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RESEARCH ARTICLE

Psychometric properties of infant electroencephalography: Developmental stability, reliability, and construct validity of frontal alpha asymmetry and delta–beta coupling

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Abstract

Resting-state electroencephalography (EEG) provides developmental neuroscientists a noninvasive view into the neural underpinnings of cognition and emotion. Recently, the psychometric properties of two widely used neural measures in early childhood—frontal alpha asymmetry and delta–beta coupling—have come under scrutiny. Despite their growing use, additional work examining how the psychometric properties of these neural signatures may change across infancy is needed. The current study examined the developmental stability, split-half reliability, and construct validity of infant frontal alpha asymmetry and delta–beta coupling. Infants provided resting-state EEG data at 8, 12, and 18 months of age ($N = 213$). Frontal alpha asymmetry and delta–beta coupling showed significant developmental change from 8 to 18 months. Reliability for alpha asymmetry, and alpha, delta, and beta power, individually, was generally good. In contrast, the reliability of delta–beta coupling scores was poor. Associations between frontal alpha asymmetry and approach tendencies generally emerged, whereas stronger (over-coupled) delta–beta coupling scores were associated with profiles of dysregulation and low inhibition. However, the individual associations varied across time and specific measures of interest. We discuss these findings with a developmental lens, highlighting the importance of repeated measures to better understand links between neural signatures and typical and atypical development.

KEYWORDS
delta–beta coupling, frontal alpha asymmetry, infant EEG, longitudinal data, psychometric properties, temperament

1 | INTRODUCTION

Electroencephalography (EEG) methods have blossomed over the last three decades as one of the most accessible techniques for psychologists and cognitive neuroscientists interested in infant brain functioning and development (Field et al., 2004; Mundy & Jarrold, 2010; Peltola et al., 2014). Although collecting EEG in infant populations poses real challenges, including task noncompliance, motion-related artifacts, and data attrition to name a few (Noreika et al., 2020), the benefits of this developmentally sensitive method far outweigh its relative disadvantages. The popularity of EEG methods in infant research is in part driven by the lower cost of EEG equipment and processing compared to other neurophysiological techniques, the relatively easy training of new users, and, most importantly, the fairly noninvasive and nonthreatening nature of the technique, which makes it infant and parent friendly. In addition, EEG is one of the few methods that

can be collected using similar protocols and procedures (e.g., standard capping, data cleaning and processing, and experimental design) across the life span (Bell & Cuevas, 2012; Csibra et al., 2008). These characteristics make EEG a particularly attractive measure to psychologists and cognitive neuroscientists focused on life span development.

Although there have been some landmark studies assessing the basic psychometric properties of EEG in young samples (Brooker et al., 2017; Hill et al., 2020; Howarth et al., 2016; Jones et al., 1997), our field would benefit from more detailed and nuanced examinations of the psychometrics of this method in infancy, both at any one time point and across time. For example, Marshall et al. (2002) reported on EEG power at the level of single hertz bins from 5 to 51 months of age in order to capture the most appropriate window for measures of infant alpha power. Their findings indicated that a dominant peak between 6 and 9 Hz consistently emerged at 10, 14, 24, and 51 months of age, supporting the use of this frequency band to represent alpha activity within this age range. Expanding basic information with respect to infant EEG is vital in order to (1) plan future research in light of known patterns of reliability, (2) interpret the generalizability or uniqueness of individual datasets, and (3) build more comprehensive models of the role that constructs captured via EEG may play in development. The current paper focuses on two measures commonly used in infant research, frontal alpha asymmetry and delta–beta coupling (Anaya et al., 2021; Harrewijn et al., 2016; Poole & Schmidt, 2020). Using a large sample of infants, we present the developmental stability, split-half reliability, and construct validity, within and across assessments (8, 12, and 18 months) for each neural signature.

1.1 | Frontal alpha asymmetry

Frontal alpha asymmetry is usually examined by comparing natural log (ln)-transformed alpha power from the right and left hemispheres of the frontal lobes, that is, modeling the difference score of cortical activity in one hemisphere relative to the other (i.e., ln-right minus ln-left; Smith et al., 2017). EEG power is inversely related to cortical activity, such that lower power values indicate greater neural activity. Thus, negative asymmetry scores indicate greater right frontal alpha asymmetry (Smith et al., 2017). Alpha power (8–12 Hz) has long been considered the dominant EEG frequency band in adults during waking states and is thought to track dispositional differences in affective responses (Mennella et al., 2017; Zhang et al., 2020). Several studies of frontal alpha asymmetry in younger samples have shown that infants have a dominant frequency band between 6 and 9 Hz, which approximates to the alpha band in adults (Diaz & Bell, 2012; Marshall et al., 2002; Smith & Bell, 2010). Indeed, researchers have expanded on the functional significance of alpha power and frontal alpha asymmetry using this 6–9 Hz band consistently across 6-, 8-, and 10-month infant samples (Marshall et al., 2002).

Some of these studies suggest that frontal alpha asymmetry can capture broad approach-avoidance motivation and behavioral tendencies, even as early as 8 months (Crespo-Llado et al., 2018). Infant studies, in particular, have linked greater right frontal alpha asymmetry to social withdrawal and the inhibited tendencies of fearful and shy temperaments (Diaz & Bell, 2012; Hane et al., 2008), negative emotionality (Smith et al., 2016), and less positive mother–infant interactions during stressful situations (Perone et al., 2020). Infant frontal alpha asymmetry may also be an important predictor of later socioemotional development, linking greater right frontal alpha asymmetry in infancy with less effortful control and more internalizing problems in early childhood, and greater left frontal alpha asymmetry with more externalizing problems (Smith & Bell, 2010; Smith et al., 2016).

More recently, researchers have begun to model repeated measures of frontal alpha asymmetry to examine its developmental stability (Brooker et al., 2017; Müller et al., 2015) and capture how developmental trends may be associated with temperament, socioemotional processes, and psychopathology risk (Gabard-Durnam et al., 2015; Gartstein et al., 2020; Howarth et al., 2016). Brooker et al. (2017) reported moderate to strong positive correlations in frontal alpha asymmetry between two assessments at 6 and 12 months, and Müller et al. (2015) reported similar correlations between two frontal alpha asymmetry assessments at 14 and 83 months. These studies provided significant contributions to our knowledge of frontal alpha asymmetry as a stable neural marker during infancy, and whether stability may extend into childhood. We wish to expand upon these landmark studies by examining the stability of frontal alpha asymmetry using multiple repeated measures, and by modeling frontal alpha asymmetry over shorter intervals in infancy, which may be more developmentally sensitive to the rapid structural and functional changes taking place in the infant brain (Johnson, 2000).

Studies with three or more assessments can model developmental trajectories and are a powerful tool to expand our knowledge of frontal alpha asymmetry as a marker of socioemotional development, psychopathology risk, and neurological disorders. For example, Gabard-Durnam et al. (2015) modeled trajectories of frontal alpha asymmetry at 6, 12, and 18 months in children at low and high risk for autism spectrum disorders. They reported significant change in frontal alpha asymmetry over time, with growth trends toward greater right frontal alpha asymmetry in the high-risk group and greater left frontal alpha asymmetry in the low-risk group. These patterns of increasing and decreasing left and right asymmetry, respectively, would not have been observed without a third assessment, underscoring the necessity of a repeated measures design.

Howarth et al. (2016) modeled trajectories of frontal alpha asymmetry in a large community sample of infants ($N = 183$), with extended repeated measures of infant EEG at 10, 24, 32, and 48 months. They reported only marginal stability in frontal alpha asymmetry between 10 and 24 months of age, and no stability between 24, 32, and 48 months. Although these studies provide some insight into the developmental trajectory of frontal alpha asymmetry, we need more repeated-measures data to assemble a full developmental account that examines change over time. Complemented with rank-order stability in frontal alpha asymmetry, these data will improve our understanding as to how change is taking place. One of our goals in the present study was to address these gaps in the literature. Furthermore, reliability analyses can also complement our examination of developmental

change, because our interpretation of frontal alpha asymmetry is consistently reliable across assessments. For example, Hill et al. (2020) recently compared frontal alpha asymmetry between mothers and infants and reported high heritability estimates, providing evidence for the theorized trait-like nature of this neural marker. Their findings were complemented by split-half reliability analyses of a 3-min task, showing that frontal alpha asymmetry had good psychometric properties (Reported αs = 0.74–0.90) across mothers and children. In the present study, we also wish to assess reliability of frontal alpha asymmetry across infancy to complement our developmental account of this neural marker and potentially provide additional information about its psychometric properties for future studies.

1.2 | Delta–beta coupling

Evolutionary theories of brain oscillations suggest that the delta EEG band reflects basic homeostatic, bottom-up processes, whereas the fast-wave activity captured in the beta EEG band reflects top-down, cognitive control processes (Engel et al., 2001; Knyazev, 2007, 2012). Developmental studies of individual EEG frequency bands support these functional interpretations, showing that delta power decreases with age and beta power increases (Clarke et al. 2001). Additionally, Clarke and colleagues reported that slow wave/fast wave ratios shift from slow wave dominated in infancy to fast wave dominated later in childhood, which is in line with age increases in cognitive function and regulation of homeostatic states. Based on these findings, researchers have posited more recently that simultaneous changes in delta and beta oscillations may reflect the crosstalk or coordinated interaction between subcortical and cortical circuitry that is involved in regulatory processes. To date, several studies have captured this neural signature through the statistical correlation between relative power in the delta and beta bands (Knyazev & Slobodskaya, 2003; Schutter & van Honk, 2004), using it as proxy for an individual's emotion regulation capacity.

In one study, van Peer et al. (2008) identified heightened levels of coupling in delta–beta activity after a cortisol administration (r = .70, p = .001) compared to a placebo group (r = .50, p = .030). Cortisol is a hormone produced by the hypothalamic–pituitary–adrenal (HPA) axis to support coordinated endocrine responses to stressors (Gunnar et al., 2009), and has been implicated in fearful states, inhibition, and anxiety. Given previous functional MRI studies linking hyperconnectivity between subcortical and cortical circuitry to sustained fear, anxiety, and inhibited temperament (Anteraper et al., 2014; Taber-Thomas et al., 2016), these findings suggest that the heightened delta–beta correlation following cortisol administration likely reflected similar hyperconnectivity between subcortical and cortical circuitry, indicative of the inhibited states induced by cortisol.

The opposite pattern of results has been reported for testosterone, a hormone implicated in fear suppression and disinhibition, which operates as an antagonist to the HPA axis. Miskovic and Schmidt (2009) compared delta–beta coupling across low- and high-testosterone groups and reported a decoupled pattern of delta–beta activity in the high-testosterone group (r = .17, p = .270) compared to the low group (r = .74, p = .001). Together, these studies suggest that heightened delta–beta coupling reflects sustained states of fear or inhibition, whereas decoupling of delta–beta activity reflects uninhibited or dysregulated states, and imply that moderate delta–beta coupling must then reflect adaptive, regulated states.

Several studies in adults and older children support this functional interpretation, linking delta–beta over-coupling to anxiety (Knyazev, 2011; Poole & Schmidt, 2019; Miskovic et al., 2011) and inhibited phenotypes (Poole et al., 2020). The only two studies of delta–beta coupling in infancy and toddlerhood partially support these hypotheses. Phelps et al. (2016) examined longitudinal links between fearful temperament and delta–beta coupling and reported that toddlers characterized with dysregulated fear (i.e., high fear in low-threat context) showed heightened delta–beta coupling at age four (r = .65, p = .050) compared to non-dysregulated toddlers (r = .19, p = .100). Brooker et al. (2016) measured delta–beta coupling and cortisol reactivity in 6-month-old infants. Infants' cortisol reactivity was operationalized as cortisol levels following emotion-eliciting episodes in the lab, after subtracting average cortisol levels collected at home over three consecutive days. They reported that infants with high cortisol reactivity displayed heightened delta–beta coupling (rs = .65–.68) compared to low-reactive infants (rs = −.17 to .21) in contexts that elicited negative affect (e.g., fear). These findings support the functional interpretation of delta–beta coupling, showing once again that greater global levels of cortisol were associated with heightened delta–beta correlation, and suggesting enhanced efforts to inhibit or regulate emotion-eliciting contexts. Although these studies have linked delta–beta coupling to infant temperament risk, several questions regarding developmental patterns of delta–beta coupling in infancy have not been asked. For instance, it is unknown whether infants who show stronger delta–beta coupling show rank-order stability throughout infancy, or whether associations with temperament risk change over time. In the present study, we hoped to begin answering these questions by examining the psychometric properties of delta–beta coupling scores in infancy.

1.3 | Current study

Our goal in the present study was to advance the infant EEG literature by examining the psychometric properties of frontal alpha asymmetry and delta–beta coupling. Particularly, exploratory analyses of sample-level changes, visit-to-visit rank-order stability, and visit-specific split-half reliability of frontal alpha asymmetry and delta–beta coupling were intended to provide actionable information for future studies of these neural correlates in infancy. We collected repeated measures of parent-reported child temperament and resting-state EEG in infants at 8 (N = 192), 12 (N = 133), and 18 (N = 108) months to compute frontal alpha asymmetry and delta–beta coupling scores. We first tested sample-level changes in frontal alpha asymmetry and delta–beta coupling complemented by visit-to-visit rank-order stability to describe change over time in these neural measures. We then examined the split-half reliability of frontal alpha asymmetry and delta–beta

coupling scores. Finally, we probed the construct validity of frontal alpha asymmetry and delta–beta coupling by testing the assessment-specific correlations between these neural measures and temperament subscales from the Infant Behavior Questionnaire—Revised (IBQ-R; Putnam et al., 2014) and Toddler Behavior Assessment Questionnaire (TBAQ; Goldsmith, 1996). Based on previous findings (Peterson et al., 2008), we looked to see if, at each assessment, asymmetry scores were correlated with IBQ and TBAQ subscales measuring temperamental aspects of approach and avoidance, such as the Fear, Activity, Approach, and Cuddliness subscales of the IBQ, and the Social and Object Fear, Activity, Inhibitory Control, and Interest subscales of the TBAQ. Based on the limited research on the temperamental correlates and functional importance of delta–beta coupling in early life, we looked to see if individual coupling scores were associated with the Negative Affect and Regulation higher-order factors of the IBQ, as well as the subscales that load onto these factors, and with Social Fear, Object Fear, and Inhibitory Control subscales of the TBAQ.

2 | METHOD

2.1 | Participants

Participants were recruited through local baby registries (40% families) and university-sponsored participant databases (13% families) as part of a large-scale study of attention, neural, and socioemotional development (Pérez-Edgar et al., 2021). In addition, we used a variety of community-level recruitment strategies, such as visiting local lactation/parenting classes, communicating with families at local community events, and talking to parents at local hospitals, healthcare centers, and Women's and Infant Centers (WIC). Community recruiting identified 38% of our families. Prospective families were contacted by letter, email, or phone explaining the motivations and methods of the study. The remaining 10% of families were recruited by word-of-mouth. The Institutional Review Boards at the Pennsylvania State University and Rutgers University approved all procedures and parents provided written consent and were compensated for their participation.

Infants and their caregivers were enrolled when the infants were 4 months of age (N = 298; 151 males, 147 females; M_{age} = 4.80 months; SD_{age} = 0.80, $range_{age}$ = 3.27–7.60 months), with an additional 46 participants enrolled at 8 months (N = 46; 19 males, 27 females; M_{age} = 8.83 months; SD_{age} = 0.73, $range_{age}$ = 7.53–10.20 months) and 13 at 12 months (N = 13; six males, seven females; M_{age} = 12.73 months; SD_{age} = 1.12, $range_{age}$ = 10.63–14.90 months), for a total enrollment of 357 infants in the full sample (176 males, 181 females). Participants were recruited from areas surrounding three sites: State College, PA (N = 167), Harrisburg, PA (N = 81) and Newark, NJ (N = 109). Caregivers identified 58 of the infants (16%) as African American/Black, nine (3%) as Asian, 78 (22%) as Latinx, 180 (50%) as White, and 27 (8%) as mixed race. Five (1%) additional caregivers declined to provide this information.

In the current sample, 110 families (56.70%) reported an income above $60,000 and the majority of mothers (N = 134) and fathers (N = 118) had a college education or beyond. Additional demographic details for the current sample are presented in Table 1.

TABLE 1 Demographic characteristics at the time of enrollment

Characteristic	Mean (SE) or % (*n*)
Parent-reported child sex	
Boys	50.75% (101)
Girls	49.25% (98)
Mother education	
No high school diploma	6.67% (13)
High school diploma	26.15% (51)
College graduate or higher	68.72% (134)
Father education	
No high school diploma	6.91% (14)
High school diploma	30.32% (57)
College graduate or higher	62.77% (118)
English home language	79.90% (159)
Marital status	
Married	78.79% (156)
Divorced	1.01% (2)
Single	9.60% (19)
Cohabitating	10.60% (21)
Nonparental child care	44.22% (88)
Child care type	
Daycare center	64.77% (57)
Home daycare	19.32% (17)
Nanny or au pair	15.91% (14)
Household income	
≤$30,000	23.20% (45)
$31,000–$60,000	20.10% (39)
≥$60,000	56.70% (110)

Note: Demographics are presented for the sample included in the present analyses.

The current study uses data from assessments at 8, 12, and 18 months. From the total enrolled sample, 192 infants contributed EEG data at 8 months, 133 at 12 months, and 103 at 18 months. During EEG processing, data were excluded from the delta–beta coupling exports due to excessive artifacts (N = 35) and insufficient number of segments (N = 54). Independently, data were also excluded from the frontal alpha asymmetry processing due to excessive artifacts in either the lights-on or lights-off condition (N = 124) and insufficient number of segments (N = 60). This resulted in the final sample included in the analyses for delta–beta coupling (N_8 = 164, N_{12} = 85, N_{18} = 90) and frontal alpha asymmetry lights-off (N_8 = 108, N_{12} = 71, N_{18} = 69) and lights-on (N_8 = 132, N_{12} = 86, N_{18} = 81). The final assessment (24 months) is not included in the current analyses because active data collection was disrupted due to COVID-19 mitigation, resulting in excessive missing data for the later time assessments.

2.2 | Procedures

Data collection was generally completed in two 2-hour visits to the lab, although some families requested different scheduling. At 8 months, 58 families completed the protocol in a 1-day visit (with a break), whereas 170 families completed the study protocol in two separate visits. At 12 months, 62 families completed the protocol in a 1-day visit (with a break), 110 families completed the study protocol in two separate visits, and one family completed the protocol in three separate visits to the lab. At 18 months, 88 families completed the protocol in a 1-day visit (with a break), whereas 42 families completed the study protocol in two separate visits. At each visit, families came into the lab and completed a series of tasks; the EEG data included in this study were usually collected during Visit 2. Parents were asked to sit their infants on a high-chair, in front of a computer monitor where a video stimulus (i.e., cave navigation video or outer space navigation video) was presented while EEG was recorded. The videos were of low contrast and muted, and both displayed slow movement on the screen to keep infants' attention while minimizing arousal. Links to view these videos are provided in the Supporting Information. Two experimenters placed and adjusted an EEG cap on the infants. Parents were instructed to remain neutral and to limit interactions with infants once the EEG collection began. Experimenters ended the EEG task early whenever infants showed high levels of distress affect or irritation. Questionnaires were primarily completed online by one parent designated as the primary caregiver at recruitment (95% mothers). Caregivers were provided a link to complete all questionnaires via Qualtrics (Qualtrics, Provo, UT) prior to the laboratory visit, but in some cases, parents completed the questionnaires in the laboratory or over the phone.

2.3 | Measures

2.3.1 | Resting-state EEG

Infant EEG was continuously recorded at 32 Ag/AgCl active scalp electrodes during a resting-state task at 8, 12, 18, and 24 months. Each infant was fitted with the active electrode cap (Brain Products actiCAP) after measuring the head circumference. A small amount of conductive gel was applied to each electrode site prior to placing the cap on the infant's head. The experimenter dispensed additional gel as needed while gently abrading the scalp until impedance levels were below 10 kΩ or the infant became fussy.

The task was divided into four 1-min blocks during which the lights in the room were either on ("lights-on") or off ("lights-off") to imitate eyes open and eyes closed conditions, respectively. This approach has been used in prior work with pediatric samples (e.g., Degnan et al., 2011). Blocks alternated between lights-on and lights-off conditions (on, off, on, off). A video showing neutral images moving continuously across the screen was presented on a 17″ computer screen to help maintain infant attention. We prioritized a subset of electrodes related to questions of interest to reduce participant burden and expedite the capping process. The following electrodes were selected: Fp1, Fp2, F3, F4, F7, F8, Cz, C3, C4, T7, T8, P3, P4, P7, P8, O1, and O2. EEG was amplified using the Brain Products ActiCHamp system and digitized at 500 Hz. EEG data were preprocessed offline using Brain Vision Analyzer 2 (Brain Products GmbH, Germany). Data were filtered using a zero-phase Butterworth infinite impulse response (IIR) filter with low and high cutoffs of 0.1 and 40 Hz (12 db/octave), respectively, and a 60-Hz notch filter. EEG data were referenced online to Fz, and then re-referenced offline to the average of P7/P8. Prioritizing particular electrodes limited our ability to meet certain assumptions of referencing schemes more commonly used in infant EEG research, such as the common average (e.g., insufficient number of electrode sites). P7/P8 were selected from the prioritized electrodes given that these sites were relatively far from scalp sites of interest, were not biased toward one hemisphere, and were minimally influenced by undesired sources of interference to the signal (Luck, 2014).

EEG data were segmented into 1-s epochs. These segments were then baseline corrected and inspected for artifacts (e.g., eye blinks). Artifacts were defined as voltage steps exceeding ± 50 μV/ms, maximum voltage difference of less than 0.50 μV within a 100 ms interval or more than 150 μV within a 200 ms interval, or amplitudes exceeding ± 200 μV. Trained research assistants visually inspected and removed the identified artifacts specific to the electrode(s) of interest, namely, F3, Fz, F4, C3, Cz, C4, Pz, P3, and P4 for delta/beta coupling, and Fp1, Fp2, F3, F4, F7, and F8 for frontal asymmetry. This semiautomated approach has been recommended as one option for limiting data loss in infant EEG/ERP studies (Hoehl & Wahl, 2012). Spectral power at each frequency was calculated via a Fast Fourier Transformation (FFT) that utilized a Hamming window with 50% overlap. Power values were then exported for additional processing in R v3.6.1 (R Core Team, 2019). The following frequency windows were used in the calculation: delta (0.5–2 Hz), alpha (6–9 Hz), and beta (11–18 Hz).

2.3.2 | Frontal alpha asymmetry

Frontal alpha asymmetry data were segmented with a 50% overlap. Only participants who provided >60 s (>120 segments) of artifact-free EEG data during either the lights-on or lights-off condition were retained in these analyses. This decision was based on findings from Hill et al. (2020), showing that adequate internal consistency in infant frontal alpha asymmetry can be achieved with this number of segments. Spectral power in the 6–9 Hz frequency range was natural log transformed for electrode channels Fp1, Fp2, F3, F4, F7, and F8. Frontal asymmetry scores were computed by first subtracting activation in the left frontal electrode from activation in the right frontal electrode (i.e., ln(right) – ln(left) = frontal asymmetry score) for specific electrode pairs, that is, Fp1/Fp2, F3/F4, and F7/F8. Frontal alpha asymmetry was calculated based on the average of these scores for each condition. A negative frontal asymmetry score reflects greater activation in the right hemisphere relative to the left hemisphere (Davidson, 1992; Fox et al., 2001; Marshall et al., 2002).

2.3.3 | Delta–beta coupling

We exported second-by-second EEG power for the delta and beta frequency bands to compute an individual delta–beta coupling score for each participant at each assessment, which allows for tracking individual patterns of change over time. This approach contrasts with averaging across the time series within a time point, which has been the common approach in the existing literature (e.g., Phelps et al., 2016). However, a similar individual score approach has been employed in several previous studies (Anaya et al., 2020; Harrewijn et al., 2016; Poole et al., 2020; Poole & Schmidt, 2019). Participants with less than 10 segments were excluded from the computation of within-person, delta–beta coupling scores. Power values across target electrodes were log transformed and then averaged to create composites for the frontal (F3, Fz, F4), central (C3, Cz, C4), and parietal (P3, Pz, P4) regions based on the 10–20 System of Electrode Placement (Herwig et al., 2003).

2.3.4 | Infant Behavior Questionnaire—Revised

The IBQ-R is a 191-item survey designed to assess general patterns of behavior associated with temperament in infancy (Parade & Leerkes, 2008; Putnam et al., 2014). Parents rated how often they observed a behavior in the past week at the 8- and 12-month assessments. Each item describes an infant behavior (e.g., *During feeding, how often did the baby lie or sit quietly?*) using a 7-point scale (1 = *never*, 2 = *very rarely*, 3 = *less than half the time*, 4 = *half the time*, 5 = *more than half the time*, 6 = *almost always*, and 7 = *always*). Parents are also given a "not applicable" response option for use when the infant has not been observed in the situation described. Each item loads onto one of 14 subscales: Activity Level, Distress to Limitations, Fear, Duration of Orienting, Smile/Laughter, High-intensity Pleasure, Low-intensity Pleasure, Soothability, Falling Reactivity, Cuddliness, Perceptual Sensitivity, Sadness, Approach, and Vocal Reactivity. Items from each subscale are averaged to obtain scale scores. Each scale, in turn, loads onto one of three broader factors (Surgency, Negativity, Orienting/Regulation). The IBQ-R has demonstrated good internal consistency, reliability, and validity, including correlations with laboratory observations (Gartstein & Marmion, 2008; Parade & Leerkes, 2008). In our sample, reliabilities across IBQ subscales were good at 8 months (Cronbach's αs = 0.728–0.943) and 12 months (Cronbach's αs = 0.767–0.922).

2.3.5 | Toddler Behavior Assessment Questionnaire

The TBAQ is a 120-item survey designed to assess general patterns of behavior associated with temperament in young children (2–3 years; Goldsmith, 1996). It was collected at 18 months. Parents rated how often their toddler displayed a specific behavior in the past month using a 7-point Likert scale (1 = *never*, 2 = *very rarely*, 3 = *less than half the time*, 4 = *half the time*, 5 = *more than half the time*, 6 = *almost always*, and 7 = *always*). Each item loads onto one of 11 subscales (Activity Level, Anger, Appropriate Attention Allocation, Inhibitory Control, Interest, Object Fear, Perceptual Sensitivity, Pleasure, Sadness, Social Fear, and Soothability). Items from each subscale are averaged to obtain scale scores. Goldsmith (1996) reported high levels of convergence with various subscales of the IBQ. In our sample, reliabilities across TBAQ subscales were good (Cronbach's αs = 0.612–0.850).

3 | ANALYTIC PLAN

Data analysis proceeded in three steps: (1) computing the split-half reliability and Cronbach's α of EEG measures, (2) modeling the mean structure and rank-order stability of EEG measures, and (3) testing the convergent and discriminant validity of EEG measures by examining associations with parent-reported infant temperament across 8, 12, and 18 months.

First, we assessed reliability of frontal alpha asymmetry separately for the lights-on and lights-off conditions. Subject-specific matrices of second-by-second asymmetry scores calculated with each electrode pair (Fp2–Fp1, F4–F3, F8–F7) and with electrode clusters (average of right – average of left) were randomly split and correlated 100 times. Mean split-half correlations and Cronbach's alpha (α) were computed for each participant's frontal alpha asymmetry data. Split-half correlations and Cronbach's alpha from the 100 iterations were then averaged across all participants within each time assessment and reported as the sample-level reliability of frontal alpha asymmetry. We employed the same 100-random split-half approach to examine the reliability of alpha power, which was carried out separately for left and right hemispheres within the lights-on and lights-off conditions. Reliability analyses were carried out using the *multicon* R package (Sherman, 2015), and reliability metrics were considered adequate when >0.65, based on previous studies that discuss the use of this metric (Bonett & Wright, 2015; Vaske et al., 2017). R scripts for deriving delta–beta coupling and frontal alpha asymmetry measures, and computing reliability metrics are available at https://github.com/bua25/manuscripts-analyses-processing.

Reliability of delta–beta coupling was assessed using a measure-specific approach, given that delta–beta coupling is in and of itself a correlation metric. Subject-specific matrices of second-by-second delta and beta power were split into odd and even segments. This resulted in two matrices per subject, one for odd segments and one for even segments. Subject-specific delta–beta coupling scores were then computed from the odd- and even-segments matrices and the sample-wide vectors of odd and even coupling scores were then correlated.

In a second step, we assessed developmental stability by examining sample-level changes in frontal alpha asymmetry and delta–beta coupling across the three assessments using repeated measures analysis of variance (ANOVAs) and rank-order stability across assessments using Pearson correlations. ANOVAs were implemented in a multilevel modeling framework using the *nlme* R package (Pinheiro et al., 2021).

In a third step, we examined the convergent and discriminant validity of frontal alpha asymmetry and delta–beta coupling by computing the zero-order correlations between these neural measures and IBQ and TBAQ subscales at each of the corresponding time assessments.

TABLE 2 Frontal alpha asymmetry reliability for lights-on EEG segments

	8 months		12 months		18 months	
	Split-half *r*	Cronbach's alpha	Split-half *r*	Cronbach's alpha	Split-half *r*	Cronbach's alpha
N	132	132	86	86	81	81
Mean	0.69	0.81	0.73	0.80	0.71	0.82
SD	0.22	0.19	0.27	0.59	0.11	0.08
Median	0.73	0.84	0.75	0.86	0.71	0.83
Min	0.11	0.07	−1.00	−4.53	0.32	0.48
Max	−1.00	−0.96	0.99	0.99	0.97	0.98

Note: Split-half average correlations are based on 100 random splits within each subject's segment-by-segment data and then averaged across the sample (*N*).

TABLE 3 Frontal alpha asymmetry reliability for lights-off EEG segments

	8 months		12 months		18 months	
	Split-half *r*	Cronbach's alpha	Split-half *r*	Cronbach's alpha	Split-half *r*	Cronbach's alpha
N	108	108	67	67	69	69
Mean	0.68	0.78	0.74	0.84	0.66	0.82
SD	0.18	0.38	0.16	0.12	0.31	0.10
Median	0.70	0.83	0.79	0.88	0.74	0.85
Min	−0.59	−2.93	0.14	0.25	−1.00	0.28
Max	0.99	1.00	0.96	0.98	0.93	0.97

Note: Split-half average correlations are based on 100 random splits within each subject's segment-by-segment data and then averaged across the sample (*N*).

Here, we provide an additional analytic approach for delta–beta coupling. Our second-by-second time series analyses of delta–beta coupling are relatively novel within the literature. This is in contrast to the difference score approach for frontal alpha asymmetry, which has been used for over 30 years (e.g., Fox & Davidson, 1988). As such, we wished to provide a comparison point for readers by also providing more common group-based approach to delta–beta correlations.

4 | RESULTS

4.1 | Reliability

Results for lights-on and lights-off conditions are presented in Tables 2 and 3. Mean split-half correlations (range *r*s = .66–.75) and Cronbach's αs (range αs = 0.78–0.84) indicated adequate reliability of frontal alpha asymmetry across the sample and across assessments, with no evidence that reliability changed over time or across lights-on and lights-off conditions. We also follow-up these analyses with reliability for alpha power separately for the left and right hemispheres. As reported in Table 4, reliability for alpha power was high (range = 0.80–0.92) across hemispheres and time assessments and seemed to be consistently better than reliability of asymmetry scores.

Reliability analyses of delta–beta coupling indicated that at 8 months, coupling scores from odd and even segments were negligibly correlated at frontal and central regions (*r*s = −.01 and −.03, respectively), and weakly correlated at the parietal region (*r* = .20). At 12 months, delta–beta coupling scores were weakly correlated at frontal and central regions (*r*s = .22 and .34, respectively), but negligible (*r* = .04) at the parietal regions. Finally, delta–beta coupling scores from odd and even segments at 18 months were weakly and negatively correlated at the central region (*r* = −.22), and negligibly correlated at frontal and parietal regions (*r*s = −.08 and −.07, respectively). We follow-up these analyses with an examination of the reliability of average delta and beta power at each assessment, given that delta and beta relative power are the actual EEG variables that constitute delta–beta

TABLE 4 Frontal alpha power reliability for lights-off and lights-on conditions

	Lights-off condition					
	8 months		12 months		18 months	
	Left	Right	Left	Right	Left	Right
M split-half correlation	0.81	0.80	0.86	0.83	0.85	0.83
Cronbach's α	0.87	0.90	0.92	0.89	0.92	0.90
SD	0.18	0.19	0.16	0.16	0.13	0.13
	Lights-on condition					
M split-half correlation	0.85	0.82	0.85	0.86	0.81	0.82
Cronbach's α	0.91	0.89	0.91	0.92	0.90	0.90
SD	0.15	0.16	0.19	0.16	0.11	0.13

Note: Split-half average correlations are based on 100 random splits within each subject's segment-by-segment power data and averaged across the sample. Left and right labels indicate brain hemispheres.

TABLE 5 Reliability of delta and beta power

		8 months		12 months		18 months	
	N	Delta power	Beta power	Delta power	Beta power	Delta power	Beta power
M split-half correlation	161	0.50	0.85	0.58	0.84	0.51	0.92
Cronbach's α	161	0.66	0.92	0.73	0.91	0.67	0.96

Note: Split-half average correlations are based on 100 random splits of average power value.

TABLE 6 Sample-level descriptives, mean differences, and rank-order stability of frontal alpha asymmetry

	Lights-off		Lights-on	
	Mean	(*SD*)	Mean	(*SD*)
Descriptive differences				
8 months	0.15*	(0.34)	0.14*	(0.30)
12 months	0.00	(0.20)	0.05*	(0.17)
18 months	−0.01	(0.13)	−0.01*	(0.17)
Rank-order stabilities				
8 months, 12 months	$r = -.06$		$r = .23$	
8 months, 18 Months	$r = -.45$*		$r = .07$	
12 months, 18 months	$r = .36$		$r = .18$	

Note: Means and standard deviations (*SD*) based on $N = 43–89$. Comparisons of frontal alpha asymmetry scores across assessments were examined with FDR correction for multiple comparisons.
*Mean values significantly differ from one another and significant correlations ($p < .05$).

coupling scores. Average correlations from assessment-specific, 100-random split halves of the delta and beta power data with Cronbach's α are presented in Table 5. Across assessments, the mean of all split-half correlations ranged from $r = .50$ to .92 and were always higher for beta compared to delta power. Cronbach's αs were adequate (αs = 0.66–0.96), and better for beta compared to delta power across all assessments.

4.2 | Developmental stability

Mean differences and variability for frontal alpha asymmetry scores are depicted in Figure 1. Repeated measures ANOVAs for frontal alpha asymmetry indicated a significant effect of age for both the lights-on ($F_{(2,66)} = 5.09$, $p = .008$) and lights-off ($F_{(2,48)} = 6.43$, $p = .003$) conditions. Post hoc, FDR-corrected comparisons, shown in Figure 1, indicated that in the lights-on condition, frontal alpha asymmetry scores significantly decreased (became more right dominant) from 8 to 12 ($p = .049$), from 12 to 18 ($p = .009$), and from 8 to 18 months ($p = .049$). Post hoc comparisons indicated that lights-off asymmetry scores significantly decreased from 8 to 12 ($p = .001$) and from 8 to 18 months ($p = .001$), but the change from 12 to 18 months was not significant ($p = .737$).

Descriptive statistics and rank-order stability of frontal alpha asymmetry across at 8, 12, and 18 months are reported in Table 6. We examined cross-assessment correlations for frontal alpha asymmetry scores to determine rank-order stability. As seen in the lower portion of this table, lights-off frontal alpha asymmetry scores exhibited weak rank-order stability from 12 to 18 months ($r = .36$, $p = .089$), and a significant, moderate change from 8 to 18 months ($r = -.45$, $p = .055$). In contrast, no significant correlations emerged for the lights-on condition (*p*s > .75).

Repeated measures ANOVAs for delta–beta coupling scores (Figure 2) indicated a significant effect of age at central ($F_{(2,118)} = 4.65$, $p = .011$) and parietal regions ($F_{(2,118)} = 3.59$, $p = .031$), but not at the frontal region ($F_{(2,117)} = 2.06$, $p = .132$). Post hoc, FDR-corrected comparisons indicated that average delta–beta coupling scores at the central region were significantly less positive at 18 months ($M_{18} = 0.02$) than at 8 months ($M_8 = 0.11$, $p = .008$), but central region coupling scores did not significantly differ between 12 and 18 months or between 8 and 12 months (*p*s > .63). These results suggested that delta–beta activity became less coupled or synchronized over time. Similarly, average delta–beta coupling scores at the parietal region were significantly less positive at 18 months ($M_{18} = 0.04$) than at 8 months ($M_8 = 0.12$, $p = .032$), but parietal region coupling scores did not significantly differ between 12 and 18 months or between 8 and 12 months (*p*s > .990). These results suggested that delta–beta activity at central and parietal regions became less coupled or synchronized over time.

Region-specific, cross-assessment correlations of delta–beta coupling scores are reported in the lower portion of Table 7 and indicated moderate change in central coupling scores from 8 to 12 months ($r = -.28$, $p = .041$), and moderate, although marginal, rank-order stability in parietal coupling scores from 8 to 12 months ($r = .23$, $p = .085$). No other correlations across assessments emerged to support rank-order stability or change (*p*s > 0.12). Correlations within time assessments indicated that delta–beta coupling scores were positively correlated between frontal and central regions ($r = .23$, $p = .003$) at 8 months, positively correlated between frontal and central ($r = .36$, $p = .001$) and frontal and parietal regions ($r = .46$, $p = .001$) at 12 months, and positively correlated between frontal and parietal regions ($r = .56$, $p = .001$) at 18 months.

4.3 | Validity

We evaluated the construct validity of frontal alpha asymmetry and delta–beta coupling by examining the assessment-specific associations between these neural measures and the subscales of the IBQ (8 and

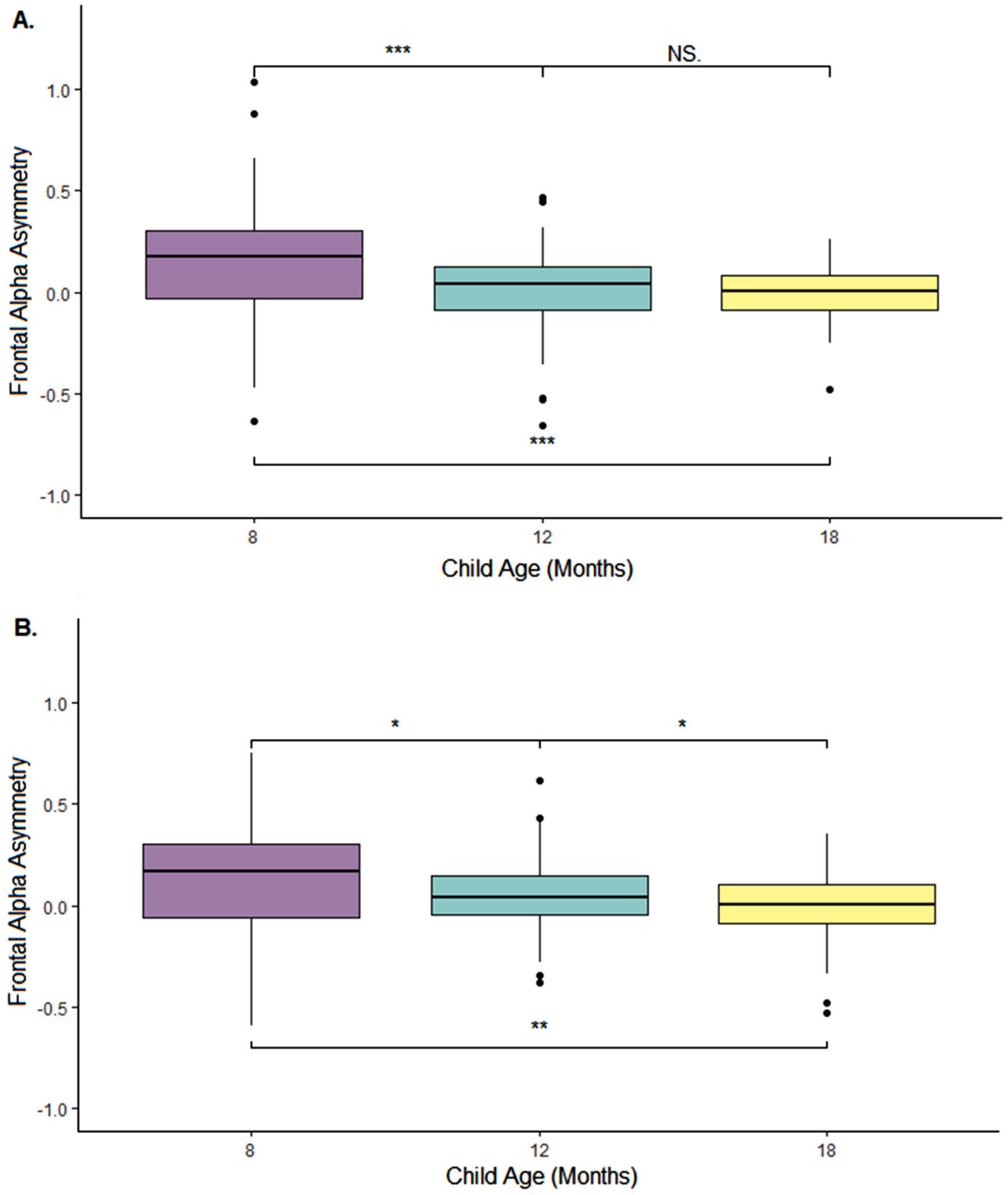
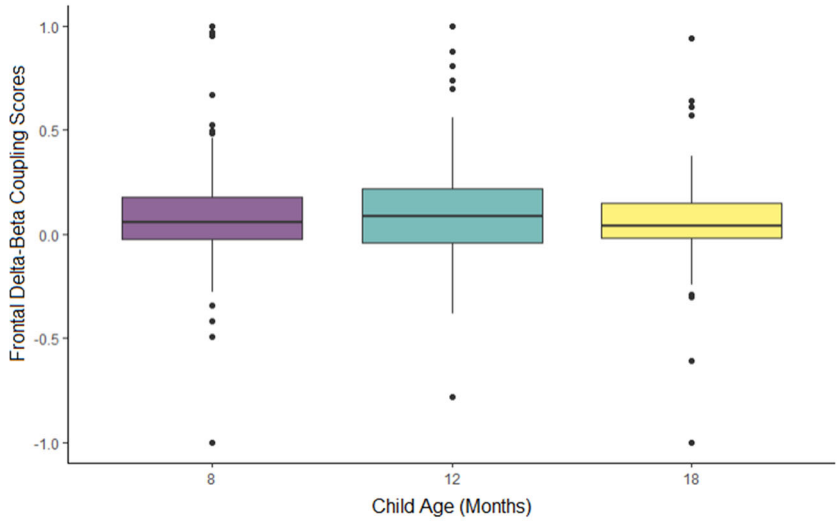
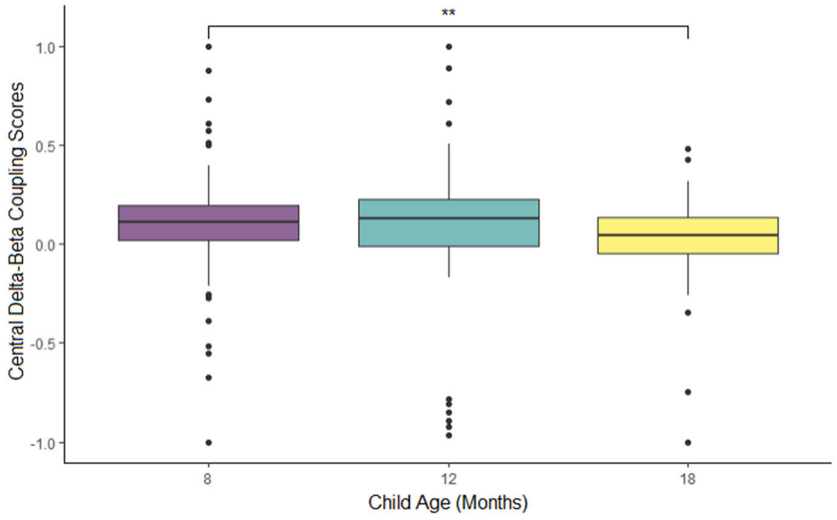


FIGURE 1 Mean differences and variability in lights-off (a) and lights-on (b) frontal alpha asymmetry scores across 8, 12, and 18 months. Positive values indicate greater left (relative to right) hemispheric activity. Negative values indicate greater right (relative to left) hemispheric activity

12 months) and the TBAQ (18 months). Specifically, we were interested in testing correlations between delta–beta coupling scores and temperament dimensions of fear and regulation, and correlations between frontal alpha asymmetry and temperament dimensions of surgency, fear, and inhibition. The temperament subscales were chosen based on the proposed relations typically noted in the existing literature for these neural measures (Brooker et al., 2016; Howarth et al., 2016; Phelps et al., 2016; Schmidt, 2008). Zero-order correlations for each time assessment are presented in Tables 8–10.

At 8 months, the only significant relation was between lights-off frontal alpha asymmetry scores and the IBQ Fear subscale (r = .37, p = .024). Contrary to expectations, infants with greater left frontal asymmetry were significantly more likely to be rated by their parent as inhibited to novelty and distressed by object or social stimulation.





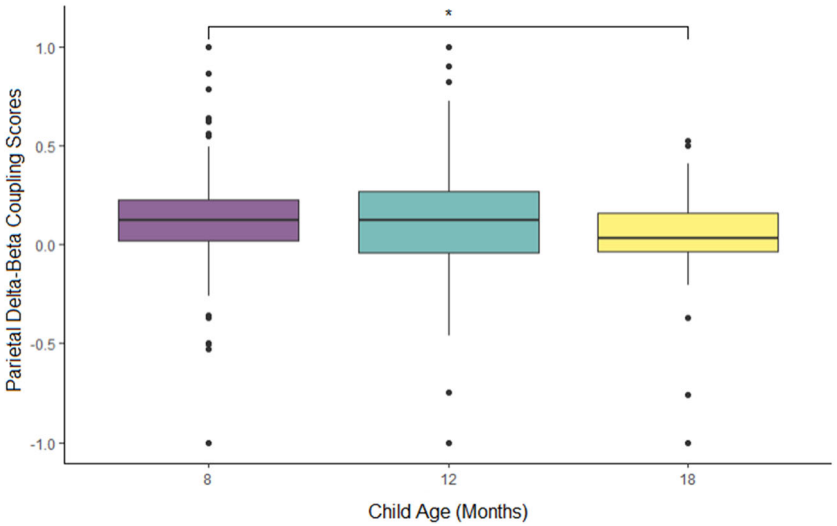


FIGURE 2 Mean differences and variability in delta–beta coupling scores across 8, 12, and 18 months

At 12 months, there were significant associations for frontal alpha asymmetry scores in both the lights-off and lights-on conditions. Lights-off asymmetry scores were significantly correlated with the IBQ Surgency higher-order factor ($r = .33$, $p = .015$), IBQ High Pleasure ($r = .35$, $p = .012$), and IBQ Approach subscales ($r = .37$, $p = .007$). These correlations suggested that infants with greater left asymmetry scores were more likely to be rated as higher in approach-related tendencies, which is directly in line with interpretations of frontal alpha asymmetry in the general literature (Peterson et al., 2008). Lights-on asymmetry scores were negatively correlated with the IBQ Fear

TABLE 7 Sample-level descriptives, mean differences, and rank-order stability of delta–beta coupling

	Frontal		Central		Parietal	
	Mean	(*SD*)	Mean	(*SD*)	Mean	(*SD*)
Descriptive differences						
8 months	0.03	(0.26)	0.11*	(0.26)	0.12*	(0.27)
12 months	0.12	(0.28)	0.09	(0.36)	0.12	(0.36)
18 months	0.05	(0.24)	0.02*	(0.23)	0.04*	(0.21)
Rank-order stabilities						
8 months, 12 months	$r = -.12$		$r = -.28$*		$r = .23$	
8 months, 18 months	$r = -.12$		$r = -.12$		$r = .08$	
12 months, 18 months	$r = .14$		$r = -.09$		$r = .19$	

Note: Means and standard deviations (*SD*) based on $N = 120$. Tukey comparisons of delta–beta coupling scores across assessments were examined with FDR correction for multiple comparisons.
*Mean values significantly differ from one another and significant correlations ($p < .05$).

subscale ($r = -.26$, $p = .051$) and positively correlated with the IBQ Cuddle subscale ($r = .29$, $p = .023$), again generally in line with the literature-based expectations.

Lastly, assessment-specific correlations between TBAQ subscales and frontal alpha asymmetry scores also emerged for both conditions at 18 months. Lights-off asymmetry scores were significantly associated with the TBAQ Pleasure subscale ($r = .28$, $p = .033$) and marginally associated with the Object Fear subscale ($r = -.23$, $p = .074$). In contrast, lights-on asymmetry scores were significantly associated with the TBAQ Activity ($r = .28$, $p = .023$) subscale, and marginally associated with the Sadness ($r = .27$, $p = .087$) and Soothability ($r = -.22$, $p = .071$) subscales.

We tested similar associations between delta–beta coupling and IBQ and TBAQ subscales. At 8 months, frontal region delta–beta coupling scores were marginally associated with the IBQ Distress subscale ($r = .16$, $p = .059$), and central region coupling scores were marginally associated with the IBQ Duration of Orienting ($r = -.15$, $p = .074$) and Activity Level subscales ($r = .15$, $p = .068$).

At 12 months, central ($r = -.22$, $p = .066$) and parietal ($r = -.30$, $p = .013$) delta–beta coupling scores were negatively associated with IBQ Duration of Orienting subscale. Parietal coupling scores were also significantly associated with the IBQ higher-order Regulation factor ($r = -.25$, $p = .033$). These associations suggested that at 12 months, infants with over-coupled delta–beta activity at central and parietal regions were rated as significantly less likely to maintain their attention on a single object, and this negative association may extend more broadly to the Regulation higher-order factor. Parietal delta–beta coupling scores were also negatively associated with the IBQ Sadness ($r = -.25$, $p = .038$) and Approach ($r = -.23$, $p = .057$).

At 18 months, only parietal region delta–beta coupling scores were marginally associated with the TBAQ Object Fear subscale ($r = -.21$, $p = .062$).

Contrary to expectations, delta–beta coupling scores were not significantly associated with the IBQ Negative Affect factor or the TBAQ Fear subscales. The link between delta–beta coupling and fearful phenotypes has mostly been supported by studies that employed group-level analyses of delta–beta coupling across categorical groups (Phelps et al., 2016; Brooker et al., 2016). For the purpose of drawing comparisons between our analyses and previous studies in infants and older populations, we examined the associations between frontal region delta–beta coupling and the IBQ Negative Affect factor (8- and 12-month) and the TBAQ Social Fear subscale (18-month) using group-based analyses. We examined frontal region delta–beta coupling only to minimize comparisons and remain consistent with the majority of previous studies, which report a link between delta–beta coupling and fearful phenotypes in this region.

At 8 months, Low ($n = 28$), Average ($n = 100$), and High ($n = 19$) categorical groups were formed based on $-1SD$ (≤ 2.39), Mean (3.07), and $+1SD$ (≥ 3.75) of the 8-month IBQ Negative Affect scores. Group-level coupling indicated that frontal region delta and beta power were significantly and positively coupled in the Low group ($r = .37$, $p = .05$), negatively coupled in the Average group ($r = -.22$, $p = .03$), and uncoupled or unrelated in the High group ($r = .22$, $p = .37$). Fisher r to z analyses of the correlation coefficients indicated that delta–beta coupling in the Low Negative Affect group was significantly different from coupling in the Average group ($z = 2.74$, $p = .01$) but not the High group ($z = 0.51$, $p = .31$), and that delta–beta coupling in the Average group was significantly different from coupling in the High group ($z = -1.66$, $p = .05$).

We repeated these analyses at 12 months, based on $-1SD$ (≤ 2.71), Mean (3.31), and $+1SD$ (≥ 3.91) of the 12-month IBQ Negative Affect scores to form Low ($n = 10$), Average ($n = 58$), and High ($n = 13$) categorical groups. Results indicated that group-level coupling was not significant for any of the Negative Affect groups ($ps > .17$).

The 18-month sample was split based on $-1SD$ (≤ 2.58), Mean (3.65), and $+1SD$ (≥ 4.72) of the TBAQ Social Fear subscales to form Low ($n = 14$), Average ($n = 52$), and High ($n = 12$) Fear groups. Again, results indicated that at 18 months, group-level coupling was not significant for any of the Fear groups ($ps > .14$).

5 | DISCUSSION

The current paper examined the psychometric properties of frontal alpha asymmetry, a widely used neural correlate of approach or motivation tendencies, and delta–beta coupling, a neural measure that has recently gained traction as a neural proxy for emotion regulation. Despite increased used of these neural measures in infant research, we can benefit from a careful description of their psychometric properties with a developmental lens that focuses on the infancy period. We modeled repeated measures of infant EEG at 8, 12, and 18 months of age to examine the developmental stability, split-half reliability, and construct validity of these neural measures in infancy. However, care must be taken to keep in mind the specific context and circumstances of data collection, as well as the secondary variables that are incorporated in analyses.

TABLE 8 Zero-order correlations between 8-month IBQ subscales and EEG measures

	Delta–beta coupling			Frontal alpha asymmetry	
8-month IBQ subscales	Frontal	Central	Parietal	Lights-on	Lights-off
Activity level	−0.01	0.15*	0.06	−0.17	−0.13
Distress to limitations	0.16*	−0.04	0.00	−0.01	0.12
Fear	0.06	0.02	−0.04	0.23	0.37*
Duration of orienting	−0.10	−0.15*	−0.06	0.08	0.01
Smiling and laughter	−0.03	−0.03	0.00	−0.11	−0.13
High-intensity pleasure	−0.12	−0.04	0.09	−0.11	−0.17
Low-intensity pleasure	−0.04	0.06	0.01	0.02	−0.07
Soothability	−0.03	−0.04	0.00	−0.10	−0.26
Falling reactivity	−0.03	−0.05	0.03	−0.06	−0.05
Cuddliness	−0.06	−0.12	−0.01	−0.08	−0.14
Perceptual sensitivity	−0.08	0.05	0.08	−0.10	−0.07
Sadness	0.15	0.08	0.05	0.05	0.19
Approach	−0.08	−0.06	−0.07	0.16	0.23
Vocal reactivity	0.01	0.03	0.02	−0.11	−0.13
Surgency factor	−0.08	0.03	0.05	−0.14	−0.12
Negative affect factor	0.13	0.03	−0.01	0.10	0.23
Regulation factor	−0.10	−0.10	−0.03	−0.01	−0.17

*$p < .05$; +$p < .07$.

TABLE 9 Zero-order correlations between 12-month IBQ subscales and EEG measures

	Delta–beta coupling			Frontal alpha asymmetry	
12-month IBQ subscales	Frontal	Central	Parietal	Lights-on	Lights-off
Activity level	−0.04	−0.14	−0.03	−0.02	0.21
Distress to limitations	0.01	−0.17	−0.13	−0.12	0.03
Fear	0.02	0.13	0.01	−0.26*	−0.22
Duration of orienting	−0.20	−0.22*	−0.30*	0.04	−0.04
Smiling and laughter	−0.07	−0.11	−0.09	0.17	0.25*
High-intensity pleasure	−0.16	−0.14	−0.17	0.10	0.35*
Low-intensity pleasure	−0.15	−0.03	−0.20	0.04	−0.12
Soothability	0.07	−0.09	−0.01	−0.04	−0.14
Falling reactivity	0.15	0.15	−0.02	0.11	0.19
Cuddliness	−0.03	0.02	0.08	0.29*	0.13
Perceptual sensitivity	−0.02	−0.04	−0.04	0.10	0.16
Sadness	−0.07	−0.19	−0.25*	−0.10	0.09
Approach	−0.07	−0.19	−0.23*	0.01	0.37*
Vocal reactivity	−0.01	−0.13	−0.10	0.12	0.04
Surgency factor	−0.08	−0.17	−0.14	0.12	0.33*
Negative affect factor	−0.07	−0.12	−0.13	−0.19*	−0.09
Regulation factor	−0.16	−0.17	−0.25*	0.14	−0.03

*$p < .05$; +$p < .07$.

TABLE 10 Zero-order correlations between 18-month TBAQ subscales and EEG measures

	Delta–beta coupling			Frontal alpha asymmetry	
18-month TBAQ subscales	Frontal	Central	Parietal	Lights-on	Lights-off
Activity level	−0.13	−0.14	0.07	0.28*	0.20
Anger	0.06	0.00	−0.04	0.12	−0.17
Appropriate attentional allocation	−0.07	0.00	0.10	−0.01	0.20
Inhibitory control	−0.07	−0.04	0.10	−0.21	0.16
Interest	−0.20	−0.04	0.20	0.06	0.17
Object fear	0.09	0.06	−0.21+	−0.04	−0.23+
Perceptual sensitivity	−0.19	−0.10	0.06	0.13	−0.04
Pleasure	0.05	0.08	0.08	0.10	0.28*
Sadness	0.05	−0.09	−0.04	0.27*	−0.02
Social fear	0.15	−0.04	−0.12	−0.01	−0.04
Soothability	0.08	0.11	0.04	−0.22+	−0.01

*$p < .05$; +$p < .07$.

The first task for the current study was to address the general reliability of our EEG-derived measures. Frontal alpha asymmetry scores, as well as relative alpha power, exhibited good reliability across lights-on and lights-off conditions, and were consistently reliable across assessments. To our knowledge, our study is the first to report these psychometric properties of frontal alpha asymmetry across multiple assessments in infancy. Our findings suggest that the internal consistency of frontal alpha asymmetry scores is not changing as a function of age, supporting the use of this metric to assess developmental patterns of infant cognitive processing. In general, there is some concern that difference scores, such as the calculation used in characterizing frontal alpha asymmetry, by necessity increase noise in a measure (van Rooijen et al., 2017). However, our data suggest that this is not as large of a concern with respect to infant frontal alpha asymmetry.

Our second goal was to describe developmental patterns of frontal alpha asymmetry from 8 to 18 months. Across lights-on and lights-off conditions, infants showed a developmental shift from greater left frontal alpha asymmetry toward greater right frontal alpha asymmetry. This shift was more pronounced in the lights-on condition, where mean differences between all assessments were statistically significant. The magnitude of change observed in our sample is in line with perhaps the only previous study to have examined mean differences in frontal alpha asymmetry throughout infancy (Gabard-Durnam et al., 2015). Gabard-Durnam and colleagues examined repeated measures of frontal alpha asymmetry in a sample of young children at high and low risk for autism, making direct comparisons of direction and shift in developmental trajectories difficult. Nevertheless, our results lend support to findings from Gabard-Durnam and colleagues, indicating that frontal alpha asymmetry scores are not stable throughout infancy. Our results may also be in line with an earlier study of temperament and frontal alpha asymmetry (Fox et al., 2001). Fox and colleagues collected repeated measures of infant frontal alpha asymmetry at 9, 14, 24, and 48 months and focused on comparing asymmetry scores between continuously inhibited, continuously uninhibited, and comparison groups within each time assessment. Although not statistically tested, their results also suggest a shift from greater left to greater right asymmetry, but only in the comparison group, which may more likely correspond to our community-recruited sample.

Developmental change from 8 to 18 months was complemented by little to no rank-order stability of frontal alpha asymmetry scores. That is, infants who showed greater left or right alpha asymmetry at 8 months did not necessarily retain this characterization at the follow-up assessments. In fact, in some cases, an infant may have completely shifted from greater left to greater right frontal asymmetry (and vice versa), as indicated by significant, negative correlations between asymmetry scores at 8 and 18 months for the lights-off condition. Previous studies have reported moderate rank-order stability between two assessments of frontal alpha asymmetry scores across short- (e.g., weeks) and long-term (e.g., 6 months) intervals within the first year in infancy (Brooker et al., 2017), and across a span of nearly 6 years (Müller et al., 2015). It is possible that when taking only two assessments, we miss shifts in frontal alpha asymmetry that are just outside the window of the short interval. Conversely, we may miss substantial developmental change taking place in between very long intervals. The weak rank-order stability we observed in this sample is consistent with another study that examined frontal alpha asymmetry with three or more assessments (Howarth et al., 2016). Here, the authors reported weak to no stability across 10, 24, and 36 months.

These data suggest that, at least in infancy, frontal alpha asymmetry is less likely to represent a stable trait marker of approach or avoidance tendencies. Rather, the measure may be most predictive of the infant's current state or the probability of a specific response to acute or immediate environmental triggers. The capability model of EEG asymmetry (Coan et al., 2006; Reznik & Allen, 2018) suggests that an individual's ability to respond or strategy in adapting to specific task demands is captured by examining EEG patterns when the individual is concurrently experiencing an emotionally evocative or motivating situation (Stewart et al., 2014). Here, of course, all infants provided EEG during

a resting-state task, which is in line with the dispositional model (Coan et al., 2006) that presumes that individuals have a general tendency to approach or withdraw across many contexts. This approach is of course an easy conceptual parallel to our view of temperamental traits as early appearing, biologically based, and relatively stable markers of behavioral trends across contexts. As can be seen in our data linking EEG asymmetry to temperament measures, this link is neither straightforward nor particularly stable.

Finally, we looked to describe the relation between frontal alpha asymmetry and parent-reported infant temperament to probe the presumed relations between asymmetry as an approach (greater left) or withdrawal (greater right) metric. In general, the specific correlations that reached statistical significance cutoffs fluctuated across time assessments. At 8 months, the relations were largely nonsignificant, and it is difficult to assess the robustness of the one positive relation. In contrast, a more expansive set of relations were evident at 12 months. That is, greater left frontal asymmetry was associated with the approach measures of Surgency, High-Intensity Pleasure, and Approach. In addition, greater right frontal asymmetry was associated with the withdrawal metric of Fear. Even here the relations emerged under some conditions (lights on) but not others (lights off), and frontal alpha asymmetry was not stably associated with any given temperament subscale over time across either condition. As such, it would appear that the relations, although logically consistent, may not be robust. Indeed, by 18 months the relations with lights-off asymmetry scores were noted for the Pleasure subscale and marginally with the Object Fear subscale, whereas lights-on asymmetry scores were significantly associated with the Activity subscale and with Soothability. The different relations across lights-on and lights-off conditions may also point to important distinctions in these experimental parameters, despite moderate to strong correlations (range *r*s = .64–.86) in alpha power between these two conditions. For example, recent work has shown that alpha peak frequency and power increase when visual input is restricted (Webster & Ro, 2020). It is possible that in our lights-off condition, a dark room would have restricted infants' visual input enough to produce individual differences in alpha peak and power, which could then permeate to produce distinct associations with temperament. Altogether, our findings suggest that future studies should test associations between temperament and frontal alpha asymmetry separately by condition.

It is not clear, a priori, why these specific relations would emerge at specific ages as a function of lights on versus lights off. However, as noted above, the fact that our measures were taken at rest may have degraded any straightforward relation between frontal alpha asymmetry and temperamental traits. Capturing frontal alpha asymmetry during active stimuli processing (as in the capability model) may have generated more consistent one-to-one relations. In addition, the pattern of data may also point to the fact that frontal alpha asymmetry, rather than serving as a stable trait-level marker, may be better conceptualized as a moderator or mediator of relations between predictors and outcomes (Gatzke-Kopp et al., 2014; Reznik & Allen, 2018). For example, infants and children with high levels of negative reactivity are more likely to show increased levels of withdrawal or social difficulties when temperamental reactivity is coupled with greater right frontal alpha asymmetry (Hane et al., 2008; Henderson et al., 2001). Risk is relatively attenuated when equally reactive infants display greater left frontal asymmetry.

We also aimed to examine, for the first time, the psychometric properties of delta–beta coupling in infancy. Internal consistency of delta–beta coupling scores was poor, and this pattern was consistent across time assessments. To our knowledge, we are the first to examine the reliability of delta–beta coupling scores computed from the repeated, second-by-second time series of delta and beta power for each participant individually. Our approach was then to compare delta–beta coupling scores computed from the odd and even segments of individual participants. These results may suggest that coupling of delta–beta activity may be changing rapidly and dynamically throughout the 4 min of resting state, but subtly enough that correlation analyses do not capture clear increasing or decreasing trends.

In fact, our recent work (Anaya et al., 2020) shows that there is substantial intraindividual variation in delta–beta coupling captured in second-by-second deviations from participants' delta and beta relative power. This variation, in turn, significantly explained differences in anxiety symptoms above and beyond average delta–beta coupling scores. Therefore, individual delta–beta coupling scores may show poor reliability because second-by-second coupling is indeed changing on a micro-longitudinal scale. This suggests that researchers should be mindful of the timescales and relations they wish to investigate through the use of dynamic delta–beta coupling measures.

Indeed, when we examined the reliability of relative delta and beta power, split-half correlations and Cronbach's α were substantially better, further supporting our conclusions that poor reliability may be originating at the level of coupling, rather than at the level of "raw" neural activity. These results also indicated that the reliability of delta power was consistently lower than the reliability of beta power. Previous studies suggest that delta power is higher in children compared to adults (Knyazev, 2012), contributing the largest source of variability in power within the frequency spectrum. Hence, delta power may reflect a large source of individual differences at this age that manifest in segment-by-segment changes in power throughout the task. These deviations may then permeate into the average power levels that are used to compute reliability metrics. Clearly, there is a need to better understand delta–beta coupling as a dynamic process, which would mirror more directly dynamic connectivity between subcortical and cortical systems of the brain. Nonetheless, reliabilities for delta and beta power were adequate (0.661–0.962) across assessments, indicating that the developmental change and stability captured in our developmental analyses were robust and reliably describe developmental patterns in delta–beta coupling over time.

Average coupling scores at the frontal region remained stable across time. In contrast, coupling scores at central and parietal regions showed significant developmental change from 8 to 18 months, decreasing over time. Furthermore, significant change over time was complemented by weak to no rank-order stability. The functional interpretation of delta–beta coupling as a correlate of emotion regulation suggests that coupling scores may increase and become more positive

over time, reflecting gradual developmental gains in infant regulation strategies (Ekas et al., 2018). Instead, our findings indicated a developmental pattern from slight positive coupling toward decoupled states or coupling scores near zero. It is possible that delta–beta coupling, like other neural systems underlying emotion regulation, may show inverted growth trajectories when examined across a longer time span, significantly decreasing throughout infancy and pointedly increasing later. This developmental pattern would map onto imaging studies reporting that amygdala–ventromedial prefrontal cortex (vmPFC) connectivity, a network implicated in the processing and updating of affective stimuli, shows inverted trajectories that change from positive connectivity in childhood to negative connectivity in adolescence and adulthood (Moreira & Silvers, 2018). Therefore, it is possible that we may see increases in delta–beta coupling scores over this same period if we capture trajectories that extend into the preschool years when regulatory processes become more overt.

Although assessed much later in development, Moreira and Silvers (2018) show that neural systems of regulation do not always follow linear trajectories that map onto linear gains in regulation abilities. It is possible that delta–beta coupling maps onto the subcortical–cortical crosstalk captured by amygdala–vmPFC connectivity, and that if measured along infancy and into preschool, a nonlinear, U-shaped trajectory of delta–beta coupling may best describe the data. Disruptions in amygdala–vmPFC circuitry are associated with emotion dysregulation symptoms that underlie anxiety (Etkin, 2012), in the same direction that decoupling and over-coupling of delta–beta activity have been associated with psychopathology risk, making this circuitry an ideal candidate for cross-validating delta–beta coupling with functional Magnetic Resonance Imgaging (fMRI) patterns. Studies that investigate developmental trajectories of amygdala–vmPFC connectivity and delta–beta coupling trajectories in the same children from childhood into adolescence will be ideally suited to test these suppositions.

Finally, we examined concurrent associations between delta–beta coupling scores and parent-reported infant temperament. These associations were only significant at 12 months, suggesting that these are not stable relations over time. Over-coupling of delta–beta activity in parietal regions seemed to be correlated with an infant behavioral profile of lower attention control, regulation, and sadness. These results are in line with most studies of delta–beta coupling that identify over-coupled patterns as a neural marker of psychopathology risk. However, the directionality of these associations is not entirely intuitive. For example, Miskovic and Schmidt (2009) suggested that negative coupling or decoupled patterns of delta–beta activity were indicative of unregulated states. Therefore, one might expect that more negative, rather than positive, coupling would be associated with temperament dimensions of low regulation.

It is possible that developmental links between delta–beta coupling and psychopathology risk along the infancy period may not be distinctive enough to capture clear directional effects between delta–beta coupling and internalizing and externalizing risk. Instead, delta–beta over-coupling in infancy may be associated with an underlying vulnerability factor that begins to differentiate later in development. Developmental studies of internalizing and externalizing behaviors may support this interpretation (Dougherty et al., 2015; Lahey & Waldman, 2007), showing high comorbidity between internalizing and externalizing tendencies in childhood (Willner et al., 2016) and greater specificity later in development (Cosgrove et al., 2011; Ormel et al., 2005). Therefore, over-coupled patterns of delta–beta power in infancy may also track risk for dysregulated and disinhibited phenotypes, broadly defined.

Alternatively, it is possible that the level of analysis at which we measure delta–beta coupling may fundamentally change the strength and direction of associations with psychopathology risk. We expected that delta–beta over-coupling would be associated with temperament tendencies of high fear, based on two previous studies that report this association in infancy (Brooker et al., 2016; Phelps et al., 2016). However, these infant studies, like most adult studies, examined delta–beta coupling through group-level correlations between average delta and beta power. This approach provides no information regarding an individual's degree of coupling in delta–beta activity. Indeed, recent work has shown that even when group-level coupling is positive, average delta–beta coupling scores for many individual participants can be negative (Poole et al., 2020), and that when average coupling scores are positive, intraindividual coupling patterns may show negative slopes (Anaya et al., 2020). Therefore, it is possible that unexpected links between positive delta–beta coupling scores and dysregulation risk may be a product of modeling delta–beta coupling at individual levels. As more studies continue to link individual variation in delta–beta coupling with individual levels of psychopathology risk, the field may start to rethink the functional interpretation of delta–beta coupling.

5.1 | Limitations

The strengths of the present study should be considered in the context of certain limitations. First, EEG data were re-referenced offline to the average of P7/P8, a less common reference scheme in developmental neurophysiological research. The broader study from which these data were collected was extensive, including repeated behavioral, eye-tracking, EEG, and questionnaire assessments at multiple research locations in the first 2 years of life. To reduce participant burden and expedite the capping process, a subset of electrodes related to our primary questions of interest were prioritized. This decision limited our ability to meet certain assumptions of more commonly used referencing schemes.

As noted above, P7/P8 were selected because these sites were not biased toward one hemisphere and were minimally influenced by undesired sources of interference to the signal (Luck, 2014). It is worth noting that these sites are adjacent to the temporalis muscle, which may introduce noise into our data. Given that no reference is truly electrically neutral, hemispherically balanced, and void of muscle influence, we determined P7/P8 to reasonably meet assumptions for an appropriate reference. In most cases, these sites were also relatively far from scalp sites of interest, with the exception of the parietal electrode sites (P3/P4) used in a subset of the delta–beta coupling analyses. The

proximity of P3/P4 to our reference electrodes may have influenced activity to appear relatively smaller than the absolute voltage may actually be. Results related to delta–beta coupling in the parietal region should therefore be interpreted with caution. Going forward, replication using alternative referencing schemes (e.g., common average, mastoid average) is warranted to determine comparability of our findings on the psychometric properties of these neural signals to the broader literature.

Another limitation of this study was our reliance on parent-reported individual differences in temperament. In doing so, we were able to use validated scales directly tailored to longitudinal use in our specific age window. However, it is not clear that our construct validity patterns would necessarily be the same were we to assess temperamental differences via direct observation. Parent-report scales typically assess broader patterns of behavior and emotion over time and across a number of contexts. In comparison, laboratory measures of temperament implement standardized and controlled observations with equivalent triggers for all infants. Future work can compare the triangulation between biological measures of trait inclination, parent report, and direct observation.

Relatedly, in assessing the temperament measures, we have focused exclusively on patterns of zero-order correlations, presented measure-by-measure and age-by-age. It may be that more comprehensive approaches that consolidate the data with variable- (e.g., factor analysis) or person-centered (e.g., latent profile analysis) techniques may find unique relations with our EEG measures, as these approaches can account for noisy measures and extract unique underlying patterns (Vallorani et al., 2021). In addition, as noted above, the EEG-derived measures may more reliably act as moderators or mediators of temperamentally linked developmental trajectories. As such, the lack of direct zero-order relations would not negatively impact these forms of emergent relations.

Lastly, it is important to note that the EEG data presented were collected from June 2017 through March 2020, at which time in-laboratory data collection was suspended due to COVID-19 mitigation. This impacted the available sample for the 18- and 24-month time assessments. Even with this unexpected data loss, sample data presented here are comparable to previous longitudinal and cross-sectional examinations of these measures.

6 | CONCLUSION

In conclusion, our data reveal new insights regarding the developmental stability, reliability, and validity of frontal alpha asymmetry and delta–beta coupling in infancy. These insights are a mere steppingstone to better understanding these neural measures in relation to both normative development and early psychopathology risk through a developmental lens. Our findings provide crucial psychometric information for future studies. Particularly, these studies may expand on the investigation of frontal alpha asymmetry and delta–beta coupling trajectories by investigating individual and environmental factors that contribute to change over time, and what specific trajectories during infancy may be linked to adaptive functioning versus greater risk for psychopathology outcomes later in development.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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