MATERNAL INFLUENCE ON EARLY INFANT EMOTIONAL REGULATION: A STUDY OF 3-MONTH INFANT BEHAVIOR, CORTISOL AND FRONTAL EEG

by

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ABSTRACT

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Prenatal maternal stress and mood, and early postnatal mother-infant interactions set the stage for the child's psychobiological, neurological and social development. While a large body of research connecting maternal depression to infant EEG asymmetry exists, the current study sought to add to the sparse literature on maternal anxiety and infant EEG. Mother-infant dyads were assessed prenatally during the third trimester, soon after birth, at 6 weeks and 3 months postnatal. Association between maternal depression and later development of right mid-frontal alpha asymmetry was confirmed, while trends suggested maternal anxiety may be associated with lateral frontal alpha asymmetry. Greater maternal sensitivity and anxiety were each associated with lower post-stressor cortisol in infants with right frontal asymmetry. Greater time spent in mutual gaze was associated with positive infant affect. Finally, quality mother-infant dynamics encourage

positive infant affect and healthy physiological stress regulation even when brain patterns associated with dysregulation have been established.

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I. INTRODUCTION

The early emotional bond made between mother and infant not only sets the stage for future parent-child interactions but also influences the trajectory of the child's psychobiological (Field, Diego & Hernandez-Reif, 2006), immunological (Marques, O'Connor, Roth, Susser, Bjørke-Monsen, 2013), cognitive and social development (Hane & Fox, 2016; Lupien, McEwen, Gunnar, Heim, 2009; Sandman, Davis, Buss & Glynn, 2012; Sullivan, Perry, Sloan, Kleinhaus & Burtchen, 2011). Bowlby concluded from his vast body of attachment research that infants are primed to not only seek proximity to a caregiver who provides for its physical survival, but also one who affords emotional support and safety (Bolwby, 1988). Importantly, Harlow (Harlow & Harlow, 1962) demonstrated that given the choice between a caregiver who provides food versus a caregiver who provides emotional and physical comfort, primate infants overwhelmingly prefer support over sustenance. While proper nutrition is a clear necessity for physical survival and function, current research continues to uncover the importance of emotional support for proper regulatory, social and cognitive development. Early experiences with a primary caregiver shape the infant's expectations for care and security and inform the infant's budding regulatory system how best to seek care and respond to stress. Given the proper physical and emotional care, infants learn that it is safe to explore their environment and to make further emotional attachments. If, however, infants are emotionally neglected or significantly stressed, stunted or aberrant physiological, neurological, psychological, cognitive and social development can occur (Bowlby, 1977;

Harlow & Zimmerman; 1959; Robertson & Robertson, 1971). While the effects of stress on development have largely been studied using animal models, this review will mainly focus on the findings of human research.

The development of regulatory behavior through the first year

Posner and Rothbart (2000) contend that self-regulation is "the single most crucial goal for advancing an understanding of development and psychopathology." Regulation is often defined as the adaptive process of modulating the intensity and duration of physiological arousal, attention and emotional states (Sroufe, Egeland, Carlson & Collins, 2005) as well as behavioral, cognitive, interpersonal or social processes (Calkins & Fox, 2002), and is, thus, often assessed through measures of behavioral, hormonal and neural activation and reactivity. It is clear that individual adults have different ways of dealing with challenge, but stable individual differences in regulatory style are also apparent in infancy and thought be present at birth (Rothbart, Derryberry & Hershey, 2000). The amniotic environment predicts postnatal infant regulation (O'Connor, Bergman, Sarkar & Glover, 2013), which in turn is associated with later childhood behavioral reactivity to stress (Leung, Tasker, Atkinson, Vaillancourt, Schulkin & Schmidt, 2010), social skills (Calkins & Fox, 2002), and cognitive development (Lawson & Ruff, 2004). Finally, childhood regulation is associated with later adult psychopathology (Althoff, Verhulst, Rettew, Hudziak, & van der Ende, 2010).

The infant psychophysiological regulatory system begins to develop prenatally and continues to mature after birth (Lupien et al., 2009). During pregnancy, the fetus' development is directly linked the mother's experiences as stress response signals can travel through the placenta (Sandman et al., 2012). Following stress, the body responds

autonomically by releasing catecholamines and by activating a regulatory circuit called the hypothalamus-pituitary-adrenal (HPA) axis which initiates a cascade of stress responses (Lupien et al., 2009). Once a stressor is detected by the brain, the paraventricular nucleus of the hypothalamus releases corticotropin-releasing hormone (CRH) and arginine vasopressin (AVP), resulting in subsequent release of adrenocotropic-releasing hormone (ACTH) by the pituitary gland. ACTH binding in the adrenal cortex, the outer layer of the adrenal gland, triggers release of steroid hormones such as cortisol, called glucocorticoids (Lupien et al., 2009). During prenatal maternal stress, glucocorticoids are released by the maternal adrenal cortex, are transmitted to the fetal environment via the placenta, at which point it can cross the fetal blood-brain barrier and uniquely influence fetal development. Glucocorticoids receptors (GRs) are ubiquitous in the brain and, given that they are involved in regulation of gene expression (Lupien et al., 2009), glucocorticoid modulation can have lifelong implications for disease vulnerability and for metabolic, neuroendocrine, cognitive and emotional function (Harris & Seckl, 2011). While glucocorticoids are necessary for normal brain development (e.g. neuronal maturation, axonal and dendritic remodeling, cell survival (Meyer, 1983; Yehuda, Fairman, & **Meyer**, 1989), myelination, synaptogenesis and neurogenesis (Seckl, 2008) in rodent models), glucocorticoid levels that are outside the normal range can disrupt normal brain maturation and impair brain function (Lupien et al., 2009).

Furthermore, while maternally released glucocorticoids act in a negative feedback loop inhibiting maternal hypothalamic, hippocampal and frontal cortex function so as to reestablish homeostasis after stress, maternal glucocorticoids in fact induce a positive

feedback loop within the placenta (Sandman, Glynn, Schetter, Wadhwa, Garite, Chicz-Demet & Hobel, 2006). The placenta senses stress responses in the form of cortisol from both mother and fetus, which triggers CRH gene (hCRHmRNA) expression within the placenta (Sandman et al., 2012). Additionally, elevated maternal stress downregulates an enzyme (11β-hydroxysteroid) that catabolizes cortisol allowing more maternal cortisol to reach the fetus (Mairesse et al., 2007). CRH levels within the placenta naturally rise across gestation, and are involved in triggering childbirth (Sandman et al., 2012). However, high levels of stress, especially early on in gestation, can lead to abnormally elevated placental CRH and cortisol levels, resulting in shortened the length of gestation (Sandman et al., 2006) and delayed fetal maturation (Sandman et al., 2012). Because the fetus is undergoing rapid growth and the developing nervous system is particularly vulnerable at this time, maternal experience and regulation is thus said to 'program', or direct, fetal and future infant development (Lupien, McEwen, Gunnar, & Heim, 2009; Sandman et al., 2012; Bolten, Nast, Skrundz, Stadler, Hellhammer, & Meinlschmidt, 2013). Furthermore, developments in all levels of the nascent infant regulatory system seem to have fundamental influence on early personality and social behavior, which interact bidirectionally with the later development of more complex levels of regulation, such as those involved in behavioral control, interpersonal processes, and metacognitions (Calkins & Fox, 2002). Thus, the developing fetal regulatory system is sensitive to maternal experience, and prenatal programming can have lifelong consequences.

Maternal influence on the development of infant regulation

Because regulatory capacities are not mature at birth, caregiver sensitivity and efficacy are critical for infant regulation and regulatory system development (Calkins and Fox, 2002; Sroufe et al., 2005). In fact, Bowlby (1973) went so far as to say that infants' behavioral, psychological and biological function are inextricably tied to parent-infant dynamics. Caregivers provide a supportive scaffolding for the developing infant regulatory system. Infants begin life quite dependent on caregiver intervention to modulate infant arousal and emotion, but over the first few years of life there is a gradual and foundational shift from dyadic co-regulation to self-regulation as the infant becomes increasingly in control of motor, attention and cognitive function and acquires skills to self-monitor and behaviorally regulate themselves (Calkins & Fox, 2002).

While maternal influence on fetal regulation is biologically mediated, maternal influence necessarily shifts after birth to more social pathways (with exception to transmission of glucocorticoids through breastfeeding (Hinde et al., 2015)). The infant regulatory system continues to develop postnatally through an interaction between the infant's innate temperament (defined by Robart and Posner (2001) as individual differences in motor control, emotional reactivity and self-regulation) and environmental factors, such as caregiver sensitivity and efficacy. Newborn infants can regulate themselves through reflexive behaviors, such as crying and sucking (Rothbart, Ziaie & O'Boyle, 1992). Crying may also be utilized by the infant as a means of signaling the caregiver to aid in regulation (Sullivan et al., 2011). Primitive self-regulation over arousal state and emotional reactivity begins to emerge by 3 months of age (Swingler, Perry, Calkins, & Bell, 2014), with more control and stability developing over time as infants are better able to regulate arousal (Blandon, Calkins, Keane, & O'Brien, 2008)

and control attention and motor function so as to visually orient to or disengage from stimuli (Rothbart & Posner, 2001) and to engage in joint attention (Mundy & Newell, 2007). Furthermore, development of functions such as the social smile allows infants to initiate and maintain positive social interactions with caregivers (Sander, 1976). Rothbart & Posner (2001) point out that regulation continues to develop throughout the early school years as the frontal cortex continues to mature and executive attention and control continue to develop through that time. However, early caregiver sensitivity has been shown to be a powerful predictor of infant regulation development over the first year of life (Conradt & Ablow, 2010).

Maternal sensitivity refers to the extent to which mothers notice, correctly interpret and quickly respond to infant cues in an appropriate manner (Ainsworth, Bell & Stayton, 1971; Beebe & Steele, 2013). It is thought that when mothers recognize infant coping strategies and respond appropriately, infants learn that they can influence dyadic states and eventually learn to regulate themselves (Swingler et al., 2014). Furthermore, when an infant learns that they can trust their caregiver to provide appropriate support in regulation when needed, the infant is more likely to test their own abilities and explore new environments (Sroufe et al., 2005). Supporting the notion that these infants have learned to use their mothers as an external source of regulation during stress (Swingler et al., 2014), infants of sensitive mothers display more mother-oriented behavior during a challenging still-face task (Kogan & Carter, 1996). Moreover, maternal sensitivity was recently found to be related to 6-month infant hippocampal volume and to the connectivity between the hippocampus and various areas in the brain responsible for emotion regulation (Rifkin-Graboi, Kong, Sim, Sanmugam, Broekman, Chen, Wong,

Kwek, Saw, Chong, Gluckman, Fortier, Pederson, Meaney & Qiu, 2015). Additionally, maternal sensitivity and mother-infant synchrony in early infancy are associated with better child regulation in later infancy and toddlerhood (Feldman, Greenbaum & Yirmiya, 1999; Kaitz, Maytal, Devor, Bergman & Mankuta, 2010; Kogan & Carter, 1996). Furthermore, Bolten, Fink & Stadler (2012) found that although prenatal stress late in gestations is positively associated with infant crying at six weeks of age, infants of mothers with high prenatal stress and high self-efficacy cried significantly less than infants of mothers with high stress and low self-efficacy. Thus, positive and appropriate maternal behavior postnatally aids in the healthy development of infant regulatory capacities and can somewhat protect infants even from the negative consequences of prenatal stress.

Conversely, mothers with depression have been found to provide less emotional support to their children, who in turn develop more emotional and behavioral dysregulation (Hoffman, Crnic & Baker, 2006). Maternal anxiety is associated with aberrant mother-infant interaction dynamics (Beebe, Steele, Jaffe, Andrews, Margolis, & Feldstein, 2011). Beebe et al. (2011) found that maternal anxiety was correlated with extreme contingency responses, more variable infant behavior, and at times either variable or overly stable maternal behavior patterns during face-to-face mother-infant play at 4 months post-gestation. Interestingly, Beebe et al. (2011) also found a pattern of dampened facial coordination in response to infant facial/vocal shifts, but heightened touch coordination with infant vocal affect for anxious mothers. The investigators suggested that these destabilizations are due to maternal hyperarousal associated with anxiety, leading to both maternal vigilance and emotional distancing (Beebe et al.,

2011). Similarly, Kaitz et al. (2010) found that although maternal anxiety was not associated with decreased overall maternal sensitivity, highly anxious mothers did act in an more exaggerated manner during play, and infants of highly anxious mothers had blunted responses to still-face and stranger challenges. Furthermore, maternal mood disorder is associated with physiological dysregulation. For example, elevated infant cortisol reactivity to stress at 9 months of age was associated with maternal mood disorder (both anxiety and depression) (Feldman, Granat, Prienta, Kanety, Kuint & Gilboa-Schechtman, 2009). Thus, maternal psychological dysfunction can lead to dysregulation of mother-infant interaction and interrupt normal infant self-regulation development. In sum, both intrinsic infant characteristics and extrinsic influence, especially via mother-infant interaction, contribute to the development of the infant regulatory system, setting the stage for adult emotion regulation.

Infant EEG and regulatory behavior

Infant EEG.

Hans Berger (1932) was the first to develop human electroencephalography (EEG), a measure of tiny, spontaneous waves of electrical activity originating from large networks of neurons in the brain, which oscillate in synchrony, and are usually measured at the scalp (Saby & Marshall, 2012). Various technologies for measuring brain activity continue to rapidly advance, however, electroencephalography (EEG) remains a popular modality for research in human developmental cognitive and affective neuroscience for several reasons. While EEG has low spatial resolution as compared to magnetic resonance imaging (MRI), it can resolve electrical signals on the order of milliseconds, essentially allowing real-time measurement of brain activity (Saby & Marshall,

2012). Furthermore, EEG is much less costly and allows much more flexibility in experimental setup in comparison to MRI (Saby & Marshall, 2012). Especially for developmental neuroscience research in very young infants, MRI is simply impractical due to its implementation constraints, and EEG allows for much more naturalistic testing environments. The ability to utilize unconstrained laboratory settings becomes especially significant when attempting to study the development of social cognition, given the impact of setting on experience.

EEG signals can be decomposed through Fourier analysis into component waves of varying frequencies. In adults, EEG is analyzed across groups of frequencies, which have been thoroughly demonstrated to be associated with distinct cognitive functions (Saby & Marshall, 2012). The groups of functional frequencies, called frequency bands, established in adults, include delta (1-3 Hz), theta (4-7 Hz), alpha and mu (8-12 Hz), beta (13-30 Hz), and gamma (30-100 Hz) (Saby & Marshall, 2012). While alpha and mu waves share the same frequency spectrum, the signals originate from different brain areas, with alpha (also referred to as posterior alpha) arising from the posterior regions and mu (also referred to as central alpha) originating from activity in the central cortex (Saby & Marshall, 2012). Given their different source locations, alpha and mu are associated with different functions. Posterior alpha rhythms are associated with visual activity, with alpha signal being strongest with eyes shut and desynchronizing with visual input (Saby & Marshall, 2012). In contrast, mu rhythms are associated with motor and somatosensory function, with mu amplitude diminishing during execution or imagining of motor movement and tactile stimulation (Paulus, Hunnius & Bekkering, 2013; Saby & Marshall, 2012). (In other words, posterior and central alpha power diminishes in

relevant regions as alpha-related activity increases.) Additionally, theta rhythms originate in frontal regions of the brain and are associated with wakefulness, memory, decision-making and emotional functions (Saby & Marshall, 2012). (For theta band activity, greater power is related to increases in theta-related activity (Saby & Marshall, 2012.)) Thus, wave frequency and source location of EEG in adults are demonstrated to be differentially associated with brain function.

However, these functional frequencies are not static across development. Given that EEG signal arises from networks of neuronal activity, it is not surprising that as functional networks take time to develop during infancy and childhood, functional EEG bands likewise evolve across development. Therefore, a given function or state will be associated with different frequency bands at different points in development. There is debate about how best to label and analyze infant EEG (Saby & Marshall, 2012). For example, while clearly discernable spectral peaks are less common in 1-week and 1-month old infants, rhythms in the range of 3 to 5 Hz appear in the occipital region in most infants by three months of age (Diego, Jones, Field, 2010), and by the end of the first year of life, activity in the occipital region is largely in the range of 7-9 Hz (Bell & Fox, 1994). Most investigators define the 6-9 Hz band as alpha for older infants (as compared to alpha in adults at 8-12 Hz), with some suggesting 4-6 Hz as the range for alpha in early infancy (Jones, Venema, Lowy, Earl & Webb, 2015). While theta ranges have been inconsistent across the literature, the modal frequency range is between approximately 3-6 Hz in infants (Saby & Marshall, 2012). Posterior alpha, mu (central alpha) and theta frequency bands have been most frequently studied in relation to social cognition. However, while posterior and central activity has been linked to social

competencies related to imitation and intention of action (Paulus, Hunnius & Bekkering, 2013; Southgate, Johnson, Karoui, & Cisbra, 2010), frontal EEG has been shown to be involved with the executive and emotional components of social cognition (Bernier, Calkins & Bell., 2016; Cuevas, Bell, Marcovitch & Calkins, 2012; Diego et al., 2010; Swingler et al., 2014).

Frontal EEG and regulation.

The frontal lobe of the neocortex is an important processing hub, as it receives inputs from all sensory cortices, from the motor area of the neocortex (Fox, 1994) and has bidirectional connections with the amygdala, an area with extensively demonstrated significance for fear processing and behavioral response to stressful stimuli (Morgan & LeDoux, 1995). Functional imaging studies have revealed that atypical patterns of fronto-amygdala activity are implicated in anxiety and depression (Heller & Casey, 2016). Because of the frontal region's broad connectivity, it is well-situated to play a significant role in emotion regulation (Fox, 1994). Frontal activity, and in particular prefrontal EEG has been found to be involved in attention, emotion regulation, cognition (Posner & Rothbart, 1994) and effortful control over behavior and emotion (Fox, 1994). Dawson (1994) suggested that power of infant frontal EEG is proportionate to the intensity of emotion expression. Furthermore, Davidson (1993) suggested that frontal asymmetries, the differences in EEG power between homologous regions of the right and left hemispheres of the frontal lobe, moderates and mediates an individual's predispositions toward approach and withdrawal behaviors. Whereas greater relative left frontal activation is associated with approach behavior and positive affect, greater right hemispheric asymmetry is associated with withdrawal tendencies and negative affect

(Davidson, 1993; Wheeler et al., 1993). Fox (1994) found that infant EEG is correlated to emotion when emotion is viewed in terms of the balance between approach and avoidance, but not when affect is viewed in terms of discrete emotions such as happiness, sadness or anger, for example. Fox (1994) further suggested that the two hemispheres are innately primed to control different sets of behaviors that underlie either approach toward or avoidance of novel or stressful stimuli. A large body of research has confirmed that frontal asymmetries, both at rest and state-dependent, are related to the individual's temperament and to changes in emotion state (Coan & Allen, 2003a; Coan & Allen, 2004). Furthermore, greater relative right activity in neonates is associated with other measures of emotion regulation such as vagal tone (Jones, Field & Almeida, 2009; Jones, Field, Fox, Davalos, Lundy & Hart, 1998). Thus, frontal asymmetry can be utilized as a non-invasive biomarker for early regulatory and behavioral predisposition, which in turn impacts later behavioral function (Swingler et al., 2014).

Maternal Influence on Infant EEG

Maternal depression has long been associated with atypical patterns of infant frontal EEG (Diego et al., 2006; Field et al., 2006; Jones et al., 1998). Depressed adults exhibit greater relative right frontal EEG activation due to reduction in left frontal activation (Jones et al., 1998). Infants of mothers with depression (both pre- and postnatal) display a similar pattern of right frontal asymmetry across infancy, beginning as early as birth (Diego et al, 2004; Jones et al., 1998). The relationship between maternal depressive symptomatology and decreased infant left frontal activity follows a somewhat dose-dependent pattern, with greater maternal depressive symptoms associated with more exaggerated infant frontal asymmetry (Dawson, Frey, Panagiotides, Osterling,

& HessI, 1997). Depression and anxiety are highly comorbid (Heller & Casey, 2016), however there are far fewer studies on the links between maternal anxiety and infant EEG than maternal depression and infant EEG. Nonetheless, Field, Diego, Hernandez-Reif, Schanberg, Kuhn, Yando & Bendell (2003) found a similar pattern of greater relative right frontal activity in the infants of mothers with depression and comorbid anxiety as compared to mother with depression only. Furthermore, while infants of anxious-only mothers had more right frontal asymmetry than infants of mothers with no mood disorder, the frontal asymmetry was most pronounced in infants of mothers with comorbid depression and anxiety (Field et al., 2010).

The relationship between maternal affect or mood disorder and infant brain activity is complex. There are mixed findings about the age at which these EEG patterns first emerge in infants of mothers with depression. While some have found evidence of neurodysregulation at birth (Diego et al, 2004; Jones et al., 1998), other studies observed infant frontal asymmetry only after 3 months of age (Field et al., 2003; Diego et al., 2006). Frontal asymmetry at birth for infants of mothers with mood disorders points to a chemical or epigenetic process influencing newborn brain activity patterns. Whereas, if asymmetry develops over the first few months of life, atypical postnatal maternal behavior may play a more significant role in the development and sustained patterns of frontal asymmetry observed through the first year of life. Two aberrant interaction styles have been observed in mothers with depression: an intrusive style, wherein the mother overstimulates her infant, and a withdrawn style, characterized by understimulation of the infant. Rather than focus on maternal psychological status, Jones, Field, Fox, Davalos, Malphurs, Carraway, Schanberg & Kuhn (1997) investigated the association between

quality of maternal interaction, focusing on intrusive and withdrawn patterns of maternal behavior, and infant EEG. The results showed that infants of withdrawn mothers had decreased left frontal and increased right frontal EEG activation, while infants of intrusive mothers displayed increased left and decreased right frontal EEG activation (Jones et al., 1997; Diego et al., 2006), and that these asymmetries developed between birth and 3-6 months of age (Diego et al., 2006). The infants of withdrawn mothers also oriented to their mothers less during face-to-face interactions (Jones et al., 1997). While additional evidence is necessary to confirm the timing of emergence of EEG asymmetry, these results point to the importance of maternal behavior for the development of infant brain activity patterns and call for further study of the interplay between infant brain activity and maternal behavior.

Swingler et al. (2014) investigated whether infant EEG at 5 months of age, viewed as an indicator of intrinsic infant approach/avoidance tendencies, might moderate the relationship between maternal sensitivity and infant regulatory behavior during a stressful arm-restraint task at that same age. The authors (Swingler et al., 2014) hypothesized that maternal sensitivity would be more critical for positive infant affect and appropriate regulatory behavior for infants with right frontal asymmetry, infants with withdrawal tendencies. While Swingler et al. (2014) found that infant EEG asymmetry did moderate the association between maternal sensitivity and infant distraction behaviors, with more distraction behaviors in infants with left frontal EEG asymmetry at baseline, the investigators also found counterintuitive interaction results between maternal sensitivity and EEG asymmetry. For infants with right frontal asymmetry, high maternal sensitivity predicted more negative affect and low sensitivity predicted less

negative affect (Swingler et al., 2014). The authors suggest that while infants with left frontal asymmetry and sensitive mothers self-distract more during stress, the displays of negative affect in infants with right frontal asymmetry are the infant's strategy for eliciting mother's help in regulation. However, these findings did not match the theory, which predicted that, for infants with right frontal asymmetry, high maternal sensitivity would result in decreased infant negative affect.

Granat, Gadassi, Gilboa-Schechtman and Feldman (2017) and have taken a closer look at the relationship between infant EEG and maternal behavior through analysis of mother-infant interaction dynamics by collecting behavioral measures in the form of time series. Granat et al. (2017) found that levels of gaze and touch synchrony (both mother and infant were engaged in the behavior at the same time) were lowest in mother-infant dyads with depressed mothers and highest in dyads with anxious mothers. They further found that maternal presence was associated with decreased negative infant affect compared with infant-stranger episodes for infants of anxious, but not depressed mothers. Given that anxious mothers were most synchronized in Granat et al. (2017), it is possible that high maternal sensitivity and maternal anxiety were confounded in the Swingler et al. (2014) study, and negative infant affect was more directly linked to maternal anxiety than to maternal sensitivity. An interaction between maternal mood and mother-infant synchrony might explain the surprising pattern of infant affect. Furthermore, Bernier et al. (2016) found that although maternal positive affect is unrelated to concurrent 5-month infant EEG frontal power, maternal affect at 5 months was positively correlated with infant frontal EEG power at 10 and 24 months. Given the unexpected results of Swingler et al. (2014) and the predictive relationship found by Bernier et al. (2016) between

maternal affect and later infant EEG activity, further longitudinal study of the relationship between maternal behavior, infant regulation and infant brain activation is necessary. Perhaps another maternal factor, such as self-efficacy, is more predictive of infant affect than maternal sensitivity. Furthermore, while Swingler et al. (2014) found a counterintuitive relationship between infant EEG, maternal sensitivity and behavioral reactivity to stress, it is possible that a biological marker for stress, such as cortisol reactivity, may reveal a different relationship between infant regulation, EEG asymmetry and maternal attunement. Lastly, it is possible that assessment of dyadic synchrony, as opposed to more static methods of assessing mother-infant interaction behavior, may help to explain the mixed and sometimes counterintuitive findings regarding the relationship between maternal anxiety and behavior and infant EEG and stress reactivity. Indeed, Beebe and Steel (2013) found that more synchrony is not always better in terms predicting later health of mother-infant attachment. Beebe and Steel (2013) contend that there is an optimal midrange of interactive contingency, wherein extreme highs or lows in contingent coordination are associated with later negative relational and behavioral outcomes. In sum, mixed and counterintuitive findings with regard to the relationship between maternal anxiety and infant bio-behavioral outcomes may be untangled through longitudinal design and using methods with more fine-grained resolution.

II. PURPOSE AND HYPOTHESES

Statement of Purpose

While there is a large body of research about maternal depression and infant EEG asymmetry, the current study seeks to add to the sparse literature on the links between maternal anxiety and infant EEG activity. The current study will assess relationships between maternal anxiety, depression and breastfeeding self-efficacy and infant regulation. As regulation involves physiological arousal, emotional, cognitive, social and behavioral processes, regulation will be assessed through infant EEG asymmetry, physiological stress regulation (as measured by cortisol stress reactivity), and maternal and infant behavioral patterns as well as the dynamic behavioral interplay between the two. Importantly, the longitudinal design of the study allows for exploration of influence of earlier maternal states on later infant regulatory activity. Finally, the current study will be the first to assess the relationship between infant-mother dynamics and infant EEG at 3 months of age.

Hypotheses

Hypothesis 1. The relationship between prenatal stress, maternal mood (anxiety and depression) and infant frontal EEG asymmetry will be analyzed. It is hypothesized that prenatal maternal stress and maternal anxiety and depression at the neonatal visit will predict 3-month infant right frontal asymmetry. Furthermore, it is hypothesized that this relationship will be moderated by maternal self-efficacy at 6 weeks post-birth.

Hypothesis 2. The relationship between prenatal stress, maternal mood and infant regulation will be analyzed. Here, regulation will be assessed as cortisol reactivity to a stressor. It is hypothesized that prenatal maternal stress, and maternal anxiety and depression at the neonatal visit will predict poor 3-month infant physiological stress regulation (high cortisol). Furthermore, it is hypothesized that this relationship will be moderated by maternal self-efficacy at 6 weeks post-birth.

Hypothesis 3. Similar to Swingler et al.'s (2014) final hypothesis, it is predicted that infants with right frontal EEG asymmetry will have lower cortisol reactivity and more positive affect if they have highly attuned mothers, as measured by synchronicity during a face-to-face interaction, but that this relationship will be moderated by maternal anxiety.

Exploratory Analysis. The relationship between maternal depression, anxiety, comorbid depression and anxiety and prenatal stress will be explored in hypotheses 1 and 2.

III. METHOD

Participants

Forty-eight mothers were recruited from early or midway through their third trimester of pregnancy (28-36 weeks gestation). Participants were recruited through flyers at local hospitals, doctors' offices and various businesses, such as yoga studios, which expectant mothers frequent. Informed consent and demographic information were obtained at a home visit before the birth of the infant. Mean gestational age (GA) at the prenatal visit when urine was collected was 34.06 weeks (*SD* = 3.09). Four percent of mothers responded that they were Hispanic, 61.5% Caucasian, 1% African American and 23.5% responded other or did not respond. This pregnancy would be the first child for 44.2% of mothers, 30.8% of mothers had one other child, and 21.6% had 2 or more other children. The mean age for mothers was 31.11 years (*SD* = 6.20) and for fathers was 32.73 (*SD* = 6.43). Average socioeconomic status (SES) as measured by the Hollingshead Two Factor Index of Social Position (Hollingshead, 1957) was 33.76 (*SD* = 7.98) which falls into middle class (social class 3).

Usable videos of mothers and infants interacting were collected for 30 of these dyads. Pearson Chi-square tests of independence were performed to examine whether infant gender, maternal ethnicity, parity and SES varied between the overall sample and the subset of participants for whom video was collected and analyzed. Results confirm that there was no significant difference for these demographic variables (χ^2_{Gender} (1, N = 48) = .006, p = n.s.; $\chi^2_{Ethnicity}$ (3, N = 48) = 4.60, p = n.s.; χ^2_{Parity} (2, N = 48) = .16, p =

n.s.). One sample t-tests comparing the subsample to the whole sample found no differences in SES ($t(27) = -.03$, p = n.s.), GA at time of urine collection ($t(28) = 1.67$, p = n.s.) or age of mother ($t(27) = -.87$, p = n.s.) or father ($t(27) = .61$, p = n.s.).

Procedure

In this longitudinal study, 48 mothers were assessed at a prenatal visit during the third trimester of pregnancy and were followed with their babies after birth at newborn, 6-week and 3-month follow-up visits. According to the mother's preference, visits took place either at the participant's home or at the WAVES lab. At the prenatal visit, after informed consent was obtained, mothers were given a packet of questionnaires pertaining to socio-economic status, social support, feelings of attachment to the fetus, stress and coping, and depression.

At the newborn visit, mothers were given a packet of questionnaires pertaining to depression and their sense of self-efficacy with regards to breastfeeding. At 6-weeks and 3-months post birth, mothers again completed depression and breastfeeding self-efficacy questionnaires, as well as a post-partum bonding questionnaire. BSES surveys were collected from 25 mothers at 6 weeks. Additionally, at the 3-month visit, infant EEG was collected, and a combination arm restraint/still-face stress procedure was conducted. With the infant sitting in a bouncy chair, the mother was asked to gently hold the infant's arms down and to look at the infant with a straight face for 90 seconds. If she was unable to hold a straight face, she was instructed to look away from the infant. This procedure was video recorded. Using a collection tube, ½ mL of infant saliva was collected at baseline before the stressor and again at 20 minutes post-stressor. Mother-infant interaction during play was also recorded during the 3-month visit. With the infant in the

bouncy chair, the mother was asked to interact with the baby as she normally would without toys. Both the mother's and the infant's faces were visible in the recording.

Measures

Maternal psychological factors.

Prenatal stress. Demographic information was assessed with a short questionnaire of five to seven questions related to each of the following: employment and education, social support, prenatal stress and coping (including questions about recent stressful life events), health information, and feeding intentions. (See Appendix A.) Following the methods used by Grizenko, Fortier, Gaudreau-Simard, Jolicoeur and Joober, R. (2015), this information was then used to score maternal stress levels from 1 to 5 based on the DSM-III and DSM-III-R axis IV scales (1 = none, 2 = mild, 3 = moderate, 4 = severe and 5 = extreme).

Depression and Anxiety. The Edinburgh Postnatal Depression Scale (EPDS) (Cox, Holden & Sagovsky, 1987) is a self-report scale specifically validated for assessing depression in the postpartum period. The scale excludes questions assessing physical symptoms of depression such as fatigue, disturbances to sleep and irritability, in order to prevent the misinterpretation of these common consequences of childbirth as depression symptomatology. The scale consists of ten questions which assess the well-being and mood of the mother in the course of the last seven days. Each question is rated on a 3-point Likert scale from 0 ("No, not at all") to 3 ("Yes, all the time") to assess the severity of a depressive symptomatology, with a total score ranging from 0 to 30. The EPDS has demonstrated good construct validity and internal reliability (Logsdon, Usui, & Nearing, 2009; Bunevicius, Kusminskas, & Buneviskas, 2009).

Matthey, Fisher and Rowe (2012) reviewed findings from studies examining the capacity of the EPDS to detect perinatal anxiety disorders. Of nine studies which investigated the factor structure of the EPDS, eight found two- and three-factor solutions. The majority of these studies found that three items load onto an "anxiety" factor. The items in question are: item 3 ("I have blamed myself unnecessarily when things went wrong."), 4 ("I have been anxious or worried for no good reason.") and 5 ("I have felt scared and panicky for no very good reason."). Furthermore, Matthey et al. (2012) present some evidence for the utility of an EPDS cut-off score as a screening tool for clinical diagnosis of anxiety disorders as determined by the DSM III-R or IV diagnostic criteria. Thus, Matthey et al. (2012) argue that these three items of the EPDS may serve as an anxiety subscale. Therefore, in this study, items 3, 4, and 5 were used as an anxiety measure and were referred to as the $EPDS_{Anx}$ subscale. The remaining seven items were used to assess postnatal depression and were referred to as the $EPDS_{Dep}$ subscale. (This seven-item shortened form of the EPDS was utilized by Tietz, Zietlow and Reck (2014)). Therefore, raw $EPDS_{Anx}$ scores could range between 0 and 9, while raw $EPDS_{Dep}$ scores ranged between 0 and 21. In order to more easily compare subscale scores, proportion of anxiety and depression were calculated by dividing an individual's score on a subscale by the maximum score possible for that subscale (9 for anxiety and 21 for depression). Thus, $EPDS_{Anx}$ and $EPDS_{Dep}$ scores reported here were proportional scores.

Self-efficacy. The Breastfeeding Self-Efficacy Scale -Short Form (BSES-SF), developed by Dennis (2003) and based on Bandura's social cognitive theory, is a 14-item scale to measure breastfeeding confidence. Possible scores range from 14-70. Each item begins with "I can always," (for example, "I can always determine that my baby gets

enough milk.") and items have a 5-point Likert scale with responses ranging from "not at all confident" to "always confident." The BSES-SF is a highly reliable measure, with high internal consistency (Cronbach's alpha = 0.94). Construct validity was validated by significant correlations between the BSES-S, the Rosenburg Self-Esteem Scale and the EPDS, $p < .001$ (Dennis, 2003). Given the importance of feeding interaction on early establishment of the mother-infant relationship, the BSES may serve as a highly relevant measure of self-efficacy with regards to motherhood.

Infant Physiology.

EEG. EEG data was collected from the infant at the beginning of the 3-month visit while the infant was in a quiet and alert state. The recording was sampled for 3-6 minutes using a stretch lycra cap (Electro Cap, Inc.) with the international 10-20 system. Impedances were brought below 5K ohms. Mid-frontal (F3 and F4), lateral frontal (F7 & F8), central (C3 and C4), parietal (P3 and P4) and occipital (O1 and O2) sites were used. All sites were referenced to the vertex (Cz), as this is a standard reference site for previous infant EEG research (see Field & Diego, 2008a for review). The EEG electrical signal was amplified using SA Instrumentation Bioamps and bandpassed from 1-100 Hz and streamed to a PC laptop. The EEG sampling online rate was 512 samples per second. The EEG recordings were saved to a computer hard drive using Snapstream v. 3.21 (HEM Data Corp, 1991).

Given the transitional nature of developing infant EEG signals, there are no universally agreed upon infant frequency bands. However, the 3-6 Hz frequency bands are typically used for young infants, and the 6-9 Hz range is used for older infants (Jones, Field, Fox, Lundy, & Davalos, 1997; Jones et al., 2015; Saby & Marshall, 2012). Thus,

this study examined the 3-6 Hz (alpha 1), 6-9 Hz (alpha 2), and 3-12 Hz (broadband) ranges. The EEG data were cleaned to exclude artifacts from eye and motor movements. Mid-frontal EEG (channels f3, f4) was successfully collected from 32 infants at the 3-month visit. Lateral-frontal EEG (channels f7, f8) was successfully collected from 28 infants at the 3-month visit. Frontal asymmetries were calculated by subtracting the natural log of the alpha power of the electrode in the left hemisphere from that of the right frontal electrode. Thus, mid-frontal asymmetry was calculated as ln(f4) - ln(f3), while lateral-frontal asymmetry was calculated as ln(f8)-ln(f7). Higher asymmetry scores indicate greater relative right power and greater relative left activation.

Cortisol. Salivary cortisol samples were collected from 34 infants at the 3-month visits. HPA reactivity was assessed by finding the difference in concentrations of salivary cortisol before and after an acute stress (Jansen, Beijers, Riksen-Walraven & de Weerth, 2010). In their review of 48 peer reviewed articles on the development of infant cortisol reactivity, Jansen et al. (2010) found that the cortisol reactivity appears to decrease with age, and because of that decrease in reactivity, the effect size of mild acute stressors attenuates after 6 months of age. Therefore, measurement of cortisol reactivity in response to a mild stressor is suitable for the 3-month age group. Salivary cortisol levels were measured at the pre-test phase (before the arm restraint/still face procedure) and 20-25 minutes after the test phase given that this is the appropriate time window for cortisol levels to peak following an acute stress (de Weerth & van Geert, 2002). Salivary cortisol concentrations for three-month-old infants generally peak in the morning and subsequently decline over the course of a day (de Weerth & van Geert, 2002). Therefore, all 3-month visits were scheduled for mornings.

Cortisol samples were obtained by collecting infant saliva using a nalgene cryogenic vial (Thermo Scientific, NY, USA) and allowing the infant to passively drool into the vial. Immediately after collection, the samples were moved to a -20°C freezer. Following the visit, samples were moved to an ultra-low temperature freezer (-80°C) and subsequently assayed using a commercial cortisol EIA kit and following assay protocols (Salimetrics, PA, USA). The test uses 25 µl of saliva per sample, has a lower sensitivity threshold of <0.007 µg/dl. Average intra- and inter-assay coefficients of variation were assessed.

Mother-infant interaction and coding.

Thirty videos of mothers and infants engaging in roughly three minutes of play interaction were collected. The mother and infant were seated facing each other, with the infant placed in an infant seat. Mothers were instructed to interact with their infants as they would normally, but without any toys. Play sessions were video recorded for later coding. For consistency across dyads and manageability of coding, videos were shortened to 150 seconds in length. All but two videos were 150 seconds long. Of the two shorter videos, one was 97 seconds in length and the other was 143 seconds long. Dyadic interactions were micro-coded using Continuous Measurement System software (CMS rating and CMS Coding packages). Coding was done on a second-by-second basis. For infant affect, coding was completed according to a scale adapted from Jones and colleagues (2004) and outlined in Table 1. Coders rated infant affect on this five-point scale, with higher scores reflecting more optimal, positive behaviors and lower scores reflecting suboptimal, more negative behaviors. Infant positivity was calculated as the percent of the total video length that the infant was in a positive state. Infant and mother

gaze were also coded on a second-by second basis using a 3-point scale: gaze towards partner, away from partner, or unclear. Each behavior was coded on separate passes for each the mother and the infant, producing four data sets for every dyad. Coders were blind to the mothers' responses to maternal mood questionnaires. Coders were assessed for interrater reliability.

Two measures of synchrony were computed using these coded videos: gaze synchrony and proportion of mutual gaze. Gaze synchrony was computed as a conditional probability: the proportion of time the mother was looking at the infant given that the infant was looking at the mother at that moment. Thus, gaze synchrony is a measure of maternal sensitivity in that it only assesses instances when the infant is gazing at the mother and determines how often the mother was attuned to her infant in these moments. This working definition of synchrony is based on Granat et al. (2017), who argue that this type of coordinated behavior is vital for general emotional and social development as well as for the formation of healthy close relationships. The proportion of the time the dyad spent in mutual gaze (the number of seconds spent in mutual gaze divided by the total number of seconds observed) was also calculated and analyzed. This measure of proportion of mutual gaze is more representative of the level of naturally occurring coordination between mother and infant.

Table 1. Infant Affect Coding Scale

Code	Behavior
5	Interest, smiling, laughing, cooing, positive vocalizations
4	Interest, bright eyed, physically reactive, cooing in response to environment in general, not necessarily in response to the mother
3	Neutral, possibly attending, non-reactive physically, no facial affect, neutral vocalizations
2	Stretching, twisting, no particular focus, whining, whimpering
1	Squinting or eyes closed, non-attentive, crying, distress

IV. RESULTS

Hypothesis 1

Validity of the EPDS anxiety subscale was confirmed by a moderate to large positive correlation between EPDS anxiety subscale scores with scores on the previously validated (Brockington, et al., 2001) Anxiety About Care subscale of the Postpartum Bonding Questionnaire (r = .485, p = .012).

To test whether prenatal maternal stress and maternal mood (depression and anxiety) at the neonatal visit predict 3-month infant right frontal asymmetry, correlations between maternal stress, postnatal (at newborn) maternal depression and anxiety, 6-week postnatal maternal self-efficacy and relative right frontal power were first assessed. Neither maternal prenatal stress nor 6-week breastfeeding self-efficacy (BSES) was significantly correlated with any measure of asymmetry (alpha1, alpha2 or BB, either lateral- or mid-frontal). Table 2 presents basic descriptive statistics for prenatal stress, anxiety and depression and BSES.

Table 2. *Questionnaire Results*

	N	*Range*	*M*	*SD*
Prenatal Stress	44	1.00-4.00	1.77	.77
EPDS Anxiety Subscale at Neonatal	42	.00-.78	.34	.23
EPDS Depression Subscale at Neonatal	42	.00-.95	.13	.18
6-week Breastfeeding Self-Efficacy	25	27-70	58.08	12.10

Lower asymmetry scores mean (greater relative left power and) greater right activation relative to left activation. Table 3 lists mean mid- and lateral frontal asymmetry scores and basic descriptive statistics for the three frequency bands tested.

As expected, higher depression subscale scores are moderately correlated with greater right mid-frontal asymmetry for alpha 1 (3-6 Hz) (r = -.40, p =.040), alpha 2 (6-9 Hz) (r = -.49, p = .010), and for broadband alpha frequencies (3-12 Hz) (r = -.42, p =.030). Similarly, there was a non-significant trend for higher anxiety subscale scores to be moderately associated with greater right lateral-frontal asymmetry for alpha 1 (r = -.38, p =.076), alpha 2 (r = -.36, p = .091) and for broadband alpha frequencies (r = -.37, p = .083). These relationships are summarized in Figure 1 and Table 4. Higher depression as measured by EPDS total scores was only significantly correlated with greater relative right mid-frontal activation for alpha 2 (r = -.43, p = .026).

Table 3. *Relative Right Frontal Alpha Asymmetry*

	N	*Range*	*M*	*SD*
Mid-frontal (alpha 1)	32	-2.66-.80	-.22	.67
Lateral frontal (alpha 1)	28	-1.67-2.81	.12	.84
Mid-frontal (alpha 2)	32	-1.71-.89	-.15	.57
Lateral frontal (alpha 2)	28	-2.26-3.35	.10	1.03
Mid-frontal (broadband)	32	-2.51-.74	-.21	.65
Lateral frontal (broadband)	28	-1.88-2.95	.11	.88

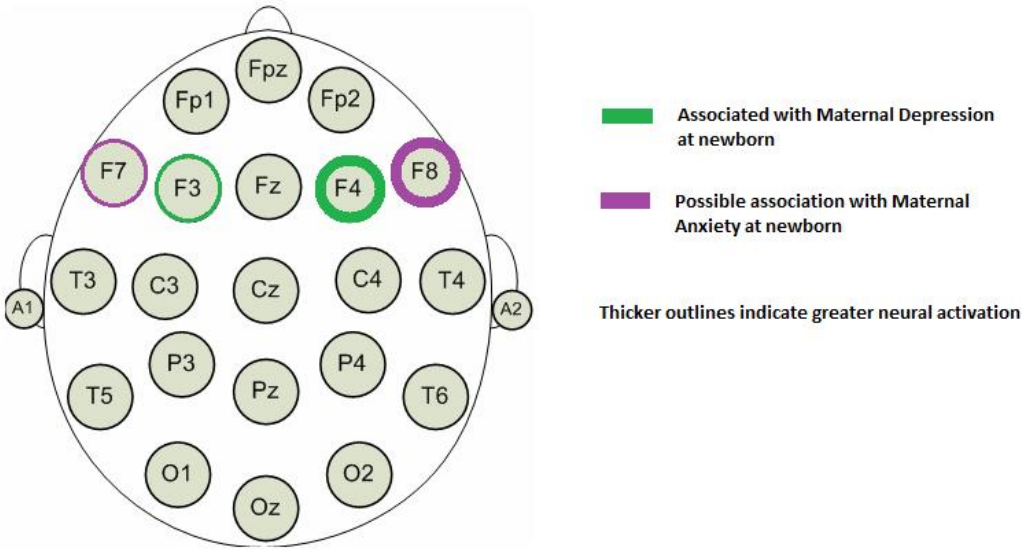

Figure 1. Higher maternal depression at the newborn visit is associated with greater mid-frontal asymmetry across all bands of alpha activity tested. A similar non-significant trend was found for maternal anxiety at newborn.

Table 4. *Correlations of maternal mood at newborn and infant EEG at 3 months*

Variables	1	2	3
1. Maternal Anxiety at Newborn	1		
2. Maternal Depression at Newborn	0.492***	1	
3. EPDS total score at Newborn	0.767***	0.936***	1
4. Right mid-frontal asymmetry (3-6 Hz)	-0.204	-0.398*	-0.34ǂ
5. Right lateral frontal asymmetry (3-6 Hz)	-0.377ǂ	-0.045	-0.245
6. Right mid-frontal asymmetry (6-9 Hz)	-0.271	-0.489**	-0.428*
7. Right lateral frontal asymmetry (6-9 Hz)	-0.36ǂ	-0.034	-0.229
8. Right mid-frontal asymmetry (3-12 Hz)	-0.223	-0.418*	-0.36ǂ
9. Right lateral frontal asymmetry (3-12 Hz)	-0.369ǂ	-0.04	-0.237

*p<.05. **p<.01. ***p<.001. ǂp near .08

To determine whether 6-week BSES scores moderated the relationship between maternal depression at newborn and right mid-frontal asymmetry, models predicting mid-

frontal alpha 1, alpha 2 and broadband asymmetry with an interaction between maternal depression at newborn (EPDS depression subscale) and maternal BSES at 6 weeks were estimated and simple slopes were calculated. There were no significant interactions between BSES and maternal depression at 6 weeks (N = 18, p=.352, p=6.07, p=.380, respectively.). (This was retested with newborn BSES because there were fewer participants missing newborn BSES than 6-week BSES, and the BSES scores for the two times were significantly correlated (r =.684, p =.001). Even with the larger sample size, no significant interactions were found for newborn BSES).

Hypothesis 2

To test whether prenatal maternal stress and maternal mood at the neonatal visit predict 3-month infant cortisol reactivity to a stressor, correlations between maternal stress, postnatal (at newborn) maternal depression and anxiety, 6-week postnatal maternal self-efficacy and relative right frontal power were conducted. Prenatal maternal stress was not significantly correlated with baseline cortisol, reactivity cortisol (cortisol level post-stressor) or change from baseline to post-stressor (p = .418, p = .753, p = .670, respectively). Basic descriptives for baseline cortisol, reactivity cortisol and change from baseline to post-stressor are listed in Table 5.

Table 5. *Cortisol Results*

	N	*Range*	*M*	*SD*
Baseline	38	.10-2.70	.71	.55
Post-stress	35	.00-2.78	.65	.51
Post-stress-Baseline Change	34	-1.28-.88	-.02	.41

Maternal anxiety and depression were tested for correlations with baseline cortisol, reactivity cortisol and with change from pre- to post-stressor. Neither maternal depression (EPDS depression subscale) nor maternal anxiety at newborn was significantly correlated to any cortisol measure (p = n.s.). Exploratory analysis found no significant correlations between prenatal stress and maternal mood at newborn.

To determine whether 6-week BSES scores moderated the relationship between maternal anxiety at newborn and cortisol reactivity, a model (N =19) predicting cortisol reactivity with an interaction between maternal anxiety and maternal BSES was estimated and simple slopes were calculated. There were no significant interactions between BSES and maternal anxiety (p=.62).

Hypothesis 3

Inter-rater reliability for mutual gaze was assessed in accordance with the guidelines described by Gwet (2014) and was found to be good (according to Fleiss's (1981) kappa benchmark) for three raters coding for gaze synchrony (Fleiss' K= .717, AC_1 =.723). Given initially poor reliability, the five infant affect categories proposed were collapsed intro three new categories: positive, neutral and negative. When using three categories, the inter-rater reliability for three raters was found to be adequate (Fleiss' K= .462, AC_1 =.653).

A number of correlations were conducted to determine whether gaze synchrony, infant affect, cortisol, right frontal asymmetry, and maternal anxiety were related. Descriptive statistics for dyadic synchrony, maternal sensitivity and infant affect are presented in Table 6.

Table 6. *Second-by-Second Coded Behavior Results*

	N	*Range*	*M*	*SD*
Gaze synchrony	30	.69-1	.92	.08
Percent time in mutual gaze	30	.67-98.67	47.63	31.85
Infant positivity	30	.00-99.33	52.32	34.51
Infant negativity	30	.00-67.33	8.19	16.07

Preliminary analysis found that gaze synchrony was uncorrelated with infant affect, while the percent of time spent in mutual gaze was correlated with infant positivity (r = .57, p = .001), but not significantly correlated with infant negativity (r = .04, p = .84). Furthermore, percent of time spent in mutual gaze was found to be multimodal. Percent of time spent in mutual gaze was plotted in intervals of 10% of time which revealed two clusters of participants (each with N=5), one falling between 10-20% of time spent in mutual gaze and another spending 80-90% of time in mutual gaze. Participants were then divided into high and low proportion mutual gaze groups based on whether they spent more or less time in mutual gaze than the median (45%). Fifteen dyads fell into both synchrony groups. T-tests were performed to compare cortisol reactivity and infant affect between the high and low proportion mutual gaze groups. Infants in the high time in mutual gaze group had significantly more positive affect (M = 68.49, SD = 32.93) than infants in the low mutual gaze group (M = 36.16, SD = 28.68), p = .01. No significant differences were found in cortisol levels between the high and low mutual gaze groups.

Additional testing revealed that greater right mid-frontal asymmetry (alpha 2) was correlated with lower cortisol reactivity (r =.398, p =.029). Correlations between gaze synchrony, infant positivity and infant cortisol (baseline, post-stressor and cortisol change) were assessed while separately selecting for participants with relative right (mid- and/or lateral-) frontal asymmetry for alpha1, alpha2 and broadband frequencies. Under these conditions, gaze synchrony was not significantly related to infant positivity or infant cortisol levels. The same procedure was performed for percent of time spent in mutual gaze with similar results.

To test whether that infants with right frontal EEG asymmetry have lower cortisol reactivity and more positive affect if they have highly attuned mothers as measured by gaze synchrony participants were split into high and low gaze synchrony groups depending on whether their conditional probability for gaze synchrony was higher or lower, respectively, than the median level. Only infants with right frontal EEG asymmetry were selected and t-tests were performed to compare cortisol reactivity and infant affect between the high and low gaze synchrony groups. No significant differences in infant affect were found. However, for infants with relative right mid-frontal asymmetry (alpha 2), participants in the high gaze synchrony group had lower cortisol levels (M = .40, SD = .20) after the stressor than participants in the low gaze synchrony group (M = .72, SD = .29), $t(11) = 2.32$, p = .04. There were similar trends for participants with relative right mid-frontal asymmetry for alpha 1 and broadband frequencies (p =.053).

Exploratory Analysis

Exploratory analysis determined that prenatal stress was uncorrelated to measures of maternal depression, anxiety or comorbid depression and anxiety (total EPDS scores). While prenatal stress was uncorrelated with infant affect, it was noted that roughly 41 percent of mothers reported no prenatal stress at all. When mothers were separated into 'no stress' (stress = 0) and 'some stress' (stress > 0) groups, a trend appeared suggesting that infants of mothers reporting no stress prenatally displayed more positive affect (M = 68.94, SD =36.24) at the three month visit during face-to-face play with their mothers than infants of mothers who reported some prenatal stress (M = 44.01, SD =31.26), $t(28) = 1.94$, $p = .06$.

Finally, for infants with mid-frontal relative right asymmetry, higher maternal anxiety at newborn was strongly correlated with significantly lower cortisol post-stressor (r = -.579, p = .038). Thus, infants with right frontal EEG asymmetry had lower cortisol levels post-stressor if they have highly anxious mothers.

V. DISCUSSION

Contrary to the predictions made, maternal prenatal stress was unrelated to either frontal EEG asymmetry or cortisol reactivity in 3-month-old infants. Given the large body of literature linking prenatal stress with infant outcomes, the null results surrounding prenatal stress point to deficiencies in the method used here to assess maternal stress. The demographic interview used to measure stress did include a number of questions relevant to stress assessment, however, many participants provided extremely short answers, making the felt impact of those stresses difficult to assess. Use of a more robust and validated stress questionnaire would likely reveal significant relationships between maternal prenatal stress and infant EEG and cortisol activity as was found in previous research. Furthermore, it would be best to employ multiple stress questionnaires in order to differentiation between general and pregnancy related stress as various types of stress may affect infant development differentially. It was predicted that the negative effects of stress on the infant might be mitigated if the mother developed high confidence her mothering capabilities (as measured by breastfeeding self-efficacy) with time after birth. However, because the measure of prenatal stress was unrelated with infant outcomes, moderation by BSES was superseded. It is also possible that confidence in mothering capability simply does not mitigate effects of prenatal stress on infants.

Nonetheless, as predicted, maternal depression at newborn did predict infant mid-frontal EEG asymmetry at 3 months. This supports previous findings linking depression and withdrawal behavior in mothers with infant frontal alpha asymmetry (Dawson et al.,

1997; Diego et al, 2004; Diego et al., 2006; Field et al., 2003; Field et al., 2006; Jones et al., 1998). There were similar non-significant trends of association between maternal anxiety at newborn and lateral frontal EEG asymmetry. While there is very little published about the relationship between maternal anxiety and infant EEG, there is some evidence of greater reductions of left frontal activity in infants of mothers with comorbid depression and anxiety compared to depression alone (Field et al., 2003; 2010), as well as in infants of anxious-only mothers compared to infants of mothers with no mood disorder (Field et al., 2010). In a review of brain asymmetry in depression, Bruder, Stewart & McGrath (2017) present EEG, behavioral (both dichotic listening and visual hemifield asymmetry), neuroimaging evidence of opposing patterns of hemispheric asymmetry for depression and anxiety. For example, Bruder et al. (2017) cite van Tol et al. (2010) who found that reduced volume of right lateral inferior frontal cortex was specific to major depression disorder, reduced left middle superior temporal volume was specific to anxiety disorder and reduced volume of rostral-dorsal ACC was common to both depression and anxiety. Interestingly, in a 2006 review of the relationship between frontal EEG activity and depression, anxiety and comorbid depression and anxiety, Thibodeau, Jorgensen & Kim found, of the thirteen infant studies reviewed, none measured at the f7/f8 (lateral frontal) scalp sites. Furthermore, two of the three adult anxiety studies (Tomarken & Davidson, 1994; Nitschke, Heller, Palmieri & Miller, 1999) included in the review which measured activity at both f3/f4 and f7/f8 sites found stronger associations between anxiety and lateral frontal asymmetry compared to mid-frontal asymmetry, similar to the results of the current study. Likewise, Gold, Fachner & Erkkila (2012) found that adult anxiety was correlated with adult frontal alpha asymmetry at f7/f8 but

not at f3/f4. Perhaps the accumulation of null results in previous research for the association between maternal anxiety and infant frontal EEG activity is simply due to an insufficient number of scalp locations at which frontal EEG was measured combined with the possibility that maternal depression and anxiety affect activity patterns of different infant brain regions.

Given that neural circuits and corresponding functional EEG bands are still fluid and developing at this time in infancy, and if infant brain activation patterns are influenced by maternal behavior, it is also possible that the current study's non-significant trends linking maternal anxiety to infant lateral frontal EEG patterns might become more pronounced and reach significance in a sample of older infants. It should be noted that Thibodeau et al. (2006) found the exact opposite trend, with larger effects for the younger samples than for the older samples. However, this was based on a relatively small number of studies and the authors provided no theoretical explanation for that finding. It is also likely that anxiety/EEG trends did not reach significance because the instrument used to measure anxiety relied on only three questions and was insufficiently sensitive. Future research should employ more robust measures to assess multiple types of anxiety. Kutsenko, Ivonin, Shuvaev, Lisyanskaya & Nozdrachev (2015) found differences in spatial structure of adult EEG depending on the type of anxiety disorder. Similarly, Bruder et al. (2017) present evidence demonstrating that, for participants with comorbid depression and anxiety, left frontal activity reduction associated with depression was suppressed for participants with an apprehensive (e.g., worry) subtype of anxiety, whereas the left frontal reduction was enhanced for participants with an arousal (e.g., panic disorder) subtype of anxiety. Ideally, long-term, transient, general state,

pregnancy specific anxiety, and apprehension versus arousal subtypes of anxiety should be measured and differentiated. It is also important for future research to distinguish mothers with anxiety alone from mothers who have comorbid depression and anxiety. Thibodeau et al. (2006) revealed that the findings surrounding the relationship between adult comorbid depression/anxiety and adult frontal alpha asymmetry are extremely mixed and inconclusive. The authors note that this is counterintuitive given that both adult depression and anxiety are separately associated with adult right frontal asymmetry, and so a combination of the two disorders would be expected to also be associated with frontal alpha asymmetry. This unexplained finding may reflect some hidden layer of complexity that is added by comorbidity of mood disorders and underscores the need to differentiate between mothers with both depression and anxiety and mothers with anxiety alone when exploring the relationship between maternal anxiety and infant brain activity. Finally, Bruder et al. (2017) point out that studies which measure EEG (adult or child) during emotionally-challenging tasks are more consistent in finding associations between depression and frontal alpha asymmetry, suggesting a functional component to the brain differences. Perhaps the same is true for anxiety. If EEG had been collected during an emotionally-charged task rather than at resting state, it is possible that anxiety would have been significantly correlated with frontal alpha asymmetry in the current study. Future studies should consider collecting EEG during an emotionally-challenging task.

Determining the relationship between maternal mood and infant brain asymmetry is important. Davidson (1995) suggested that greater right than left frontal activation may indicate vulnerability for developing depression and anxiety disorders. Blackhart, Minnix & Kline (2006) found evidence supporting this in adults with anxiety. Greater relative

right frontal activation was associated with greater trait anxiety one year later. Furthermore, Hannesdóttir, Doxie, Bell, Ollendick & Wolfe (2010) found that right frontal asymmetry in early childhood was associated with more physiological arousal during stress and poorer ability to regulate emotions (according to parental report) at nine years of age. Additionally, Bruder et al. (2017) stated that asymmetry of resting state alpha in adults relates to clinical response to antidepressants and could, therefore, be important for developing biomarkers for clinical treatment. Thus, elucidation of the relationship between infant EEG asymmetry and maternal mood (taking into account subtypes and comorbidities of anxiety and depression) could potentially be useful for early detection of risk for later development of mood disorder and emotional regulation deficiencies as well as inform intervention procedures for mothers with mood disorder. Furthermore, the basic relationship between maternal mood and infant EEG must be clarified for the effects of maternal behavior and sensitivity on infant outcomes such as infant EEG to be fully understood.

While maternal depression and anxiety at newborn were unrelated to infant cortisol reactivity, greater relative right mid-frontal asymmetry was correlated with lower cortisol reactivity, and when only infants with greater right mid-frontal asymmetry were selected, elevated maternal anxiety at newborn was significantly associated with blunted cortisol post-stressor. Again, relative right mid-frontal asymmetry at 3 months was strongly associated with maternal depression at newborn. Thus, it seems there is a complex relationship between comorbid depression/anxiety, infant EEG and stress regulation. These results indicate that infants with frontal alpha asymmetry (which is predicted by maternal depression) have lower cortisol levels post-stress if their mothers

reported high levels of anxiety. It is possible that most anxious mothers behaved differently from the other mothers and effectively soothed their infants after the stressor, thus preventing the predicted stress-related spike in cortisol. Alternatively, Gunnar and Vazquez (2001) found a pattern of hypocortisolism, suppression of HPA axis function, is associated with adverse early life conditions. Lastly, it is possible that the experimental environment influenced these results. Given that a number of the study visits were conducted in the lab, being in this new setting may have been stressful for infants which created high baseline levels of cortisol, and as the infant acclimated to the lab their cortisol levels reduced. In fact, just over 47 percent of infants had a baseline cortisol level that was higher than their cortisol level after the stressor. The environmental stress may have overshadowed the experimental stressor for some infants.

As predicted, high maternal sensitivity as measured by gaze synchrony was associated with lower infant post-stressor cortisol levels in infants with relative right frontal EEG (alpha 2) asymmetry, supporting Swingler et al.'s (2014) hypothesis that maternal sensitivity is especially important for appropriate regulatory behavior for infants with right frontal asymmetry. However, infant affect was unrelated to gaze synchrony in this group of infants. Again, Swingler et al. (2014) found that for infants with right frontal asymmetry, high maternal sensitivity predicted more negative affect and low sensitivity predicted less negative affect. Given the null results produced in the current study with regards to maternal sensitivity (as measured by gaze synchrony) and infant affect, and high dyadic synchrony being associated with increased infant positivity, an explanation to the counter-intuitive findings linking increased maternal sensitivity to increased negative infant affect remains elusive. In contrast, high dyadic synchrony (as

measured by proportion of time in mutual gaze) was unrelated to infant cortisol reactivity but was associated with more positive infant affect. It should be noted, however, that the infants assessed here were two months younger than those assessed in Swingler et al. (2014), and it is quite possible that the relationship between infant EEG, maternal sensitivity and infant behavior is simply more loosely connected at this younger age. For example, Diego, et al. (2006) found more pronounced relative right frontal asymmetry in babies of withdrawn and depressed mothers later in infancy than within the first few days after birth. If maternal behavior influences infant behavior and brain patterns, it would make sense to find stronger statistical relationships between maternal and infant factors given that older infants have had more exposure to their mothers and more time for those patterns to develop. Future studies should include a 6-month visit to determine whether the current null results regarding maternal sensitivity and infant affect are due to timing and developmental confounds.

Taken together, these results suggest that for infants with brain activity patterns consistent with avoidant tendencies, maternal sensitivity is critical for physiological stress regulation, while the quality of infant-mother relationship as measured by the proportion of gaze synchrony is more closely related to positive infant affect. In other words, even if infants have more relative right frontal activity (a pattern associated with poorer infant behavioral outcomes and with maternal depression), those infants that are able to synchronize well with their mother display more positive behavior and those with sensitive mothers are also better equipped to regulate their physiological reaction to stress. This underscores the importance of quality mother-infant relationship dynamics

for the healthy development of infant behavioral and physiological stress regulation, even in the face of developmental risk as indicated by brain activity.

A limitation of the current study was the quality of videos coded. The videos were not originally recorded with this type of micro-analytical coding in mind. There was some variability in angle and clarity, making rating more difficult and more inconsistent, especially for fleeting or particularly fluid affective states. Subpar inter-rater reliability for the coding of infant affect may be partially to blame for the null infant affect results. Therefore, future studies assessing mother/infant behavior dynamics need to ensure more uniform behavior recording settings and arrange the cameras to collect clear and unobstructed views of both the infant and mother. Furthermore, more robust measures for prenatal stress and maternal anxiety should be considered for future studies. Differentiation between subtypes of depression and anxiety, as well as differentiation between mothers with only depression or anxiety versus those with comorbid depression and anxiety appears to be paramount for clarifying the aggregation of mixed results concerning maternal mood and infant frontal alpha asymmetry.

In sum, the current study was the first to explore the relationship between infant-mother dynamics and infant EEG at 3 months of age. Furthermore, the results suggest that the relationship between maternal anxiety and later infant brain activity may be clarified and established in the future by taking lateral frontal alpha activity into account. Lastly, these results suggest that while poor maternal mood does predict brain patterns associated with aberrant infant behavior, quality mother-infant relationship dynamics may nonetheless encourage positive infant affect and healthy physiological stress regulation even when suboptimal brain patterns have already been established.

VI. APPENDIX OF QUESTIONNAIRES

Stress Questionnaire

1. How many times have you moved in the last year?

2. Do you or anyone else in your household have any health problems?

3. Do you or anyone else in your household have a drinking or drug problem?

4. If so, does this cause problems for you?

5. Have you or any of your family experienced some sort of stress or major life change which caused you to feel sad or stressed?

6. Have you or any of your family been diagnosed with a mood disorder?

Breastfeeding Self Efficacy Scale

PLEASE CIRCLE THE ANSWER THAT BEST DESCRIBES HOW CONFIDENT YOU ARE WITH BREASTFEEDING YOUR BABY.
(There is no right or wrong answer)

1 = Not At All Confident
2 = Not Very Confident
3 = Sometimes Confident
4 = Confident
5 = Very Confident

1. I can always determine that my baby gets enough milk.

2. I can always cope with breastfeeding like I have other challenging tasks.

3. I can always breastfeed my baby without using formula as a supplement.

4. I can always ensure that my baby is properly latched on for the whole feeding.

5. I can always manage the breastfeeding situation to my satisfaction.

6. I can always manage to breastfeed even if my baby is crying.

7. I can always keep wanting to breastfeed.

8. I can always comfortably breastfeed with my family members present.

9. I can always be satisfied with my breastfeeding experience.

10. I can always deal with the fact that breastfeeding can be time-consuming.

11. I can always finish feeding my baby on one breast before switching to the other.

12. I can always continue to breastfeed my baby for every feeding.

13. I can always manage to keep up with my baby's breastfeeding demands.

14. I can always tell when my baby is finished breastfeeding.

Edinburgh Postnatal Depression Scale (EPDS)

Date: _____ Clinic Name/Number: _____

Your Age: _____ Weeks of Pregnancy/Age of Baby: _____

Since you are either pregnant or have recently had a baby, we want to know how you feel. Please place a **CHECK MARK** (✔) on the blank by the answer that comes closest to how you have felt **IN THE PAST 7 DAYS**—*not just how you feel today.* Complete all 10 items and find your score by adding each number that appears in parentheses (#) by your checked answer. This is a screening test; not a medical diagnosis. If something doesn't seem right, *call your health care provider regardless of your score.*

Below is an example already completed.

I have felt happy:
Yes, all of the time ____ (0)
Yes, most of the time ✔ (1)
No, not very often ____ (2)
No, not at all ____ (3)

This would mean: "I have felt happy most of the time" in the past week. Please complete the other questions in the same way.

1. I have been able to laugh and see the funny side of things:
   As much as I always could ____ (0)
   Not quite so much now ____ (1)
   Definitely not so much now ____ (2)
   Not at all ____ (3)

2. I have looked forward with enjoyment to things:
   As much as I ever did ____ (0)
   Rather less than I used to ____ (1)
   Definitely less than I used to ____ (2)
   Hardly at all ____ (3)

3. I have blamed myself unnecessarily when things went wrong:
   Yes, most of the time ____ (3)
   Yes, some of the time ____ (2)
   Not very often ____ (1)
   No, never ____ (0)

4. I have been anxious or worried for no good reason:
   No, not at all ____ (0)
   Hardly ever ____ (1)
   Yes, sometimes ____ (2)
   Yes, very often ____ (3)

5. I have felt scared or panicky for no good reason:
   Yes, quite a lot ____ (3)
   Yes, sometimes ____ (2)
   No, not much ____ (1)
   No, not at all ____ (0)

6. Things have been getting to me:
   Yes, most of the time I haven't been able to cope at all ____ (3)
   Yes, sometimes I haven't been coping as well as usual ____ (2)
   No, most of the time I have coped quite well ____ (1)
   No, I have been coping as well as ever ____ (0)

7. I have been so unhappy that I have had difficulty sleeping:
   Yes, most of the time ____ (3)
   Yes, sometimes ____ (2)
   No, not very often ____ (1)
   No, not at all ____ (0)

8. I have felt sad or miserable:
   Yes, most of the time ____ (3)
   Yes, quite often ____ (2)
   Not very often ____ (1)
   No, not at all ____ (0)

9. I have been so unhappy that I have been crying:
   Yes, most of the time ____ (3)
   Yes, quite often ____ (2)
   Only occasionally ____ (1)
   No, never ____ (0)

10. The thought of harming myself has occurred to me:*
    Yes, quite often ____ (3)
    Sometimes ____ (2)
    Hardly ever ____ (1)
    Never ____ (0)

TOTAL YOUR SCORE HERE ▶ ____

*** If you scored a 1, 2 or 3 on question 10, PLEASE CALL YOUR HEALTH CARE PROVIDER (OB/Gyn, family doctor or nurse-midwife) OR GO TO THE EMERGENCY ROOM NOW** to ensure your own safety and that of your baby.

If your total score is 11 or more, you could be experiencing postpartum depression (PPD) or anxiety. **PLEASE CALL YOUR HEALTH CARE PROVIDER (OB/Gyn, family doctor or nurse-midwife) now** to keep you and your baby safe.

If your total score is 9-10, we suggest you **repeat this test in one week or call your health care provider** (OB/Gyn, family doctor or nurse-midwife).

If your total score is 1-8, new mothers often have mood swings that make them cry or get angry easily. Your feelings may be normal. However, if they worsen or continue for more than a week or two, call your health care provider (OB/Gyn, family doctor or nurse-midwife). Being a mother can be a new and stressful experience. Take care of yourself by:

- ▶ Getting sleep—nap when the baby naps.
- ▶ Asking friends and family for help.
- ▶ Drinking plenty of fluids.
- ▶ Eating a good diet.
- ▶ Getting exercise, even if it's just walking outside.

Regardless of your score, if you have concerns about depression or anxiety, please contact your health care provider.

Please note: The Edinburgh Postnatal Depression Scale (EPDS) is a screening tool that does not diagnose postpartum depression (PPD) or anxiety.

See more information on reverse. ▶

Edinburgh Postnatal Depression Scale (EPDS). Adapted from the *British Journal of Psychiatry*, June, 1987, vol. 150 by J.L. Cox, J.M. Holden, R. Segovsky.

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