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Prenatal maternal depression predicts neural maturation and negative emotion in infants

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ABSTRACT

Despite widespread acceptance that prenatal symptoms of depression in mothers are detrimental to infants' long-term emotional and cognitive development, little is known about the mechanisms that may integrate outcomes across these domains. Rooted in the integrative perspective that emotional development is grounded in developing cognitive processes, we hypothesized that prenatal symptoms of depression in mothers would be associated with delays in neural maturation that support sociocognitive function in infants, leading to more problematic behaviors. We used a prospective longitudinal study of mothers ($N = 92$) and their infants to test whether self-reported symptoms of depression in mothers during the second and third trimesters were associated with neural development and infant outcomes at 4 months of age. While controlling for postpartum symptoms of depression, more prenatal symptoms of depression in mothers predicted less neural maturation in the parietal region of 4-month-old infants. Less neural maturation, in turn, was associated with greater infant negativity, suggesting neural maturation as a putative mechanism linking maternal symptoms of depression with infant outcomes. Differences in neural regions and developmental timing are also discussed.

1. Introduction

Early environmental characteristics play a significant role in infant cognition-emotion interactions, ultimately leading to both short- and long-term infant outcomes. The prenatal environment is particularly salient, as prenatal stress in the form of maternal depressive symptoms positively predicts negative emotional reactivity (Davis et al., 2007; Diego et al., 2005) and negatively predicts cognitive function in infants (Barker et al., 2013), effects which can persist well into adolescence (de Bruijn et al., 2009; Luoma et al., 2001; Luoma et al., 2004). Research aimed at understanding the effects of maternal depression on infant outcomes have traditionally tested emotion and cognition as separate effects in streams of development. Yet, contemporary theory and mounting empirical work underscore both the overlap and the interplay between putatively cognitive and emotional trajectories of development, particularly early infancy (Bell & Wolfe, 2004; Calkins & Bell, 2010). Nonetheless, the mechanisms for such overlap remain largely unknown. During infancy, the maturation of neural architecture that supports cognitive and emotional processing may provide insight into their integration during a sensitive period of development (Deave et al., 2008; Luoma et al., 2001). Thus, the current study was designed to test an integrative pathway, infant neural maturation, by which maternal symptoms of prenatal depression may impact infant

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development.

Though the precise mechanistic pathways by which maternal depression impacts infants' emotional and cognitive development remain unknown, theoretical and empirical work highlights a prominent role for developing biological systems. Prenatal Programming (Barker, 1998) and Developmental Origins (Sandman et al., 2011) perspectives suggest that the conditions of the intrauterine environment "program" biological systems in the fetus for a specific mode of function following birth. Extending this position, the Adaptive Calibration Model identifies cortisol, an end product of the hypothalamic-adrenal-pituitary (HPA) response to stress or challenge, as a mechanism by which programming may occur (Del Giudice et al., 2011). Indeed, maternal depressive symptoms are linked to increases in maternal cortisol (Diego et al., 2004; Harris et al., 2000) and cortisol is known to cross the placenta (Field, 1998).

Each of these theories suggests that although in-utero alterations to development are intended to support long-term viability of the fetus in a challenging environment, such changes may have significant negative consequences for long-term outcomes. Greater intrauterine stress is associated with delayed emotional and cognitive development in infants. More maternal symptoms of depression in the prenatal period predict greater trait-level negative emotion by infant age 3 months (Davis et al., 2007; Diego et al., 2005), and greater negative reactivity by infant age 4 months (Davis et al., 2004). More symptoms of prenatal depression in mothers are similarly associated with poorer cognitive functioning during this same period (Davis & Sandman, 2010; Hernandez-Reif et al., 2006) and into early childhood (Barker et al., 2013). Importantly, greater infant negative emotionality and poorer function predict greater socioemotional difficulties and risk for impairment as early as 4 months of age (Kagan, 1997; Posner & Rothbart, 2000). Notably, the timing of maternal-based stressors, like depressive symptoms, appears to be important for the course of infant cognitive and emotional development. Earlier, rather than later stressors, predict greater deficits in infant cognitive function (Davis & Sandman, 2010; Sandman et al., 2012) and postnatal symptoms have been linked to attenuated, rather than enhanced negative emotional reactivity (Field et al., 2005, 2007).

Though emotion and cognition were historically tested as separate streams of development, concurrent alterations to function related to prenatal stressors like maternal depression highlight their overlap, particularly in infancy. An integrative perspective, acknowledging infant emotional development as grounded in concurrently-developing cognitive processes (Bell et al., 2019) offers an opportunity for identifying mechanisms involved in linking prenatal maternal depression to individual differences in infant function. Indeed, historical theoretical accounts view emotion and cognition as two inseparable aspects of an integrated system (Piaget, 1981), explicitly suggesting that cognitive abilities can be linked to the maturation of neural architecture that will support both cognitive and socioemotional processing (Bell, 2015; Bell & Deater-Deckard, 2007). Well-regulated emotional responses will, in turn, provide a foundation for emerging developing abilities over time (Bell et al., 2019; Bell & Diaz, 2012; Fredrickson, 2004). This symbiotic interplay perpetuates across childhood and into early adulthood (Bell & Wolfe, 2004; Thompson, 1994). Thus, infant neural systems are likely mechanisms for overlapping effects in cognitive and emotional development. As the fetal brain undergoes rapid development during pregnancy (Charil et. al., 2010), it is also a likely mechanism by which maternal depression may exert simultaneous influence on infant socioemotional and cognitive function.

Electroencephalography (EEG) offers an opportunity to test the role of neural development in the cascade by which prenatal stressors, like maternal depression, impact infant development. Initial work in this area has focused on differences in alpha power (8–12 Hz in adults), the dominant frequency in infants beginning in the second half of the first year of life, between the right and left hemispheres. Alpha asymmetry has been linked to differences in propensities toward positive and negative affect (Davidson & Fox, 1982; Gartstein et. al., 2020) as well as approach and avoidance motivation (Harmon-Jones & Allen, 1997), making it a particularly useful tool for understanding individual differences in trait-level emotion. Infants exposed to prenatal maternal depression display greater relative right frontal asymmetry (Lusby et al., 2014; Field et al., 2004), putatively indexing greater propensities for negativity and avoidance and underscoring the possibility that individual differences in infant neural function are linked to mothers' prenatal symptoms. However, unlike changes in raw alpha power (Cuevas & Bell, 2011), links between alpha asymmetry and infant cognition are less apparent, perhaps due to the functional significance of asymmetry being largely affective in nature.

Yet non-asymmetric measures of EEG power remain useful. Greater alpha power, particularly at frontal electrode sites, is associated with cortical deactivation or inactivity (Bell & Wolfe, 2004; Thompson, 1994). Alpha power may also denote an aptitude for selectively inhibiting neural networks or regions that are not immediately needed (Klimesch et al., 2007) and/or a state of readiness for impending challenges (Knyazev et al., 2006). In the infant brain, both alpha power and the frontal cortex are underdeveloped relative to adults. Instead, changes in spatial distribution (crudely measured through EEG) and relative power across frequencies follow a predictable developmental course. Indeed, during early infancy neural activity is less localized (Thatcher et al., 1987), with greatest levels of neural activity being observed in the occipital, parietal, and temporal cortices (Chugani & Phelps, 1986). Consistent with a caudal-to-rostral pattern of neural development (Brody et al., 1987), alpha-like oscillations are visible by 4 months of age, peaking largely in parietal regions of the brain before shifting to largely central regions around 10 months of age (Hagne, 1968, 1972).

As alpha is developing across the first months of life, the power in the infant EEG is predominantly seen in the range of the delta band (1–4 Hz in adults; Bell, 1998). Developmental shifts from low- to high- frequencies as the dominant power spectra parallel advances in cognitive and emotional skills, including self- and emotional regulation (Bell, 1998; Bell & Wolfe, 2007; Hagne, 1968). Moreover, given the predictable developmental pattern (increasing alpha power and decreasing delta power) across infancy and early childhood (Bell & Wolfe, 2007; Marshall et al., 2002), ratio scores (e.g., ratio of high-frequency/alpha power to low-frequency/delta power) can be created to proxy neural maturation. Because greater high-frequency, relative to low-frequency, power is expected as the brain matures, greater ratio scores suggest more advanced neural maturation. Alpha/delta ratio scores are associated with both emotional development (Schmidt & Poole, 2018, 2021; Schmidt et. al., 2022) and cognitive abilities (Schleiger et al., 2014), indicating their utility for investigating neural maturation as a mechanism by which maternal symptoms of depression may affect the co-development of cognitive and emotion processes. Specifically, prenatal stressors like maternal depression likely impair the

maturation of neural systems that support the development of more adaptive cognitive and emotional capacities, leading to expectations for smaller alpha-delta ratios, poorer infant regulation, and less negative reactivity. These patterns are likely to be visible in parietal regions of the brain during early infancy and shift to frontal regions over time.

However, the relatively recent advancement of the Stress Acceleration Hypothesis presents an equally plausible competing possibility that early exposure to adversity, including prenatal depression, triggers the premature maturation of the biological systems supporting emotional and cognitive abilities (Callaghan & Tottenham, 2016). As is true for theories of Prenatal Programming and Adaptive Calibration, the early maturation of these systems is intended to offer short-term survival benefits in that the early termination of a plastic period may limit the negative consequences of harmful environmental input. However, it is also likely to prohibit the acquisition of the highest level of emotional and cognitive function by limiting windows of developmental plasticity (Tottenham, 2020), resulting in long-term impairment. Consistent with these expectations, children who experience early life adversity show precocious patterns of neural function, akin to that of typically-developing adolescents, in response to emotional cues yet have long-term problems with regulation (Callaghan et al., 2019; Gee et al., 2013). Such a pattern raises the possibility that prenatal stress, in the form of maternal depression, may predict accelerated neural maturation in the form of higher alpha-delta ratios in infants, making an explicit test of this association even more necessary.

In the current study, we tested neural maturation as a mechanism linking mothers' symptoms of prenatal depression with infant outcomes. Because we know of no work that has previously investigated alpha-delta ratios in infants beyond the first 72 h of development (Conde et al., 2017), we parsed this effort into two steps. First, we tested whether symptoms of depression during pregnancy predicted neural maturation in the first year of life as indexed by ratios of alpha-delta power. This effort reflected a test of competing hypotheses that allow for greater maternal symptoms to predict either less neural maturation or greater neural maturation in 4-month-old infants. We hypothesized that greater maternal symptoms of prenatal depression would be associated with less neural maturation in 4-month-old infants, with effects being largely visible at parietal electrodes given the stage of infant development under study. We conducted this test in a prospective longitudinal study of mothers from pregnancy to the early postnatal period. The inclusion of a postnatal assessment enabled us to control for postnatal symptoms, allowing for the isolation of effects of prenatal symptoms. Furthermore, this allowed for the examination of timing differences in the impact of maternal symptoms of prenatal depression on infant neural maturation. Given substantial evidence in the literature supporting the second trimester being a particularly sensitive period for the effects of maternal depression on fetal development (Sandman et. al., 2012, Davis et. al., 2006; Nyman et. al., 2020), we hypothesize maternal symptoms of depression during the second trimester will be a stronger predictor for infant neural maturation than maternal symptoms of depression during the third trimester.

Then, to clarify the nature of the association between prenatal symptoms of depression and infant neural development, we conducted an additional set of analyses that included infant behavior. Because the grounding theories for this work posit an initial adaptive period followed by long-term maladaptive effects, this step is necessary to understand whether any associations between maternal symptoms of depression and alpha-delta ratio should be considered adaptive or maladaptive for infant development. First, we tested whether infants' neural maturation was associated with infant behaviors. We focused on infant negativity given that it is a joint product of infant emotion and cognition (Campos et. al., 1989; Cole et. al., 2004; Hoemann et al., 2020; Rothbart et. al., 2006). We hypothesized that more maternal symptoms of prenatal depression would be associated with greater infant negativity and less regulation. In addition, we also included a measure of infant positive affect. The inclusion of this variable thus permitted a test of an alternative hypothesis that more maternal symptoms of prenatal depression would be associated with greater infant positive affect, as opposed to the maladaptive nature that our core hypothesis specifically emphasizes.

Second, we conducted a mediation analysis that tested infant neural maturation as a mediator of the association between maternal symptoms of prenatal depression and infant outcomes. We hypothesized that infant neural maturation will mediate this relation, such that more prenatal symptoms of depression will predict less neural maturation, which in turn will predict a greater level of infant negativity.

2. Method

2.1. Participants

Expectant mothers ($N = 92$; hereafter "mothers") enrolled in a longitudinal examination of peripartum emotion in mothers and offspring from 2015 to 2017 (Brooker et al., 2020; Nyman et al., 2020). Procedures were approved by the human subjects committee of the Institutional Review Board at (IRB #RB011615-FC). Mothers provided informed consent and were compensated for their participation after each study visit. Participants were recruited from the rural Mountain West region of Montana through local hospitals (26 %), university listserv emails (25 %), flyers posted at schools and businesses (13 %), Women, Infants, and Children office referrals (7 %), mother groups (2 %), and word of mouth (27 %). Eligibility for the present study required that participants be in their second or third trimester of pregnancy with no reported medical stimulant usage or neurological impairment. Both primigravida and multigravida women were enrolled.

Participation included a laboratory visit during the second trimester (maternal assessment; $M = 21.15$ weeks; $SD = 3.79$), third trimester (maternal assessment; $M = 35.92$ weeks; $SD = 1.47$), and at 4 months postpartum (mother and infant assessment; M infant age $= 4.27$ months; $SD = 0.62$). Targeted gestational age for each prenatal visit, based on the broader study aims, were 20 weeks (second trimester), 36 weeks (third trimester), and 4 months (postpartum). Targets were selected to enable the delineation of prenatal (e.g., early vs. late) and postpartum effects, and to allow for the assessment of neural bases of infant emotional development (Davis et al., 2004; Davis & Sandman, 2010). Rolling recruitment resulted in 81 mothers participating during the second-trimester visit (12 of

the full sample were not yet enrolled), 86 mothers participating during the third-trimester visit (6 withdrawals, 1 skipped phase), and 75 mothers participating during the 4-month postpartum visit (8 discontinuations, 7 withdrawals from the second phase, 3 withdrawals due to babies not meeting age requirement prior to study conclusion; PI changed institutions). Participants were primarily non-Hispanic (96 %) and White (89 %; Asian = 8 %, American Indian = 1 %, African American = 1 %, and mixed-race = 1 %), ranging in age from 21 to 41 years upon enrollment (M =30.49, SD = 4.22). Family income ranged from less than $15,000 to more than $91,000 annually, with the families most frequently reporting gross annual incomes between $51,000-$60,000 (17 %), or above $91,000 (25 %). The median and mode level of years of education were equivalent to a college degree (36 %), though parents' years of education ranged from 11 (less than a high school degree) to more than 20 (advanced or professional degree).

Self-reports of obstetric complications (Marceau et al., 2013) indicated low rates of pregnancy and delivery complications (M = 3.35, SD = 2.22), and medication use (M = 1.02, SD = 1.13); most infants were full-term (M = 39.64, SD = 1.75 weeks; 34–44 weeks; 6 preterm infants). Parity information was not available for all mothers. Given the low-risk nature of the sample, pregnancy and delivery complications were not considered further.

2.2. Procedure

Mothers completed identical prenatal visits during their second and third trimesters of pregnancy. Visits were scheduled via an online booking website or over the phone with the research staff. Two weeks prior to the scheduled visits, participants were mailed questionnaire packets which included assessments of self-reported symptoms of depression and general demographic information. Participants were instructed to bring completed packets to the laboratory visit.

Research staff contacted mothers to confirm the child's birth approximately one month after their anticipated due date and to schedule the postnatal laboratory visit. Mothers were mailed questionnaires in an identical fashion to the prenatal assessments, though postnatal questionnaires also included reports of pregnancy and delivery complications and infant temperament. EEG data were collected from infants during a baseline episode in the laboratory. Infants also completed a behavioral episode intended to assess motoric and affective reactivity. Mothers were present with their infants for all episodes.

A complete list of the measures used in this study can be accessed via the Open Science Framework (https://osf.io/tuv8s/?view_only=8789217ba74c4c2d9de7f6d08bc1cb83). Given the study hypotheses, this work focuses on mothers' self-reported symptoms of depression and anxiety during pregnancy and at 4 months postpartum along with infant neural activity and observed behaviors at age 4 months.

2.3. Measures

2.3.1. Maternal symptoms of depression

Mothers self-reported their depressive symptoms at each assessment using the Edinburgh Postnatal Depression Scale (EPDS; Cox et al., 1996). The EPDS is a 10-item survey designed to detect symptoms of emotional distress characteristic of depression ("I have felt scared or panicky for no very good reason"). The EPDS has been validated for use prenatally (Cox et al., 1996). Mothers indicated the degree to which they had experienced each symptom within the past week on a 4-point Likert scale (0 = absence of the symptoms, 3 = most frequent experience of the symptoms). Individual items were summed to create a total score (maximum possible score = 30). Scores of 12 or greater on the EPDS are consistent with the likely presence of depression. Ten mothers during the second trimester, 6 mothers during the third trimester, and 7 mothers at 4 months postpartum met the threshold for clinical levels of depression. Scores in the current data set ranged from 1 to 25. The EPDS showed acceptable internal consistency (second trimester $\alpha = 0.82$, third trimester $\alpha = 0.88$, postnatal $\alpha = 0.82$).

2.3.2. Infant characteristics

Characteristic cognitive and emotional behaviors in infants were measured during the postnatal assessment through both maternal reports and laboratory observations. Mothers reported their infant's regulatory ability and negative reactivity using the Infant Behavior Questionnaire (IBQ-R; Gartstein & Rothbart, 2003), a 195-item parent-report measure that asked mothers to rate, on a 7-point Likert scale, the degree to which certain behaviors were characteristic of their infant over the previous 2 weeks (0 = Never, 7 = Always). The IBQ-R has demonstrated validity for assessing 14 domains of temperament in infants aged 3–12 months, which are then composited to reflect three broader factors of infant temperament: Surgency/Extraversion, Negative Affectivity, and Orienting/-Regulation. Considering the aims of the current study, we focused on Negative Affectivity (e.g., fusses or cries immediately after sleeping, clings to a parent when introduced to an unfamiliar adult; $\alpha = 0.97$) and Orienting/Regulation (e.g., enjoys being sung to, plays with one toy or object for 5–10 min or longer; $\alpha = 0.95$) dimensions.

Observed infant reactivity at 4 months of age was assessed using a *Mobile Task* (Kagan, 1997) designed to assess temperamental reactivity in infants. For this episode, infants were buckled into a car seat facing an experimenter. Mothers were instructed to sit close to their infant but out of sight. The experimenter placed one small, stuffed toy on a mobile and displayed it to the infant for 20 s. Following this, they added two additional stuffed toys to the mobile, displaying all the three toys to the infant for 20 s. These steps were repeated for a total of three trials (six toy combinations) in all. Toys displayed were counterbalanced across trials between jungle animals and teddy bears. Mothers were asked to remain uninvolved throughout the procedure but were told that they could intervene to end the episode if infants became overly distressed (n = 1). Infant behaviors were recorded from a video camera set up behind the experimenter.

Infant behaviors were coded offline in 20-second epochs, consistent with Thistle coding procedures (Fox et al., 2015; Pérez-Edgar

et al., 2021). Positive affect (smiling and neutral or positive vocalizations) and negative affect (fussing and crying) were coded for each epoch. Coders were two graduate research assistants who demonstrated reliability for each code (k = 0.70) prior to coding independently. Half (50 %) of episodes were double-coded to protect against coding drift. Discrepancies in codes were resolved through verbal conference that included a review of videos by coders and consensus on a final score. Frequency counts were summed across epochs and divided by the total number of epochs completed by the infant in order to account for between-subject differences in episode length. Scores that were more than + /- 3 SD from the mean were windsorized (negative affect: $n = 3$; positive affect: $n = 2$) to a value of 3 SD from the mean (Tukey, 1962).

2.3.3. Infant Alpha-Delta Ratios

Continuous EEG was recorded from infants during a 5-minute baseline period. Mothers were given a small set of low-intensity (e.g., small, did not make noise, did not have lights, etc.), age-appropriate toys and asked to keep their infant as calm and still as possible for 5 min of recording.

Prior to beginning the baseline recording, infants were fitted with a 32-channel Bio-Semi elastic head cap. Electroconductive gel was placed into plastic electrode holders, followed by Ag-AgCl-tipped active electrodes, arranged according to the 10–20 labeling system. Data were recorded using a BioSemi Active 2 system (Cortech Solutions, LLC; Wilmington, NC), with a sampling rate of 2048 Hz. A combination of the common-mode sense active electrode and driven right leg passive electrode were used to form the ground during recording. Horizontal and vertical eye movements were recorded via passive electrodes placed at the outer canthus of the left eye and right eye, and supra and infraorbital sites of the left eye, respectively.

EEG data were processed offline using Brain Vision Analyzer (Brain Products: Gilching, Germany). Following standardized internal procedures for data processing ((Brooker et al., 2020; Nyman et al., 2020), continuous EEG was re-referenced to the average of all 32 channels, high-pass filtered at 0.1 Hz, low-pass filtered at 30 Hz, and corrected for eye movement or blinks (Gratton et al., 1983). Segments of 1.000 s were extracted from the continuous EEG and baseline corrected using the average of the entire data segment. Artifacts were identified using an automated procedure when one of the following criteria were met: a voltage step of more than 75 μV between data points, a difference of 150 μV within 200 ms, amplitudes below 0.5 μV within a 50 ms period, and activity that exceeded + 100 μV or − 100 μV. The remaining segments were visually inspected for artifacts.

Artifact-free data were submitted to a fast-Fourier transform using a hamming window with 50 % overlap. Consistent with previous work (Bell & Cuevas, 2012; Najjar & Brooker, 2017; Schmidt & Poole, 2021), regional EEG power (in μV^2) was derived in the alpha (6.0–9.0 Hz) and delta (0.5–2.9 Hz) frequency bands. Power was not derived for six infants due to excessive artifacts in the recording. All remaining infants had a minimum of 121 segments included in averages ($M = 464.87$, $SD = 161.59$). Mean alpha and delta power were composited separately at frontal (F3, Fz, F4) and parietal (P3, Pz, P4) electrode sites. Upon inspection for univariate outliers, one case was found to have extreme values for all EEG measures; EEG data were not retained for this case. Alpha-delta ratios were formed by dividing power in the alpha band by power in the delta band. Ratio values were log transformed to correct for skew. Ratio values were not significantly correlated with infant age (frontal: $r = 0.21$, $p = .13$; parietal $r = 0.22$, $p = .11$) or number of segments of clean EEG data (frontal: $r = 0.05$, $p = .73$; parietal $r = 0.19$, $p = .16$).

2.4. Covariate

Given the overlap between maternal symptoms of depression and maternal symptoms of anxiety across the prenatal and postpartum periods, we assessed maternal anxiety symptoms to use as a covariate in analyses. Mothers self-reported their anxiety symptoms at each assessment using the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990). The PSWQ is a 16-item self-report measure designed to assess the trait of worry (“ I’ve been a worrier all my life”) through participants’ endorsement of positively and negatively worded statements using a 5-point Likert-type scale; 1 being “not at all typical” and 5 being “very typical.” We focused on the assessment of worry given prior demonstrations that increases in anxiety during the prenatal period frequently center around worries related to the pregnancy, delivery, and being able to take care of a new infant (Meyer et al., 1990; Ross & McLean, 2006). The PSWQ has previously been validated for use in maternal samples across the perinatal period (Voegtline et al., 2021), and demonstrated high internal consistency across all assessments (second trimester α = 0.91, third trimester α = 0.93, postnatal α = 0.93).

2.5. Missing data

Most mothers in the study ($n = 75$) completed at least 1 prenatal assessment and a postnatal assessment. Of these, the majority ($n = 65$) completed all three assessments (second trimester, third trimester, and postnatal), though a subset joined the study in their third trimester ($n = 9$) and thus completed only the third trimester and prenatal assessments. One mother completed only the second trimester and postnatal assessments because she went into early labor. Of the 17 remaining participants, six left the study after the first assessment (second trimester data only), 2 participants enrolled in their third trimester but did not complete a postnatal assessment, and 9 completed the second and third trimester assessments but did not complete a postnatal assessment. Most prenatal assessments were missed because the primary investigator changed institutions and the laboratory was closed before infants were born. We have added these additional details regarding missing data to our missing data section.

Overall, of the 92 participants in the study, maternal depression (EPDS) scores included in the analysis varied across the second trimester ($n = 77$), third trimester ($n = 82$), and postnatal ($n = 67$) visits, with total missing data across visits ($n = 53$) largely attributed to participant drop out or failing to return the questionnaire. Mothers’ self-reported anxiety data (PSWQ), included as a covariate in the analysis, varied similarly across the second trimester ($n = 78$), third trimester ($n = 81$), and postnatal ($n = 67$) visits.

Table 1
Descriptive statistics and bivariate correlations for primary variables.

	n	M	SD	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
1. Mom second trimester depression symptoms	77	6.39	4.12															
2. Mom third trimester depression symptoms	82	5.46	4.74	0.48**														
3. Mom postnatal depression symptoms	67	5.30	4.26	0.35**	.32**													
4. Mom second trimester anxiety symptoms	78	40.63	11.01	0.42**	.27*	.44**												
5. Mom third trimester anxiety symptoms	81	40.70	12.04	0.38**	.55**	.36**	.74**											
6. Mom postnatal anxiety symptoms	67	40.90	12.24	0.53**	.39**	.72**	.73**	.69**										
7. Infant frontal alpha-delta ratio	57	-4.15	0.54	-0.42**	-.01	-0.10	-0.15	-0.34*	-.24									
8. Infant parietal alpha-delta ratio	57	-4.18	0.49	-0.23	0.07	0.00	-0.06	-0.17	-0.06	0.85**								
9. Infant frontal alpha power	57	1.48	0.74	-0.16	-0.14	0.12	-0.07	-0.16	0.07	0.44**	.37**							
10. Infant frontal delta power	57	97.62	51.16	-0.02	-0.20	-0.15	-0.01	0.00	-0.02	-0.20	-0.37**	.67**						
11. Infant parietal alpha power	57	1.79	1.13	-0.30*	-.19	0.15	-0.13	-0.25	0.22	0.50**	.24	0.79**	.56**					
12. Infant parietal delta power	57	108.58	55.57	-0.03	-0.21	-0.14	0.00	0.07	-0.04	0.27**	-.40**	.52**	.88**	.60**				
13. Mom-reported infant negative affect	66	2.94	0.60	0.10	0.13	0.23	0.06	-0.01	0.19	-0.10	0.07	-0.17	-0.19	-0.20	-0.15			
14. Mom-reported infant regulation	66	5.33	0.42	-0.11	-0.06	-0.03	-0.28	-0.25	-0.17	0.16	0.18	-0.09	-0.30*	.01	-0.23	-0.25*		
15. Observed infant negative affect	67	0.51	1.13	0.33*	.12	0.27*	.17	0.04	0.33**	-.07	0.05	-0.14	-14	-0.26	-0.21	0.04	-0.07	
16. Observed infant positive affect	67	0.24	0.46	-0.11	-0.19	-0.16	-0.15	-0.14	-0.03	0.03	0.08	-0.11	-0.11	-0.14	-0.08	0.09	-0.01	-0.02

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

Regarding infant characteristic data collected during postnatal visits, mother-reported infant behavior (IBQ) scores were available for 66 participants with missing data ($n = 27$) similarly attributed to discontinuation or failure to return packets. Total observed infant reactivity data were available for 67 infants and missing for 26 which was attributed to infants being too young to participate before the study ended ($n = 3$), mothers opting out of the study during the prenatal phase ($n = 7$), postnatal phase ($n = 8$), or corrupted/unknown missing video data ($n = 8$). Lastly, alpha-delta ratios were available for 57 infants at 4 months postpartum with missing data ($n = 36$).

Analysis of patterns of missing data from all measured variables suggested that data were missing at random (Little's MCAR: $\chi 2(66) = 81.255$, $p = .098$). Consistent with recommendations for MAR data, all variables were included in the full-information maximum likelihood (FIML) procedures used to handle missing data (Enders, 2010). Because all participants had some subset of available data, the final analytic sample comprised 92 participants.

Preliminary analyses were conducted in SPSS version 28 (IBM SPSS Statistics, 2021). Data were examined for the presence of outliers and to ensure that variables were normally distributed. Primary hypotheses were tested in Mplus. Given substantial overlap between maternal symptoms of anxiety and depression, and evidence that maternal anxiety may also predict infants' postpartum emotional development (O'Connor et. al., 2014a,2014b), anxiety symptoms were entered as covariates for depressive symptoms. First, we tested whether maternal depressive symptoms predicted frontal and parietal alpha-delta ratios in infants at age 4 months. Separate scores of maternal depressive symptoms were included for each assessment given evidence for timing differences in the impact of maternal emotion on infant outcomes across pregnancy (Brooker et al., 2020; Davis et al., 2007).

To further understand the potential effects of maternal symptoms of depression on infants' neural activity, we also planned follow up tests that would test associations between alpha-delta ratio and infant emotion characteristics. These analyses were intended to help us understand whether any individual differences in neural function might be understood as adaptive or maladaptive at infant age 4 months. A full mediation model was not examined given our limited sample size.

3. Results

3.1. Preliminary analyses

Descriptive statistics and correlations among study variables are reported in Table 1. Symptoms of maternal depression showed moderate stability over time, with the greatest stability observed across the two prenatal assessments. At the bivariate level, greater numbers of maternal depressive symptoms in the second trimester of pregnancy were associated with smaller frontal alpha-delta ratio scores at age 4 months but were unrelated to parietal alpha-delta ratio scores. Maternal depressive symptoms in the third trimester did not show bivariate associations with infant alpha-delta ratios at frontal or parietal sites. Greater negative affect in infants at 4 months of age was associated with greater numbers of maternal depressive symptoms in the second trimester and at the postpartum assessment. Maternal symptoms of depression were not associated with mother-reported infant behaviors or observed positivity in infants at the bivariate level.

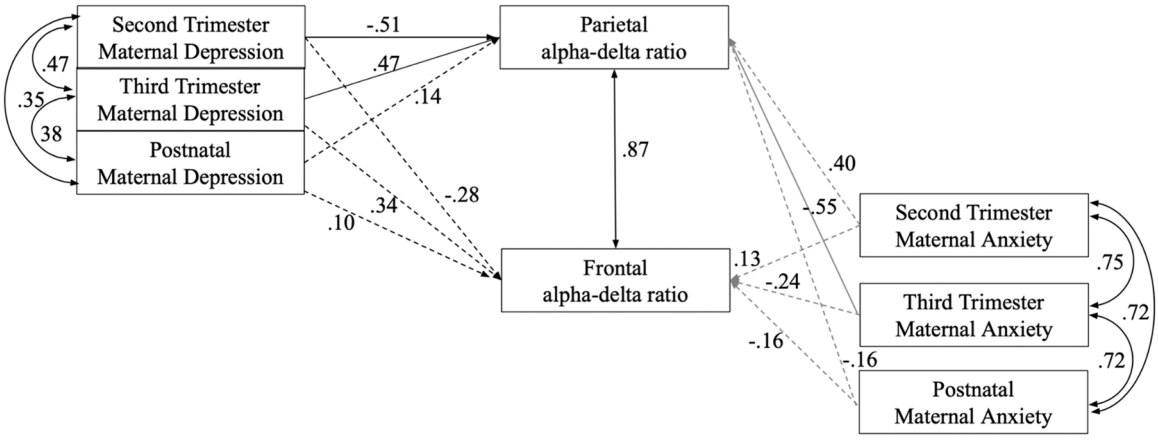

Fig. 1. *Symptoms of maternal depression as predictors of frontal and parietal alpha-delta ratio. Note:* All values reflect standardized estimates; solid lines reflect values that are significant at $p < .05$; dashed lines reflect nonsignificant associations. The following correlations have been omitted from the figure to enhance readability: second trimester depression was significantly correlated with second trimester ($r = 0.44$), third trimester ($r = 0.41$), and postnatal ($r = 0.45$) anxiety; third trimester depression was significantly correlated with second trimester ($r = 0.27$), third trimester ($r = 0.55$), and postnatal ($r = 0.46$) anxiety; postnatal depression was significantly correlated with second trimester ($r = 0.44$), third trimester ($r = 0.39$), and postnatal ($r = 0.73$) anxiety.

3.2. Maternal symptoms of depression as a predictor of infant neural maturation

Frontal and parietal alpha-delta ratio scores were simultaneously regressed onto maternal depressive symptoms at the second trimester, third trimester, and postpartum assessments. The analytic model allowed maternal symptoms of depression and anxiety to correlate within and across assessments. The model also allowed frontal alpha-delta ratio to correlate with parietal alpha-delta ratio.

As shown in Fig. 1, greater depressive symptoms in the second trimester were associated with a smaller alpha-delta ratio at parietal electrodes ($B = -0.07$, $SE(B) = 0.02$, 95 % CI [− 0.11,.–03], $p < .01$), while greater depressive symptoms in the third trimester ($B = 0.05$, $SE(B) = 0.02$, 95 % CI [0.01,0.10], $p = .02$) were associated with larger alpha-delta ratio scores at parietal electrodes. Postnatal depressive symptoms ($B = 0.02$, $SE(B) = 0.03$, 95 % CI [− 0.03,0.07], $p = .49$) were unrelated to parietal alpha-delta ratio scores.

Infant alpha-delta ratio at frontal electrodes was unrelated to maternal depressive symptoms in the second trimester ($B = -0.03$, $SE(B) = 0.02$, 95 % CI [− 0.08,0.01], $p = .14$), third trimester ($B = 0.04$, $SE(B) = 0.03$, 95 % CI [− 0.01,0.08], $p = .16$), and postnatal period ($B = 0.01$, $SE(B) = 0.03$, 95 % CI [− 0.04,0.02], $p = .64$).

3.3. Infant neural maturation ratio as a predictor of infant characteristics

We then proceeded to test whether infant characteristics were associated with alpha-delta ratios. Because maternal-reported and observed characteristics were uncorrelated, they were tested in separate models. In both cases, infant characteristics were regressed onto alpha-delta ratios at frontal and parietal electrodes. Maternal symptoms of depression and anxiety were included in models to test whether alpha-delta ratio scores were uniquely associated with infant characteristics, above and beyond the influence of maternal emotion. All predictor variables were allowed to covary. Larger alpha-delta ratios at frontal sites ($B = 0.72$, $SE(B) = 0.34$, 95 % CI [0.07, 1.38], $p = .03$) and smaller alpha-delta ratios at parietal sites ($B = -0.71$, $SE(B) = 0.36$, 95 % CI [− 1.42,0.01], $p = .05$) were associated with greater mother-reported negativity in infants. Neither alpha-delta ratio scores at the frontal site ($B = 0.34$, $SE(B) = 0.26$, 95 % CI [− 0.17,0.84], $p = .19$) nor alpha delta ratio scores at the parietal site ($B = -0.26$, $SE(B) = 0.28$, 95 % CI [− 0.80,0.29], $p = .36$) was associated with mother-reported infant regulation.

Observed infant negativity was unrelated to alpha-delta ratios at frontal ($B = 0.43$, $SE(B) = 0.57$, 95 % CI [− 0.69, 1.55], $p = .45$) and parietal ($B = -0.33$, $SE(B) = 0.61$, 95 % CI [− 1.54,0.87], $p = .59$) sites. Greater observed infant positivity was also unrelated to alpha-delta ratios at frontal ($B = 0.26$, $SE(B) = 0.24$, 95 % CI [− 0.21,0.72], $p < .01$) and parietal sites ($B = -0.24$, $SE(B) = 0.25$, 95 % CI [− 0.73,0.26], $p = .82$).

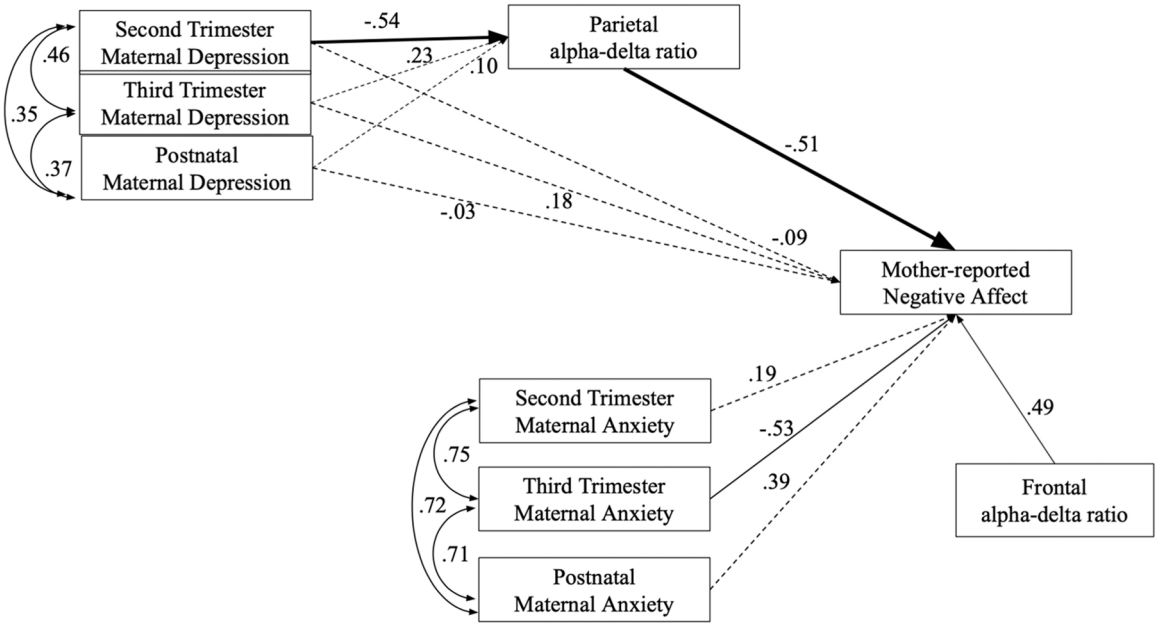

Fig. 2. *Indirect effects of maternal depression on mother-reported negative affect* via *parietal alpha-delta ratio*. *Note:* All values reflect standardized estimates; solid lines reflect values that are significant at $p < .05$; dashed lines reflect nonsignificant associations. The following correlations have been omitted from the figure to enhance readability: second trimester depression was significantly correlated with second trimester ($r = 0.44$), third trimester ($r = 0.41$), and postnatal ($r = 0.46$) anxiety; third trimester depression was significantly correlated with second trimester ($r = 0.28$), third trimester ($r = 0.55$), and postnatal ($r = 0.45$) anxiety; postnatal depression was significantly correlated with second trimester ($r = 0.44$), third trimester ($r = 0.40$), and postnatal ($r = 0.73$) anxiety. The indirect effect of second trimester depression (beta = 0.28, SE(beta) = 0.10, p < .01) on infant regulation is shown as bolded solid lines.

3.4. Neural maturation as a mechanism linking maternal symptoms of depression to infant characteristics

Given demonstrated associations between maternal symptoms of depression and parietal alpha-delta ratio, as well as associations between parietal alpha-delta ratio and mother-reported infant negativity, we explored a mediation model that tested the full mechanistic association. Specifically, we tested whether the alpha-delta ratio at parietal electrodes mediated the association between maternal symptoms of depression and infant outcomes. Although it is rooted in developmental theory and supported by the results reported above, we stress the nature of this analysis as exploratory given our small sample size and the fact that infant neural activity and behaviors were assessed concurrently. That is, it is intended as a preliminary examination that may aid in the justification of future work on such pathways.

Our pathways of interest were the paths from prenatal symptoms of depression (second and third trimesters) to parietal neural maturation in infants, although we retained our measure of mothers' postnatal depressive symptoms in the model in order to isolate tests of prenatal effects. Similarly, we retained measures of maternal anxiety to isolate effects of prenatal symptoms of depression. Because frontal alpha-delta ratios were also related to infant negativity, we also included this measure as a covariate in our model. The full analytic model is shown in Fig. 2.

There was a significant indirect effect of second trimester depressive symptoms on mother-reported infant negative affect (B =0.05, $SE(B) = 0.02$, CI [0.01,0.09], $p = .01$). Greater maternal symptoms of depression in the second trimester predicted smaller alpha-delta ratios at age 4 months ($B = -0.07$, $SE(B) = 0.02$, CI [− 0.11, − 0.03], $p < .01$), which in turn predicted greater infant negativity ($B = -0.69$, $SE(B) = 0.21$, CI [− 1.10, − 0.28], $p < .01$). There was no significant indirect effect of third trimester depressive symptoms on infant negativity via alpha-delta power ratio ($B = -0.02$, $SE(B) = 0.02$, CI [− 0.05,0.01], $p = .27$).

4. Discussion

We prospectively examined whether prenatal symptoms of depression in mothers predicted neural maturation as indexed by alpha-delta ratio scores in 4-month-old infants. We found that mothers' prenatal symptoms of depression predicted neural maturation, largely in the parietal region of the infant brain. To date, alpha-delta ratio scores have predominantly been linked to frontal brain maturation in samples of older children (Schmidt & Poole, 2021; Schmidt et al., 2022). This focus on the frontal alpha-delta ratios has largely resulted from a priori hypotheses regarding frontal lobe development rather than a specificity of previous results to frontal sites. While such a focus is sensible in older samples, alpha-delta ratio scores at parietal sites have a unique utility in infants, for whom neural activity is less localized while the frontal cortex matures (Thatcher et al., 1987). Indeed, for at least the first 3 months of life, the greatest levels of neural activity are observed in the occipital, parietal, and temporal cortices (Chugani & Phelps, 1986). Over time, infant alpha rhythms show a normative topographical shift from posterior to anterior regions (Bell, 1998; Dreyfus-Brisac & Curzi-Dascalova, 1975; Stroganova et al., 1999). Increases in frontal alpha power become particularly noticeable between 6 and 8 months of age (Chugani et al., 1987), preceding normative global increases in alpha power (Bell & Fox, 1994) and declines in delta power between roughly 8 and 12 months of age (Hagne, 1968). Thus, given the young age of infants in our sample, it is perhaps not surprising that individual differences in the alpha-delta ratio were most meaningful at parietal sites. That is, limited overall alpha power at frontal sites during this stage of development would result in a restricted range and limit our ability to capture meaningful individual differences. Indeed, our raw measures suggest both less power and less variability in alpha at frontal compared to parietal electrodes. Increases in the relative alpha-delta ratio would be expected overall, but most notably at frontal recording sites during the second half of the first year of life based on normative patterns of development in the EEG (Bell, 1998; Bell & Fox, 1994). Before that time, examinations of activity at parietal electrodes may be most fruitful.

Most directly linked to our hypotheses, we found evidence for less neural maturation in infants when mothers had reported more symptoms during the second trimester of pregnancy. The experience of depressive symptoms is associated with increased cortisol production (Diego et al., 2004; Harris et al., 2000); higher levels of cortisol during pregnancy have been linked to both cognitive (Davis & Sandman, 2010) and affective (de Weerth et. al., 2003) outcomes in infants. Maternal serum cortisol during pregnancy is also inversely associated with fetal brain growth (Li et al., 2012). Critically, inconsistencies in findings linking prenatal cortisol to infant outcomes have led to suggestions that the process is mediated through other aspects of development (Zijlmans et. al., 2015). Consistent with our hypotheses and the aims of the special issue, the current findings set forth neural maturation as one putative mediator of links between mothers' prenatal depressive symptoms and infant social and cognitive outcomes as they simultaneously unfold. Indeed, greater maternal symptoms during the second trimester of pregnancy were associated with less neural maturation, which then predicted greater trait-level infant negativity. Thus, it is possible that when mothers experience high levels of depressive symptoms, associated increases in maternal cortisol levels may enhance the potential for stress hormones to cross the placental barrier (Gitau et. al., 1998), limiting fetal brain development.

Precisely why ratios may be small is known, though symptoms of maternal depression are associated with slowed neural maturation reflected as a delayed decline in delta power. Persistent high levels of delta power would result in smaller alpha-delta ratio scores. Our understanding of the functional significance of delta power remains limited, though a prominent theory is that excessive low-frequency power reflects a delay in the maturation of the central nervous system (Barry et al., 2003). Children (5–31 months of age) who were institutionalized during infancy show greater slow-wave activity at parietal sites relative to never-institutionalized children (Marshall et al., 2004). Though it is not clear whether findings related to such extreme early circumstances can be directly applied to results in our typically-developing sample, they are consistent with the possibility that early stress in the form of maternal symptoms of depression impact infant outcomes by inhibiting neural maturation and delaying normative developmental shifts from low-frequency to high-frequency neural rhythms.

The timing of exposure to maternal symptoms appears to be critical. In contrast to findings in the second trimester, greater maternal symptoms of depression later in pregnancy were associated with larger alpha-delta ratio scores, indicating more maturation by age 4 months. These findings offer a conceptual replication of other investigations (Davis & Sandman, 2010). Similarly, patterns of results from work using alpha-delta ratio scores suggest that early (prenatal) adversity is linked with less neural maturation while later (postnatal) adversity predicts greater neural maturation (Hassan et. al., 2021). If parturition is not a precise temporal cutoff, these findings along with our own suggest that, in general, earlier experiences of adversity are associated with delays in maturation while later experiences of adversity are associated with expedited maturation. Such a pattern is reasonable from a prenatal programming perspective as a fetus earlier in development would benefit less from a biological "push" toward independent function relative to a fetus that is almost fully developed (Barker, 1998; Callaghan & Tottenham, 2016). These theories may be further extrapolated to anticipate expedited frontal maturation at even later assessments. In this way, our results are partially consistent with both the Fetal Programming perspective and the Stress Acceleration Hypothesis, but suggest developmental windows for delayed versus precocious development. This will be an important avenue for future work.

It should be noted that the association between maternal depression later in pregnancy and increased alpha-delta ratio scores, putatively indexing greater neural maturation, was only visible when earlier levels of prenatal depression were controlled. That is, second trimester symptoms of depression appeared to act as a suppressor variable, unmasking an association between third trimester depressive symptoms and neural maturation that was invisible at the bivariate level. Classically, suppressor variables are recognized to restrain variance in other predictors that is irrelevant to the outcome (Cohen et al., 2003). In this study, when stability in maternal prenatal symptoms (i.e., variance in symptoms of depression that is shared across trimesters) is controlled, a positive association between third trimester symptoms and neural maturation emerges. That is, greater symptoms of depression in the third trimester predict greater neural maturation, as measured by alpha-delta ratio, assuming equal numbers of symptoms across participants during the second trimester. This finding highlights the need for additional work that accounts for stability and instability of maternal symptoms over time in the prediction of infant neural development.

Consistent with previous work (Davis et al., 2004; Field, 2017), maternal symptoms of depression were positively associated with infant negativity at 4 months of age. However, links between neural maturation and infant negativity were less consistent. Specifically, greater frontal maturation and less parietal maturation were linked to greater infant negativity at 4 months of age. Associations were only evident through maternal reports. As such, maternal perceptions almost certainly play a role in this link; however, it is important to note that because maternal symptoms of anxiety and depression were included in the model, the association cannot solely be a product of maternal symptoms. It is possible that the specificity of the association to maternal reports may be the product of maternal reports capturing a broader range of behavior relative to the behavioral task used here, which is primarily intended to reflect immediate reactivity to novelty (Kagan, 1994; Zentner & Bates, 2008). As such, this link may reflect an association between neural maturation and the broad deployment of negativity, rather than fearful inhibition, in infants.

Although our cross-sectional design limits the conclusions that can be drawn from a test of mediation, the inclusion of infant behavior in the final model is critical for understanding whether less neural maturation in relation to maternal symptoms may be protective or problematic. We found that less neural maturation appears to serve as a mechanism for the association between second trimester maternal depressive symptoms and increased infant negativity. Although increased postnatal depressive symptoms were also associated with greater negativity in infants, maternal postnatal depressive symptoms were not associated with infant neural maturation, making it an unlikely mechanism for this effect. Thus, it is possible that apparent differences in the impact of maternal depressive symptoms on infant neural development reflect different pathways of development. It will be critical for this possibility to be addressed in future longitudinal work.

The links to infant negativity in the current work likely reflect a mechanism of neural maturation that simultaneously impacts infants' cognitive and emotional development. Specifically, infant negativity likely reflects the cognitive-affective intersection of high levels of affective reactivity and low levels of cognitive regulation (Cole et. al., 2004; Rothbart et. al., 2006). Emotion and cognition are well-integrated systems; displays of negativity may reflect infants' inability to reorient in order to avoid or diminish experiences of distress (Robthart et al., 2006), or affective behavioral strategies that emerge from cognitively-based efforts to elicit help (Hoemann et al., 2020; Campos et. al., 1989). As such, it is sensible that - as highlighted in this special issue - affective and cognitive development co-occur. Our work suggests neural development as one possible mechanism for this co-occurrence and further suggests that this mechanistic association is impacted by prenatal factors like maternal depression. As such, our work lays the foundation for future studies to more clearly delineate the developmental process(es) by which prenatal experiences begin a developmental cascade that can predict emotional and cognitive development in infants' first year of life.

The current study is not without limitations. Although there was a notable prevalence of symptoms of depression in our participants, analyses are focused on a community sample whose risk for cognitive and emotional problems may be low. Similarly, although this sample as a whole reflects a range of sociodemographic characteristics, participants were predominantly White, non-Hispanic, and from moderately high-income households on average. Future work is needed to understand the degree to which results are generalizable to other populations.

In addition, our study did not track parity. Previous links between parity and maternal distress have been inconsistent, with some work reporting no differences in maternal levels of depression based on parity (e.g., Westdahl et al., 2007), while others report both greater maternal distress in primiparous than in multiparous mothers (Gillespie et al., 2018; Dørheim et al., 2009), and more depressive symptoms in multiparous relative to primiparous mothers (Tronick & Reck, 2009). Consequently, future studies should include parity to better understand possible deviations from the current pattern of findings.

Finally, alpha-delta ratio was measured concurrently with infant behaviors. Though this is useful for understanding direct associations, this study design is not conducive for mediation analysis. Therefore, results should be interpreted cautiously considering

inherent restraints on statistical power and the noted limitations above. Future work utilizing a longitudinal, relative to cross-sectional, design with a larger sample size may capture more stable effect size estimates and increase the generalizability of results.

Despite limitations, this work offers an important advancement to our understanding of the mechanisms that may underlie the interplay among developing systems of cognition and emotion, consistent with the aims of the special issue. Namely, results suggest neural development as a mechanism that links prenatal maternal symptoms with infants' sociocognitive function during a critical period for emotional and cognitive development in young children.

CRediT authorship contribution statement

Jennifer Kling: Conceptualization, Methodology, Data curation, Writing – review & editing. **Sejal Mistry-Patel:** Methodology, Data curation, Writing – review & editing. **Sarah G. Peoples:** Methodology, Data curation, Writing – review & editing. **Daniel R. Caldera:** Methodology, Data curation, Writing – review & editing. **Rebecca J. Brooker:** Conceptualization, Methodology, Data curation, Writing – review & editing.

Conflicts of interest

There are no conflicts of interest to disclose.

Data Availability

Data will be made available on request.

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