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The Association between EEG Asymmetry and Negative Affectivity in Infants of Depressed Mothers

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Abstract

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The present study extended prior research on the effect of maternal depression on the infants' vulnerabilities to develop depression by examining predictors of vulnerabilities in two domains of functioning. Recently, research showed that infants who index more than a single vulnerability are most likely to develop psychopathology, particularly if the multiple vulnerabilities cross biological and affective domains. We considered how prenatal and postpartum depression might be associated with infants' interacting vulnerabilities of electroencephalogram (EEG) asymmetry and the temperament domain of negative affectivity, and whether infants whose mothers are depressed both pre- and postnatally may be at highest risk. Participants were 46 women with a history of Major Depressive Episode or an anxiety disorder, who were studied during pregnancy or postpartum through 3 months and their 3-month-old infants. When the infants were 3-months-old, mothers completed the Infant Behavior Questionnaire-Revised (IBQ-R) and infant EEG activity was recorded. For the sample as a whole, the correlation between relative right frontal EEG activity and negative affectivity was small and not statistically significant. In addition, concurrent maternal depressive symptom levels did not have an effect on the degree of association between the two variables. However, infants of mothers who were either prenatally depressed or postpartum depressed showed a small, albeit non significant correlation in the expected direction, consistent with the idea that exposure to perinatal depression may increase the likelihood of interacting vulnerabilities to developing depression.

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Table of Contents

The Association between EEG Asymmetry and Negative Affectivity in Infants of Depressed Mothers

Major Depressive Disorder (MDD) is a psychopathological disorder that is prevalent in the general population. MDD in women is twice as common as in men (Accortt, Freeman, & Allen, 2008) and the lifetime prevalence rate for women is approximately 21% (Kessler et al., 1994). Similar rates of depression are found during pregnancy and postpartum and some women experience depression at both periods. Along with these high rates are theoretical and empirical reasons to be concerned about children of mothers with perinatal depression, and especially children who are exposed both pre- and postnatally. Goodman and Gotlib's (1999) integrative model to understand maternal depression as a risk factor in the development of psychopathology in the child proposes four mechanisms as pathways to the development of vulnerability. They are (a) heritability of depression, (b) innate dysfunctional neuroregulatory mechanisms, (c) exposure to negative maternal cognitions, behaviors, and affect, and (d) stressful context of a child's life. The researchers proposed that these mechanisms are likely to interact with each other, further increasing the vulnerability to the development of psychopathology in the offspring.

Recently, researchers have expanded on the notion of interacting vulnerabilities by suggesting that infants who manifest more than a single vulnerability are most likely to develop psychopathology, particularly if those multiple processes cross either affective/behavioral or biological domains (Ingram & Price, 2001). Thus, examining more than one domain of infant functioning may reveal those who are most vulnerable. In this paper, we consider how antenatal depression and, later, postnatal depression might

be associated with infants' interacting vulnerabilities of negative affectivity and particular patterns of brain activity, and whether infants whose mothers are depressed both pre- and postnatally may be at highest risk for these dual vulnerabilities.

Although some studies have shown associations between antenatal depression and both negative temperament (McGrath, Records, & Rice, 2008) and relative right frontal electroencephalogram (EEG) asymmetry (Fernandez et al., 2004; Field, Fox, Pickens, & Nawrocki, 1995; Jones, Field, Davalos, & Pickens, 1997), further examining their interaction in infants of perinatally depressed mothers might reveal essential steps in the pathway from risk to disorder (Gottesman & Gould, 2003). It is particularly important to focus on infants of mothers with depression because genetic and environmental influences of maternal depression may amplify infants' vulnerability to depression. We expected that higher levels of depression symptoms during pregnancy and postpartum would be associated with both more negative affectivity and greater relative right frontal EEG asymmetry.

EEG as a Vulnerability to Depression

Researchers have been interested in EEG in infants of depressed mothers given that EEG is a promising index of depression vulnerability in infants. Support for this point comes, first, from studies of adults, in whom left frontal hemisphere activation has been found to represent positive affect and approach-related behavior whereas right frontal activation has been found to be related to negative affect and withdrawal-related behavior (Davidson, 1998). This association between the left and right hemisphere activation and approach and withdrawal has been replicated in many studies (Coan &

Allen, 2003; Davidson, 1998; Fox, 1991; Harmon-Jones & Allen, 1998; Mathersul, Williams, Hopkinson, & Kemp, 2008).

Second, several researchers have shown the association between relative right frontal EEG activation and general negative emotions in adults. For instance, greater relative right frontal activation was correlated with self-reports of more negative emotions in reaction to negative affect eliciting film clips (Wheeler, Davidson, & Tomarken, 1993). Similarly, individuals with relative right frontal EEG asymmetry rated more negative responses to films that elicited both positive and negative affect, with a particularly strong relation between frontal asymmetry and fear responses (Tomarken, Davidson, & Henriques, 1990). The mood ratings were controlled, indicating that relative right frontal asymmetry significantly predicted more negative ratings regardless of current mood. Also, mothers who scored above the cut-off score for depression on the Center for Epidemiological Studies Depression (CES-D) Inventory displayed relative right frontal EEG asymmetry and this was positively associated with greater inhibition scores on the Behavioral Inhibition and Behavioral Approach System Questionnaire (BIS/BAS) (Diego, Field, & Hernandez-Reif, 2001a). Positive and negative affectivity/emotionality are known to be indexes of the approach and inhibition systems, with inhibition, or negative emotionality, defined as deficits in approach motivation and the inability to experience positive affect, which are the characteristics of depression (Putnam & Stifter, 2005). Overall, relative right frontal EEG asymmetry is associated with negative mood in adults.

Third, studies of adults further show that relative right frontal EEG activation is associated with depression symptoms or clinically diagnosed depression in community

and clinical samples. For example, undergraduate students and mothers who exceeded the clinical cut-off to indicate high levels of depression symptoms on self-report measures such as the Beck Depression Inventory (BDI) and Center for Epidemiologic Studies Depression Scale (CES-D) showed greater relative right frontal activation at a resting baseline than those with low scores on the BDI and the CES-D (Diego, Field, & Hernandez-Reif, 2001b; Schaffer, Davidson, & Saron, 1983). Correlational studies similarly found EEG asymmetry values to be associated with depression scores, such that women with higher depressive symptoms showed greater right frontal EEG activity (Diego et al., 2001b). That is, regardless of research design, EEG asymmetry has been found to be significantly associated with depressive symptoms in adults. Similarly, samples of adults who were clinically diagnosed with depression, relative to controls, were found to have greater relative right frontal EEG activity (Allen, Urry, Hitt, & Coan, 2004; Gotlib & Rosenfeld, 1998; Henriques & Davidson, 1990). Thus, relative right frontal brain activation is associated with higher levels of depressive symptoms in community samples and with diagnosed depression in clinically depressed samples.

Fourth, researchers have found significantly greater relative right frontal activation during the resting state not only in currently depressed adults but also in remitted depressed adults compared to controls (Davidson, 1998). Depressed adults clinically diagnosed through Research Diagnostic Criteria (RDC) who were free from depressive symptoms for at least a year showed relative right frontal EEG activity at baseline compared to never-depressed adults, despite showing no difference in their self-reported emotional states which measured interest, amusement, happiness, fear, sadness, disgust, and anger (Henriques & Davidson, 1990). Similarly, researchers categorized

college students as currently depressed or previously depressed based on the Inventory to Diagnose Depression (IDD), the lifetime version of the IDD, and the Structural Interview for the DSMIII-R (SCID) and found that both groups showed higher relative right frontal EEG activity compared to the never-depressed group and also displayed no significant difference in relative right frontal EEG activity from one another (Gotlib & Rosenfeld, 1998). These findings indicate that the relationship between depression and right frontal EEG asymmetry continues beyond depression episodes into the period of remission. While one hypothesis about the relationship is that EEG patterns remain after a depressive episode, it is also possible that the asymmetry is a stable trait marker of depression vulnerability, which may have been present even before the depressive episodes. Indeed, evidence for the stability of brain asymmetry has led some researchers to conclude that frontal asymmetry is a trait-marker of depression that is stable, regardless of the presence or absence of active depression (Coan & Allen, 2004). Thus it is compelling to examine the earliest occurrence of such a trait marker and observe if this brain pattern is present beginning in infancy in infants of depressed mothers.

Fifth, research has shown infants in the general population who display relative right frontal EEG asymmetry to exhibit negative emotionality. A correlational study between EEG asymmetry and behavior during maternal separation in 10-month-old infants found that those with right frontal EEG asymmetry patterns had a greater proneness to cry and had higher levels of distress (Davidson & Fox, 1989). In a longitudinal test of these associations, 4-month-old infants who were classified as high on motor activity and negative affect exhibited greater relative activity in the right frontal hemisphere at 9-months of age and also displayed inhibited behavior toward novelty at

14-months of age, compared to infants characterized as high on motor activity and positive affect and those who were low on motor activity and affect (Calkins, Fox, & Marshall, 1996). In another longitudinal study, EEG asymmetry measured at 7-months predicted 12-month-olds' greater amount of crying in response to separation with the mother (Fox, Bell, & Jones, 1992). Additionally, 3- to 6-month-old infants of mothers who scored higher than the BDI cut-off score displayed right frontal EEG asymmetry compared to a non-depressed group that scored lower, and when these infants were observed again at 3 years, they displayed the same asymmetry pattern and simultaneously showed inhibited behaviors, such as staying in close proximity to the mother, showing distress to novel events and people, and failing to approach and reach out for toys (Jones et al., 1997). These studies show that infants with relative right frontal EEG asymmetry are prone to exhibit withdrawal behaviors, consistent with the studies that have shown the right hemisphere to be associated with emotions related to withdrawal and inhibition.

Whether through genetics or fetal or postpartum exposures, infants of depressed mothers are vulnerable to the later development of depression. Therefore, based on the notion that greater relative right frontal EEG asymmetry may be a trait marker for depression, a few researchers have examined EEG patterns in infants of depressed mothers. Greater relative right frontal EEG activation in a baseline condition has been found in infants of concurrently depressed mothers at one week, one month, three months, and 3- to 6-months of age, compared to same-age infants of mothers low in depressive symptoms (Diego, Jones, & Field, 2009; Field et al., 1995). In addition, Dawson and colleagues extended these findings to 11- to 17-month-old infants of postpartum depressed mothers and also found the same pattern during mother-infant interaction

compared to infants of non-depressed mothers (Dawson, Frey, Panagiotides, Osterling, & Hessl, 1997a; Dawson, Panagiotides, Klinger, & Spieker, 1997b). Further, in a longitudinal study, seven out of eight infants of mothers with high depressive symptoms who had displayed relative right frontal EEG activity at 3- to 6-months of age, when observed again at 3 years of age, continued to show the same EEG pattern (Jones et al., 1997). This study suggested that EEG asymmetry is a stable brain pattern until the toddler years in infants of mothers with high depressive symptoms. Along with evidence for stability of EEG asymmetry in remitted depressed adults, these findings show the consistency of this asymmetrical brain pattern in both infants and adults who once had depression. This in turn supports the theory that relative right frontal EEG asymmetry is an indicator of vulnerability to the later development of depression in infants.

In summary, evidence supporting the notion of EEG asymmetry as a stable index of vulnerability to depression is accumulating across studies demonstrating EEG asymmetry in community samples of adults with depressive symptoms, clinically diagnosed adults, remitted depressed adults, and infants of mothers with high depressive symptoms. In terms of the latter, however, studies have been limited to depression in the postpartum. It is important to note that a variety of risk factors associated with maternal depression, such as factors in the postpartum environment, genetic vulnerabilities, and prenatal differences in the intrauterine environment, may mediate the relationship between maternal depression and infant's psychophysiological responses, in this case, EEG brain patterns (Dawson et al., 2001). Given that prenatal depression occurs at least as frequently as postpartum depression (Accortt et al., 2008; Weissman & Olfson, 1995) and that prenatal depression is a strong predictor of postpartum depression (Beck, 1996a;

Rahman & Creed, 2006), many infants who are exposed to postpartum depression are likely to have also been exposed to depression during their fetal development. Thus it is important to extend the findings on postpartum depression to examine the link between prenatal depression and EEG asymmetry.

Temperament as a Vulnerability to Depression

While frontal asymmetry is considered a biological marker for depression or a vulnerability to the later development of depression, temperament offers a more behavioral approach to conceptualizing and measuring vulnerabilities to the later development of psychopathology, although temperament also has a strong biological basis. Temperament is defined as individual differences in emotional, motor, and attentional reactivity and self-regulation (Gartstein & Rothbart, 2003) which are affected by the interaction of heredity and life experiences (Rothbart, 1981). Of particular interest as a vulnerability to the later development of depression is the temperament construct of Negative Affectivity, one of three temperament factors identified by Rothbart, along with Surgency/Extraversion and Affiliation or Orienting/Regulation.

Why is negative affectivity important? Studies have shown an association between this domain in children and the later development of psychopathology. Several theorists proposed that early individual trait differences are similar to later personality and social development in children and adults, with direct relevance to the later development of psychopathology in childhood and adolescence (Rothbart & Bates, 1998; Shiner & Caspi, 2003). Negative affectivity (on infant and childhood measures) has strong conceptual similarities to the dimension of neuroticism/negative emotionality (N/NE), one of the Big Five domains of adult personality that is most prevalent in

depression, which encompasses characteristics such as negative self-perception and experiencing negative emotions (Digman & Inouye, 1986; Watson & Clark, 1984). Supporting this idea, researchers theorized that N/NE is a basic factor for all disorders within the Diagnostic and Statistical Manual of Mental Disorders (DSM) but is most strongly linked with depression whereas Extraversion/Positive Emotionality (E/PE) is also significantly and modestly present in mood disorders, with low E/PE being related to depression (Mineka, Watson, & Clark, 1998; Watson, Gamez, & Simms, 2005). Thus negative affectivity in infants may act as a precursor to negative emotionality or neuroticism, with its strong link to depression.

Based on the emphasis on the influence of life experiences, temperament researchers have investigated the influence of postpartum environment on the infant, particularly with the assumption that maternal depression may disturb mother-child interactions (Dawson et al., 2001; McGrath et al., 2008). Depressed mothers may fail to provide stimulation for her infant's emotional development or respond according to her infant's needs (Goodman & Gotlib, 1999). This consistent, repeated behavior of the mother would lead to infant withdrawal or increase in negative affect (Tronick & Gianino, 1986). These theories and findings may explain the results of empirical studies that have shown high negative affectivity in infants of postpartum depressed mothers. For instance, in a longitudinal study which examined maternal depression and anxiety during the postpartum period, women who were depressed on the second post-birth day and scored high on depressive symptoms had infants with more negative emotionality compared to infants of anxious mothers and controls who reported low anxiety and depressive symptoms on the State-Trait Anxiety Inventory and BDI at 9-months of age (Feldman et

al., 2009). Whiffen and Gotlib (1989) found that 2-month-old infants of mothers with postpartum depression had difficult temperament, with mothers reporting longer and more frequent crying in these infants. Negative affectivity's association with maternal depressive symptoms was also found in school-aged children (M = 12.9 years) and also at a time point seven months later (Lonigan, Phillips, & Hooe, 2003). Also, Beck (1996b) conducted a meta-analysis using 17 studies to determine the relationship between infant temperament and postpartum depression during the first year of the infant's life. All studies used depression symptom scales, such as the BDI, CES-D, Edinburgh Postnatal Depression Scale, and others. With a 95% confidence interval ranging from 0.261 to 0.369, a significant relationship was found between infant temperament and postpartum depression. Together, the studies show that postpartum maternal depression is significantly associated with negative infant temperament. Although genetics is known to play a role (Rothbart & Bates, 1998), the findings suggest that maternal postpartum depression may negatively influence the infant's temperament as a function of a deficient rearing environment.

Concerns about genetics or prenatal environmental exposures associated with depression have led researchers to also study negative affectivity in infants of antenatally depressed mothers to determine the extent to which prenatal depression is associated with negative affectivity in infants. For instance, Davis et al. (2004) found that elevation in prenatal maternal depression was associated with an increase in behavioral inhibition in infants independent of the mother's postpartum depression. A few years later, Davis and colleagues (2007) found an increase in negative affectivity associated with prenatal exposure to maternal depression. They proposed that the intrauterine environment put

forth ‘programming’ effects on fetuses of depressed mothers, a concept theorized by Nathanielsz (1999) indicating that a stimulus or event during the critical development period of the fetus has long-lasting influence. Huot, Brennan, Stowe, Plotsky, and Walker (2004) also revealed that maternal depression during pregnancy predicted negative affectivity in offspring. These studies support temperament’s strong biological basis and that negative temperament, in particular, is developed through influences of prenatal maternal depression on the intrauterine environment.

Evidence for a biological basis may also be shown through findings of stability from infancy to childhood (Propper & Moore, 2006). Neonatal distress and negative affectivity were each correlated with levels of distress in infants at 9-months of age (Matheny, Riese, & Wilson, 1985) and with levels of negative affectivity at 15-months of age (di Pietro, Larson, & Porges, 1987). Some researchers have studied negative affectivity in infants and young children to determine associations with later indices of psychopathology. For example, temperament has been found to be a mediator of associations between depression in parents (mothers or fathers) and the later development of depression in offspring (Bruder-Costello et al., 2007). Also, when measured in a general population sample of 3- and 12-month-old infants, the negative affectivity domain was found to be predictive of depressive symptoms approximately 14 months later (Gartstein & Bateman, 2008). Overall, studies that involved prenatally depressed mothers and stability studies support the biological basis for temperament development in the infant.

Although temperament has a strong biological basis, the behavioral outcome of infants depends on genes and the environment in operation together (Rothbart & Bates,

1998). However, it is unknown to what extent genetics or environment contributes to the formation of infant temperament. Zuckerman (1995) stated that individuals do not inherit personality traits or behavior mechanisms, but inherit chemical templates that produce and regulate proteins that build nervous systems, neurotransmitters, enzymes, and hormones. However, these templates do not define individual differences in temperament. Temperament, which later on develops into personality, is formed through the interaction between genetics and environment; it is this interaction that determines the temperament outcome. It is also difficult to determine which of the two, genetics that reflect depression or a disturbed postpartum environment which lacks physical and emotional support, contributes more towards the infant becoming vulnerable to develop depression in the future. Therefore, further research is needed to determine the prenatal and postpartum influences of temperament.

Association between EEG and Temperament in Infants of Depressed Mothers

Given the support for both EEG and temperament as indicators of vulnerability to depression, it is not surprising that researchers have begun to examine the associations between the two indices. A few studies have found an association between EEG asymmetry and negative temperament. For example, 9-month-old infants who were rated by their mothers as temperamentally distressed based on the Infant Behavior Questionnaire (IBQ) showed greater relative right frontal brain activity during a baseline condition as well as during infant-directed speech conditions, with the exception of a surprise condition (Santesso, Schmidt, & Trainor, 2007). Schmidt (2008) found that infants who had stable right EEG asymmetry exhibited higher scores on the IBQ Fear subscale, one of the subscales under the Negative Affectivity domain. Although many

EEG studies have shown an association between behaviors of withdrawal (such as distress, crying, and inhibition) and relative right frontal EEG asymmetry in infants of the general population, no study has studied the association between EEG asymmetry and the Negative Affectivity domain itself, especially in infants of depressed mothers. According to Derryberry and Rothbart (2001), the Negative Affectivity domain is the element of temperament that comprises negative emotions such as fear, distress, anger, anxiety, and sadness. Although withdrawal behaviors may seem to be indices of negative affectivity, the withdrawal (and approach) system is based on motivational aspects of the individual's response whereas negative affectivity is a part of the emotionality aspect of temperament (Rothbart & Bates, 1998). Rothbart and Bates stressed temperament to be an evolutionarily conserved affective-motivational system. Thus, withdrawal reflected by EEG asymmetry and negative temperament are two constructs that constitute temperament that differ in structure.

Based on the fact that negative affectivity is a precursor to N/NE in adults and it is the major personality trait for depression, it is important to know the extent to which EEG asymmetry and negative affectivity are associated. Findings of an association between EEG asymmetry and negative temperament in infants may suggest that the two vulnerabilities reflect an underlying process that includes both physiological/motivational and behavioral/affective domains of functioning. Thus it is particularly important to identify predictors of not only each of these individual vulnerabilities but also of their co-occurrence.

Yet the extent to which EEG asymmetry and negative temperament are correlated in infants of depressed mothers has rarely been the subject of study. Thus we extended

the findings on the EEG and withdrawal/inhibited behavior in infants of the normative population (Calkins et al., 1996; Santesso et al., 2007) to hypothesize that relative right frontal EEG asymmetry and negative temperament would be significantly associated in infants of depressed mothers.

Influence of Prenatal and Postpartum Depression on the Infant

Studies of both EEG and temperament have stressed the influence of the prenatal environment on the physiological and psychological development of the infant. Vulnerabilities to developing depression may be genetically linked through the influence of prenatal maternal depression while postpartum maternal depression may negatively influence the environment, acting as an additive factor that would yield even worse outcome in infants. This is evident in temperament studies. For instance, infants of mothers who had both prenatal (measured during the third trimester of pregnancy) and postpartum depression had more difficult temperament at 2-months of age and later at 6-months of age compared to infants with non-depressed mothers at both times (McGrath et al., 2008). A study of minority ethnicity women (Diego, Field, & Hernandez-Reif, 2005) and their 5- to 13-day-old infants found no significant difference between levels of fussing/crying, movement, or behaviors related to stress in infants who were exposed to prenatal depression only (between 23 and 27 weeks of gestation) and those who were exposed to both prenatal and postpartum depression (measured concurrently with the newborn measures). However, both were significantly worse than infants of non-depressed mothers and those who were only exposed postpartum. Although this study did not display postpartum depression as an additive factor, the researchers concluded that this outcome may be due to the very young age of the infants, resulting in a limited effect

of maternal interactions on infant behavior. On the other hand, the results emphasized the influence of prenatal depression on the infant. Overall, these studies highlighted prenatal maternal depression as an underlying barrier for healthy infant growth. Therefore, it can be hypothesized that infants of mothers who were prenatally and postpartum depressed will display the highest levels of negative affectivity; this will confirm the notion that the intrauterine environment is critical to explain for infant's vulnerability to depression and that maternal postpartum depression may worsen the outcome.

Although studies have established temperament to have a strong genetic/biological basis, thus indicating the importance of the prenatal environment, EEG studies have focused on the influence of the postpartum environment. Most EEG studies only used infants of postpartum depressed mothers as samples and have found a significant association between relative EEG asymmetry and withdrawal behaviors. One of the rare studies that have incorporated a prenatally depressed mother sample was conducted by Diego et al. (2004). Infants with mothers who experienced both prenatal and postpartum depression showed the highest relative right frontal EEG asymmetry, followed by infants with postnatal exposure only, then infants with prenatal exposure only, and then infants with no depression exposure. Given that prenatal depression is a strong predictor for postpartum depression, it is highly likely that EEG patterns have a biological basis as well and it is affected by the prenatal environment. This suggests that, similar to temperament, EEG asymmetry in infants of prenatally depressed mothers would be influenced by the intrauterine environment and that postpartum depression will strengthen the chances for EEG asymmetry to be a vulnerability to developing depression in the future. Based on these findings, we hypothesized that infants of mothers with both

prenatal and postpartum depression will have the strongest degree of association between EEG asymmetry and negative affectivity, with infants of prenatally depressed mothers scoring in between the dually exposed group and those with postpartum depression only or no depression.

In summary, the goal of this present study was to further examine the relationship between EEG and temperament based on infants' level of exposure to their mothers' depression. Studies showed that EEG asymmetry is positively correlated to N/NE (or depressive symptoms) in community and clinical samples of adults as well as withdrawal/inhibited behaviors in infants in the normative population. However, no studies have found the association between EEG asymmetry and negative temperament, a construct that later develops into N/NE, in infants of depressed mothers. Also, we expected depression during pregnancy to act as a moderating variable that will influence the degree of relationship between relative right frontal EEG asymmetry and negative affectivity in infants. Based on these premises, we expected that infants of mothers who experienced depression both prenatally and postpartum would have the strongest correlation between the two domains compared to prenatally depressed mothers and postpartum mothers. Hence we hypothesize that infants of depressed mothers will have a correlation between relative right frontal EEG asymmetry and negative temperament and infants of mothers who experienced both prenatal and postpartum depression will have the strongest correlation between EEG asymmetry and scores on the negative affect domain of the IBQ-R.

Method

Participants

The data for this study were derived from the longitudinal study *Perinatal Stress and Gene Influences: Pathways to Infant Vulnerability*. Women were recruited through several sources. Most were referred in response to advertisement to the referring practitioners and the Women's Mental Health Program (WMHP) of the Department of Psychiatry and Behavioral Sciences at Emory University website. Also, 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 also were referred to the study. Various additional recruitment strategies were employed: annual mailing, posting at WMHP, posting 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.

Eligibility criteria were: meeting DSM-IV criteria for a previous Major Depressive Episode (MDE), Obsessive Compulsive Disorder (OCD), Generalized Anxiety Disorder (GAD), or Post-Traumatic Stress Disorder (PTSD), as well as being less than 16 weeks pregnant measured from last menstrual period, or planning pregnancy. Participants needed to be between ages 18 and 45, be fluent in both written and verbal English, be able to give informed consent and abide by study procedures, and be able to identify the biological father of the infant. Exclusion criteria were as follows: 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.

Potential patients who were interested in the study filled out checklists and a phone interview followed. Women who continued to qualify for the study were administered the full Structured Clinical Interview for the Diagnostic and Statistical Manual- IV Axis I Disorders – Patient Edition (SCID) (First, Spitzer, Gibbon, & Williams, 1995) in-person at the first visit during pregnancy. The screen and mood disorders sections were assessed during subsequent visits, as were other modules as required to follow up on positive screens.

For this study, 46 women participated. Of these 46, one participant was missing data on age, marital status, and ethnicity. Participants ranged from 25 to 41 years of age (M = 34 years, SD = 3.9). Approximately 91% were married and on average had completed 16.7 years (SD = 1.9) of education. Forty-four percent of mothers were primiparous and 87% were European-American, with the remaining 9% being African American and 4% Native American. Excluding eight participants who were missing Hollingshead scores, the sample was, on average, of middle socioeconomic status with M = 53.33 based on the Hollingshead scale (Hollingshead, 1975).

Procedure

The study design was longitudinal, with data collected at different time points throughout pregnancy and postpartum. Postpartum data were collected on the infants at 3-, 6-, and 12-months of age. Mothers and infants visited the laboratory at each of these infant ages, including an additional pediatrician visit when the infant was 6-months of

age. A number of measures were assessed. The full SCID was administered to assess any DSM-IV (American Psychiatric Association, 1994) diagnoses, the Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) was administered in pregnancy and postpartum, and the BDI-II (Beck, Steer, & Brown, 1997) was administered at infant age 3-, 6-, and 12-months postpartum to assess depressive symptom levels. At infant ages 3-, 6-, and 12-months, participating mothers completed the Infant Behavior Questionnaire –Revised (IBQ-R) and infants' electroencephalogram (EEG) was recorded. The data from pregnancy and the three month postpartum visit were the focus of this study. During pregnancy, women completed an average of five SCID interviews, ranging from 2 to 9 times (*SD* = 1.56) and five BDIs, with a range from 1 to 10 times (*SD* = 1.95). One participant was missing prenatal SCID data and prenatal BDI data. From birth through infant age at 3-months, women completed an average of two SCID interviews, with a range from 0 (n = 3) to 3 times (*SD* = 0.64) and two BDIs, ranging from 0 (n=5) to 4 times (*SD* = 0.77), and 96% of the participants completed the BDI-II when the infants were 3-months of age.

At the 3-month laboratory visit, mothers brought in completed IBQ-R questionnaires that had been sent to their homes prior to the visit or completed them at the laboratory. Mothers and their infants were video-recorded and infants' EEG was recorded during a 3-minute baseline, 5-minute feeding, and 5-minute freeplay segment. Prior to baseline recording, a research assistant manipulated toys in order to distract the infant while the EEG cap was fastened on the infant's head. Baseline EEG was recorded for 3 minutes while infants sat on their mothers' laps. A research assistant blew bubbles during the segment. This procedure was designed to quiet the infants and to minimize eye

movements and gross motor movements. Mothers were instructed not to talk to infants during this segment of the EEG recording. During the feeding segment, mothers were allowed to breastfeed or use a bottle to feed her infant. Finally, during the freeplay 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.

Previously, researchers have found resting frontal EEG asymmetry to be related to individual differences in infant temperament (Fox, 1991; Santesso et al., 2007). Therefore, in order to capture the resting frontal asymmetry patterns, only the baseline segment of the EEG recording was utilized in this study.

Measures

Negative affectivity measure.

The Infant Behavior Questionnaire - Revised(Gartstein & Rothbart, 2003). Based on the definition of temperament determined by Rothbart and Derryberry (1981), the IBQ-R is a factor-analytically derived measure of infant temperament. The questionnaire contains 191 items and, as is typical, was completed by the mother. The items ask the participant to rate the infant's behavior during the past week in a variety of situations. Sample questions include: *When the baby was upset about something, how often did s/he stay upset for up to 10 minutes or longer? When being dressed or undressed during the last week, how often did the baby squirm and/or try to roll away? After sleeping, how often did the baby cry if someone doesn't come within a few minutes?* These items are scored on a 7-point scale, from 1 (*Never*) to 7 (*Always*). The questionnaire yields scores on 14 scales (Activity Level, Approach, Cuddliness/Affiliation, Duration of Orienting, Falling Reactivity, Fear, Frustration/

Distress to Limitations, High Intensity Pleasure, Low Intensity Pleasure, Perceptual Sensitivity, Sadness, Smiling and Laughter, Soothability, and Vocal Reactivity) that cluster into three overarching factor scores: Surgency/Extraversion, Negative Affectivity, and Affiliation or Orienting/Regulation. Each scale is scored by calculating the mean of items determined that correspond to the construct. The number of items for each scale range from 10 to 18. Negative Affectivity, the variable of interest in this study, is determined by the mean of four scales: Falling Reactivity, Fear, Frustration/Distress to Limitations, and Sadness. The range of possible scores for Negative Affectivity is 1, corresponding with low negative affectivity to 7, corresponding with high Negative Affectivity.

For each of the four scales that comprise Negative Affectivity, internal consistency for the 3-6 month normative sampling group was high, with Cronbach's alphas as follows: Falling Reactivity (0.84), Fear (0.90), Frustration/Distress to Limitations (0.81), and Sadness (0.85) (Gartstein & Rothbart, 2003). Gartstein and Rothbart also reported high inter-rater reliability between primary and secondary caregivers from a small sample (n = 26) for Falling Reactivity (r = 0.69), Fear (r = 0.75), and Frustration/Distress to Limitations (r = 0.57), but low and not significant for Sadness (r = 0.27). For our sample, the internal consistency scores for Negative Affectivity were: Falling Reactivity (r = 0.83), Fear (r = 0.91), Frustration/Distress to Limitations (r = 0.73), and Sadness (r = 0.80).

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). Specifically, 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 bandpassed 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 (Fox, 1994). Thus, a negative asymmetry score reflects greater right frontal activation, whereas a positive asymmetry score reflects greater left frontal activation.

Depression measures.

Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1995). The SCID is a semi-structured diagnostic interview designed to assess for a past history of any Axis I disorders as well as for current diagnostic status based on the Diagnostic and Statistical Manual- Fourth Edition (DSM-IV) (American Psychiatric Association, 1994). During the initial screening and following visits, trained research assistants administered the SCID, with reliability determined by a senior psychiatric nurse who listened to the audiotapes of each interview and independently assigned diagnoses.

Beck Depression Inventory (Beck et al., 1961). The original BDI is a self-report measure of depression symptom severity with 21 questions about how the subject was

feeling during the past week. Each question has four possible choices, ranging from 0 to 3. A total score is calculated by adding the ratings across items, with higher scores indicating greater severity of depression symptoms. The scores can also be interpreted as follows: 0-9 signifies no depression, 10-18 signifies mild-moderate depression, 19-29 signifies moderate-severe depression, and 30-63 signifies severe depression. 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 (Beck et al., 1961). The BDI-I was administered at multiple time periods during pregnancy.

Beck Depression Inventory-Second Edition (Beck et al., 1997). The BDI-II is a 21-item self-report measure of depression symptom severity, which was designed to be more compatible with the DSM-IV definition of major depression. Respondents are instructed to base their answers on the past two weeks. 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 depression 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. 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(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). The BDI-II was administered during the postpartum laboratory visits.

Data Analytic Strategy

First we ran descriptive statistics on the demographic characteristics of the sample. To characterize depression, we ran descriptive statistics based on the clinician-based and self-reported measures of depression (SCID and BDI) during pregnancy and postpartum. Through this, we defined mothers as meeting diagnostic criteria for a Major Depressive Episode (MDE) or having clinically-significant depressive symptom scores during pregnancy or at postpartum.

Then, to test the first hypothesis that infants of depressed mothers will have a significant association between relative right frontal EEG asymmetry and negative affectivity, we examined descriptive statistics on the measures of these two variables: the EEG asymmetry and negative affectivity scores. Subsequently, Pearson's *r* was utilized to test the degree of association between the two variables for the whole sample. We then conducted correlation analyses between the two variables on mothers who met DSM-IV criteria for major depression during pregnancy and also for those who met DSM-IV criteria for major depression during the first three months postpartum. If these correlations of prenatally depressed mothers and those of mothers with postpartum depression yield non-significant results, the prenatal and postpartum BDI scores calculated by adjusted area under the curve (AUC), mean, and peak scores, were to be used as control variables in a partial correlation analysis to see if severity of depressive symptoms either pre- or post-natally significantly influenced the degree of relationship between the EEG asymmetry and negative affectivity. In addition, to test the role of

concurrent depression symptom levels, we tested the association between EEG and negative affectivity with the concurrent BDI-II scores as the control variable in a partial correlation.

Results

Descriptive and Preliminary Analyses

Depression. Based on DSM-IV diagnostic criteria for major depression and data from the SCID, 24% (n = 11) of the mothers were depressed during pregnancy. Of these 11 mothers, roughly 45% met criteria twice, followed by once (36%), 5 times (9%), and 7 times (9%). During the first three months postpartum, 13% (n = 6) of the mothers were depressed. Of those 6 women, half met criteria once and half met criteria twice.

The descriptive statistics for the prenatal and postpartum BDI were based on adjusted area under the curve (AUC), mean, and peak score (see Table 1).

EEG and negative affectivity variables. Descriptive statistics for EEG and negative affectivity are presented in Table 1. Fifty percent of the sample had negative scores, which indicates more relative right frontal EEG asymmetry.

Hypothesis Testing

Hypothesis one: Infants of depressed mothers will have a significant association between relative right frontal EEG asymmetry and negative affectivity. Based on correlational analyses for the sample as a whole, EEG asymmetry and negative affectivity had a small, non-significant correlation, $r(44) = 0.14$, $p = 0.37$. This represented a small effect size, based on Cohen's d (Cohen, 1988). The association was in the opposite of the predicted direction, with more negative affectivity (not significantly) associated with less right frontal EEG activity. To test the role of prenatal or postpartum

depression on these associations, Pearson's *r* was calculated separately for women with prenatal or postpartum depression. For mothers who met diagnostic criteria for major depression during pregnancy, the association was negative (in the expected direction) but small and non-significant, with a small effect size, $r(44) = -0.16$, $p = 0.63$. For those who did not meet diagnostic criteria for major depression during pregnancy, the association was positive, but also small and non-significant, with a medium effect size, $r(44) = 0.27$, $p = 0.20$.

In order to observe the influence of prenatal depressive symptom levels on the correlation between EEG asymmetry and negative affectivity, we next tested the adjusted AUC, mean, and peak prenatal BDI scores as control variables in partial correlation analysis (see Table 2). Even after controlling for the three variables, EEG asymmetry and negative affectivity yielded very small positive correlations. The BDI adjusted AUC, $r(44) = 0.40$, $p = 0.01$, and mean, $r(44) = 0.48$, $p = 0.001$, but not peak scores $r(44) = 0.29$, $p = 0.07$, were associated with Negative Affectivity. Adjusted AUC and mean scores had large effect sizes whereas peak scores had medium effect size. Overall, the results indicated that prenatal depression significantly predicted negative affectivity but did not have a significant effect on the relationship between EEG asymmetry and negative affectivity.

The same analyses were conducted for postpartum depression. For infants of mothers who were diagnosed with postpartum depression through 3 months based on the SCID, the association between EEG asymmetry and negative affectivity was small and negative (in the predicted direction) but not significant, $r(44) = -0.23$, $p = 0.66$. The direction of the association suggested that more negative affectivity was associated with

more relative right frontal EEG asymmetry. Those who were not postpartum depressed through 3 months also showed non-significant results and had a positive correlation with small effect size between the two variables, $r(44) = 0.15$, $p = 0.39$.

Then, to observe the influence of postpartum depressive symptoms on the correlation between EEG asymmetry and negative affectivity, we tested the adjusted AUC, mean, and peak postpartum BDI scores as control variables in a partial correlation analysis (see Table 2). As for postpartum depressive symptoms, the association between EEG asymmetry and negative affectivity was small and positive, even after controlling for the depressive symptom variables. For the postpartum depressed mothers, there was no significant correlation between negative affectivity and BDI adjusted AUC, $r(44) = 0.02$, $p = 0.92$, mean, $r(44) = 0.06$, $p = 0.71$, and peak $r(44) = 0.05$, $p = 0.77$. All had small effect sizes.

Finally, the BDI-II assessment was used as the control variable in a partial correlation analysis to test the role of concurrent depressive symptoms on the association between EEG asymmetry and negative affectivity scores in mothers at the 3-month laboratory visit. The results were not significant $r(44) = 0.14$, $p = 0.37$, with a small effect size. Overall, results displayed that concurrent levels of postpartum depression symptoms did not affect the association between EEG asymmetry and negative affectivity.

Antidepressants and other exposures. At least at some point in pregnancy, 80% of the women took caffeine, 58% of women took Selective Serotonin Reuptake Inhibitors (SSRIs), and 33% consumed alcohol. Rates of use of other antidepressants and tobacco were small. In order to see if the use of antidepressants affected the relationship between

EEG asymmetry and negative affectivity scores, we first conducted a bivariate correlation analysis using Pearson's *r*. All analyses were two-tailed. Then, the three exposure variables were used in a partial correlation analysis as control variables. For caffeine, the correlation was $r(44) = 0.14$, $p = 0.38$, which revealed no significance and no difference in the relationship between EEG asymmetry and negative affectivity. SSRIs, $r(44) = 0.14$, $p = 0.36$, and alcohol, $r(44) = 0.14$, $p = 0.37$, yielded the same results. All three exposures had small effect sizes. Therefore, we concluded to disregard the use of antidepressants and exposures as an influence due to no significant effect on the relationship between the EEG asymmetry and negative affectivity variables.

In summary, although all correlations were non-significant, the correlations between EEG asymmetry and negative affectivity were in the expected direction for mothers who had either prenatal or postpartum depression based on the SCID assessments. Due to the very small number of mothers who were clinically diagnosed as prenatally or postpartum depressed, the second hypothesis could not be tested.

Discussion

Although research has verified a number of vulnerabilities that make infants susceptible to the development of psychopathology and also documented that interactions between these vulnerabilities may increase the possibility for psychopathologic disorder, less is known about these interactions in infants of depressed mothers. In order to observe if maternal depression augments infants' vulnerability to depression in the form of EEG asymmetry or negative affectivity, this study examined the interaction between affective/behavioral and biological domains via EEG asymmetry and negative

temperament in infants of mothers with history of depression, some of whom further experienced depression prenatally and postpartum.

Overall, our results indicated that EEG asymmetry and negative affectivity were not significantly associated. Furthermore, partial correlations showed that maternal prenatal and postpartum depressive symptoms had no significant influence on the degree of association between the two variables. Maternal depressive symptoms measured when infants were 3-months of age also did not influence the degree of association between EEG and negative affectivity. Consistent with our hypothesis, however, results displayed a non-significant trend in which infants of prenatally and postpartum depressed mothers showed a small correlation in the expected direction. That is, more negative EEG asymmetry scores trended toward association with higher negative affectivity scores on the IBQ in infants of depressed mothers. Due to non-significant results in the first hypothesis, we were unable to test the second hypothesis, which predicted infants of both prenatally and postpartum depressed mothers to have the strongest correlation between the two variables compared to infants of only prenatally depressed mothers, those of only postpartum depressed mothers, and those of non-depressed mothers.

Although the results were non-significant, it was interesting to see the tendencies of both infants of prenatally depressed mothers and those of postpartum depressed mothers displaying a correlation between EEG asymmetry and negative affectivity. In previous literature, the association between relative right frontal EEG asymmetry and negative temperament is found in infants of the general population (Santesso et al., 2007; Schmidt, 2008) but not in infants of depressed mothers. More specifically, studies have shown infants of the general population who reflected relative right frontal EEG

asymmetry to display withdrawal behaviors, such as fussing, crying, and heightened levels of distress (Calkins et al., 1996; Davidson & Fox, 1989; Fox et al., 1992), which are motivational indices of negative temperament (Derryberry & Rothbart, 2001). Based on the fact that both EEG and temperament have biological/genetic bases and both have stability throughout infancy to toddlerhood, it is interesting to see no significant association and such a low correlation in our study. However, the fact that both the prenatal depressed group and postpartum depressed group displayed negative correlations suggests that infants of depressed mothers may be more likely to simultaneously manifest vulnerabilities across the biological and affective/behavioral domains, relative to those of non-depressed mothers.

Similarly, controlling for depressive symptoms did not make a significant difference in the correlation between EEG asymmetry and negative temperament. Three different methods of measuring the level of depression symptoms reported on the BDI were calculated (adjusted AUC, mean, and peak). The partial correlations verified that neither prenatal nor postpartum depressive symptoms had a significant influence on the correlation between the two variables. This contrasted the literature that has demonstrated a significant correlation between relative right frontal EEG asymmetry and behaviors that implicate negative temperament in infants of mothers with high depressive symptoms (Jones et al., 1997). In addition, the BDI-II assessed at the 3-month laboratory visit did not yield significance either.

Previous literature has shown infant EEG asymmetry and negative temperament to be independently associated with maternal depression. In this study, we observed that the EEG asymmetry results did not significantly correlate with prenatal and postpartum

BDI adjusted AUC, mean, and peak whereas negative affectivity had a significant correlation with the BDI AUC and mean for prenatal depression. A possibility that may explain the non-significant association between EEG asymmetry and maternal depression symptoms is presented by Orekhova, Stroganova, Posikera, and Malykh (2003). They suggested that there is an increase in EEG frequency heritability during the second half-year of life. Based on the fact that the infants in our sample were 3-months-old, heritability of EEG asymmetry patterns from mothers to infants may not have been fully represented. Although EEG literature has found relative right frontal EEG asymmetry in a sample as young as 1-week-old infants of depressed mothers (Diego et al., 2009), the frontal asymmetry patterns were found in different frequency bins. One-week-old infants could only be differentiated at 3-7 Hz whereas 3-month-old infants displayed differences at 4-12 Hz. The researchers found the narrow 4-9 Hz band to be the best frequency band in distinguishing EEG asymmetry in infants of depressed mothers from those of non-depressed mothers. Although much literature used a frequency of 6-9 Hz similar to our study (Bell & Fox, 1994; Marshall, et al., 2002), using a wider frequency band may have had a better possibility to yield significant differences in infant EEG asymmetry patterns.

On the other hand, negative temperament was significantly associated with the BDI adjusted AUC and mean for prenatal depression but not for postpartum depression. This supports either the strong biological/genetic basis of temperament that has been continuously established in previous research (Austin, Hadzi-Pavlovic, Leader, Saint, & Parker, 2005; Davis et al., 2007; Propper & Moore, 2006; Rothbart & Bates, 1998) or that negative temperament is developed through influence of the prenatal maternal depression on the intrauterine environment. Also, the average number of BDI assessments

administered during pregnancy (M = 5.49) was higher than that of postpartum (M = 2.14), which may have contributed to the significant relationship between negative temperament and prenatal depressive symptoms but not for postpartum depressive symptoms.

Several limitations are noted. One primary limitation of this study is the small sample size. The mothers were recruited from a longitudinal study that is currently in progress, so the number of infants who had both EEG asymmetry and IBQ scores is steadily increasing, leaving open the possibility of re-testing the hypotheses with a larger sample. A larger sample size may have generated significant correlations or more apparent trends in the results. Another possible limitation is the demographic characteristics of the sample. The mothers were predominantly European-American, mostly married, and came from middle to high socioeconomic status. Most of these mothers were being treated with antidepressant medications, which may have alleviated the depressive symptoms. This explains the generally low depressive symptom scores on the BDI and BDI-II assessments and that it may have masked the predicted associations.

Future studies might also consider alternative means of working with the EEG data. Instead of using a pre-defined window of time such as our EEG segments, Schmidt (2008) proposed a method of collecting EEG data based on a computation of overlapping second-by-second frontal EEG asymmetry scores across a 90-second baseline condition. Highly dynamic neural processes and moment-to-moment changes in the balance of hemispheric activity result in continuously changing EEG asymmetry. Schmidt argued that EEG asymmetry measures using power summed across a few minutes may not be sensitive to short-term changes in EEG activity. Using 9-month-old infants of the general

population, he found that those with relative right frontal EEG asymmetry significantly correlated with IBQ fear ratings.

Overall, this study did not support the hypothesis that perinatal depression would be associated with more co-occurring infant vulnerabilities to develop psychopathology in the future. With the planned larger sample size to increase statistical power for more robust statistical testing, a fuller test of the hypotheses will be possible.

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Table 1

Descriptive Statistics on Core Variables

Variables	*M*	*SD*	Min	Max
EEG	-0.05	0.31	-1	1.06
Negative Affectivity	3.44	0.42	2.78	4.39
Prenatal BDI Adjusted AUC	345.27	258.27	40.91	1207.82
Prenatal BDI Mean	7.92	5.95	0.90	27
Prenatal BDI Peak	13.39	9.67	2	42
Postpartum BDI Adjusted AUC	95.07	89.37	0	462
Postpartum BDI Mean	7.66	7.22	0	39
Postpartum BDI Peak	10.69	8.96	0	42
BDI-II	8.68	8.63	0	44

Note. Min = minimum score; Max = maximum score

Table 2

Partial Correlation Analysis Adjusted for BDI, between EEG Asymmetry and Negative Affectivity

	Prenatal		Postpartum Through 3 Months	
Measurement	*r*	Sig.[1]	*r*	Sig.[1]
Adjusted AUC	0.08	0.63	0.10	0.56
Mean	0.06	0.71	0.10	0.57
Peak	0.09	0.58	0.10	0.57

[1]Two-tailed