). Approximately 86% were married and on average had completed 16.91 years (SD = 1.88) of education. Nearly half (45.5%) of mothers were primiparous. The majority of the women were European-American (87.9%), with the remaining 7.6% being African American, 1.5% Native American, and 3.0% Asian. Excluding nine participants who were missing Hollingshead total scores (a measure of socioeconomic status), the sample was, on average, of middle socioeconomic status with M = 54.63 based on the Hollingshead scale (Hollingshead, 1975). This average score represents people who are medium level business persons or minor professionals. Of the 66 infants, 27 (41%) were female and 39 (59%) were male.

Procedure

Data were collected from the women at multiple time points throughout pregnancy and the first six months postpartum. During pregnancy, women completed an average of 5.55 BDIs, with a range from 1 to 10 times (SD = 1.87). From birth through infant age 6 months, women completed an average of 5.58 BDIs, ranging from 2 to 15 times (SD = 2.37). Women also completed the Beck Depression Inventory – Second Edition (BDI-II) (A.T. Beck, Steer, & Brown, 1997) at infant ages 3 and 6 months in order to assess for concurrent depressive symptoms. Most (n = 55; 83%) completed the BDI-II at both infant ages 3 and 6 months; an additional 11 women completed the BDI-II at either 3 or 6 months.

Mothers and infants visited the laboratory at infant ages 3 and 6 months. A total of 60 mother-infant dyads participated at 3 months and 61 at 6 months. In all, 55 mother-infant dyads completed visits at both time points. At each visit, mothers and their infants were video-recorded and infants' EEG was recorded during a 3-minute baseline, 5-minute feeding, and 5-minute free play segment. 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. Mothers were instructed not to talk to their infant during this segment of the EEG recording. During the feeding segment, the mother was allowed to breast- or bottle-feed her infant. Finally, during the free play segment, the mother was provided with toys from the laboratory to use to play with the infant and instructed to play with her child in any way she would like. The data from pregnancy and the 3 and 6 month postpartum visits were the focus of this study. At 3 months of age, 60 infants (100%) had usable baseline EEG data, 59 (98%) had usable feeding EEG data, and 57 (95%) had usable free play EEG data. All 61 infants who visited the laboratory at 6 months of age had usable data in all three segments.

Measures

EEG measures (M. A. Bell, personal communication, November 18, 2009).

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.) with electrodes in the 10/20 system pattern. After the cap was placed on the head, recommended procedures regarding EEG data collection with infants and young children were followed (Pivik, Broughton, Coppola, Davidson, Fox, & Nuwer, 1993). Following this, conductive gel provided by the cap manufacturer was placed in each site. Using a blunt tip syringe, the gel was pushed onto the scalp with the edge of a Q-tip. Electrode impedances were measured and accepted if they were below 5K ohms. The electrical activity from each lead was amplified using separate SA Instrumentation Bioamps 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.) 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, with the 16 electrode sites evenly distributed across the head (Hagemann, Naumann, & Thayer, 2001). Then, the average reference EEG data were artifact scored for eye movements and gross motor movements. 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 one-second width and 50% overlap. Power was computed for the 6 to 9 Hz frequency band. Infants and young children have a dominant frequency between 6 to 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, Malmstadt, Dolski, Kalin, Goldsmith, & Davidson, 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.

Depression measures.

Beck Depression Inventory (A. T. Beck et al., 1961). The original BDI is a self-report measure of depression symptom severity with 21 questions. Respondents are asked to answer the questions based on how they were feeling during the past week. Each item on this instrument is rated on a 4-point scale, ranging from 0 to 3. A total score is calculated by adding the ratings across items, with higher scores indicating greater severity of depressive symptoms. The scores can be interpreted as follows: 0-9 indicates 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. Beck et al., 1961).

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 were based on the past two weeks, thus paralleling the DSM-IV criteria of duration for a major depressive episode, this measure was designed to be more compatible with the DSM-IV definition of major depression. Each item on this instrument is rated on a 4-point scale, ranging from 0 to 3. A total score is calculated by adding the ratings across items, with higher scores indicating greater severity of depressive symptoms. Depression scores ranging from 0 to 13 indicate a non-depressed individual or one with 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 be depressed (A.T. Beck, Steer, & Brown, 1997). The BDI-II has been found to be both 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 been shown to have good concurrent validity with measures of both antenatal and postpartum depression (Boyd, Le, & Somberg, 2005; Steer, Scholl, & Beck, 1990) (see Table 1 for descriptive statistics of depression and EEG measures).

Results

Preliminary Analyses

Preliminary analyses were conducted to test for any associations between demographic variables and infant EEG asymmetry scores at 3 and 6 months of age. Results indicated that maternal age at delivery was not associated with any measures of infant EEG asymmetry at either 3 or 6 months of age (see Table 2). Socioeconomic status was significantly associated with infant EEG asymmetry scores during feeding and

free play only at 6 months, such that higher socioeconomic status was associated with greater relative right frontal EEG asymmetry scores (see Table 2). Independent samples t-tests revealed that infant gender was not significantly related to infant baseline EEG asymmetry scores at 3 months of age ($t(58) = .28$, $p = .78$) or 6 months of age ($t(59) = 1.51$, $p = .14$). Similarly, infant gender was not significantly related to infant EEG asymmetry scores during the feeding context at 3 months of age ($t(57) = -.30$, $p = .77$) or 6 months of age ($t(59) = .45$, $p = .65$), nor was it associated with infant EEG asymmetry scores during the free play context at 3 months of age ($t(55) = .36$, $p = .72$) or 6 months of age ($t(59) = .40$, $p = .69$). Further, findings from One-Way ANOVAs revealed that race/ethnicity was not significantly related to infant baseline EEG asymmetry scores at 3 months ($F(3, 56) = .94$, $p = .43$) or at 6 months ($F(3, 57) = 2.72$, $p = .05$); to infant EEG asymmetry scores during feeding at 3 months ($F(3, 55) = .73$, $p = .54$) or at 6 months ($F(3, 57) = .10$, $p = .96$); or to infant EEG asymmetry scores during free play at 3 months ($F(3, 53) = 1.36$, $p = .26$) or at 6 months ($F(3, 57) = .40$, $p = .75$). Finally, the number of prenatal weeks that infants were exposed to maternal antidepressant use was only significantly associated with 6 month baseline infant EEG asymmetry scores and not to scores in either of the other contexts at 6 months, or to scores in any of the contexts at 3 months of age (see Table 2). Thus none of these variables were controlled for in the analyses.

Hypothesis Testing

Hypothesis 1. The first aim of the study was to test for the replication of the finding of an association between maternal postpartum depressive symptoms and infants' greater relative right frontal EEG asymmetry scores at both ages. We tested this

hypothesis by examining associations between EEG asymmetry scores and both maternal depressive symptom levels at the time of the EEG measure and a cumulative, area under the curve (AUC) measure of depressive symptom levels over the first 3 or 6 months postpartum. Contrary to prediction, Pearson product moment correlations revealed no significant association between maternal concurrent depressive symptom levels (BDI-II) at infant age 3 months and infant baseline frontal EEG asymmetry scores at 3 months of age ($r(57) = -.05$, $p = .36$). Similarly, maternal postpartum depressive symptoms measured at multiple time points from birth through infant age 3 months (BDI AUC postpartum) and infant baseline EEG asymmetry scores at 3 months of age were not significantly associated ($r(57) = .02$, $p = .43$). That is, contrary to prediction, postpartum depressive symptoms were not significantly associated with infant 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) ($r(56) = -.18$, $p = .09$), or maternal postpartum depressive symptoms from birth through infant age 6 months (BDI AUC postpartum) ($r(59) = -.06$, $p = .33$). Again, contrary to prediction, postpartum depressive symptoms were not significantly associated with infant EEG asymmetry scores at 6 months of age.

Hypothesis 2. We also examined the additive and interactive effect of maternal prenatal depressive symptoms and postpartum depressive symptoms in predicting infant baseline EEG asymmetry scores at 3 and 6 months of age. 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 scores specifically among infants whose mothers had more (or increasing) depressive symptoms postnatally. Regression analyses revealed that neither maternal prenatal nor postpartum depressive symptoms separately predicted 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 EEG asymmetry scores at either 3 or 6 months of age. There was a trend for the interaction between prenatal and postpartum maternal depressive symptoms through infant age 3 months predicting infant baseline frontal EEG asymmetry scores at 3 months of age (see Table 3).

At infant age 6 months, maternal prenatal and postpartum depressive symptoms through infant age 6 months interacted to predict infant baseline frontal EEG asymmetry scores (see Figure 1). Among women with low postpartum depressive symptom levels, prenatal depressive symptoms and infant EEG asymmetry scores were not significantly associated ($r(29) = .12$, $p = .27$). In contrast, prenatal depressive symptoms and infant EEG asymmetry scores were significantly associated among women with high postpartum depressive symptoms ($r(25) = -.46$, $p < .01$).

Hypotheses 3, 4, and 5. Our third hypothesis was that infant EEG asymmetry scores would be consistent across contexts at both 3 and 6 months of age. Results of Pearson product moment correlations revealed significant positive associations between all three contexts at both ages (see Table 4). Our fourth hypothesis was that infant baseline EEG asymmetry scores would be stable from 3 to 6 months of age. Pearson correlations revealed that baseline frontal EEG asymmetry scores from 3 to 6 months of

age were significantly associated ($r(53) = .26$, $p < .05$), such that greater relative right frontal EEG asymmetry at 3 months of age was associated with greater relative right frontal EEG asymmetry at 6 months of age. Our fifth hypothesis was that infant EEG asymmetry scores in maternal-interaction contexts (feeding and free play) would be stable from 3 to 6 months of age. Contrary to our hypothesis, only infant frontal EEG asymmetry scores in the free play condition were significantly associated across age ($r(50) = .30$, $p < .05$), such that greater relative right 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 ($r(52) = .05$, $p = .37$).

Hypothesis 6. Finally, we hypothesized 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. In order to test this association, a correlation between the difference score in maternal concurrent depressive symptoms and the difference score in infant EEG asymmetry scores from 3 to 6 months was conducted. Results revealed 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 ($r(50) = -.16$, $p = .27$), though the association was in the predicted direction, with increasing maternal depressive symptoms associated with decreasing infant EEG asymmetry scores. A regression was run in order to examine this association in a different way. 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 revealed that only infant 3 month 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 Table 5).

Discussion

The current longitudinal study investigated the relationship between maternal prenatal and postpartum depressive symptoms and infant EEG asymmetry scores measured at 3 and 6 months of age in three contexts (baseline, play with mother, and feeding by mother). The first hypothesis was an attempt to extend upon previous findings, which indicated that there was a significant negative association between both concurrent maternal diagnosed depression and depressive symptoms and infants' frontal EEG asymmetry scores at various time points throughout infancy (Dawson et al., 2001; Diego et al., 2006; Jones, Field, Davalos, et al., 1997; Jones, Field, Fox, et al., 1997). Our findings did not support this hypothesis. Instead, the current findings indicate that neither maternal concurrent depressive symptoms nor a more comprehensive measure of maternal postpartum depressive symptoms (collected during the period from the infant's birth through the time of the lab visit) were significantly associated with infant EEG asymmetry scores in our sample.

There are several possible explanations for the lack of significance in our findings. For one, the current sample was more broadly middle class and predominately Caucasian, as compared to samples described in the studies from the Field lab (previously cited), which included low to middle socioeconomic, primarily ethnic minority samples.

Although further analyses revealed that there were no significant associations between maternal age, SES, or race, and infant EEG asymmetry scores in our sample, 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 those studies. A second possible explanation for the different findings is that previous studies included community samples and did not select mothers based on their history of depression, whereas our sample was selected based on having a lifetime history of depression or anxiety. Thus the common history of depression in the women in the current sample may better explain the infant EEG asymmetry scores, while the postpartum depressive symptoms may not add additional variance. Future studies might compare more diverse samples, as well as those with and without histories of depression to help further determine what may account for the differences in findings.

The second specific aim of the current study was to examine the potential additive or interactive associations between both maternal prenatal and postpartum depressive symptoms 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 (as discussed above) or the relationship between maternal prenatal depressive symptoms and newborns' infant 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).

We hypothesized that maternal prenatal and postpartum depressive symptoms would either additively or interactively predict significant variance in infant EEG asymmetry scores. Neither hypothesis was supported at infant age 3 months, though the interaction term approached significance. At infant age 6 months, there was not a significant additive effect of maternal prenatal and postpartum depressive symptoms in predicting infant EEG asymmetry scores. However, our hypothesis that maternal prenatal and postpartum depressive symptoms would interact to predict infant EEG asymmetry scores at 6 months was supported. In particular, we found that higher levels of prenatal depressive symptoms were associated with greater relative right frontal EEG asymmetry scores specifically among infants whose mothers had higher levels of depressive symptoms postnatally. In contrast, the relationship 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 suggest a moderating role of postpartum depressive symptom levels in the association between prenatal depressive symptom levels and infant EEG asymmetry scores at 6 months of age. If the interaction effect is also found at 3 months of age with a larger sample, then findings would suggest that an interactive rather than additive effect of maternal prenatal and postpartum depressive symptoms is more predictive of infant EEG asymmetry scores. Alternatively, the findings may simply suggest that infant EEG

asymmetry scores at 3 and 6 months of age have different predictors; in particular, the effects of maternal prenatal and postpartum depressive symptoms on infant EEG asymmetry scores appear to be limited to these scores at 6 months of age.

The third specific aim was to extend the findings of the consistency of infant EEG asymmetry scores across contexts down to younger infants. Consistent with Dawson's findings in older infants (Dawson et al., 2001; Dawson et al., 1997), the current findings supported our hypothesis that infant EEG asymmetry scores would be consistent across contexts at both 3 and 6 months of age. These findings suggest that consistency of infant EEG patterns across contexts emerges even earlier than had previously been shown.

As infant baseline EEG asymmetry scores have been found to be stable across ages (Diego et al., 2006; Jones, Field, Davalos, et al., 1997; Jones, Field, Fox, et al., 1997), the current study tested for the replication of these findings of stability in baseline EEG asymmetry scores, as well as extend these findings to an examination of the stability of EEG asymmetry scores across ages to maternal interaction contexts. With regards to this aim, the findings supported our hypotheses that infant EEG asymmetry scores during baseline and free play would be stable; specifically, greater infant EEG asymmetry scores at 3 months were significantly associated with greater infant EEG asymmetry scores at 6 months in both contexts. In contrast, infant EEG asymmetry scores during feeding were not stable across ages, which may reflect important differences in the feeding context as compared to the baseline and free play contexts. When all three findings, as well as those of the significant association of infant EEG asymmetry scores across contexts at both ages, are considered together, the associations that were significant and demonstrated stability were moderate (in effect size) at best. Further, in our sample, only 34 infants

(62%) demonstrated a stable pattern of either left or right frontal EEG asymmetry from 3 to 6 months of age, whereas the other 38% (n=21) showed a change from either left to right or right to left frontal asymmetry across ages. This places even further importance on exploring the variables (in our case, maternal depressive symptoms), which are associated with these patterns at multiple time points during infancy in order to determine for which subsets of infants these associations might be stronger.

Finally, we examined the association between changes in maternal postpartum depressive symptoms and the stability of infant baseline EEG asymmetry scores from 3 to 6 months of age. Contrary to our prediction, the relationship 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 increases 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 exploration of this question indicated similar findings, such that only 3 month infant EEG asymmetry scores significantly predicted 6 month infant EEG asymmetry scores, while maternal postpartum depressive symptoms did not. The lack of significance may be accounted for by sample size, or by the fact that the monthly trajectory of maternal depressive symptoms from infant age 3 to 6 months was not examined. Therefore, a change in maternal concurrent depressive symptoms from 3 to 6 months may not accurately reflect the exposure that infants are receiving in the months between these assessments. More frequent sampling of mothers' depressive symptom levels may be important to help explicate the observed changes in infant EEG

asymmetry scores across ages. Also, there may be other variables which are predicting this association and are needed in order to understand which infants' scores stay similar and which diverge.

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

Overall, the findings of the current study suggest that infant EEG patterns are consistent across contexts even as early as 3 months of age, and that they are stable from 3 to 6 months of age, although only in baseline and free play contexts. Taken together, these findings provide evidence for individual variability in infant EEG patterns. The current findings also indicate that, at least in predicting infant EEG asymmetry scores at 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.

Strengths, Limitations, and Future Directions

The current study had several notable strengths, the first of which was the longitudinal study design, during which data was collected beginning in pregnancy and through 6 months postpartum. Depressive symptoms were measured at multiple time points throughout and infant EEG was collected at two ages and in three contexts at each age. Also, the sample size was comparable to other studies of infant EEG. It consisted of predominately middle class, Caucasian women and their infants, which allowed for the examination of the association between maternal depressive symptoms and infant EEG asymmetry scores among women with histories of depression, while controlling for stressors such as poverty, maternal age, SES, etc. In conjunction, however, is the limitation that findings from this sample may not be generalizable to non-clinical or more diverse samples.

These data are part of an ongoing study, with which planned analyses are to re-test these hypotheses with a larger sample. The complete dataset will allow us to test the role concurrent infant affect and parenting quality, which may help to explain associations between maternal depressive symptoms and infant EEG asymmetry scores. Finally, given the preliminary findings of the interactive role of prenatal and postpartum maternal depressive symptoms in predicting infant EEG asymmetry scores at 6 months of age, future research should measure the trajectories of maternal depressive symptoms in order to gain a clearer understanding of the impact of changes in maternal depressive symptoms on changes in infant EEG asymmetry scores.

Conclusion

The current study demonstrated that although previous research would initially suggest that infant EEG asymmetry scores represent trait-like markers of vulnerability, in

actuality there is much variability in infant EEG patterns. The importance of both maternal prenatal and postpartum depressive symptoms was investigated, and results suggested that there was a moderated relationship between these two variables, such that higher levels of maternal prenatal depression was associated with greater relative right frontal infant EEG asymmetry scores only in the context of higher levels of postpartum depressive symptoms. Further research is needed in order to investigate the mechanisms by which maternal 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 infant EEG patterns.

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Table 1

Descriptive Statistics of Maternal Depression and Infant EEG Variables

Variables	*M*	*SD*	Minimum	Maximum	N
Prenatal depression AUC	361.72	268.18	40.91	1207.82	61
Postpartum depression AUC through 3 months	112.83	97.19	0	519	65
Postpartum depression AUC through 6 months	222.66	171.01	0	926.80	65
Concurrent depression at 3 months	9.28	8.67	0	44	60
Concurrent depression at 6 months	9.02	8.66	0	43	60
Baseline EEG 3 months	-.003	.26	-.89	1.02	60
Feeding EEG 3 months	-.03	.18	-.46	.39	59
Free Play EEG 3 months	.04	.25	-.58	.78	57
Baseline EEG 6 months	.12	.28	-.40	1.15	61
Feeding EEG 6 months	.05	.20	-.45	.59	61
Free Play EEG 6 months	.17	.34	-.31	1.86	61

Note. AUC stands for Area under the Curve.

Table 2

Intercorrelations among Maternal Demographic Variables and Infant EEG Asymmetry Scores in All Three Contexts at Both Ages

	Mother age at delivery	Hollingshead total	Prenatal antidepressant exposure number of weeks
3 month EEG			
Baseline	.06	-.15	.10
Feeding	-.22	.01	-.04
Free Play	-.07	.06	.08
6 month EEG			
Baseline	-.02	-.03	-.31*
Feeding	-.21	-.32*	-.24
Free Play	-.05	-.27*	-.10

** $p < .01$ level (2-tailed). * $p < .05$ level (2-tailed).

Table 3

Summary of Hierarchical Regression Analyses for Maternal Depression Variables Predicting Infant EEG Asymmetry Scores

	Baseline frontal infant EEG asymmetry scores			
	3 months		6 months	
Predictor	ΔR^2	β	ΔR^2	β
Step 1	.002		.018	
Prenatal depression AUC		.039		-.133
Step 2	.008		.004	
Postpartum depression AUC		.128		.092
Step 3	.059		.083*	
Interaction term		-.730		-.804*
Total R^2	.068		.105	
n	54		58	

* $p < .05$ level. ** $p < .01$ level.

Table 4

Intercorrelations among Infant EEG Asymmetry Scores in All Three Contexts at Both Ages

	1	2	3	4	5	6
1. Baseline 3 months	__	.30*	.44**			
2. Feeding 3 months		__	.52**			
3. Free Play 3 months			__			
4. Baseline 6 months				__	.34**	.31**
5. Feeding 6 months					__	.40**
6. Free Play 6 months						__

** $p < .01$ level (1-tailed). * $p < .05$ level (1-tailed).

Table 5

Summary of Hierarchical Regression Analyses for Maternal Depression Variables and 3 Month Infant EEG Asymmetry Scores Predicting 6 Month Infant EEG Asymmetry Scores

	Baseline infant EEG asymmetry scores at 6 months	
Predictor	ΔR^2	β
Step 1	.095	
3 month infant baseline EEG asymmetry scores		.281*
Postpartum depression AUC through 3 months		-.147
Step 2	.018	
Concurrent depression at 6 months		.-.226
Total R^2	.114	
n	53	

* $p < .05$ level. ** $p < .01$ level.

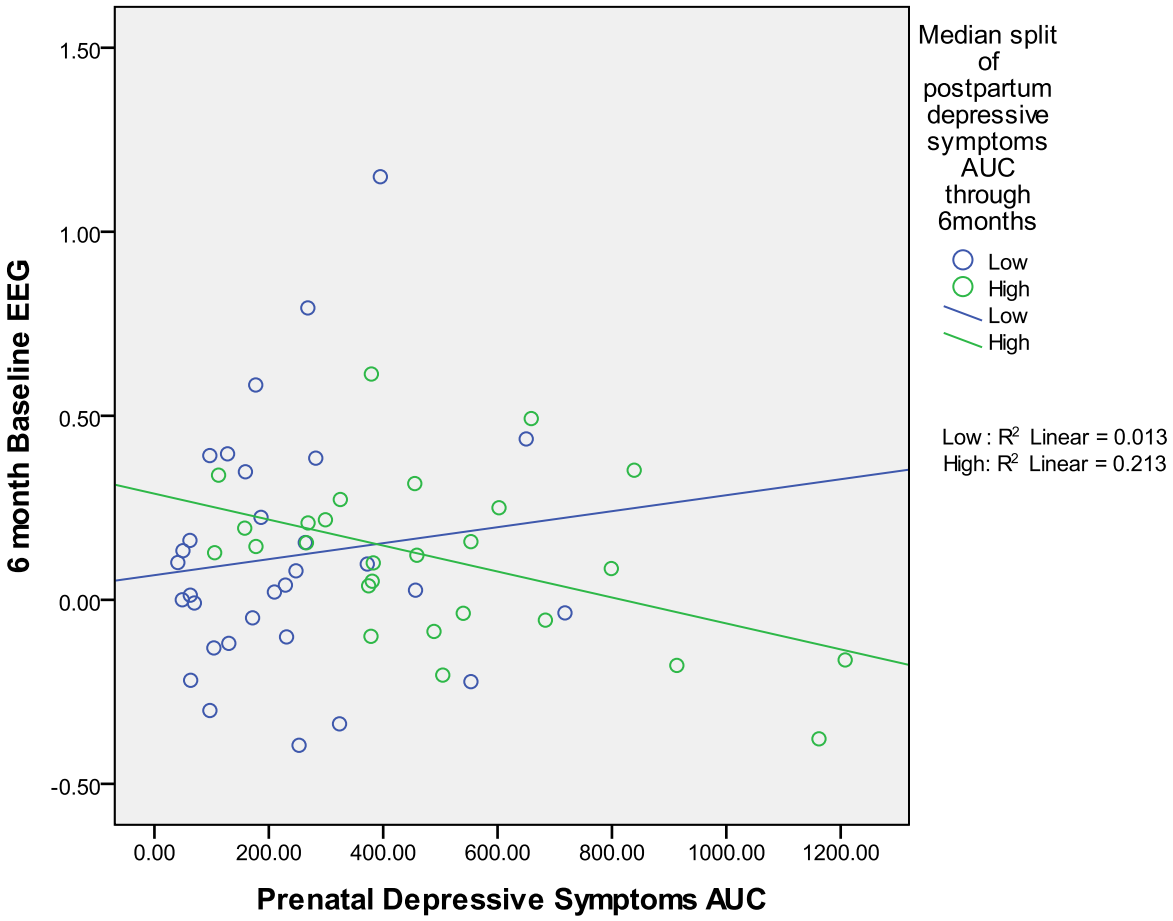

Figure 1. The association between infant 6 month baseline EEG asymmetry scores and maternal prenatal and postpartum depressive symptoms. The association between maternal prenatal depressive symptoms and infant baseline EEG asymmetry scores at 6 months of age is moderated by levels of maternal postpartum depressive symptoms. In particular, infants whose mothers have higher levels of both prenatal and postpartum depressive symptoms display the greatest relative right frontal EEG asymmetry.