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Molly Davis 3/30/2012

The Role of Infant EEG, Mothers' Prenatal Anxiety and Parenting Behaviors in the Association Between Mothers' Perinatal Depression and Infant Positive Affect

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a thesis submitted to the Faculty of Emory College of Arts and Sciences
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Abstract

The Role of Infant EEG, Mothers' Prenatal Anxiety and Parenting Behaviors in the Association Between Mothers' Perinatal Depression and Infant Positive Affect

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The present study sought to examine the relationship between mothers' cumulative depression symptom levels during the prenatal and postpartum periods and infant positive affect (PA) at 3 and 6 months of age. Participants were 194 mothers with a history of depression and/or anxiety and their 3- and 6-month old infants. Mothers' depression symptom levels were assessed during the prenatal and postpartum periods using the Beck Depression Inventory (BDI). When the infants were 3 and 6 months of age, mothers and their infants visited the lab to complete a series of videotaped segments. State PA was assessed using continuous coding of affect exhibited during the mother-infant free play interactions and trait PA was measured using the Surgency/Extraversion factor on the Infant Behavior Questionnaire-Revised (IBQ-R). We tested mediators for the relationship between mothers' depression and infant PA: infant electroencephalography (EEG) asymmetry scores and parenting quality behaviors (ratings of observed sensitivity/responsiveness, withdrawal and time spent in PA). Mothers' postpartum depression symptom levels for the first 3 months and first 6 months postpartum were each significantly negatively correlated with infant trait PA at 6 months ($r(159) = -.23$, $p < .01$; $r(132) = -.26$, $p < .01$), but were not significantly associated with any of the other PA indices. Mothers' prenatal symptom levels were not significantly associated with infant PA at 3 or 6 months. Also, neither infant EEG nor parenting quality mediated the association between mothers' depression symptoms during the first 6 months postpartum and infant trait PA at 6 months. Mothers' prenatal anxiety symptom levels, did not significantly predict infant PA. Results suggest mothers' cumulative postpartum depression symptom levels, particularly during the first 3 months of the postpartum period, influence the temperament form of infant PA at 6 months. Future research should examine the PA trajectories of infants of depressed mothers during the first 6 months and beyond.

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Depression in the perinatal period is not uncommon; about 13% of women experience depression during the postpartum period (O'Hara & Swain, 1996) and rates during the prenatal period are comparable (J. Evans et al., 2001). Infants of depressed mothers are at increased risk for adverse outcomes, including low levels of positive affect (for a review, see Goodman & Gotlib, 1999). Positive affect (PA) involves a "set of hedonic, behavioral, motivational, and physiologic features" (Forbes & Dahl, 2005). PA functions as a subjective state (Forbes & Dahl, 2005) as well as a trait (Clark & Watson, 1991), or temperamental form of positive affect, often referred to as positive affectivity (Forbes & Dahl, 2005). For the purposes of this study, we will refer to these concepts as state and trait PA respectively. Low levels of these state and trait components of PA serve as possible vulnerabilities to the later onset of depression.

Deficits in PA have been found in depressed individuals (Forbes & Dahl, 2005) and low PA early in childhood has been found to be associated with depressive symptoms later in childhood (Dougherty, Klein, Durbin, Hayden, & Olino, 2010). Moreover, low PA has been found in offspring of depressed mothers (Cohn, Campbell, Matias, & Hopkins, 1990; Kovacs & Lopez-Duran, 2010). Though some research has examined PA in infants of depressed mothers, most has focused on negative affect (NA). As a largely unexplored early vulnerability to depression and a component differentiating depression from other internalizing disorders such as anxiety (L. A. Clark & Watson, 1991), PA deserves to be studied further. Despite some findings indicating a relationship between mothers' depression and low infant PA, many questions remain unanswered. In particular, the mechanisms by which infants of depressed mothers develop low PA remain unclear. Theory and research suggest that unfavorable parenting qualities and

infant EEG may serve as mediators of the relationship between mothers' depression and infant PA, though no known studies have tested these variables as mediators for the proposed association. Therefore, we expanded upon literature in the field by creating a model that tested infant EEG asymmetry and several parenting qualities as mediators for the relationship between mothers' prenatal and postpartum depression and infant state and trait PA (see Figure 1). Developing a greater understanding of the relationship between perinatal depression and infant PA can aid in the formation of interventions to help mitigate the risk for psychopathology in infants of depressed mothers.

Definitions of PA

To begin, it is important to not only understand the ways state and trait components of PA have been defined in the literature but also to examine the relationship between the two constructs and their significance in the context of offspring of depressed mothers. State PA includes observable positive emotional displays such as smiling and laughing (Olino et al., 2011) and involves the "subjective experience of pleasant emotions" (Forbes & Dahl, 2005). State PA can be thought of as observable behaviors meant to mirror trait PA. Because low state PA has been found in offspring of depressed mothers (Cohn et al., 1990; Field, Healy, Goldstein, & Guthertz, 1990; Righetti-Veltema, Conne-Perréard, Bousquet, & Manzano, 2002) and in depressed individuals (Forbes & Dahl, 2005), low state PA can be regarded as a potential marker for the later onset of depression.

Whereas one component of PA is an emotional state, another is an emotional trait (L. A. Clark & Watson, 1991), or a factor of temperament (Derryberry & Rothbart, 1988). Rothbart and Derryberry (1981) defined temperament as constitutionally based

individual differences in reactivity and self-regulation. Trait PA, though more enduring than state PA, also includes behaviors such as smiling and laughing (Gartstein & Marmion, 2008), which in the case of trait PA, represent inherent behavioral tendencies rather than a subjective state. Children at familial risk for depression often show lower levels of trait PA (Kovacs & Lopez-Duran, 2010). Additionally, low trait PA in early childhood has been found to predict depressive symptoms later in childhood (Dougherty et al., 2010) as well as later depressotypic cognitive styles (Hayden, Klein, Durbin, & Olino, 2006). We will explore trait PA as a potential early vulnerability to psychopathology in infants of depressed mothers.

Typical Development of Infant PA

Researchers have explored the typical developmental trajectory of PA. Some stability has been found for infant state (Forbes, Cohn, Allen, & Lewinsohn, 2004) and trait PA (Lemery, Goldsmith, Klinnert, & Mrazek, 1999) from 3 to 6 months of age. As early as the first 2 to 3 months of life, individual differences in trait PA can be detected (Rothbart, 1989) and the probability of smiling and laughter, and the intensity and duration of these behaviors, increases throughout the first 13.5 months of life (Derryberry & Rothbart, 2001). Linear increases have been found for other aspects of PA such as happiness and interest (Denham, Lehman, Moser, & Reeves, 1995).

Origins of Infant PA

Some researchers have theorized about hereditary components of temperament. Monozygotic twins have been shown to be more similar than dizygotic twins on several temperament dimensions, including PA (for a review, see Saudino, 2005). Thus far, findings on the heritability of temperament are inconclusive (Ivorra-Martinez, Gilabert-

Juan, Molto-Ruiz, & Sanjuan, 2007). Although it is likely that genetics contribute to individual differences in PA, it is necessary to also assess environmental factors in order to better understand these individual differences.

Environmental factors have been discussed in relation to PA and temperament in general. For instance, non-shared environmental factors (Tronick, 2003) and culture (Gartstein et al., 2006) can influence temperament. We will explore the role of several environmental factors in the PA of infants of depressed mothers.

Postpartum Depression and Infant PA

One question of interest is what might disrupt infants' typical development of PA. Based on research, there is support for the fact that mothers' postpartum depression may contribute to the development of PA deficits in infants. Though mothers' depression at various time points could impact infants' PA, most research has focused on the relationship between postpartum depression and infants' state PA. Several studies have found that when interacting with their mothers, infants of postnatally depressed mothers have lower state PA than infants of nondepressed mothers. These results have been found in home (Cohn et al., 1990; Righetti-Veltema et al., 2002) and lab settings (Field et al., 1990) at various points during infants' first six months of life. However, most researchers in the field study depression as a dichotomous variable, which can detract from the power in statistical analysis and gives a very narrow view of depression.

Associations between mothers' depression and infant PA may be particularly strong when the depression is chronic. For example, Campbell, Cohn and Meyers (1995) found evidence that suggests infants of mothers with depression lasting through six months postpartum were less positive during interactions compared to infants of mothers

with more short-lived depression. To better understand the association between mothers' depression and infant PA, we aimed to not only replicate the previously found association between these variables but also to expand upon existing literature by studying depression symptom levels as a continuous variable. This allowed us to study the role of varying levels of symptom severity, including subclinical depression, in the association between mothers' depression and infant PA. We also recruited mothers with a history of anxiety and/or depression, thus increasing the likelihood of studying women who experienced postpartum, and/or prenatal, depression.

Though the relationship between mothers' postpartum depression and infant trait PA has not been explicitly determined, there is reason to believe a relationship similar to that identified between postpartum depression and infant state PA would exist. As the more innate form of state PA and as a component of the same construct, it is expected that both trait and state PA would be lower in infants of postnatally depressed mothers compared to infants of nondepressed mothers. Also, the correlation between state and trait PA (Tellegen, 1985) suggests that because postpartum depression has been found to predict low state PA it would also predict low trait PA.

Research on mothers' postpartum depression and infant temperament has primarily focused on NA and temperament in general. These studies suggest that there is a relationship between mothers' postpartum depression and infant temperament, thus justifying the exploration of the specific association between maternal postpartum depression and infant trait PA. In a meta-analysis of the literature in this field up until 1993, C.T. Beck (1996) noted that there is a moderate, statistically significant association between mothers' postpartum depression and negative infant temperament. Also,

mothers' depression during the prenatal and postnatal periods has been associated with mothers' reports of more difficult infant temperament at 2 and 6 months of age (McGrath, Records, & Rice, 2008). In contrast to these findings, maternal depression/anxiety, sensitivity and social support did not predict infants' later development of trait PA (Pauli-Pott, Mertesacker, & Beckmann, 2004). This study assumed that the depression and anxiety scales were intercorrelated and therefore the authors computed principle component analyses for every age to limit the number of variables in the study (Pauli-Pott et al., 2004). One aim of the current study was to tease apart depression and anxiety in order to assess the relative contributions of each to infant PA instead of assessing both as a combined variable. In addition, since infants of postnatally depressed women often exhibit disruptions in temperament and because NA and PA may arise from some of the same neural structures (Derryberry & Rothbart, 2001), we will examine the association between infants' trait PA and mothers' postpartum depression.

Prenatal Depression and Infant PA

Researchers have primarily focused on the relationship between mothers' prenatal depression and temperament factors such as NA, leaving the relationship between prenatal depression and infant PA largely unexplored. Also, researchers have focused on trait rather than state NA. The limited research in this field suggests there is an association between mothers' prenatal depression and infant temperament (Davis et al., 2004; Huot, Brennan, Stowe, Plotsky, & Walker, 2004). Furthermore, potential early signs of low PA have been found in newborns of prenatally depressed mothers (for a review, see Field, Diego, & Hernandez-Reif, 2006) such as fewer interest expressions on

the Brazelton Neonatal Behavior Assessment Scale as compared with newborns of nondepressed mothers (Lundy, Field, & Pickens, 1996). Similar to studies involving mothers with postpartum depression, Lundy et al. (1996) divided mothers into the depressed and nondepressed groups based on cut scores. We will study mothers' prenatal depression as a continuous variable to capture a range of symptom severities and will also assess infant PA beyond the neonatal period to find out if mothers' prenatal depression influences infant PA later in infancy.

In addition, the literature suggests biological correlates of prenatal depression may impact infant temperament. For example, women with prenatal depression symptoms have been found to have higher rates of cortisol, a glucocorticoid stress hormone, relative to women without such symptoms (Field, Hernandez-Reif, et al., 2006), however, there have been findings indicating this is only the case when anxiety and depression are comorbid, meaning they co-occur (L. M. Evans, Myers, & Monk, 2008). Elevated glucocorticoid exposure could result in the programming of the HPA axis in the fetus in a way that can impact temperament, by means of making the infant reactive to stressors (Cicchetti & Rogosch, 1996). Elevated cortisol at certain points in pregnancy has also been found to be associated with higher levels of infant fear, with prenatal depression and anxiety additionally predicting this outcome (Davis et al., 2007). Because of its impact on temperament and on early signs of PA during the neonatal period, and due to the overlapping neural structures involved in NA and PA (Derryberry & Rothbart, 2001), we will examine whether prenatal depression leads to deficits in infant PA. Also, as a component of PA, state PA is meant to be an observable representation of trait PA. So, we will examine the relationship between prenatal

depression and infant state and trait PA with the expectation that prenatal depression is similarly associated with both forms of infant PA.

Possible Mediators for the Relationship Between Mothers' Depression and Infant PA

There are several mechanisms by which infants of depressed mothers can develop low state and trait PA and these mechanisms are important to understand in order to determine the origins of early markers of depression. Whereas theoretical models generally focus broadly on adverse outcomes that offspring of depressed mothers face, Kovacs and Lopez-Duran (2010) proposed some physiological mechanisms for the development of low PA in children of depressed mothers. However, because this was a review paper, and since Kovacs and Lopez-Duran (2010) did not focus on infants specifically, a model is still needed to explain mechanisms for the predicted association between mothers' depression and infant PA. Possible mechanisms for the development of adverse outcomes in infants of depressed mothers that have been proposed in the literature include modeling (Goodman & Gotlib, 1999), heritability (Goodman & Brand, 2009) and maternal prenatal cortisol (Davis et al., 2007; Field, Hernandez-Reif, et al., 2006). In order to contribute to the understanding of mechanisms that lead to low PA in infants of depressed mothers, we will examine previously unexplored mediators of this relationship: infant EEG and parenting qualities. Figure 1 displays the theoretical model for these proposed mediators of the association between mothers' depression symptom levels and infant PA. In Figure 1, mothers' prenatal depression severity, postpartum depression severity and prenatal anxiety severity serve as predictors, or independent

variables and the mediators are meant to explain the relationship between these predictors and the dependent variables (infant state and trait PA).

Mechanism 1: Infant EEG

Researchers have proposed that temperament "arises from individual differences in distinct neural systems related to positive and negative emotions" and that these neural systems are likely housed in limbic structures such as the amygdala, hippocampus and hypothalamus (Derryberry & Rothbart, 2001). Researchers have developed specific theories about the neurological correlates of PA. Neural circuits involved in reward processing such as the striatum, orbitofrontal cortex, amygdala and dorsolateral prefrontal cortex are associated with PA (Forbes & Dahl, 2005). These findings indicate that studying certain neurological patterns, which we will examine using infant EEG, may help explain individuals' levels of PA.

We will test infant EEG, a measure of brain activity determined to be associated with emotion, as a possible mediator of the predicted association between mothers' depression and infant PA. Though research has examined the relationship between mothers' depression and infant EEG, in addition to the association between infant EEG and PA, infant EEG has not been looked at as a mechanism by which mothers' depression can impact infant PA. It is important to study infant EEG in order to better understand neurological mechanisms that contribute to vulnerabilities to the later development of psychopathology in offspring of depressed mothers.

It is crucial to note the meaning of different EEG patterns. Relative right frontal EEG asymmetry is associated with withdrawal emotions such as negative affect whereas positive, approach emotions such as joy are associated with relative left frontal EEG

asymmetry (Fox, 1991)**.** Cerebral hemispheric asymmetry, measured using EEG, is a potential precursor to depression in the pre-adult years (Kovacs & Lopez-Duran, 2010), with young offspring at familial risk for depression exhibiting atypical asymmetry patterns. Cerebral hemispheric asymmetry is "implicated in affectivity and mood repair" (Kovacs & Lopez-Duran, 2010). EEG patterns are important for understanding neurological tendencies of those at risk for depression, particularly since depression has been linked to greater relative right frontal (and thus lower relative left frontal) EEG activation (Henriques & Davidson, 1991).

Not only has relative right frontal EEG asymmetry been found in depressed adults but this asymmetry has also been observed in infants of depressed mothers. A large portion of these studies has relied on mothers who are of low socioeconomic status, adolescent mothers and ethnic minority status. Such environmental factors could confound the relationship between mothers' depression and infant outcomes by exacerbating the negative effects of mothers' depression on infant development. Despite these potential confounds, infants of depressed mothers have shown relative right frontal (or lower left frontal) EEG asymmetry compared to infants of nondepressed mothers during the first six months of life (Field, Fox, Pickens, & Nawrocki, 1995; Jones, Field, Fox, Lundy, & Davalos, 1997). Similar results have been found for infants ages 11-17 months (Dawson, Klinger, Panagiotides, & Hill, 1992) and infants of prenatally depressed women (Field et al., 2004). These findings suggest infants often develop EEG patterns similar to their depressed mothers, indicating that mothers may be transmitting neurological, and other, depressive tendencies to their infants.

Research suggests an association between EEG asymmetry and levels of PA. Greater relative left frontal activation has been found to be associated with PA (Fox, 1991). Research has shown that infants watching happy video segments exhibit greater relative left than right frontal EEG activation (Davidson & Fox, 1982). Associations between left frontal EEG asymmetry and PA have been found in young adults as well (e.g. Wheeler, Davidson, & Tomarken, 1993). Low PA in early childhood has been found to predict right EEG asymmetry a few years later (Shankman et al., 2005). Infants of depressed mothers exhibited less PA and greater relative right frontal EEG activation, compared to infants of nondepressed mothers when interacting with mothers and strangers (Diego et al., 2004). The data on typical populations and infants of depressed mothers suggest there is likely a relationship between EEG asymmetry and state PA. As a result, we will explore infant EEG as a potential mediator and marker of the predicted relationship between mothers' depression and infant state PA.

Several researchers have studied the relationship between infant EEG and trait PA. Infants determined to have stable left frontal EEG asymmetry patterns were rated significantly higher for pleasure on the original IBQ compared with infants in the stable right frontal EEG asymmetry group (Schmidt, 2008). Infants of the same age who were rated by their mothers as temperamentally distressed based on the original IBQ exhibited greater relative right frontal brain activity in multiple conditions (Santesso, Schmidt, & Trainor, 2007). In sum, research suggests an association between EEG asymmetry patterns and state and trait PA. Due to this association, as well as the relative right frontal EEG asymmetry and low PA found in infants of depressed mothers, we will assess infant

EEG as a mediator and marker of the relationship between mothers' depression and infant PA.

Mechanism 2: Parenting

Parenting quality has been studied in the context of general adverse outcomes for offspring of depressed mothers but its role as a mediator of the relationship between mothers' depression and infant PA has yet to be determined. Goodman and Gotlib (1999) emphasized that depressed mothers tend to be inadequate social partners for their children and may not be able to fulfill their child's social and emotional needs. Such deficits in parenting negatively impact children's development of social and cognitive abilities (Goodman & Gotlib, 1999). Also, the emotional interactions between depressed mothers and their infants are likely to be atypical and shape the infants' early affect development, thus sparking the development of a depressotypic organization (Cicchetti & Toth, 1998). Further research is needed to determine if the parenting qualities of depressed mothers negatively impact their infants' PA.

We intend to contribute to the limited empirical research on the role of parenting in the relationship between mothers' depression and infant PA by assessing parenting quality as a mediator of this association. Research has shown that mothers with depression have many unfavorable parenting qualities. For instance, depressed mothers have demonstrated fewer affectionate contact behaviors (Fleming, Ruble, Flett, & Shaul, 1988) and have exhibited more insensitive parenting (Cooper et al., 1999; Hoffman & Drotar, 1991) toward their infants compared to nondepressed mothers. Depressed mothers have been found to display lower PA when interacting with their offspring than nondepressed mothers (Field et al., 1990; Lovejoy, Graczyk, O'Hare, & Neuman, 2000;

Righetti-Veltema et al., 2002), especially when their depression lasted through 6 months postpartum (S.B. Campbell et al., 1995). We will explore the impact of parenting qualities on the PA of infants of depressed mothers.

Based on theory, and some empirical evidence, it appears parenting plays a role in infants' affective outcomes. The literature in this field has largely focused on temperament in general and NA, rather than PA. Parents have been found to influence their infants' emotionality, or temperament, particularly by impacting their development of emotion regulation (Propper & Moore, 2006). Research suggests parents may impact infants' emotional dispositions via an affective communication system, which influences infants' emotional experiences and developmental outcomes (Tronick, 2003). Infants appreciate the emotional meaning of their caretakers' affect expressions and the emotional expressions of both the infant and the caretaker serve as factors that "allow them to mutually regulate their interactions" (Tronick, 2003). Such coordination, in addition to the ability to change NA into PA, is crucial for positive development (Tronick, 2003). Through this affective communication system, parents impact their children's emotional experiences (Tronick, 2003) and, therefore, potentially their levels of PA.

Though focused on NA, research has found a relationship between parenting quality and infant temperament. For example, infants' fussing and crying has been found to be related to unresponsive maternal attitudes and behavior (S. B. Crockenberg & Smith, 2002). Because NA and PA seem to arise from some similar neurological structures (Derryberry & Rothbart, 2001), it is likely that the parenting qualities of depressed mothers found to impact infant NA may also influence infant PA.

Although not previously tested as a mediator, research has found an association between parents' and infants' PA (Forbes et al., 2004), suggesting depressed mothers' PA may influence infants' PA. Several studies have found depressed mothers and their infants exhibit lower state PA than nondepressed mother-infant dyads (S.B. Campbell et al., 1995; Cohn et al., 1990; Field et al., 1990; Righetti-Veltema et al., 2002). Given the unfavorable parenting qualities found among depressed mothers and the impact of parenting behaviors on infants' emotional development, we will study parenting qualities, specifically mothers' observed PA, sensitivity and withdrawal, as mediators for the predicted association between mothers' depression and infant state and trait PA.

Role of Anxiety

To assess whether mothers' depression predicts infant PA beyond any contributions of mothers' anxiety, we will assess the role of mothers' prenatal anxiety in infant state and trait PA. Longitudinal, familial and epidemiological studies have found high levels of comorbidity between affective and anxiety disorders (Kasper, Boer, & Sitsen, 2003). Furthermore, during both the prenatal and postpartum periods, depressive and anxiety symptoms are highly prevalent (Lee et al., 2007; Stuart, Couser, Schilder, O'Hara, & Gorman, 1998). High anxiety during the prenatal period has even been found to increase the likelihood of postpartum depression (Austin, Tully, & Parker, 2007). Also, depressed pregnant women have been found to have higher levels of cortisol, a stress hormone associated with anxiety, compared to nondepressed women (Diego et al., 2004; Field, Hernandez-Reif, et al., 2006).

Prenatal anxiety, which is highly correlated with depression, even in pregnancy (Goodman & Tully, 2009), has been studied in relation to infant NA and other

temperament factors, but not PA. Given that anxiety and depression are closely related and that prenatal anxiety has been found to impact infant temperament, it is essential to examine whether prenatal anxiety may impact infant PA. Both prenatal depression and anxiety have been found to be associated with infant temperament (Davis et al., 2007; Davis et al., 2004). Further, maternal pregnancy-specific and general prenatal and postnatal anxiety have been found to be independently related to the mothers' perceptions of their infants' temperamental difficulties, with chronically high anxiety amongst mothers predicting the highest levels of infant activity and NA (Henrichs et al., 2010). The limited research in this area demonstrates not only a close association between prenatal depression and anxiety but also an association between mothers' prenatal anxiety and infant temperament. Therefore, we will compare mothers' prenatal and postpartum depression with mothers' anxiety levels in terms of the ability of these variables to predict infant trait PA levels. Additionally, as a component of the overall construct of PA, we will examine the role of infant state PA in this relationship.

The Present Study

Studies on the relationship between mothers' depression and infant PA have left important questions unanswered, particularly in terms of mediators of this relationship and the role of prenatal depression and trait PA. Also, studies in this field generally focus on NA. The literature on mothers' depression and their offspring's PA has mainly examined the relationship between history of depression and trait PA (Kovacs & Lopez-Duran, 2010) and between postpartum depression and state PA (S.B. Campbell et al., 1995; Cohn et al., 1990; Field et al., 1990; Righetti-Veltema et al., 2002). Research has not simultaneously examined the association between depression during the prenatal and

postpartum periods and both state and trait PA. So, it remains unclear as to how exactly perinatal maternal depression may be associated with infants' PA. Also, though several models have theorized about the mechanisms by which adverse outcomes arise for offspring of mothers who experience depression during the perinatal period, there is limited empirical evidence regarding these mechanisms and an even smaller body of literature that specifically looks at the mechanisms by which offspring of depressed mothers develop low PA. Without an understanding of the role of perinatal depression in infants' development of low PA, it is difficult to fully determine the origins of this early vulnerability to psychopathology and therefore difficult to develop effective intervention and prevention strategies.

The current study improved upon several aspects of the literature. We studied the impact of both prenatal and postpartum depression on infant state and trait PA. This allowed us to gain a better understanding of the role of mothers' depression in infants' development of PA. We sampled women of primarily middle socioeconomic status, which eliminated the confounds present in several previous studies. Also, we sampled women with a history of depression and/or anxiety, thus enhancing the likelihood of finding participants who experienced perinatal depression. We examined depressive symptoms on a continuum, which gave us the opportunity to determine how the varying severity of depressive symptoms affects infant PA. In addition, we tested infant EEG asymmetry and parenting qualities as possible mediators for the relationship between mothers' depression and infant PA. We also examined infant EEG asymmetry as a potential marker for low PA and assessed the role of mothers' prenatal anxiety relative to mothers' depression in predicting infant PA. Studying infant EEG and parenting

behaviors as mediators aided in the understanding of mechanisms contributing to levels of state and trait PA in infants of mothers who have experienced depression. Further, we were able to tease apart the impact of mothers' anxiety and depression on infant PA in order to gain a better sense of which aspects of mothers' psychopathology primarily contribute to infant PA levels.

Overall, the present study sought to provide a more comprehensive examination of the association between mothers' perinatal depression and their infants' PA. We relied on a sample of mothers with a history of depression and/or anxiety and their 3- and 6-month old infants to test these associations. Since individual differences in trait PA have been found to emerge at around 2 to 3 months of age (Rothbart, 1989) and because studies have found support for PA deficits in infants of depressed mothers during the first six months of life, in the current study, infants were studied at both 3 and 6 months of age. Additionally, examining infant PA at multiple points during the first six months of life gave us a unique understanding of the early individual differences of this vulnerability to depression in at-risk infants and allowed us to compare infant PA levels at 3 and 6 months of age.

Hypotheses

1. Mothers' cumulative prenatal depression symptom levels and cumulative depression symptom levels during the first 3 months postpartum would be significantly, negatively correlated with infant state and trait PA at 3 and 6 months of age. Mothers' cumulative depression symptom levels during the first 6 months postpartum would be significantly, negatively correlated with infant state and trait PA at 6 months of age.

2. Infant EEG (the pattern of relatively greater right frontal EEG asymmetry) would mediate the relationship between mothers' depression symptom levels (at each point in time) and infants' concurrent and prospective state and trait PA.

3. Mothers' observed parenting qualities: levels of sensitivity/responsiveness, withdrawal and time spent in displays of positive affect during face-to-face interactions with their infants would also mediate the relationship between mothers' depression symptom levels and infant PA.

4. Maternal prenatal anxiety levels would be significantly, negatively correlated with infant state and trait PA at 3 and 6 months but, relative to mothers' prenatal anxiety, mothers' prenatal and postpartum depression levels would account for more of the variance in infant PA.

Method

Participants

All participants were part of a longitudinal study titled *Perinatal Stress and Gene Influences: Pathways to Infant Vulnerability*. Women were primarily recruited from the Women's Mental Health Program (WMHP) of the Department of Psychiatry and Behavioral Sciences at Emory University through referrals from their doctors. Other women who were recruited included those who had clinical evaluations at the WMHP and Grady Satellite Clinic as well as women screened to participate in research at the Emory Mood and Anxiety Disorders Program who were excluded as a result of a positive pregnancy test. Several other strategies including annual mailings, flyers at WMHP and Atlanta obstetrics practices, and educating staff at the WMHP and other clinical programs

at Emory about this research, in addition to an annual referral dinner for community clinicians that WMHP hosted, were used for recruitment.

The women in this study 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). The participants were also required to be less than 16 weeks pregnant from their last menstrual period, between the ages of 18 and 45, fluent in written and verbal English, able to give informed consent and follow the study procedures, capable of identifying their infant's biological father and willing to complete a minimum of one prenatal assessment. Women were excluded for exhibiting active suicidality or homicidality, having psychotic symptoms, meeting DSM-IV criteria for bipolar disorder, schizophrenia, and/or a currently active eating disorder, having an active substance use disorder within 6 months before her last menstrual period and/or a positive urine drug screen, having an illness requiring treatment that could influence the study, such as epilepsy, asthma, autoimmune disorders, and having abnormal thyroid stimulating hormone or anemia. We used the Structured Clinical Interview for the Diagnostic and Statistical Manual- IV Axis I Disorders – Patient Edition (SCID; First, Spitzer, Gibbon, & Williams, 1995), a drug screening, a self-report of participants' illnesses and a blood test to screen for these exclusion criteria.

The present study included data from 194 women with a history of depression and/or anxiety. Women were seen in the lab when their infants were 3 months of age and again when the infants were 6 months of age. Of the infants in the study, 46.60% were female and 53.40% were male. The women were between the ages of 20.70 and 44.5 at the time of delivery (M = 33.88 years, SD = 4.29). The mean educational attainment was

16.47 years. Most women identified themselves as White (87.10%) while 9.30% were Black, 2.90% were Asian, and 0.70% were Native American. About 43.10% of the mothers were primiparous and 81.90% were married. The sample was primarily of middle socioeconomic status (M = 51.93), as measured by the Hollingshead scale (Hollingshead, 1975).

Procedures

Data were collected during pregnancy and the postpartum period. Throughout pregnancy, the participants filled out The Beck Depression Inventory (BDI; (A. T. Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) an average of 5.25 times, with a range of 1 to 13 (SD = 1.82) and also completed the State-Trait Anxiety Inventory (STAI) (Spielberger, Gorsuch, & Lushene, 1970) to measure anxiety an average of 4.53 times, with a range of 1 to 14 (SD = 2.34). Women completed the BDI during the postpartum period until the infant was 6-months old, filling out an average of 4.92 BDI's ranging from 1 to 16 (SD = 2.647).

Mothers visited the laboratory with their infants when their infants were 3 months of age and then 6 months of age. When the infants were 3 months of age, 194 mothers completed the lab visit. At 6 months, 181 women completed the visit and 171 completed the visits at both 3 and 6 months. During each lab visit, mothers and their infants participated in a 3-minute baseline, 5-minute feeding, and 5-minute free play segment. At the beginning of the visit, an EEG cap was placed on the infant's head and research assistants played with the infants to distract them from the capping. EEG activity was recorded during each segment. During the baseline segment, a research assistant blew bubbles while the infant sat on his or her mother's lap. The mothers were told not to

speak during this segment; the baseline portion of the study was meant to elicit a quiet, alert state for the infant and minimize eye and gross motor movements. For the feeding segment, the mother breast- or bottle-fed the infants. During the free play segment, the mother was instructed to play with her infant however she wanted and was given a standard set of toys from the laboratory to play with. These segments were all video-recorded. The women completed the Infant Behavior Questionnaire-Revised (IBQ-R; Gartstein & Rothbart, 2003) at each lab visit.

The present study included analyses of the EEG recordings and free play segments for the 3- and 6-month visits. At 3 months of age, 71 infants had usable baseline EEG data and one outlier 3 standard deviations below the mean was excluded from analyses. At 6 months of age, 71 infants had usable baseline EEG data. Because editing EEG files is a time-intensive process, these numbers reflect the segments that were available for analyses at the time the present study was conducted.

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 measure

Beck Depression Inventory (A. T. Beck et al., 1961). This is a self-report measure of depression symptoms that has been found to be valid and reliable and has particularly high content validity and internal consistency (A. T. Beck et al., 1961). Participants are asked 21 questions based on how they were feeling during the previous week. All items are given scores on a 4-point rating scale from 0 to 3. The individual scores are added to yield an overall score and higher scores translate to increased severity of depressive symptoms. Scores of 0-9 signify no depression, 10-18 suggests mild-moderate depression, 19-29 indicates moderate-severe depression, and 30-63 suggests severe depression (A. T. Beck et al., 1961). For mothers' prenatal depression, we utilized area under the curve (AUC) scores on the BDI during pregnancy since AUC scores allowed us to assess mothers' cumulative symptom levels, and therefore infants' total exposure to their mothers' symptoms. AUC scores were calculated according to methods used by Pruessner, Kirschbaum, Meinlschmid, and Hellhammer (2003). Similarly, for postpartum depression, we used mothers' BDI AUC scores for the first 3 months, months 4 through 6 and for the first 6 months of the postpartum period.

Parenting Quality Measures

Maternal Interactive Quality Ratings. Mothers' quality of interaction with their infants during face-to-face play interactions was rated on the Maternal Interactive Quality Rating Scale, composed of 14 scales derived from the standardized rating scales of Ainsworth (Ainsworth, Blehar, Waters, & Wall, 1978), Clark (1985), and Campbell (1991). The scales measure the mother's interactive behavior with her infant and particularly assess behaviors known to be early life stressors. The scales are broken up into five categories: 1) Insensitive Parenting, 2) Intrusiveness, 3) Withdrawal, 4) Positive Affect and 5) Negative Affect. Insensitive Parenting included two scales from Campbell: *sensitivity/responsiveness to distress* and *sensitivity/responsiveness to nondistress.* Intrusiveness was assessed using two scales: Campbell's *intrusiveness* and Ainsworth and colleagues' *cooperation vs. interference*. The Withdrawal scales measured *detachment/disengagement* and *flatness of affect*. Positive Affect included Campbell's scales for *positive regard for the child*, *warmth,* and *stimulation of development*. *Quality of verbalizations* and *structures and mediates environment* were scales created by Clark that were added to the maternal interactive quality ratings after the initial use of the rating scales. The Negative Affect scales were also from Clark and included *quality and amount of physical contact: negative*; *angry, hostile mood*; and *displeasure, disapproval, criticism*. Each scale is scored on 4- or 5-point Likert scale. Ratings take into account quality and quantity/intensity of the maternal behaviors that each scale measured. To obtain the sensitivity/responsiveness scores for 3 and 6 months used in the present study, we calculated the mean of the *sensitivity/responsiveness to distress* and *sensitivity/responsiveness to nondistress* scales. For the withdrawal scores for 3 and 6 months, we calculated the mean of *positive regard for the child*, *warmth, stimulation of*

development, q*uality of verbalizations, structures and mediates environment,* and reverse scores of *detachment/disengagement* and *flatness of affect.* Higher scores on the mean sensitivity/responsiveness and withdrawal variables indicated more positive parenting qualities.

Raters were trained by rating free play segments from a previously conducted study and discussing those ratings with a senior clinical psychologist until reaching agreement on the ratings. Ratings for the current study began once there was consistently high inter-rater reliability among the team members, meaning the members disagreed by no more than one point on no more than a few scales. The raters were blind to the depression status of the mothers in the segments they rated. Reliability was checked each week for randomly selected segments. Reliability involved two members independently rating the same segment. Reliability ratings were completed for 20% of the segments.

For the mean withdrawal scores at 3 and 6 months, Pearson correlations revealed interrater reliability of .90 and .71 respectively. For the mean sensitivity scores at 3 and 6 months, Pearson correlations revealed interrater reliability of .54 and .27 respectively. In addition to computing exact interrater reliability, we assessed the degree of agreement within 1 point on the scales by recoding the sensitivity mean and withdrawal mean ratings into low and high scores. So, for the 4-point scales, ratings of 1 and 2 were recoded to a rating of 1 so they could be grouped together and scores of 3 and 4 were recoded into a score of 2 for the purpose of these reliability statistics. For the 5-point scales, scores of 1 and 2 were recoded as a rating of 1 and scores of 4 and 5 were recoded as a 2, while the ratings of 3 were not recoded. This yielded a Spearman correlation coefficient of .55 for the withdrawal mean score at 6 months. For the sensitivity mean

scores at 6 months, the raters agreed within 1 point on 86% of the segments rated for reliability. A correlation coefficient could not be calculated for the sensitivity mean scores at 6 months because of the limited variability in the data used for reliability. Research assistants were assigned segments to rate in a way that allowed each rater to be paired with each of the others an equal number of times while also ensuring each team member was the reliability and primary rater an equal number of times. Raters met each week to discuss the segments that had been rated for reliability, in addition to any questions about other segments rated that week.

Mother Observed Affect Coding. The videotaped free play segments were coded continuously for maternal affect following procedures similar to those used for the infant affect coding. The affect behaviors coded were Laughter, Smile, Interest, None, Worry/Tension, Brief Marked Distress and Marked Distress. These codes were modified versions of Dawson's Wilson-Rubesch-Clark Global Affect Rating Scale for Mother and Experimenter in the EEG. The data were considered "uncodeable" when the mother was out of the view of the camera and in other similar situations that could not be clearly coded. All coding was done independently and observers were blind to the past and current levels of depression of each of the women.

To train for maternal affect coding, each coder independently rated the same segment and then discussed that segment until reaching agreement. This was done until all team members coded several segments with high degrees of agreement. For reliability, 20% of segments were randomly selected and two coders independently coded the same segment. Kappa coefficients for reliability were calculated based on exact agreement, while providing a 1-second time frame for inter-rater reaction time. Coders discussed

disagreements on reliability segments until agreement was reached, however, scores were not changed following these discussions. Kappa coefficients were .80 and .82 for the maternal affect coding at 3 and 6 months respectively. The relative duration (total percentage of time) the mother spent in positive affect states, which was a combination of the Laughter, Smile and Interest codes, was used in analyses for the present study. By studying relative duration, we statistically controlled for portions of the segment that were uncodeable. Previous studies have analyzed percentage of time as a means to study the amount of time an individual exhibited a particular affect state (S. C. Crockenberg, Leerkes, & Lekka, 2007).

Infant Affect Measures

Infant observed affect coding. The videotaped free play segments were coded continuously for infant affect, with coders noting the exact second an affective change took place. The affect behaviors coded were Approach, Withdrawal, and Neutral for the 3-month-old infants and High Level Approach, Low Level Approach, Neutral, Low Level Withdrawal and High Level Withdrawal for the 6-month-old infants. Codes were modified versions of Dawson's Wilson-Rubesch-Clark Global Affect Rating Scale for Mother and Experimenter in the EEG. The data were considered "uncodeable" when the infant was out of the view of the camera and in other similar situations that could not be clearly coded. Observers were blind to the past and current levels of depression of each of the women.

Training for infant affect coding consisted of each coder independently coding the same segment and then discussing the segment until reaching a consensus. Once all members of the team independently coded several segments with high degrees of inter-

rater agreement, training was complete. Thereafter, in weekly meetings, segments were randomly selected for reliability, which meant two coders would independently code the same segment and allowed for the calculation of interrater reliability. In all, 20% of segments were coded for reliability. Kappa coefficients were calculated for each segment based on exact agreement, while providing a 1-second time frame for inter-rater reaction time. Disagreements were discussed until a consensus was achieved, though scores were not changed. Kappa coefficients were .77 and .78 for the infant affect coding at 3 and 6 months respectively.

For the present study, we examined the relative duration (total percentage of time) the infant spent in positive affect states, which was coded as Approach at 3 months and a combination of High and Low level Approach at 6 months.

The Infant Behavior Questionnaire-Revised (Gartstein & Rothbart, 2003). The IBQ-R measures infant temperament, according to Rothbart and Derryberry's (1981) definition of temperament. The factor-analytically derived measure is composed of 191 items that the mothers answered just prior to, or during, each of the two lab visits. The questionnaire calls for the respondent to rate the infant's behavior during the past week. Items on the IBQ-R include questions such as *how often during the last week did the baby laugh aloud in play* and *when playing quietly with one of her/his favorite toys, how often did the baby show pleasure*. All items are scored on a 7-point scale, from 1 (Never) to 7 (Always). The IBQ-R includes 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) and

yields three overall factor scores: Orienting/Regulatory Capacity, Surgency/Extraversion, and Negative Affectivity. The mean for items on each individual scale is calculated. Each scale contains a different number of items, with the number of items per scale ranging from 10 to 18.

The present study relied on the Surgency/Extraverison factor score to measure infant trait PA at both 3- and 6-months of age. This overall factor is comprised from these scales: Approach, Vocal Reactivity, High Intensity Pleasure, Smiling and Laughter, Activity Level and Perceptual Sensitivity. The mean of these scales comprised the overall Surgency/Extraversion scores, which range from 1 to 7, with higher scores indicating higher levels of infant PA. All 14 scales of the IBQ-R have demonstrated adequate internal consistency (Gartstein & Rothbart, 2003). Gartstein and Rothbart (2003) found inter-rater agreement between primary and secondary caregivers for Surgency/Extraversion (r = 0.49)**.**

Anxiety Measure

The State-Trait Anxiety Inventory (Spielberger et al., 1970). The STAI measures trait and state markers of anxiety, with 20 items assessing each. Respondents answered the items based on how well the statement described their current feelings along a 4-point Likert scale (1 = not at all, 4 = very much so). The scores for all items were added to compute the total score, which ranges from 20-80. Higher scores indicated higher levels of anxiety. We used scores from the state items rather than the trait items on the STAI since they were more in line with the depression measures we used. Research has found sufficient concurrent validity and internal consistency for the STAI (Spielberger et al., 1970). We used AUC scores during pregnancy for the STAI in our analyses.

Analytic Plan

To test our first hypothesis, that mothers' cumulative depression symptom levels during the prenatal period and the first 3 and 6 months of the postpartum period would be prospectively and concurrently negatively correlated with infant state and trait PA at 3 and 6 months of age, we tested Pearson bivariate correlations between each of the independent and dependent variables. We performed bivariate correlations for the association between mothers' prenatal BDI AUC levels and infant state PA at 3 months (measured using relative duration of observed infant Approach at three months from the coded affect) and 6 months (using relative duration of observed infant High and Low level Approach). The associations between prenatal BDI AUC levels and infant trait PA (measured using the Surgency/Extraversion factor of the IBQ-R) both at 3 and 6 months were also tested. Pearson bivariate correlations were then run using the postpartum BDI AUC scores for the first 3 months and all of the infant PA measures at 3 and 6 months. Pearson correlations were also run between mothers' BDI AUC levels for the first 6 months and infant relative duration of observed positive affect at 6 months and infant Surgency/Extraversion at 6 months.

Next, we tested the hypothesis that infant baseline EEG (the pattern of relatively greater right frontal EEG asymmetry) would mediate the relationship between mothers' depression (at each point in time) and infants' PA. We conducted bivariate correlational analyses between mothers' prenatal depression AUC scores on the BDI and infant EEG at 3 months and at 6 months (measured using asymmetry scores) and between mothers' BDI AUC levels for the first 3 months postpartum and infant EEG asymmetry scores at both ages. We also conducted Pearson correlations between mothers' BDI AUC levels

for the first 6 months postpartum and infant EEG asymmetry at 6 months of age. Next, we examined the association between each of the mediators and the dependent variables for this hypothesis. We conducted bivariate correlations between infant 3 month EEG and infant relative duration of observed Approach at 3 months; between 3 month infant EEG and infant relative duration of observed positive affect at 6 months; between infant 3 month EEG and infant Surgency/Extraversion scores on the IBQ-R at 3 months; between infant 3 month EEG and infant Surgency/Extraversion at 6 months; between infant 6 month EEG and infant relative duration of observed positive affect at 6 months; and between infant 6 month EEG and infant Surgency/Extraversion at 6 months.

To test the hypothesis that infant EEG would mediate the relationship between mother's depression and infant state and trait PA, we used the MEDIATE macro plug-in for SPSS, which follows statistical procedures outlined in Hayes and Preacher (2011). Though the Baron and Kenny (1986) method of testing mediation has been widely used, there are many advantages to the Hayes and Preacher (2011) method. While the Baron and Kenny procedure will show whether there is a significant mediator or not, the methods used in the MEDIATE macro will not only indicate the presence or absence of a significant mediator, but will also yield an estimate of the magnitude of the mediated effect (Preacher & Kelley, 2011). In addition, the Baron and Kenny method is usually low-powered (MacKinnon, Fairchild, & Fritz, 2007). The macro plug-in, on the other hand, uses bootstrapping to estimate the standard error of the mediated effect and this procedure requires fewer subjects than the Baron and Kenny method to find a mediated effect of the same magnitude (Fritz & MacKinnon, 2007).

For the hypothesis that mothers' observed parenting qualities (measured using average scores for ratings of mothers' sensitivity/responsiveness to distress and nondistress during free play at both 3 and 6 months, average scores for ratings of mothers' withdrawal at both 3 and 6 months, and relative duration of time mothers spent in displays of PA during face-to-face play interactions with the infants at both 3 and 6 months) would mediate the relationship between mothers' depression symptom levels and infant PA, we ran statistical tests similar to those used for the hypothesis regarding infant EEG. We performed Pearson bivariate correlations between our independent variables (mothers' prenatal BDI AUC scores and mothers' BDI AUC scores for the first 3 months postpartum) and each of our 3- and 6-month parenting quality mediators. We also conducted Pearson correlations between mothers' postpartum BDI AUC levels for the first 6 months and each of the 6-month parenting quality mediators. To test the association between these mediators and the dependent variables, we performed Pearson correlations between our 3-month parenting quality variables and each of the 3- and 6-month PA indices and between our 6-month parenting quality variables and the 6-month PA indices. Finally, we tested mediation by using the MEDIATE macro plug-in for SPSS.

For our final hypothesis, we predicted that mothers' prenatal anxiety levels would be significantly, negatively correlated with infant state and trait PA at 3 and 6 months but that, relative to prenatal anxiety, mothers' prenatal and postpartum depression levels would account for more of the variance in infant PA. We first ran Pearson bivariate correlations between mothers' prenatal anxiety (measured using the STAI AUC levels) and each of the depression and infant PA variables. In order to assess whether mothers'

depression predicted infant PA beyond any contributions of prenatal anxiety to these PA levels, we tested hierarchical multiple regressions involving mothers' prenatal depression and anxiety and mother's postpartum BDI AUC scores for the first 3 months as predictors and each of the 3-month infant PA indices as the dependent variables. We also tested a hierarchical multiple regression involving mothers' prenatal depression and anxiety and mother's postpartum BDI AUC scores for the first 3 months and for months 4 through 6 as predictors and each of the 6-month infant PA indices as the dependent variables. This allowed us to compare the variance in infant PA accounted for by mothers' anxiety relative to mothers' depression.

Results

Preliminary Analyses and Descriptive Statistics

Associations between demographics and infant PA variables were assessed to determine if any demographic variables needed to be controlled for in the analyses (see Tables 1 and 2). A Bonferroni correction was used as a result of the large number of tests run with demographic variables, thus yielding a significance level of .002. Only one demographic variable was associated with any of the PA variables at this significance level: mothers' age at time of delivery was significantly, negatively correlated with infant Surgency/Extraversion at 3 months of age (see table 2). Therefore, we did not control for any demographic variables.

Table 3 shows the Pearson bivariate correlations among the predictor variables. Given what is known about the continuity of depression during the perinatal period, these correlations show the expected associations between the BDI AUC scores at each time point. Further, due to the comorbidity of depression and anxiety, our findings regarding

the association between prenatal STAI AUC levels and the BDI AUC prenatal and postpartum levels are in line with what was expected. Table 4 shows the Pearson bivariate correlations among the parenting variables. We found much consistency within parenting variables over time and also across the different variables.

Hypothesis Testing

Hypothesis 1. We hypothesized that mothers' cumulative prenatal depression symptoms and cumulative depression symptom levels during the first 3 months postpartum would be significantly, negatively correlated with infant state and trait PA at 3 and 6 months. We further predicted that mothers' cumulative postpartum depression symptom levels during the first 6 months postpartum would be significantly, negatively correlated with infant state and trait PA at 6 months. To test our first hypothesis, we ran Pearson bivariate correlations for each of the proposed associations. Results are shown in Table 5. Contrary to our hypothesis, mothers' prenatal depression levels were not significantly associated with any of the infant indices of PA: infant relative duration of observed Approach at 3 months, infant relative duration of observed positive affect at 6 months, or Surgency/Extraverison scores on the IBQ-R at 3 or 6 months.

Contrary to our hypothesis, mothers' postpartum BDI AUC scores for the first 3 months postpartum were not significantly correlated with any of the scores of infant PA at 3 months, but they were significantly correlated with one of the scores of infant PA at 6 months. Consistent with our hypothesis, mothers' postpartum BDI AUC scores for the first 3 months postpartum were significantly negatively correlated with infant Surgency/Extraversion scores at 6 months of age ($r(159) = -.23$, $p < .01$). That is, higher levels of mothers' postpartum depression symptoms for the first 3 months were

significantly associated with lower infant Surgency/Extraversion scores on the IBQ-R at 6 months.

Also consistent with our hypothesis, mothers' postpartum BDI AUC scores through 6 months were significantly, negatively correlated with infant Surgency/Extraversion scores on the IBQ-R at 6 months ($r(132) = -.26$, $p < .01$). Higher BDI AUC symptom levels during the first 6 months postpartum were related to 6-month old infants' lower levels of Surgency/Extraversion scores on the IBQ-R. Contrary to our hypothesis, mothers' postpartum depression AUC scores through 6 months postpartum were not significantly associated with relative duration of observed infant positive affect at 6 months of age (see Table 5).

In order to tease apart the impact of exposure to mothers' depression during the first 3 months from exposure to mothers' depression during months 4 through 6, we also ran a Pearson bivariate correlation between mothers' BDI AUC scores during months 4 through 6 and infant Surgency/Extraversion at 6 months of age. We found a significant association between mothers' postpartum BDI AUC scores during months 4 through 6 and infant Surgency/Extraversion at 6 months ($r(159) = -.20$, $p < .05$). Higher postpartum BDI AUC scores for months 4 through 6 of the postpartum period were related to lower infant Surgency/Extraversion scores at 6 months.

To examine if mothers' prenatal depression symptom levels played a role in infant Surgency/Extraversion at 6 months in the context of mothers' postpartum depression, and to compare the impact of mothers' depression symptom levels during the first 3 months postpartum with mothers' depression symptom levels during months 4 through 6, we conducted a hierarchical multiple regression. For the first step, mothers' prenatal BDI

AUC was entered. Mother's postpartum BDI AUC for the first 3 months was entered for step two and mother's postpartum BDI AUC for the second 3 months was entered for step three to predict infant Surgency/Extraversion scores on the IBQ-R at 6 months. Results indicated that mothers' postpartum BDI AUC levels for the first 3 months alone significantly predicted infant Surgency/Extraversion at 6 months, accounting for 4.50% of the variance in infant Surgency/Extraversion scores. Mothers' prenatal BDI AUC levels and mothers' postpartum BDI AUC levels for months 4 through 6 did not account for significant additional variance in infant 6-month Surgency/Extraversion scores (see Table 6).

Hypothesis 2. We predicted that infant EEG asymmetry would mediate the relationship between mothers' depression (at each point in time) and infants' concurrent and prospective state and trait PA. First, we examined the association between mothers' BDI AUC levels, during pregnancy and the first 3 months of the postpartum period, and infant EEG asymmetry scores at 3 and 6 months. We then tested the association between mothers' postpartum BDI AUC levels during the first 6 months and infant EEG asymmetry scores at 6 months of age. Pearson correlations revealed that mothers' prenatal BDI AUC levels were not significantly associated with infant EEG asymmetry scores at 3 months ($r(66) = .11$, $p = .38$) or at 6 months ($r(65) = .11$, $p = .39$). Mothers' postpartum BDI AUC levels for the first 3 months were also not significantly associated with infant EEG asymmetry scores at 3 months ($r(69) = .05$, $p = .66$) or 6 months ($r(66) = .06$, $p = .64$). Finally, mothers' postpartum BDI AUC levels for the first 6 months were not significantly associated with infant EEG asymmetry scores at 6 months ($r(70) = -.04$, $p = .72$)

Second, we tested the associations between infant EEG asymmetry scores at 3 months of age and infant relative duration of observed Approach at 3 months, infant relative duration of observed positive affect at 6 months, and infant Surgency/Extraversion scores on the IBQ-R at 3 and 6 months. We examined the association between infant EEG asymmetry scores at 6 months and infant relative duration of observed positive affect at 6 months and infant Surgency/Extraversion at 6 months as well. Infant EEG asymmetry scores at 3 months were significantly associated with infant relative duration of Approach at 3 months ($r(65) = .28$, $p = .03$). Relatively greater left frontal EEG asymmetry at 3 months of age was associated with higher levels of infant relative duration of Approach at 3 months of age. Infant EEG asymmetry scores at 3 months were not significantly associated with infant relative duration of observed positive affect at 6 months ($r(58) = -.05$, $p = .72$), or with infant Surgency/Extraversion at either 3 months ($r(69) = .14$, $p = .25$) or 6 months ($r(60) = .05$, $p = .69$). In addition, infant EEG asymmetry scores at 6 months were not significantly associated with infant relative duration of observed positive affect at 6 months ($r(62) = -.08$, $p = .53$) or infant Surgency/Extraversion at 6 months ($r(65) = -.06$, $p = .62$).

Next, we used the MEDIATE macro plug-in for SPSS, which utilizes statistical procedures outlined in Hayes and Preacher (2011), to assess whether infant EEG asymmetry scores mediated the relationship between mothers' BDI AUC symptom levels for the first 6 months of the postpartum period and infant Surgency/Extraversion scores on the IBQ-R at 6 months. We used the BDI AUC scores for the first 6 months postpartum when testing this mediation rather than the BDI AUC scores for the first 3 months postpartum in order to assess the role of the infants' total exposure to the

mothers' depression symptoms during the first 6 months in this relationship. Contrary to our hypothesis, we did not find evidence for mediation (see Table 7).

Hypothesis 3. In order to test the hypothesis that mothers' observed parenting qualities (levels of sensitivity/responsiveness, withdrawal and time spent in displays of positive affect during face-to-face interactions with their infants) would mediate the relationship between mothers' depression symptoms and infant PA at each time point, we ran Pearson bivariate correlations between the mediators and the depression scores and between the mediators and infant PA variables. Mother's prenatal BDI AUC levels, postpartum BDI AUC levels for the first 3 months, and postpartum BDI AUC levels for the first 6 months were not significantly correlated with any of the parenting quality variables (see Table 8). Some parenting quality variables were significantly associated with observed infant PA variables (see Table 9). For instance, mothers' sensitivity/responsiveness at 3 months, withdrawal at 3 months and relative duration of observed positive affect at 3 months were significantly associated with infant relative duration of Approach at 3 months. Also, mothers' sensitivity/responsiveness at 3 months and mothers' relative duration of observed positive affect at 6 months were both significantly, positively correlated with infant relative duration of observed positive affect at 6 months of age.

We used the MEDIATE macro plug-in for SPSS to assess whether the observed parenting qualities mediated the relationship between mother's BDI AUC symptom levels for the first 6 months of the postpartum period and infant Surgency/Extraversion scores on the IBQ-R at 6 months. Contrary to our hypothesis, we did not find significant mediating effects for these associations (see Table 7).

Hypothesis 4. We hypothesized that mothers' prenatal anxiety levels would be significantly, negatively correlated with infant state and trait PA at 3 and 6 months but that mothers' prenatal and postpartum depression symptom levels would account for more of the variance in infant PA at 3 and 6 months. Mothers' prenatal STAI AUC levels were significantly, positively correlated with mothers' BDI AUC levels for the prenatal period and the first 3 and 6 months postpartum (see Table 3). Contrary to our hypothesis, Pearson bivariate correlations revealed mothers' prenatal STAI AUC scores were not significantly associated with infant observed relative duration of Approach at 3 months ($r(111) = .10$, $p = .30$), infant observed relative duration of positive affect at 6 months ($r(118) = .11$, $p = .23$), infant Surgency/Extraversion scores at 3 months ($r(138) = .05$, $p = .54$) or infant Surgency/Extraversion scores at 6 months ($r(128) = -.02$, $p = .81$).

To assess the contributions of mothers' prenatal anxiety to infant PA in the context of mothers' prenatal and postpartum depression, we conducted hierarchical multiple regressions. For the first step, mothers' prenatal BDI AUC and mothers' prenatal STAI AUC were both entered. Mothers' postpartum BDI AUC scores for the first 3 months was entered for the second step and mothers' postpartum BDI AUC for the second 3 months was entered for the third step to predict infant Surgency/Extraversion scores on the IBQ-R at 6 months. Results indicated that mother's postpartum BDI AUC levels during the first 3 months alone significantly predicted infant Surgency/Extraversion at 6 months, which was partially in line with our hypothesis, accounting for 4.5% of the variance in infant Surgency/Extraversion scores. Mothers' prenatal STAI AUC scores, prenatal BDI AUC levels and mothers' postpartum BDI AUC levels for months 4 through 6 did not account for significant additional variance in

infant 6-month Surgency/Extraversion scores (see Table 11). Similar regressions were run for infant Surgency/Extraversion at 3 months (see Table 10), infant relative duration of Approach at 3 months (see Table 10), and infant relative duration of observed positive affect at 6 months (see Table 11). The hierarchical regressions involving the 3-month infant PA measures only included the first and second steps since we were not assessing retrospective associations between mothers' depression symptom levels and infant PA. These regression models did not support our hypothesis.

Discussion

The present study was the first, to our knowledge, to not only examine the role of both prenatal and postpartum depression symptoms in the association between mothers' depression symptom levels and infant state and trait PA but also to propose, and empirically test, a model for mediators of this association. We found support for the prediction that mothers' cumulative postpartum depression symptom levels during both the first 3 and 6 months would be significantly negatively correlated with infant trait PA at 6 months. That is, higher levels of depression symptoms for the first 3 and 6 months postpartum were associated with lower levels of infant trait PA at 6 months. We did not find support for the predicted association between mothers' cumulative postpartum depression symptom levels during the first 3 months and infant trait PA at 3 months. We did not find support for the predicted association between mothers' cumulative postpartum depression symptoms during the first 3 months and infant state PA scores at 3 or 6 months. Also, our findings did not support the predicted association between mothers' cumulative postpartum depression symptoms during the first 6 months and infant state PA scores 6 months. Our findings did not support the hypothesis that

mothers' cumulative prenatal depression symptoms would be significantly, negatively correlated with infant state and trait PA at 3 and 6 months. Contrary to our hypotheses, infant EEG asymmetry, mothers' sensitivity, mothers' withdrawal and mothers' relative duration of observed positive affect did not mediate the relationship between mothers' depression and infant PA. Also contrary to our hypothesis, mothers' prenatal anxiety severity was not significantly, negatively correlated with infant state or trait PA at 3 or 6 months. However, in support of our hypothesis, we found mothers' postpartum depression levels during the first 3 months significantly predicted infant trait PA at 6 months beyond the contribution of mothers' prenatal anxiety. Contrary to our hypothesis, mothers' depression levels during the prenatal and postpartum periods did not significantly account for more of the variance in the other PA indices as compared with mothers' prenatal anxiety levels.

Our first hypothesis was intended to replicate findings that mothers' postpartum depression was associated with infant state PA (S.B. Campbell et al., 1995; Cohn et al., 1990; Field et al., 1990; Righetti-Veltema et al., 2002). In addition, we sought to extend these findings by also examining the role of cumulative maternal prenatal depression symptom levels and infant trait PA in this association, while also testing possible mediators. Our results did not support previous findings that indicated that mothers' postpartum depression is associated with lower infant state PA (S.B. Campbell et al., 1995; Cohn et al., 1990; Field et al., 1990; Righetti-Veltema et al., 2002). Unlike previous studies, which analyzed depression as a dichotomous variable based on diagnoses (S.B. Campbell et al., 1995; Cohn et al., 1990) or cut points (Field et al., 1990; Righetti-Veltema et al., 2002), the present study examined depression symptom levels on

a continuum in order to study women with a wider variety of symptom levels. Moreover, Field et al. (1990) excluded women whose BDI scores fell between the depressed and nondepressed cut scores, thus focusing on the more extreme scores rather than the full range of symptom levels. Also, some researchers observed mother-infant interactions in the home (S.B. Campbell et al., 1995; Cohn et al., 1990; Righetti-Veltema et al., 2002), whereas the present study utilized lab visits to assess state PA during mother-infant interactions so that we could assess PA in a more controlled, standardized environment than the home. Future studies should include measures of PA of infants of depressed mothers in both settings to ensure the mother's depression symptoms are impacting infant PA independent of the setting. Although other studies have found that mothers with postpartum depression have infants with lower state PA than mothers without depression, our findings may suggest that this association may only occur among a subset of women with high levels of clinical symptoms or depression that meets diagnostic criteria or cut scores.

Mothers' cumulative prenatal depression symptoms were not significantly associated with any infant PA variables. Perhaps, even though mothers' prenatal depression has been found to impact infant temperament (e.g. Davis et al., 2004; Huot et al., 2004) and newborns' expressions of interest (Lundy et al., 1996), cumulative prenatal depression symptom levels in the broader sense may not significantly impact infant PA, particularly beyond the neonatal period. There may, however, be biological correlates or other components of mothers' prenatal depression that influence infant PA. Future research should focus on elevated cortisol and other known factors associated with prenatal depression to determine if such factors are associated with infant PA. In

comparing our findings with others in the field that have found an association between mothers' prenatal depression and temperament factors such as infant NA (Huot et al., 2004) and behavioral reactivity (Davis et al., 2004), our findings suggest there may be separate pathways by which mothers' prenatal depression influences different factors of infant temperament. Researchers should study these pathways in order to gain a better understanding of the unique influences of mothers' prenatal depression on factors of temperament.

In support of our hypothesis, mothers' cumulative depression symptom levels during both the first 3 months and months 4 through 6 of the postpartum period were significantly, negatively correlated with infant trait PA at 6 months. Supplemental analyses revealed that mothers' cumulative depression symptoms during the first 3 months accounted for more of the variance in infant trait PA at 6 months than mothers' depression symptom levels during months 4 through 6. This finding suggests that the first 3 months of the postpartum period represent a critical time for infant trait PA development and a time when exposure to mothers' depression symptoms is particularly salient. Given that temperament has been discussed as a precursor to personality, and because temperament is conceptualized as an enduring trait, it is important that we found that mothers' postpartum depression symptoms influence infant trait PA as early as the first few months of life. These findings support results from studies that have found an association between mothers' postpartum depression and infant temperament (C. T. Beck, 1996; McGrath et al., 2008). Our results are contrary to those of Pauli-Pott et al. (2004), the one study we found to date that has examined the role of mothers' postpartum depression in the development of infant trait PA. Pauli-Pott et al. (2004) found maternal

depression/anxiety, sensitivity and social support did not predict infants' later development of trait PA. Because Pauli-Pott et al. (2004) assumed that the depression and anxiety scales were intercorrelated and therefore computed principle component analyses, they were not able to fully assess the impact of mothers' depression on infant trait PA independent of the impact of anxiety. Our study, however, teased apart mothers' depression and anxiety and found that higher postpartum depression alone is associated with lower infant trait PA as early as the first 6 months of life. Based on our findings, and those of Pauli-Pott et al. (2004), it seems that mothers' depression, when studied in conjunction with anxiety, may not influence infant trait PA but that mothers' depression on its own is associated with lower trait PA, particularly at 6 months of age.

Unlike 6-month trait PA, 3-month trait PA, contrary to our hypothesis, was not associated with any of the depression measures. Since individual differences in trait PA do not emerge until around 2 to 3 months of age (Rothbart, 1989), it may not be until later in the first year that these individual differences are solidified. Also, it is possible that as PA increases over time (Denham et al., 1995; Derryberry & Rothbart, 2001), mothers' depression symptoms have an increasing influence on infant PA, thus potentially explaining the difference in our findings for 3- and 6-month trait PA. Future research should longitudinally assess the relationship between mothers' postpartum depression symptom levels and infant trait PA during the first 6 months and beyond.

Despite the fact that previous studies have demonstrated a relationship between mothers' depression and infant relative right frontal EEG asymmetry (Field et al., 1995; Jones et al., 1997) during the first 6 months of life and between EEG asymmetry and PA (Diego et al., 2004; Fox, 1991; Schmidt, 2008), infant EEG did not mediate the

association between mothers' postpartum depression symptom levels for the first 6 months and infant trait PA at 6 months. Moreover, though infant EEG asymmetry at 3 months was significantly, positively correlated with relative duration of infant Approach at 3 months, infant EEG asymmetry was not significantly associated with any of the other indices of PA or any of the predictors. Much of the research on the EEG of infants of depressed mothers includes samples of women who were of low socioeconomic status, adolescent mothers and an ethnic minority. Our sample was primarily composed of women of middle socioeconomic status and did not include adolescent mothers. The stressors that mothers in other studies in this field likely faced, in addition to the depression itself, may be necessary for infant EEG to be associated with mothers' depression symptoms and infant PA and for the predicted mediation to occur. Future studies should include women with more diverse socioeconomic backgrounds. Because this is the first known study that empirically assessed infant EEG asymmetry as a mediator for the relationship between mothers' depression and infant state and trait PA, researchers should study this relationship further, and potentially assess infant EEG asymmetry as a moderator as well as a mediator. Studying infant EEG as a moderator would entail comparing PA of infants in the sample who had relatively greater right frontal EEG with those who had greater relative left frontal EEG.

Though several maternal parenting qualities were found to be associated with infant PA, we did not find significant associations between any of our depression and parenting quality variables and therefore did not find that mothers' sensitivity, withdrawal or observed positive affect at 6 months mediated the association between mothers' postpartum depression levels for the first 6 months and infant trait PA at 6

months. This is contrary to our hypothesis and does not support many findings in the field, which have indicated that depressed mothers exhibit unfavorable parenting qualities such as fewer affectionate contact behaviors (Fleming et al., 1988) and more insensitive parenting (Cooper et al., 1999; Hoffman & Drotar, 1991) compared to nondepressed mothers. We are in the midst of working to identify the best variables to capture parenting qualities out of the rich data set we currently have available. Further, it may be necessary to take into account other environmental factors such as the fathers' parenting qualities.

Finally, our results did not support the hypothesis that mothers' prenatal anxiety levels would predict infant PA. Mother's prenatal anxiety symptom levels were not significantly, negatively correlated with infant state or trait PA at 3 or 6 months. However, our findings partially supported the prediction that mothers' depression symptoms would predict variance in infant PA beyond the contribution of mothers' prenatal anxiety. So, it seems that mother's depression, rather than anxiety, is related to infant trait PA. It also appears that mothers' prenatal anxiety on its own does not significantly influence infant state or trait PA. This coincides with the tripartite model of anxiety and depression (L. A. Clark & Watson, 1991), which posits that low PA is specific to depressive symptomatology and differentiates depression from other internalizing disorders such as anxiety.

In sum, results from the present study indicate that higher cumulative maternal postpartum depression symptom levels during the first 6 months, and particularly during the first 3 months, predict lower infant trait PA at 6 months of age. We contributed to the very limited research on this topic since this was the first study that we found to date that

showed that mothers' depression during the postpartum period can influence the inherent, enduring form of infant PA during first few months of life. This suggests that infants of mothers with postpartum depression symptoms exhibit low trait PA, and thus a vulnerability to the later development of psychopathology early in the first year. This early identification of risk factors for the later development of psychopathology can aid future researchers in the creation of prevention and intervention techniques for mitigating negative developmental trajectories in at-risk individuals. Further, this study suggests mothers' postpartum depression symptoms impact infant trait PA at 6 months beyond potential contributions of prenatal anxiety. So it seems that it is mothers' postpartum depression symptoms, rather than anxiety symptoms, that impact infant trait PA.

Strengths, Limitations, and Future Directions

The present study has many strengths. The study utilized a longitudinal design, which allowed for the comparison of infant PA at multiple points throughout the first year and also provided the opportunity to assess mothers' depression many times during the prenatal and postpartum periods. In addition, having multiple measures of depression during the prenatal and postpartum periods meant we were able to gain a better sense of the infants' exposure to the mothers' depression symptoms than could be garnered by assessing depression at a single point in time. We also were able to use measures of both state and trait PA to better understand the role of mothers' depression symptoms in infant PA. We relied on a sample of women with a history of depression and/or anxiety, which increased the likelihood of studying women who experienced postpartum, and/or prenatal, depression. The fact that our sample contained primarily women of middle

socioeconomic status helped eliminate many of the confounds that could occur due to the stresses of being of low socioeconomic status.

One possible limitation of the current study is the fact that not all of the EEG data was available at the time analyses were conducted for this study. As previously mentioned, preparing EEG data for analysis is a time-intensive process. Though this study had a sample size comparable to, or larger than, many other infant EEG studies (Field et al., 1995; Jones et al., 1997), once more data is available for analyses, we will be able to gain a better understanding of the role of infant EEG in the association between mothers' depression and infant PA. In addition, the kappa coefficients for our sensitivity variables were not as high as we would have hoped. We will continue to investigate ways to measure mothers' parenting qualities.

The present study is part of ongoing, longitudinal data collection that will culminate this summer. We will re-test our hypotheses with larger sample sizes, which will be particularly helpful for infant EEG asymmetry, since this measure had a small sample size compared to our other variables. Because the limited research in this field has mainly focused on PA of infants of postnatally depressed mothers during the first 6 months of life, it is important to expand upon existing research to study the developmental trajectories of PA of infants of mothers with depression symptoms through the first year and throughout the lifespan. We plan to conduct a follow-up study using 12-month data that has been collected. We also plan to study the role of other variables such as attachment style and contingent responsiveness, meaning the amount of time a mother-infant dyad spends in matching affective states, in the association between mothers' depression symptom levels and infant trait PA. These future studies will allow

for a better understanding of how this potential vulnerability to depression develops over time and which points in the developmental trajectory are most amenable to prevention and intervention strategies.

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Table 1

ANOVAs for Maternal Demographic Variables and Infant PA Variables and an Independent Samples T-Test for Infant Gender and Infant PA

	Mothers' Marital Status	Mothers' Race	Infant Gender
3 Month Infant PA			
Relative Duration of Observed Approach	2.85	.70	-.71
Surgency/Extraversion on the Infant Behavior Questionnaire-Revised	2.7	1.92	-.19
6 Month Infant PA			
Relative Duration of Observed Positive Affect	.24	.07	.13
Surgency/Extraversion on the Infant Behavior Questionnaire-Revised	.28	.66	-.25

* $p < .002$ level (2-tailed).

Table 2

Intercorrelations Among Maternal Demographic Variables and Infant PA at 3 and 6 Months

	Maternal age at delivery	Maternal years of education	Parity	Hollingshead
3 Month Infant PA				
Relative Duration of Observed Approach	-0.03	-0.08	-0.02	-0.07
Surgency/Extraversion on the Infant Behavior Questionnaire-Revised	-0.26*	-0.21	-0.03	-0.14
6 Month Infant PA				
Relative Duration of Observed Positive Affect	-0.08	-0.01	-0.02	0.03
Surgency/Extraversion on the Infant Behavior Questionnaire-Revised	-0.21	-0.19	-0.01	-0.16

* $p < .002$ level (2-tailed).

Table 3

Intercorrelations among Mothers' Beck Depression Inventory (BDI) Area Under the Curve (AUC) Scores and Mother's State-Trait Anxiety Inventory (STAI) Scores

	1	2	3	4
1. Prenatal BDI AUC	—	.71**	.74**	.78**
2. Postpartum BDI AUC for first 3 months		—	.96**	.55**
3. Postpartum BDI AUC for first 6 months			—	.55**
4. Prenatal STAI AUC				—

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

Table 4

Intercorrelations Among Mothers' Observed Parenting Quality Variables

	1	2	3	4	5	6
1. Relative Duration of Positive Affect at 3 months	—	.58**	-.02	.17	.60**	.40**
2. Relative Duration of Positive Affect at 6 months		—	.114	.26**	.38**	.60**
3. Sensitivity at 3 months			—	.22*	.29**	.11
4. Sensitivity at 6 months				—	.30**	.63**
5. Withdrawal at 3 months					—	.46**
6. Withdrawal at 6 months						—

Note. Higher parenting quality scores indicate more positive parenting behaviors.
** $p < .01$ level (2-tailed). * $p < .05$ level (2-tailed).

Table 5

Correlations Between Mothers' Prenatal and Postpartum Beck Depression Inventory (BDI) Area Under the Curve (AUC) Scores and Infant PA at 3 and 6 Months

	Prenatal BDI AUC	Postpartum BDI AUC for first 3 months	Postpartum BDI AUC for first 6 months
3 month Relative Duration of Observed Infant Approach	.09	.078	__
3 month Surgency/ Extraversion Scores on IBQ-R	-.02	-.02	__
6 month Relative Duration of Observed Infant PA	.06	-.04	-.04
6 month Surgency/ Extraversion Scores on IBQ-R	-.11	-.23**	-.26**

Note. BDI AUC levels at 6 months were examined in relation to 6-month PA outcomes but not 3-month outcomes since we were not assessing retrospective associations.
** $p < .01$ level (2-tailed). * $p < .05$ level (2-tailed).

Table 6

Hierarchical Regression Analyses for Mothers' Beck Depression Inventory (BDI) Area Under the Curve (AUC) Scores Predicting Infant Surgency/Extraversion Scores on the Infant Behavior Questionnaire-Revised (IBQ-R) at 6 Months of Age

	Infant Surgency/ Extraversion Scores	
	6 months	
Predictor	ΔR^2	β
Step 1	.013	
Prenatal BDI AUC		.108
Step 2	.045**	
Postpartum BDI AUC for first 3 months		-.269
Step 3	.001	
Postpartum BDI AUC for months 4 through 6		-.045
Total R^2	.058	
n	158	

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

Table 7

Mediators for the Relationship Between Mothers' Beck Depression Inventory (BDI) Area Under the Curve (AUC) Levels for the First 6 Months Postpartum and Infant Surgency/Extraversion Scores at 6 Months

			Bootstrapping (95% CI)	
	Effect	SE	Lower	Upper
Mediator: Infant EEG Asymmetry				
	0.0000	0.0001	-0.0001	0.0002
Mediator: Mother's Sensitivity				
	0.0000	0.0000	-0.0001	0.0001
Mediator Mother's Withdrawal				
	0.0000	0.0000	-0.0001	0.0001
Mediator Mother's Observed PA				
	0.0000	0.0001	-0.0001	0.0001

Note. 10,000 bootstrap samples

Table 8

Intercorrelations Among Prenatal and Postpartum Beck Depression Inventory (BDI) Area Under the Curve (AUC) Scores and Parenting Variables at 3 and 6 Months

	Prenatal BDI AUC	Postpartum BDI AUC for first 3 months	Postpartum BDI AUC for first 6 months
3 Month Maternal Parenting Qualities			
Sensitivity/ Responsiveness	-.026	-.014	__
Withdrawal/Low Positive	-.009	-.006	__
Relative Duration of Observed Positive Affect	.058	.044	__
6 month Parenting Qualities			
Sensitivity/ Responsiveness	-.086	-.106	-.112
Withdrawal/Low Positive	-.031	-.104	-.129
Relative Duration of Observed Positive Affect	.056	-.017	-.056

Note. Depression variables at 6 months were examined in relation to 6-month parenting qualities but not 3-month parenting qualities since we were not assessing retrospective associations. Higher parenting quality scores indicate more positive parenting behaviors. ** $p < .01$ level (2-tailed). * $p < .05$ level (2-tailed).

Table 9

Intercorrelations among Mothers' Observed Parenting Quality Variables and Infant PA Measures at 3 and 6 Months

	Parenting Qualities					
	Sensitivity		Withdrawal		Positive Affect	
	3 Months	6 Months	3 Months	6 Months	3 Months	6 Months
3 Month Infant PA Relative Duration of Observed Approach	.298**	__	.252**	__	.184*	__
Surgency/Extraversion on the Infant Behavior Questionnaire-Revised	-.003	__	-.085	__	-.002	__
6 Month Infant PA Relative Duration of Observed Positive Affect	.216*	.032	.072	.093	.129	.216*
Surgency/Extraversion on the Infant Behavior Questionnaire-Revised	.010	-.061	-.078	-.078	-.034	.048

Note. Parenting quality variables at 6 months were examined in relation to 6-month PA outcomes but not 3-month outcomes since we were not assessing retrospective associations. Higher parenting quality scores indicate more positive parenting behaviors.
** $p < .01$ level (2-tailed). * $p < .05$ level (2-tailed).

Table 10

Hierarchical Regression Analyses for Mothers' Beck Depression Inventory (BDI) Area Under the Curve (AUC) Scores and Mother's State-Trait Anxiety Inventory (STAI) Scores Predicting Infant Surgency/Extraversion Scores on the Infant Behavior Questionnaire-Revised (IBQ-R) at 3 Months of Age and Infant Relative Duration of Approach at 3 Months of Age

	Infant PA at 3 Months			
	Surgency/Extraversion		Relative Duration of Approach	
Predictor	ΔR^2	β	ΔR^2	β
Step 1	.013		.01	
Prenatal BDI AUC		-.165		.02
Prenatal STAI AUC		.184		.07
Step 2	.000		.000	
Postpartum BDI AUC for First 3 Months		.103		.02
Total R^2	.013		.01	
n	137		110	

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

Table 11

Hierarchical Regression Analyses for Mothers' Beck Depression Inventory (BDI) Area Under the Curve (AUC) Scores and Mother's State-Trait Anxiety Inventory (STAI) Scores Predicting Infant Surgency/Extraversion Scores on the Infant Behavior Questionnaire-Revised (IBQ-R) at 6 Months of Age and Infant Relative Duration of Observed Positive Affect at 6 Months of Age

	Infant PA at 6 Months			
	Surgency/Extraversion		Relative Duration of Positive Affect	
Predictor	ΔR^2	β	ΔR^2	β
Step 1	.025		.01	
Prenatal BDI AUC		-.029		.06
Prenatal STAI AUC		.174		.17
Step 2	.045*		.01	
Postpartum BDI AUC for First 3 Months		-.267		-.08
Step 3	.001		.004	
Postpartum BDI AUC for Months 4 through 6		-.046		-.12
Total R^2	.071		.024	
n	127		117	

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

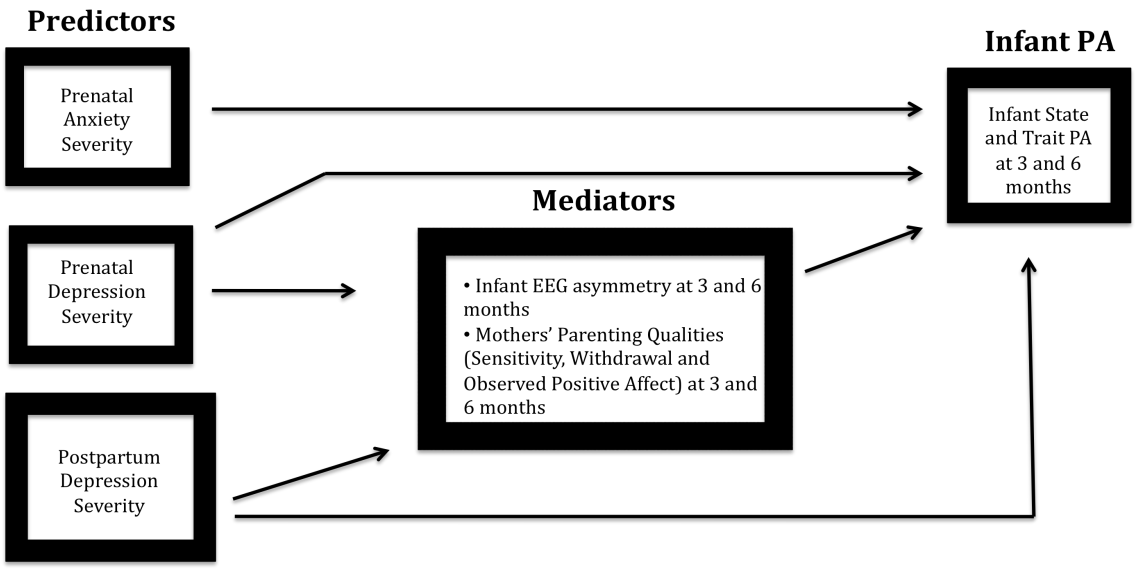

Figure 1. Diagram of the proposed relationship between mothers' prenatal anxiety, prenatal depression and postpartum depression symptom levels and infant state and trait PA, with infant EEG asymmetry and parenting variables mediating the relationship between mothers' depression and infant PA.