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Developmental Trajectories of Delta–Beta EEG Coupling Across Infancy: The Influence of Infant Temperament and Maternal Anxiety Over Time

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ABSTRACT

Coupling, or the correlation, between delta and beta EEG power may underly regulatory processes. Stronger, positive delta–beta coupling is associated with anxiety and fearful temperament, potentially tracking dysregulation. However, most studies have reported on cross-sectional investigations of delta–beta coupling in children. Thus, the normative development of delta–beta coupling remains poorly understood. Here, we leveraged a diverse sample ($N = 165$) of infants with assessments of EEG, negative affect, and maternal anxiety across 4, 8, 12, 18, and 24 months to model trajectories of delta–beta coupling and associations with infant and maternal traits over time. Multilevel models of delta (1–2.5 Hz) and beta (11–18 Hz) power indicated that delta–beta coupling increased from 8 to 24 months at the Frontal region but decreased over time at the Central and Parietal regions. Increasing negative affect over time (slope) was associated with steeper decreases in Parietal coupling. Higher maternal anxiety levels over time were associated with increasing trajectories of Parietal delta–beta coupling, but only for infants with decreasing negative affect trajectories. We show that delta–beta coupling trajectories are differentially modulated by infant temperament and anxiety levels in the infants' proximal environment.

1 | Introduction

Delta–beta coupling captures the coordination between simultaneous changes in delta and beta electroencephalogram (EEG) oscillations, operationalized through the statistical correlation between power in the delta and beta frequency bands (Miskovic and Schmidt 2009; Myruski et al. 2022; Poole and Schmidt 2019; van Peer et al. 2008). Delta power may reflect bottom-up processes, while beta power reflects top-down, cognitive control (Riddle et al. 2021). Developmental studies of EEG power indicate that delta power decreases, whereas beta power increases, as a function of age (Clarke 2001), in line with known age-related gains in cognitive control (Diamond 2002). While not empirically tested, these findings suggest that delta–beta coupling may reflect the interaction between subcortical and cortical circuitry involved in regulatory processes, such as amygdala–mPFC connectivity, which has been implicated in fMRI studies of emotion regulation (Gee et al. 2013; Silvers et al. 2017). In adults, positive delta–beta coupling has been associated with better attentional control (Morillas-Romero et al. 2015) and cognitive control (Riddle et al. 2021). Among children 5–7 years old, positive delta–beta coupling has been associated with adaptive

emotion regulation concurrently and 2 years later (Myruski et al. 2022).

These studies converge on the utility of delta–beta coupling as a candidate measure for investigating the neural foundations of infant emotion regulation. Behavioral studies show that infants begin to rudimentarily regulate their emotions as early as 3 months (Ekas et al. 2018). However, we know little about the neural processes underlying early regulatory efforts due to methodological limitations to simultaneously capture infant regulation and neural activity. Quantifying trajectories of delta–beta coupling as a function of socioemotional characteristics (i.e., infant temperament) may expand our understanding of neural systems of infant emotion regulation. This approach may also help contextualize previous behavioral findings by fine-tuning our characterization of when and for whom delta–beta coupling trajectories signal maladaptive or resilient profiles. In the present study, we wished to advance current knowledge in infant neurodevelopment by modeling developmental change in EEG-derived delta–beta coupling. Additionally, we examined relations with infant temperament and maternal anxiety levels as a way to examine individual and contextual relations previously linked to the development of emotion regulation (Suurland et al. 2017).

The extant literature suggests that moderate, positive delta–beta coupling reflects adaptive, regulated states (Myruski et al. 2022; Riddle et al. 2021), while both heightened coupling and decoupled delta–beta activity may indicate maladaptive states of overcontrol and dysregulation, respectively (Miskovic and Schmidt 2009; Ramos et al. 2024). Indeed, most studies reporting stronger coupling in high-risk groups also report significant positive coupling in low-risk groups, albeit of lower or moderate magnitude (Phelps et al. 2016; Poole and Schmidt 2019). Moderate coupling may be indicative of efficient coordination between subcortical and cortical systems, necessary for adaptive regulation, without becoming overly rigid. However, most of these studies have examined and compared delta–beta correlations between groups (e.g., high vs. low anxiety), which provides little information regarding an individual's degree of connectivity between subcortical and cortical systems that delta–beta coupling has been theorized to capture. Understanding thresholds of coupling within any one person will help us contextualize delta–beta coupling patterns as adaptive or maladaptive.

Delta–beta coupling can be investigated at the individual level by using the full time series of EEG delta and beta power to compute an individual's coupling score (Brooker et al. 2021; Poole et al. 2020; Poppelaars et al. 2018). Additionally, intraindividual coupling scores can also be computed by modeling deviations in the delta and beta power time series from the average delta and beta power scores (Anaya, Vallorani, et al. 2021). Studies of individual delta–beta coupling have reported different associations with anxiety (Poppelaars et al. 2018) and temperament (Anaya, Vallorani, et al. 2021; Poole et al. 2020) compared to results from group-level studies, suggesting that the level of analysis can shift observed patterns. Individual coupling scores may provide more nuanced information about the level of connectivity between cortical and subcortical systems over time, increasing predictive power in our studies.

1.1 | Delta–Beta Coupling and Infant Temperament

Infant temperament and emotion regulation have been conceptualized as interrelated processes, where temperamental predispositions may capture early tendencies in emotion reactivity and regulation (Rothbart et al. 2006). Emotion reactivity appears earlier and, over time, is thought to be modulated by later emerging regulatory processes that become increasingly more efficient and complex, and this interplay may tether emotion regulation trajectories (Rothbart et al. 2011). For example, higher levels of infant negative affect—a temperament profile characterized by higher fear and inhibition—have been associated with limited exposure to novel environments (Almas et al. 2011) and fewer positive caregiver–infant interactions (Kim et al. 2014), which may, in turn, distinctly shape the development of emotion regulation and associated neural circuitry.

Indeed, fearful and inhibited temperament has been associated with heightened positive delta–beta coupling in childhood. For example, toddlers characterized by dysregulated fear at age 2 exhibited heightened delta–beta coupling at age 4 (Phelps et al. 2016). Similarly, children 9–12 years old characterized as behaviorally inhibited exhibited heightened delta–beta coupling compared to noninhibited peers (Poole et al. 2020). Fearful temperament is a robust, early risk factor for anxiety (Clauss and Blackford 2012); thus, several studies have also linked heightened delta–beta coupling to higher anxiety in children 8 years old (Poole and Schmidt 2019), preadolescents 9–12 years of age (Anaya, Vallorani, et al. 2021), and adults (Knyazev 2011; Knyazev et al. 2006). Together, these findings suggest that heightened delta–beta coupling may be associated with neural mechanisms of overcontrol, which usually characterize anxiety phenotypes and fearful temperament profiles later in childhood and adolescence (Lamm et al. 2014; White et al. 2011). However, no study has examined the link between delta–beta coupling, fearful temperament, and anxiety risk over time. Infancy captures a window of rapid changes in neural mechanisms, which may increase sensitivity to anxiety levels in the proximal environment. Concomitantly, temperamental fear crystalizes into more stable, individual patterns for some infants. Thus, investigating delta–beta coupling during infancy may prove critical for understanding early disruptions in emotion processing and psychopathology risk.

1.2 | Delta–Beta Coupling and Maternal Anxiety

The link between heightened delta–beta coupling and anxiety in adults suggests that infant delta–beta coupling trajectories may vary as a function of caregiver anxiety levels. Maternal anxiety is associated with self-reports of lower responsiveness (Nicol-Harper et al. 2007), lower warmth, and higher maternal hostility (Seymour et al. 2015), which may hinder caregiving interactions critical for neurodevelopment (Luby et al. 2020) and emotion socialization (Crugnola et al. 2016). Therefore, maternal anxiety may shape the neural mechanisms of emotion regulation during infancy, which may be reflected in distinct trajectories of infant delta–beta coupling. In a pilot study (n = 16), Miskovic et al. (2011) reported significantly stronger delta–beta coupling in children and adolescents (7–14 years) of parents with social

phobia compared to no social phobia. However, to our knowledge, no study has reported on delta–beta coupling as a function of maternal anxiety during infancy. Maternal anxiety can change between 3 and 24 months postpartum (Agrati et al. 2015) and may only carry risk to infant neural development with increasing levels over time. Thus, we examined maternal anxiety trajectories over the first 2 years postpartum to capture links with infant delta–beta coupling over time.

1.3 | The Present Study

The present study leveraged repeated measures of infant resting-state EEG at 8, 12, 18, and 24 months to model trajectories of intraindividual delta–beta coupling. We examined (1) the contributions of individual- and visit-level differences to the overall variance in delta–beta coupling, along with growth models of delta and beta power individually to contextualize potential visit-level differences, and (2) delta–beta coupling trajectories as a function of infant age, infant negative affect, and maternal anxiety over time.

In line with our recent psychometric study of average delta–beta coupling in this sample (Anaya, Ostlund, et al. 2021), we predicted that intraindividual delta–beta coupling would be stable at the Frontal region, and its variance would be attributed to individual-level differences. While changes in the Frontal lobe during infancy and developmental gains in regulation abilities may suggest that delta–beta coupling should increase over time, at least one previous study modeling delta–beta coupling trajectories in children from age 3 to 5 failed to show such increases over time (Brooker et al. 2021). Based on our previous publication, we also predicted that intraindividual delta–beta coupling would decrease over time at the Central and Parietal regions and that variance here would be primarily attributed to visit-level differences. Our previous publication (Anaya, Ostlund, et al. 2021) only focused on average coupling scores and cross-sectional correlations with infant temperament. The current paper expands on this previous publication by adding an assessment at 24 months, modeling intraindividual delta–beta coupling, and investigating infant temperament and maternal anxiety over time.

Based on previous studies of delta–beta coupling and fearful temperament (Phelps et al. 2016; Poole et al. 2020), we hypothesized that higher infant negative affect over time would be associated with higher and more stable delta–beta coupling trajectories. Similarly, based on previous studies of anxiety risk and delta–beta coupling (De Pascalis et al. 2020; Poole and Schmidt 2019), we hypothesized that higher maternal anxiety over time would be associated with higher and more stable delta–beta coupling trajectories.

Finally, we conducted exploratory analyses that may help us understand associations between infant negative affect and maternal anxiety with delta–beta coupling. First, based on previous studies showing that maternal anxiety levels can be exacerbated by infant negative affect (Agrati et al. 2015; Brooker et al. 2015), we tested whether the interaction between these variables would predict delta–beta coupling trajectories. Additionally, given the conceptualization of infant temperament as early tendencies in reactivity and regulation, we tested whether maternal reports of infant regulation moderated any associations between infant negative affect and delta–beta coupling.

2 | Materials and Method

2.1 | Participants

Participants were enrolled (N = 357) as part of a large-scale study of attention, neural, and socioemotional development (Pérez-Edgar et al. 2021). The Institutional Review Boards at the Pennsylvania State University and Rutgers University approved all procedures. Parents provided written consent and were compensated for their participation.

Families visited the lab when infants were 4, 8, 12, 18, and 24 months of age. Questionnaire data were collected at all five visits, while EEG data were only collected at the last four visits. From the total enrolled sample, 192 infants contributed EEG data at 8 months, 133 at 12 months, 103 at 18 months, and 65 at 24 months. Data collection was disrupted due to COVID-19 mitigation, resulting in higher rates of missing data for the later assessments.

Caregivers identified 18 of the infants (10.9%) as African American/Black, nine (5.4%) as Asian, 32 (19.4%) as Latinx, 101 (61.2%) as White, three (1.8%) as Native American, and two (1.2%) as mixed race. Across the sample, 84 caregivers (50.9%) reported an income above $60,000, and most mothers (N = 100) and fathers (N = 84) had a college education. Demographic details are presented in Table 1.

2.2 | Procedures

Most families completed data collection in two 2-h visits to the lab, and EEG data were usually collected during the second visit. Questionnaires were primarily completed online via Qualtrics (Qualtrics, Provo, UT) and only by one caregiver (95% biological mothers). Questionnaire details and psychometric properties are reported in Table S1.

2.3 | Measures

2.3.1 | Resting-State EEG

Infant EEG was continuously recorded at 32 Ag/AgCl active scalp electrodes (Brain Products actiCAP) during a resting-state task. Caregivers sat their infants on a highchair and were instructed to remain neutral during the task. EEG collection was divided into four 1-min blocks of "lights-on," "lights-off" to imitate eyes-open and eyes-closed conditions (e.g., Degnan et al. 2011). Infants watched a low-contrast, muted video (i.e., cave navigation video or Wall-E video) on a 17″ computer screen. Impedances were kept below 10 kΩ. EEG was amplified and digitized at 500 Hz. Data were preprocessed using the Harvard Automated Processing Pipeline in Low Electrode Encephalography (HAPPILEE; Lopez et al. 2022). Preprocessing included 1-Hz high-pass filtering, 100-Hz low-pass filtering, offline re-referencing to the average of all channels, removal of 60-Hz line noise, bad channel detection

TABLE 1 | Demographic characteristics at time of enrollment.

Characteristic	Mean (*SE*) or % (*n*)
Parent-reported child sex	
Boys	49% (81)
Girls	51% (84)
Mother education	
No high school diploma	8.48% (12)
High school diploma	20.61% (34)
College graduate or higher	70.91% (117)
Father education	
No high school diploma	6.06% (10)
High school diploma	29.70% (49)
College Graduate or Higher	59.39% (98)
Married or cohabiting parents	78.79% (130)
Child care type	
Parent (home)	53.94% (89)
Daycare center	31.52% (52)
Home daycare	7.88% (13)
Nanny or au pair	6.66% (11)
Childcare days if enrolled (days/week)	3.21 (1.41)
Household income	
≤$30,000	22.42% (37)
$31,000–$60,000	26.06% (43)
≥$60,000	46.67% (77)

Note: Family demographics were collected at time of enrollment.

and interpolation, and artifact correction. Artifact correction was carried out via the wavelet transform, a method that is optimal for low-density infant EEG and outperforms ICA methods in infant samples (Lopez et al. 2022). Data were then excluded ($N = 182$) based on the cross-correlation before and after wavelet transformation ($r < 0.15$) in line with recommendations for infant EEG. Data were also excluded due to technical issues ($N = 24$) and insufficient number of segments ($N = 12$). This resulted in the final sample included in the current analyses ($N_8 = 96$, $N_{12} = 66$, $N_{18} = 61$, $N_{24} = 51$). Infants included in the analysis did not differ from infants with missing EEG data on ethnicity, sex assigned at birth, birth weight, or daycare arrangement ($ps > 0.22$). Further, these infants did not differ on the IBQ Negative Affect factor at any of the visits ($ps > 0.09$). However, mothers of infants with missing EEG data were more likely to report higher BAI scores at the 4-month visit ($t(214) = -3.3957$, $p = 0.001$), as well as lower education ($t(380) = 2.6483$, $p = 0.008$) and income ($t(371) = 3.262$, $p = 0.001$) at enrollment. Data quality metrics are reported in Table S2. Data were segmented at 1-s intervals, and the spectral power of delta (1–2.5 Hz) and beta (11–18 Hz) frequency bands was generated via the *genPower* script. Power values were then exported for additional processing in R v4.4.1 (R Core Team 2024).

We exported second-by-second delta and beta relative power for each participant at each assessment. Previous studies of individual delta–beta coupling have used a similar approach, exporting segments at the level of 4 or 20 s (Harrewijn et al. 2016; Poole et al. 2020). Participants with less than 10 segments were excluded from the analyses. Epoch-specific power values across target electrodes were log transformed and then averaged to create composites for the Frontal (F3, F4), Central (C3, C4), and Parietal (P3, P4) regions based on the 10–20 System of Electrode Placement (Herwig et al. 2003).

2.3.2 | Infant Behavior Questionnaire—Revised (IBQ-R)

Infant negative affect and regulation at 4, 8, and 12 months were operationalized via the Negative Affect and the Regulation factors, respectively, from the IBQ-R, a 191-item survey designed to assess temperament-linked emotional and behavioral tendencies in infancy (Putnam et al. 2014). Parents rated infant behavior using a 7-point scale ranging from 1 = *Never* to 7 = *Always*. The Negative Affect factor consisted of averaged scores across the Fear, Distress to Limitations, Sadness, and Falling Reactivity subscales, while the Regulation factor consisted of averaged scores across the Duration of Orienting, Cuddliness, Soothability, and Low-Intensity Pleasure subscales. Reliabilities for our sample were good across time (Cronbach's α range: 0.728–0.953).

2.3.3 | Toddler Behavior Assessment Questionnaire (TBAQ)

Infant negative affect and regulation at 18 and 24 months were operationalized via the Negative Affect and Regulation factors from the TBAQ, a 120-item survey designed to assess temperament in toddlers from maternal reports (Goldsmith 1996). Parents rated how often their toddler displayed a specific behavior in the past month using a 7-point Likert scale ranging from 1 = *Never* to 7 = *Always*. The TBAQ Negative Affect factor reflects the averaged scores across the Anger, Sadness, Object Fear, and Social Fear subscales, capturing similar underlying temperament tendencies as captured in the IBQ Negative Affect factor. Similarly, the Regulation factor reflects the averaged scores of the Attention Focusing, Low-Intensity Pleasure, Soothability, and Cuddliness subscale, capturing similar tendencies as the IBQ Regulation factor. Previous studies have established continuity between IBQ and TBAQ subscales (Carranza et al. 2013). Reliabilities in our sample were good across both assessments (Cronbach's α range: 0.612–0.850).

2.3.4 | Beck Anxiety Inventory (BAI)

Mothers reported on their anxiety levels using the BAI (Beck et al. 1988) at 4, 8, 12, 18, and 24 months. The BAI is a 21-item questionnaire designed to evaluate the severity of anxiety in healthy and psychiatric populations and to distinguish cognitive and somatic symptoms of anxiety from depression symptoms. Mother rated individual symptoms of anxiety (e.g., fear of losing control) in the past month using a 4-point Likert scale ranging from 0 = *Not at all* to 3 = *Severely*. Higher scores (range: 0–63) indicate greater symptom severity, with scores above 16 indicating moderate anxiety. Reliabilities for this sample were excellent across time (Cronbach's α range: 0.891–0.937).

2.3.5 | Covariates

We included parent-reported child sex, parent education, birth weight, and medication, alcohol, and drug use during pregnancy as covariates. Caregiver education was included as the mean of mothers' and fathers' education levels. Medication, alcohol, and drug use were binary variables of maternal endorsement of each behavior.

3 | Data Analysis

We first used three-level multilevel models to examine within-person delta–beta coupling and to determine the extent of variability in delta–beta coupling that may be attributed to visit and individual differences. Across all analyses, delta–beta coupling was modeled as the within-person association between delta and beta epochs, and variance in coupling was partitioned using models of the form

$$\text{Delta}_{ivt} = b_{0iv} + b_{1iv}\text{Beta}_{ivt} + e_{ivt}, \quad (1)$$

$$b_{0iv} = g_{00i} + r_{0iv}, \quad (2)$$

$$g_{00i} = P_{000} + u_{00i}, \quad (3)$$

$$b_{1iv} = g_{10i} + r_{1iv}, \quad (4)$$

$$g_{10i} = P_{100} + u_{10i}, \quad (5)$$

separate for the Frontal, Central, and Parietal regions. In these models, up to 240 epoch-specific observations of delta power obtained from individual i at visit v during epoch t are modeled as a function of a visit-specific intercept b_{0iv}, a visit-specific coupling coefficient b_{1iv}, and residual. Visit-specific coupling coefficients are modeled as deviations from individual-specific and sample-level means. The extent of deviation across visits and between individuals is given by the variance $\sigma^2{}_{r1}$ and $\sigma^2{}_{u0}$, and was quantified using intraclass correlations such that proportion of total variance accounted for at these levels was calculated as $\frac{\sigma^2_{r1}}{\sigma^2_{r1}+\sigma^2_{u10}}$, $\frac{\sigma^2_{u0}}{\sigma^2_{r1}+\sigma^2_{u10}}$ and interpreted as the extent of observed variance in delta–beta coupling that could be attributed to visit and individual differences.

These models were complemented by individual growth models for delta and beta power to examine reductions and increases in power previously reported for these bands (Clarke et al. 2001; Gómez et al. 2013).

Multilevel models were then reconfigured such that within-person associations between delta and beta power were moderated by continuous infant age (in months). This model was reconfigured with an intercept of zero, in order to keep the focus on coupling. Covariates of interest were then added to the models. Zero-order correlations between outcome variables, predictors, and covariates are reported in Table 2.

Finally, we examined whether delta–beta coupling trajectories were moderated by infant negative affect and maternal anxiety over time. We first used a growth model to examine Negative Affect trajectories. Negative affect increased over time ($\gamma_{10} = 0.064, SE = 0.010, p = 0.001$), in line with previous literature (Gartstein et al. 2018). To capture this change, we extracted the random intercept and slope parameters for each participant to use as predictors of delta–beta coupling. Similarly, we used a growth model to examine maternal anxiety trajectories. Maternal anxiety did not significantly change over time ($p = 0.521$). Therefore, BAI scores were averaged over time, and we used these composite scores as predictors of delta–beta coupling. In a set of exploratory analyses, we also examined the interaction between infant negative affect and maternal anxiety, as well as the role of infant regulation using the IBQ Regulation factor in moderating the effect of infant negative affect on delta–beta coupling. Details for exploratory models and results are provided in Supporting Information S1.

All models were fitted with the *lme4* package in R (Bates et al. 2015). Missing data were accommodated using restricted maxi-

TABLE 2 | Zero-order correlations between study variables.

	M (*SD*)	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Negative affect intercept	−0.01 (0.48)	—											
1. Negative affect slope	0.00 (0.05)	**−0.22**	—										
1. Average maternal anxiety	6.03 (5.93)	**0.23**	−0.13	—									
1. Infant birth weight (lbs.)	7.52 (1.05)	−0.04	−0.02	−0.08	—								
1. Parent education (years)	16.34 (2.93)	−0.10	0.00	**−0.16**	0.08	—							
1. Daycare (days/week)	1.92 (1.91)	−0.11	0.06	−0.06	0.12	**0.34**	—						
1. Average income	5.36 (2.22)	**−0.24**	0.08	−0.13	**0.24**	**0.64**	**0.39**	—					
1. Average Frontal delta	0.00 (0.56)	0.00	0.05	0.14	0.01	−0.07	0.07	−0.09	—				
1. Average Central delta	0.00 (0.50)	−0.10	0.13	−0.08	0.00	0.03	0.06	0.00	0.04	—			
1. Average Parietal delta	0.00 (0.51)	0.00	0.09	**0.19**	0.02	−0.02	−0.10	−0.12	0.10	−0.08	—		
1. Average Frontal beta	0.00 (0.41)	0.03	0.06	−0.01	0.05	−0.14	−0.11	−0.14	−0.02	0.02	0.07	—	
1. Average Central beta	0.00 (0.37)	−0.06	0.16	0.08	0.00	0.05	**0.19**	0.07	−0.03	0.09	0.12	0.06	—
1. Average Parietal beta	0.00 (0.49)	0.07	−0.02	−0.05	−0.11	−0.02	−0.05	0.07	−0.06	−0.04	−0.02	−0.16	**−0.17**

Note: Average variables indicate the mean across assessments between 4 and 24 months. Delta and beta variables are the average delta and beta power deviations. Parent education indicates the mean between mother and father education level. Significant correlations ($p < 0.05$) are indicated in bold.

TABLE 3 | Variance decomposition in delta–beta coupling at two levels: Persons and visits.

	Delta–beta coupling		
	Frontal	**Central**	**Parietal**
Intercept, π_{000}	5.014*	3.786*	4.256*
Person variance, σ^2_{u00}	0.845	0.632	0.772
Visit variance, σ^2_{r0}	0.158	0.227	0.147
Residual variance, σ^2_e	0.308	0.257	0.257
Coordination, π_{100}	0.019*	0.101*	0.070*
Person variance, σ^2_{u10}	0.001 (1.3%)	0.010 (26.6%)	0.005 (28%)
Visit variance, σ^2_{r1}	0.028 (98.7%)	0.028 (73.4%)	0.012 (72%)
Total variance coordination, $\sigma^2_{r1} + \sigma^2_{u10}$	0.029	0.038	0.017

Note: Models based on up to 240 epochs per person, nested within 274 visits, nested within 165 persons. Parameter estimates from three-level bivariate models for delta–beta coupling across Frontal, Central, and Parietal regions. Percentages of total variance in coupling given in parenthesis for visit and person levels.
*$p < 0.001$.

mum likelihood (REML) in these models, which provides bias correction of parameter estimates in linear mixed-effects models with case-wise deletion of missing data (Kenward and Roger 1997; Vallejo et al. 2004). Statistical significance was evaluated at $\alpha = 0.05$ and FDR-corrected across all multilevel models. Only FDR-corrected *p*-values are reported. Higher order interactions were trimmed when nonsignificant. Significant interactions were probed using regions of significance analysis with the *interactions* package (Long 2019).

4 | Results

4.1 | Variance in Intraindividual Delta–Beta Coupling Across Assessments

Multilevel models of delta–beta coupling variance decomposition are presented in Table 3. Average delta–beta coupling at the Frontal region was positive ($\pi_{100} = 0.226$, $SE = 0.012$, $p = 0.001$), suggesting that increases in epoch-centered deviations of delta power were significantly associated with increases in beta power. Variance at the Frontal region was almost entirely attributed to visit-level differences (95.5%, $\sigma^2_{u10} = 0.026$), suggesting that individual-level factors played virtually no role in explaining intraindividual delta–beta coupling at the Frontal region.

Average delta–beta coupling at the Central region was also positive ($\pi_{100} = 0.217$, $SE = 0.013$, $p = 0.001$), and variance was majorly attributed to visit-level differences (87.2%, $\sigma^2_{r1} = 0.024$), with only 12.8% ($\sigma^2_{u10} = 0.003$) attributed to individual-level differences.

Average delta–beta coupling at the Parietal region was positive ($\pi_{100} = 0.248$, $SE = 0.005$, $p = 0.001$). Variance at this region was primarily attributed to visit-level differences (79%, $\sigma^2_{r1} = 0.009$), with only 21% ($\sigma^2_{u10} = 0.003$) attributed to individual-level differences.

Growth models of delta and beta power indicated that delta power did not change over time at the Frontal region ($\gamma_{10} = 0.001$, $SE = 0.004$, $p = 0.760$), but significantly decreased over time at the Central ($\gamma_{10} = -0.015$, $SE = 0.005$, $p = 0.003$) and Parietal ($\gamma_{10} = -0.013$, $SE = 0.005$, $p = 0.002$) regions. Beta power did not change over time across any region (all *p*s > 0.892). Individual trajectories of delta and beta power are depicted in Figure 1A,B.

4.2 | Developmental Trajectories of Infant Delta–Beta Coupling

Intraindividual delta–beta coupling systematically increased from 8 to 24 months at the Frontal region ($\gamma_{20} = 0.006$, $SE = 0.001$, $p = 0.001$). At this region, the prototypical infant exhibited an intraindividual delta–beta coupling score of 0.139 at 8 months, and for every 1-month increase in age, delta–beta coupling increased by 0.006.

In contrast, intraindividual delta–beta coupling systematically decreased as a function of age at the Central ($\gamma_{20} = -0.005$, $SE = 0.001$, $p = 0.001$) and Parietal ($\gamma_{20} = -0.004$, $SE = 0.001$, $p = 0.001$) regions. At the Central region, the prototypical infant exhibited a delta–beta coupling score of 0.302 at 8 months, and for every 1-month increase in age, delta–beta coupling decreased by 0.005. Similarly at the Parietal region, the prototypical infant exhibited a delta–beta coupling score of 0.296 at 8 months, which decreased every month at a rate of 0.004. Figure 2 illustrates dynamic, second-by-second changes in intraindividual delta and beta power for one participant within and across assessments to illustrate increases or decreases in coupling over time.

Covariates were then regressed on these models. Infant birthweight, pregnancy smoking, parent education, and child biological sex were significant covariates and retained in all follow-up analyses.

4.3 | Conditional Effect of Infant Negative Affect Over Time

Infant negative affect was not significantly associated with delta–beta coupling trajectories at the Frontal or Central regions (*p*s > 0.933).

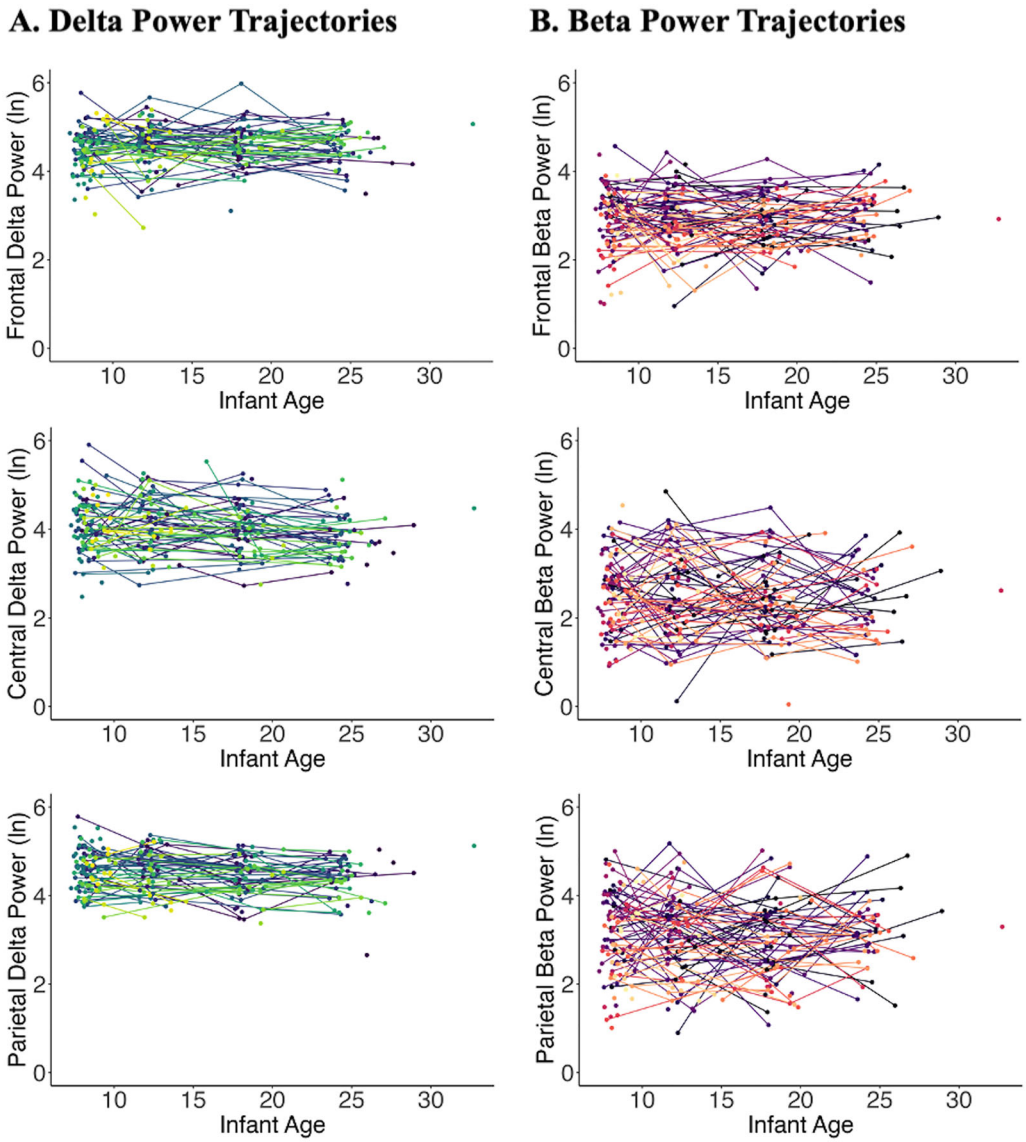

FIGURE 1 | Trajectories of delta (A) and beta (B) power.

At the Parietal region, the slope of infant negative affect significantly predicted delta–beta coupling over time ($\gamma_{150} = -0.052$, $SE = 0.018$, $p = 0.032$). Regions of significance analysis (Figure 3) indicated that infants with stable (Slope = 0) and increasing (Slope > 0.059) negative affect trajectories exhibited a steeper rate of decrease in delta–beta coupling compared to infants with decreasing (Slope < −0.059) negative affect trajectories.

4.4 | Conditional Effect of Maternal Anxiety Over Time

There was no main effect of average maternal anxiety on delta–beta coupling trajectories across any of the cortical regions (*p*s > 0.968), and the effect of infant negative affect slope at the Parietal region remained significant in these models.

4.5 | Exploratory Analyses

4.5.1 | Interaction Between Infant Negative Affect and Maternal Anxiety

At the Parietal region, trajectories of delta–beta coupling significantly varied as a function of the interaction between average maternal anxiety levels over time and infant negative affect slope ($\gamma_{220} = -0.006$, $SE = 0.002$, $p = 0.014$). We probed this interaction with regions of significance analyses at decreasing ($M_{\text{Slope}} < -0.059$; $n = 23$), stable ($M_{\text{Slope}} = 0$; $n = 94$), and increasing negative affect trajectories ($M_{\text{Slope}} > 0.059$; $n = 28$) in tandem with −1 *SD*, mean, and +1 *SD* of average maternal anxiety levels. Results indicated that maternal anxiety was associated with delta–beta coupling trajectories only for infants with decreasing negative affect trajectories ($\beta = 0.012$, $p = 0.037$).

As illustrated in Figure 4, when mothers reported typically lower (−1 *SD* = −0.537) levels of anxiety across time, Parietal delta–beta coupling gradually decreased over time, similar to

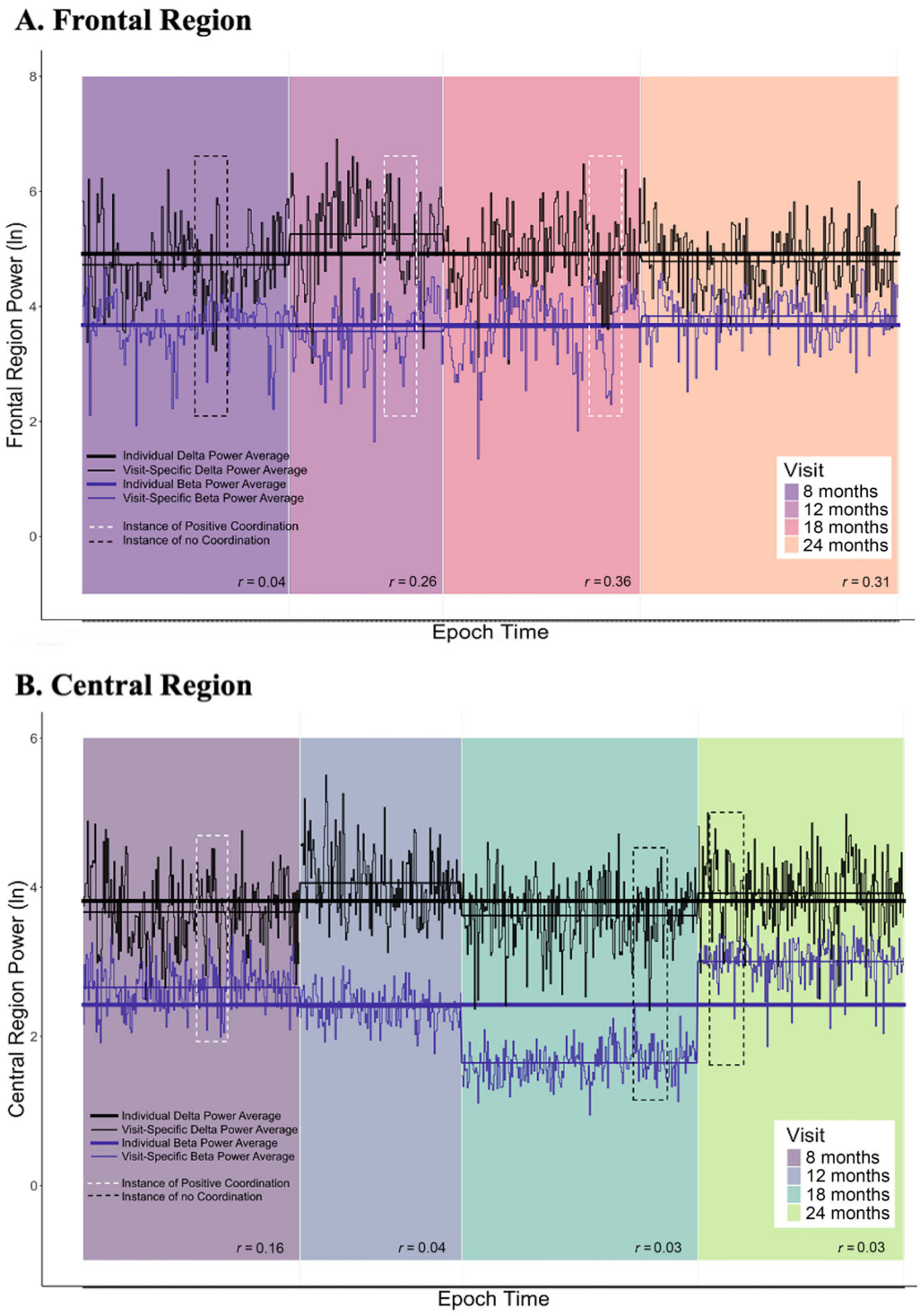

FIGURE 2 | Dynamic, second-by-second changes in delta and beta power at Frontal (A) and Central (B) regions for a single participant in the context of developmental change between 8 and 24 months. Selected windows indicate instances when increases or decreases in delta power from the individual's visit-specific delta average were met by similar increases or decreases in beta power (white dash) or were not coordinated (black dash).

the sample average trajectory. However, when mothers reported average ($M = 0$) levels of anxiety over time, Parietal delta–beta coupling remained stable over time. Finally, when mothers reported generally higher (+1 $SD = 0.537$) anxiety levels over time, Parietal delta–beta coupling increased over time, a pattern most divergent from the average decrease seen for the sample.

4.5.2 | Moderating Effect of Infant Regulation

Growth models of the IBQ Regulation factor indicated that infant regulation significantly decreased over time (Intercept = 5.089, $SE = 0.033$, $p = 0.001$; Slope = −0.131, $SE = 0.018$, $p = 0.001$). Here too, we extracted random intercept and slope values to include

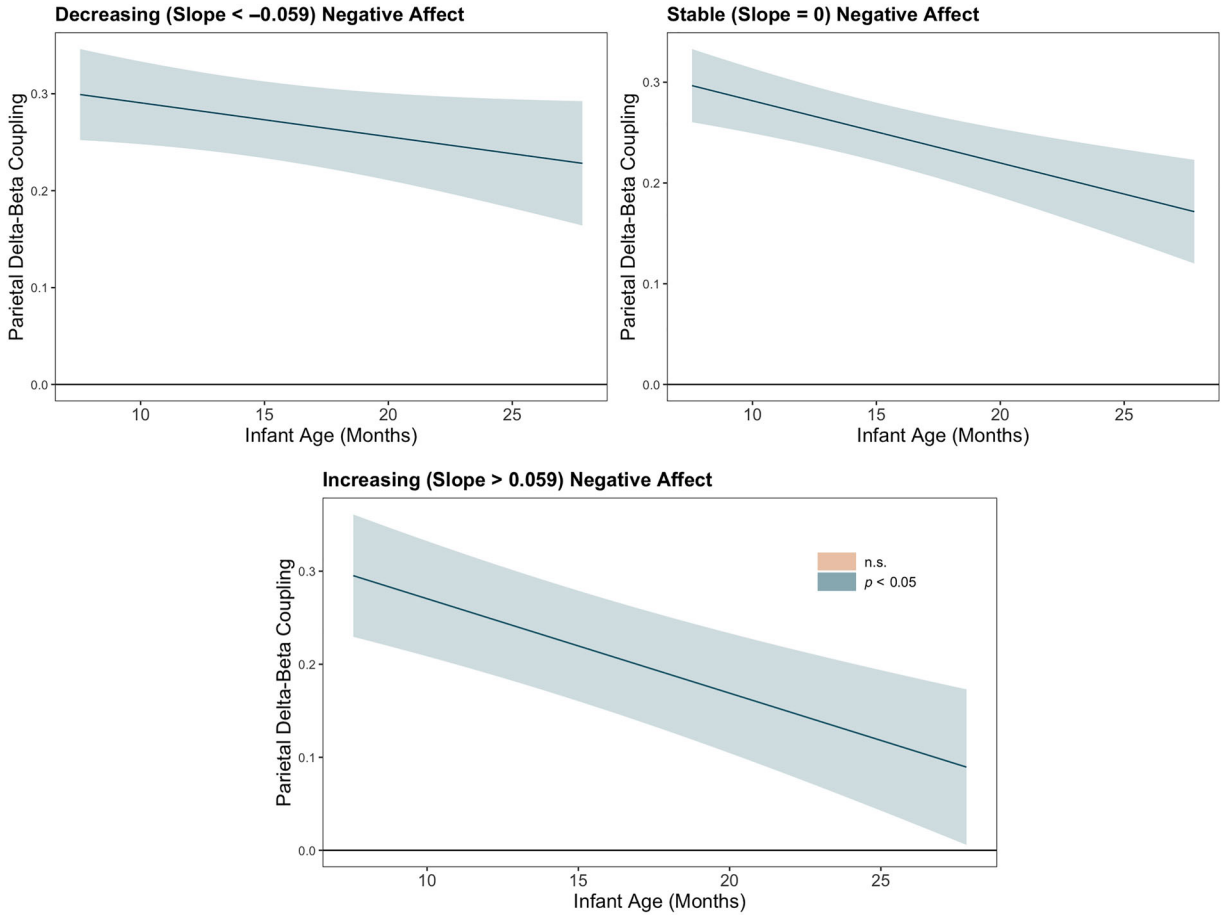

FIGURE 3 | Parietal region intraindividual delta–beta coupling trajectories as a function of decreasing, stable, and increasing infant negative affect over time.

in the multilevel models. The intercept of infant regulation (i.e., 4-month IBQ Regulation scores), but not the slope, significantly interacted with the intercept of infant negative affect to predict 8-month Parietal delta–beta coupling ($\gamma_{110} = 0.399$, $SE = 0.002$, $p = 0.015$). Regions of significance analysis (Figure S1) indicated that delta–beta activity at 8 months was decoupled for infants with low levels of regulation and the highest levels of negative affect at 4 months.

5 | Discussion

In the present study, we used repeated measures of EEG delta–beta coupling during infancy to examine (1) person-level and visit-level contributions to variance in intraindividual delta–beta coupling, (2) developmental change in delta–beta coupling from 8 to 24 months, and (3) whether delta–beta coupling trajectories systematically varied as a function of infant temperament and maternal anxiety over time. Several important findings emerged.

Our results first indicate that delta–beta coupling between 8 and 24 months was positive, moderate, and comparable across cortical regions—a finding that aligns with previous studies in older children (Brooker et al. 2016; Phelps et al. 2016). Our study expands on this literature by providing average, individual coupling levels ($r = 0.217$–0.248) that may help set thresholds for normative or typical coupling levels during infancy. In doing so, we hope to inform future studies in better contextualizing when over-coupling or under-coupling patterns are present in individuals. Indeed, a better understanding of atypical and typical coupling levels is necessary for advancing the use of delta–beta coupling as a potential target in neurofeedback treatments of psychiatric conditions.

We found virtually no individual-specific variance at the Frontal region ($\sigma^2 = 0.5\%$), in contrast to previous findings of significant group differences in Frontal coupling as a function of trait-like markers (Phelps et al. 2016; Poole and Schmidt 2019). It is possible that individual differences in Frontal delta–beta coupling emerge later in infancy. Alternatively, it is possible that these results are specific to our intraindividual approach, which yielded lower coupling levels compared to group-level studies ($r = 0.40$–0.60), potentially reflecting differences in how we may capture biological functioning in individuals versus groups. Future research using this approach to compare delta–beta coupling in adults and children may expand our understanding of trait-like factors and Frontal coupling.

We also found that intraindividual coupling at the Frontal region was positive and gradually increased over time, while Central and Parietal coupling for the prototypical infant was positive and moderate at 8 months, then gradually decreased over time. These

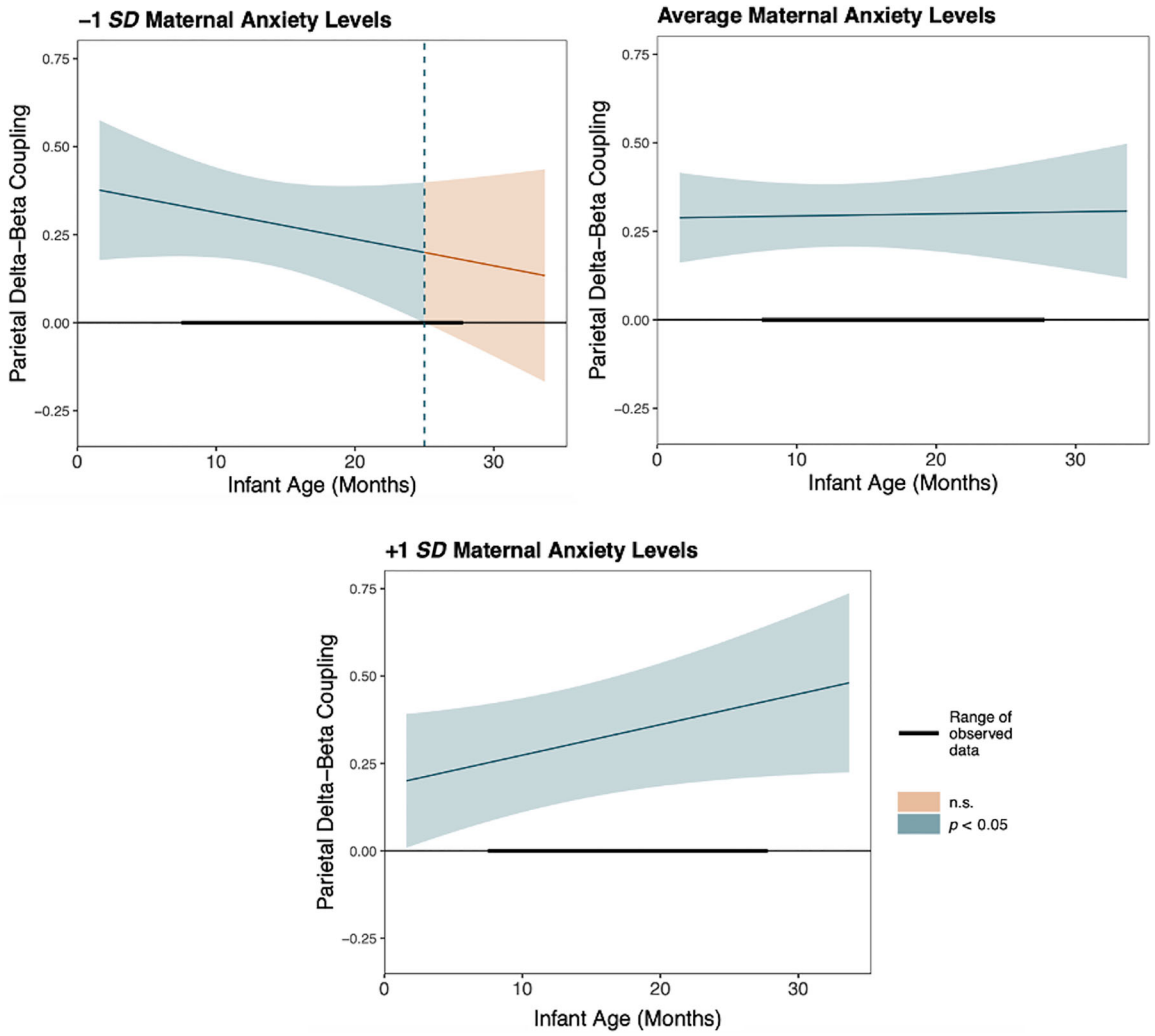

FIGURE 4 | Intraindividual delta–beta coupling at the Parietal region for infants with decreasing negative affect trajectories as a function of maternal anxiety levels over time.

developmental patterns indicate that the source of visit-to-visit influences on delta–beta coupling across regions is, in part, due to general, age-related changes in cognitive function. These patterns partially replicate the mean changes between 8 and 18 months we previously reported for this sample's average coupling scores (Anaya, Ostlund, et al. 2021), supporting some consistency between intraindividual dynamics and interindividual coupling.

Developmental change in delta–beta coupling across regions noted here aligns with Brooker et al. (2021), who reported that person-specific profiles of delta–beta coupling significantly changed from ages 3 to 5 years, and developmental changes also differed across regions. Our study joins Brooker et al. (2021) to begin piecing together the developmental course of delta–beta coupling from infancy through childhood, showing early dynamic changes and potential malleability of this neural process. The gradual decrease in coupling at the Central and Parietal regions complemented by increases in coupling at the Frontal region aligns with neurodevelopmental accounts of parietal-to-frontal maturation (Gogtay et al. 2004), which suggest that posterior functioning involved in regulation progressively shifts to more anterior structures as a function of age.

Interestingly, these developmental patterns were not driven by trajectories of delta or beta power, with the latter being stable over time across all regions. While some previous studies report increases in beta power in tandem with decreases in delta power over time, these studies have included older children or cross-sectional samples (Clarke et al. 2001; Ogawa et al. 1989; Somsen et al. 1997), suggesting that changes in delta and beta power may emerge after infancy. At least one previous study reported stable trajectories of delta power at the Frontal region in typically developing infants (Tierney et al. 2012), similar to our results. Another study reported increases in beta power during infancy (Wilkinson et al. 2024). However, it is worth noting that this pattern only emerged at the Central region, and not at the Frontal or Posterior regions, partially aligning with our results. Furthermore, Wilkinson et al. (2024) included a large sample of infants ($N = 592$) who were followed later into 36 and 44 months. Thus, it is possible that our smaller sample size and final assessment at 24 months may have limited our ability to detect a linear increase in beta power at the Central region.

Together, these results suggest that intraindividual coupling captures dynamic coordination between changes in the time series of delta and beta power, and this coupling seems to be inde-

pendent of average power and to follow different developmental trajectories. This concept aligns with previous investigations of delta–beta coupling showing that groups who differ in delta–beta coupling (e.g., high vs. low anxiety) do not differ in their average delta or beta power (Poole and Schmidt 2019). These findings support the functional significance of delta–beta coupling as a neural marker that is modulating across development, potentially reflecting current state-level functioning. Further, the presence of lawful variation suggests a metric that may be sensitive to intervention and modification, and holds potential as a future target in treatments of psychiatric disorders, such as neurofeedback.

When we examined the effect of infant negative affect, we found that opposite to our hypothesis, more stable and increasing negative affect over time predicted accelerated decreases in Parietal coupling. Heightened delta–beta coupling, conceptualized as a propensity for overcontrol or hyperresponsiveness, has been previously associated with fearful temperament profiles (Phelps et al. 2016; Poole et al. 2020). Instead, our findings suggest that during infancy, increasing negative affect is associated with decoupling of delta–beta activity, which may indicate disruptions in the downregulation of the limbic system (Riddle et al. 2021). Notably, previous studies focused on dysregulated fear and behavioral inhibition, which are unique manifestations of extreme and stable fear patterns. Thus, it is possible that our discrepant findings are a result of using the IBQ Negative Affect factor, which captures broader aspects of negative emotionality (e.g., distress, sadness) rather than temperamental fear specifically. However, parallel analyses with the IBQ Fear subscales (Supporting Information S1.2) indicated that delta–beta coupling trajectories did not vary as a function of maternal reports of infant fear. Thus, our results indicate that more comprehensive aspects of negative emotionality, potentially signaling broad disruptions in emotion processing during infancy, may better track the interaction between subcortical and cortical systems involved in regulation this early in infancy, rather than maternal reports of fear specifically. Alternatively, the lack of association with infant fear may be a factor of our individual approach, since previous studies of fearful temperament and delta–beta coupling have only examined group-level coupling (Brooker et al. 2016; Phelps et al. 2016), while more recent studies using an individual approach also report no associations with IBQ fear (Brooker et al. 2021).

Concomitantly, by capturing associations between negative affect and delta–beta coupling this early in infancy, we may be modeling broad neural risk that has yet to morph into specific patterns of overcontrol or undercontrol. Indeed, both perspectives have been argued to underly disruption in emotion processing (Eisenberg et al. 2001; Murray and Kochanska 2002), and higher infant negative affect has been shown to predict higher problems across internalizing, externalizing, and dysregulation domains (Behrendt et al. 2020). Further, associations between negative affect and delta–beta coupling may also differ between resting-state and task-based EEG. Resting-state EEG correlates may reflect infants' *dispositions*, or how they process and react to the environment (Coan and Allen 2004), while task-based correlates may reflect infants' *capabilities*, or how they respond to challenge or stress in the moment (Coan et al. 2006). Thus, delta–beta coupling profiles derived from task-based EEG may further elucidate when this neural pattern reflects overcontrol or undercontrol. Future studies that model individual delta–beta coupling in tandem with emotion regulation EEG tasks and later-emerging psychopathology risk may shed light on the timing and specificity of associations.

We also observed that higher average levels of maternal anxiety between 4 and 24 months were associated with infants' increasing Parietal delta–beta coupling trajectories, but this effect was specific to infants who displayed decreasing negative affect. This moderation seems counterintuitive to the well-documented phenotypic continuity between infant negative reactivity, fearful temperament, and anxiety risk (Clauss and Blackford 2012; Kagan et al. 1984; Fox et al. 2001). However, upon further inspection, this group of infants exhibited higher negative affect at 4 months (i.e., intercept), and decreases over time did not necessarily translate into lower negative affect levels compared to the sample average at 24 months. Thus, this interaction may align with previous studies showing that higher maternal anxiety in tandem with higher negative affect during infancy increases risk for psychopathology, captured by heightened, positive delta–beta coupling (De Pascalis et al. 2020; Poole and Schmidt 2019). This pattern may signal potential disruptions in neural systems of emotion regulation toward overcontrol tendencies. However, this interaction should be interpreted with caution and replicated in future studies given that only a small subsample exhibited this pattern ($n = 23$). Additionally, future studies may wish to examine this association in tandem with measures of mother–infant interactions to understand mechanistic pathways from early anxiety risk to delta–beta coupling.

Our findings should be interpreted considering several limitations. First, infant emotion regulation was not directly measured. Instead, we relied on infant temperament, tracking early tendencies in emotion reactivity and regulation. Future studies should assess infant delta–beta coupling and observations of emotion regulation behaviors. Second, maternal anxiety and infant temperament were measured via maternal reports, potentially increasing shared method variance. Relatedly, because caregivers reported on infant temperament, our findings should be interpreted in the context of caregivers' perceptions of their infants. Third, we relied on resting-state EEG to assess delta–beta coupling in line with previous studies. However, future studies that can elicit changes in infant emotion and emotion regulation while collecting EEG may further elucidate how delta–beta coupling is implicated in propensities for overcontrol and psychopathology risk. Finally, we did not assess childhood socioemotional outcomes, limiting our interpretation of delta–beta coupling trajectories and risk or resilience for psychopathology.

In conclusion, the present study expands the current literature by modeling developmental change in delta–beta coupling during infancy and showing that infant temperament and maternal anxiety predict systematic differences in delta–beta coupling trajectories. Indeed, this study is the first to examine how delta–beta coupling trajectories may be modulated by anxiety levels in the infants' proximal environment and suggests that, for some infants, higher maternal anxiety levels during the postpartum period may lead to early tendencies for neural mechanisms of overcontrol. Our findings inform the functional significance of delta–beta coupling at the level of the individual and highlight the importance of modeling maternal anxiety and infant temperament across the postpartum period.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.

Supplementary Materials: dev70087-sup-0001-SuppMat.pdf