Regular Article

Electrophysiological correlates of inhibitory control in children: Relations with prenatal maternal risk factors and child psychopathology

Xiaoye Xu[1,9], George A. Buzzell[2,3], Maureen E. Bowers[4], Lauren C. Shuffrey[5], Stephanie C. Leach[4], Marco McSweeney[4], Lydia Yoder[4], William P. Fifer[6], Michael M. Myers[6], Amy J. Elliott[7,8], Nathan A. Fox[4] and Santiago Morales[9]

[1]Department of Psychology, State University of New York at Cortland, Cortland, NY, USA, [2]Department of Psychology, Florida International University, Miami, FL, USA, [3]Center for Children and Families, Miami, FL, USA, [4]Department of Human Development and Quantitative Methodology, The University of Maryland at College Park, College Park, MD, USA, [5]Department of Child and Adolescent Psychiatry, NYU Grossman School of Medicine, New York, NY, USA, [6]Department of Psychiatry, Columbia University, New York, NY, USA, [7]Avera Research Institute, Sioux Falls, SD, USA, [8]Department of Pediatrics, University of South Dakota School of Medicine, Sioux Falls, SD, USA and [9]Department of Psychology, University of Southern California, Los Angeles, CA, USA

Abstract

Inhibitory control plays an important role in children's cognitive and socioemotional development, including their psychopathology. It has been established that contextual factors such as socioeconomic status (SES) and parents' psychopathology are associated with children's inhibitory control. However, the relations between the neural correlates of inhibitory control and contextual factors have been rarely examined in longitudinal studies. In the present study, we used both event-related potential (ERP) components and time-frequency measures of inhibitory control to evaluate the neural pathways between contextual factors, including prenatal SES and maternal psychopathology, and children's behavioral and emotional problems in a large sample of children ($N = 560$; 51.75% females; $M_{age} = 7.13$ years; Range$_{age} = $ 4–11 years). Results showed that theta power, which was positively predicted by prenatal SES and was negatively related to children's externalizing problems, mediated the longitudinal and negative relation between them. ERP amplitudes and latencies did not mediate the longitudinal association between prenatal risk factors (i.e., prenatal SES and maternal psychopathology) and children's internalizing and externalizing problems. Our findings increase our understanding of the neural pathways linking early risk factors to children's psychopathology.

Keywords: child psychopathology; electrophysiological assessments; inhibitory control; mediation; prenatal risk factors

(Received 10 May 2023; revised 15 March 2024; accepted 18 March 2024; First Published online 24 April 2024)

Inhibitory control, the cognitive ability of controlling attention and behavior to suppress a dominant response in favor of a subdominant response, plays an important role in children's cognitive and socioemotional development, including the development of psychopathology (e.g., Olson et al., 2011; Yavuz et al., 2022). The development of inhibitory control is influenced by biological and experiential factors. Increasing evidence building on the developmental origins of health and disease (DOHaD) framework has established the role of in-utero experiences in shaping children's developmental outcomes (Doyle & Cicchetti, 2018), including both inhibitory control and psychopathology. Although the link between variations in prenatal SES and mood and children's risk for psychopathology has been established, the developmental pathways linking these prenatal risk factors to later socioemotional development remain not well understood

(e.g., Shackman & Gee, 2023). One possible pathway is linked to the neural correlates of inhibitory control. Neural correlates of inhibitory control index the neurobiological mechanisms underlying the development of inhibitory control and may provide a unique perspective on the developmental pathway linking prenatal risk to children's psychopathology that cannot be captured with behavioral measures (Buzzell et al., 2020). In the present study, we examined the roles of ERP components and time-frequency measures of inhibitory control in the longitudinal relations between prenatal risk factors (i.e., SES and maternal psychopathology) and children's psychopathology.

Neurophysiological correlates of inhibitory control

ERPs

The most studied neurophysiological correlates of inhibitory control are N2 and P3 ERP components, which have been found to be involved in children's inhibitory control and have also been related to children's psychopathology (Hoyniak & Petersen, 2019; Riggins & Scott, 2020). The N2 component is the second negative

deflection in the waveform and occurs approximately 200–400 ms post-stimulus in children over frontal areas (Bruin & Wijers, 2002). This component has been associated with conflict monitoring and response inhibition (Hoyniak, 2017). Specifically, the literature has mostly examined N2 amplitude and N2 latency to peak as measures of the N2 component. Elicited by tasks that require inhibitory control (e.g., the Go/No-go task), N2 amplitude is significantly larger in magnitude to stimuli that require inhibition compared to stimuli that require a prepotent response (Conejero et al., 2023; Hoyniak & Petersen, 2019). As children grow older, they become better at ignoring distracting information, so decreases in the difference between the N2 amplitude of stimuli that require inhibition and the N2 amplitude of stimuli that require a prepotent response could indicate a decrease in their processing of irrelevant information (Lo, 2018). However, recent studies with young children have not found a significant difference between N2 amplitudes of the two types of stimuli (Algarín et al., 2013; Andreu et al., 2023; Hosch et al., 2024; Lamm et al., 2012; Sullivan et al., 2022), suggesting inconsistencies in the presence of the differences in N2 amplitudes in early development (Hoyniak, 2017). Besides N2 amplitude, N2 latency to the peak is another measure, which is defined as the interval between the onset of the stimulus and the most negative point of N2 (Jodo & Kayama, 1992). Shorter N2 latency to peak may represent more efficient response inhibition in response to the inhibition stimuli compared to the activation stimuli (Smith et al., 2010).

The P3 component is a positive-going wave peaking between 300 and 600 ms for adults and is maximal over central-parietal areas (Riggins & Scott, 2020). Similar to the N2 component, the literature has focused on the P3 amplitude and P3 latency to peak as measures of the P3 component. Children's P3 latency to peak is delayed or longer than adults, and as children age, their latency to peak can decrease by 3.6–18.4 ms per year (Riggins & Scott, 2020). The P3 is primarily elicited in tasks that engage attention, memory, and problem solving (Polich, 2007). Similar to the N2 component, the P3 could be elicited by response inhibition tasks like the Go/No-go task due to the P3's direct or indirect relations with inhibition (Huster et al., 2013; Luijten et al., 2014). However, compared to the N2, the P3 is thought to be more closely related to cognitive evaluation processes related to decision making, rather than conflict monitoring (Polich, 2007). The P3 amplitude is larger in magnitude in response to the inhibition stimuli (No-go) compared to stimuli that require a prepotent response (Go) (Conejero et al., 2023; Eimer, 1993; Kopp et al., 1996). More efficient response inhibition could also be represented by shorter P3 latency to peak in response to the inhibition stimuli compared with the prepotent stimuli (Johnstone et al., 2005).

Time-frequency analyses

While ERPs have proven to be productive for our understanding of inhibitory control across development, they have several important limitations. ERP analyses do not fully utilize the information contained in the EEG signal, as they do not distinguish different frequencies of oscillations and assume the component of interest is temporally consistent across trials (Cohen, 2014; Morales & Bowers, 2022). In contrast, time-frequency measures separately measure signal strength (EEG power) and signal consistency across trials (intertrial phase synchrony; ITPS). Importantly, it has been well documented that signal consistency increases with age across childhood, such that low consistency may be a characteristic of immature neural systems (Bowers et al., 2018; DuPuis et al., 2015; Morales et al., 2022, 2023). Moreover, given that neuronal

oscillations are a fundamental property of brain function (Buzsaki & Draguhn, 2004), time-frequency measures have been proposed to provide a neurophysiological mechanism underlying the processes captured by the EEG data like inhibitory control (Cavanagh & Frank, 2014). In this way, compared with ERP measures, time-frequency measures provide a more direct measure of the neural mechanisms underlying inhibitory control, linking early prenatal risk to later psychopathology.

Although time-frequency analyses and ERPs are complementary as measures of inhibitory control, most research to date on inhibitory control has exclusively focused on ERPs. The relatively few time-frequency analyses of children's inhibitory control have primarily focused on the theta frequency band (4–8 Hz; Adam et al., 2020; Nigbur et al., 2011). Theta oscillations have been related to the N2 component (Cavanagh & Frank, 2014). Signal strength (power) in the theta band in midfrontal areas is increased when children and adults engage in cognitive control to detect and signal the need for control to other brain systems that are involved in attention or action selection (Adam et al., 2020; Meyer et al., 2019; Nigbur et al., 2011; Watson & Bell, 2013). Moreover, similar to ERPs, the latency to peak of midfrontal theta power in response to inhibitory control has been found to be faster among older children, potentially indicating increased efficiency in information processing and inhibitory control implementation. In addition, signal consistency (ITPS) in the theta band has been found to be higher for conditions that require inhibition (e.g., No-go) compared with other conditions (e.g., Go) (Papenberg et al., 2013), suggesting that theta consistency could also be a time-frequency measure of inhibitory control in childhood.

Similar to theta oscillations, delta (1–4 Hz) oscillations have also been studied as an electrophysiological measure related to inhibitory control (Prada et al., 2014). Theta oscillations are thought to contribute to the N2 component, and delta oscillations are thought to contribute to the P3 component (Cavanagh & Frank, 2014; Prada et al., 2014). Using Go/No-go and stop trial tasks, researchers found that enhanced signal strength (power) in the delta band could index response inhibition (Schmiedt-Fehr & Basar-Eroglu, 2011). In addition, delta signal consistency (ITPS) has been found to be higher for conditions that require inhibition (e.g., No-go) compared with other conditions (e.g., Go) (Papenberg et al., 2013; Schmiedt-Fehr & Basar-Eroglu, 2011), suggesting that consistency in the delta band could also be a time-frequency measure related to inhibitory control.

Relations between risk factors and children's inhibitory control

Although there has been limited work linking prenatal risk factors to inhibitory control, several risk factors, including both SES and maternal psychopathology, have been well established as important predictors of children's inhibitory control. For instance, SES and maternal psychopathology are thought to impact children's self-regulation through several mechanisms, including parenting behaviors (Warnock et al., 2016) and stress-related pathways in the postnatal period (Evans & Kim, 2013). Mothers with psychopathology may find parenting challenging, particularly during children's early years, resulting in lower supportive parenting behaviors like sensitivity (NICHD Early Child Care Research Network, 1999). Parenting behaviors, in turn, could predict children's inhibitory control (Geeraerts et al., 2021). For SES, greater financial resources can reduce stress in the household and increase parental investment, providing a more cognitively

enriching environment for children to develop inhibitory control (Brody et al., 2002; Hoff et al., 2002). The relatively few studies examining prenatal mechanisms support the DOHaD framework by suggesting potential prenatal long-term influences (Lewis et al., 2015; Monk et al., 2019). Specifically, prenatal and perinatal maternal psychopathology and distress could influence child development via fetal brain development and programing effects, including hormonal priming effects, alteration of placental function and perfusion, and epigenetic mechanisms (Lewis et al., 2015; Monk et al., 2019). Although prenatal psychopathology has been found to predict children's inhibitory control at 6–9 years (Buss et al., 2011), to our knowledge, no studies have examined prenatal effects on electrophysiological markers of inhibitory control. Instead, existing studies have documented relations between SES and maternal psychopathology and electrophysiological markers of inhibitory control from infancy to adolescence. For instance, SES had positive and concurrent associations with children's N2 and P3 amplitudes (John et al., 2019; Kishiyama et al., 2009). Theta power evoked in response to errors was smaller in toddlers from low-SES families, compared with toddlers from high-SES families (Conejero et al., 2016). Maternal education was longitudinally and positively related to the resting theta power at 10 and 24 months (Bernier et al., 2016). For findings on maternal psychopathology, Connell et al. (2019) found that compared with adolescent girls (10–14 years) of non-depressed mothers, daughters of depressed mothers exhibited a less negative N2 amplitude and a smaller P3 amplitude. Although it has been documented that prenatal risk factors (e.g., maternal SES and distress) is related to electrophysiological markers in children (e.g., Hernandez et al., 2024; Pierce et al., 2019), these studies have been done using EEG at rest and have mostly collected data during infancy. To our knowledge, few studies have examined the longitudinal predictions from earlier SES and maternal psychopathology to later electrophysiological markers of children's inhibitory control, especially when SES and maternal psychopathology were measured during the prenatal period to support the DOHaD framework. To fill this gap in the literature and better understand the development of children's inhibitory control, we investigated relations between early risk factors, including prenatal SES and maternal psychopathology, and children's later event-related electrophysiological measures, including ERP components and power and consistency in the theta and delta bands.

Inhibitory control's relation to psychopathology

Children's inhibitory control has been found to be an important predictor of children's psychopathology (see Lipszyc & Schachar, 2010 for a meta-analytic review). Regarding associations with ERP measures, the magnitude of N2 amplitude has been found to be negatively associated with externalizing psychopathology in childhood (see Hoyniak & Petersen, 2019 for a meta-analytic review). Additionally, children with a smaller P3 amplitude displayed more aggression symptoms (Petersen et al., 2018). Similarly, children with high internalizing problems had a smaller P3 amplitude than children with low internalizing problems, and their P3 amplitude was the smallest when they had both internalizing and externalizing problems (Hill et al., 1999). For time-frequency measures, reduced mediofrontal theta is thought to be an endophenotype for externalizing disorders for adolescents and young adults (Gilmore et al., 2010; Kamarajan et al., 2015).

Moreover, because earlier factors like SES and maternal psychopathology have both been found to be important predictors

of children's psychopathology (e.g., see Goodman et al., 2011 and Peverill et al., 2021 for meta-analytic reviews), inhibitory control could act as a mediator in the prediction from earlier factors to children's psychopathology. For example, data from the Bucharest Early Intervention Project suggest that the development of event-related mediofrontal theta across the adolescent period (12–16 years) was negatively predicted by the early neglect children experienced in Romanian institutions (Buzzell et al., 2020). Moreover, changes in mediofrontal theta development were related to adolescents' psychopathology during the same period, and importantly, reduced theta power acted as a mediator in the positive relation between neglect and adolescents' psychopathology (Buzzell et al., 2020). This finding is similar to studies with rodents that revealed that repeated exposure to maternal neglect during a sensitive period of rodent growth is related to reduced mediofrontal theta power during the juvenile period (Reincke & Hanganu-Opatz, 2017). In the current study, we would like to extend the existing literature by examining the mediating role of both the ERP components and time-frequency measures of children's inhibitory control in the relations between prenatal risk factors and children's psychopathology. Supporting the DOHaD framework (Doyle & Cicchetti, 2018), prenatal risk factors, including maternal mood and SES, have been widely investigated in their predictions of children's internalizing and externalizing problems (Doyle & Cicchetti, 2018). Besides other potential biological mechanisms related to hormones and the immune system (Tien et al., 2020), neurophysiological indices of children's inhibitory control could serve as a developmental pathway in the associations between prenatal risk factors and children's internalizing and externalizing problems.

The current study

In the current study, we used children's ERPs and time-frequency measures of inhibitory control to examine their associations with maternal prenatal risk factors and examine if they predicted children's behavioral problems. Specifically, we had three aims in the current study. The first aim was to examine if individual differences in ERP measures and time-frequency measures of inhibitory control could be predicted by prenatal maternal factors. Based on prior research, we hypothesized that higher prenatal SES would be related to better inhibitory control skills, indexed by larger N2 magnitude, P3 amplitude, and theta/delta power and ITPS, as well as shorter latency to peak for all the ERPs and time-frequency measures. We also expected that higher prenatal maternal psychopathology would be related to lower levels of inhibitory control, indexed by smaller N2 magnitude, P3 amplitude, and theta/delta power and ITPS and longer latency to peak for all the ERPs and time-frequency measures.

The second aim was to examine if inhibitory control and children's emotional and behavioral problems had concurrent associations. We hypothesized that higher N2 magnitude, P3 amplitude, and theta/delta power and ITPS would be related to less internalizing and externalizing problems, as well as longer latency to peak for all the ERP measures and time-frequency measures would be related to more internalizing and externalizing problems.

The third aim directly builds on the first two aims. We investigated if any of the ERPs and time-frequency measures of inhibitory control could act as mediators in the relations between prenatal risk factors and children's behavioral problems. We hypothesized that both ERPs (i.e., N2 amplitude and latency to peak, P3 amplitude and latency to peak) and time-frequency

measures (i.e., theta/delta power, consistency across trials, and latency to peak) would act as mediators of the negative relations between SES and children's internalizing and externalizing problems, and the positive relations between maternal psychopathology and children's internalizing and externalizing problems.

Method

Participants

Children ($N = 560$; age range = 4–11 years ($M_{age} = 7.13$ years); 51.75% females) and their biological mothers were recruited as part of a large longitudinal study that examined how early environmental exposures could influence children's development. Participants included in this analysis were originally recruited as part of the Safe Passage Study (Dukes et al., 2014) and are now participants in the NIH-funded Environment Influences on Child Health Outcomes network (Gillman & Blaisdell, 2018). Data were collected in two sites in South Dakota. Most participants were White (81.23%), followed by American Indian (12.98%), and other (5.79%). Most participants were non-Latinx (95.79%). Data on prenatal risk factors, including SES and maternal psychopathology, were reported by mothers during the prenatal period. Mothers reported on average 14.87 years of education (Range = 7–17 years) and $3297 as average monthly income (Range = $250–$5000) during the prenatal period. Data on children's EEG were collected at the following ages: 4 ($n = 70$; $M_{age} = 4.23$; $SD_{age} = .15$), 5 ($n = 122$; $M_{age} = 5.23$; $SD_{age} = .15$), 7 ($n = 185$; $M_{age} = 7.22$; $SD_{age} = .14$), 9 ($n = 97$; $M_{age} = 9.24$; $SD_{age} = .13$), and 11 ($n = 86$; $M_{age} = 11.24$; $SD_{age} = .14$). Internalizing and externalizing behaviors were collected concurrently with the EEG assessment or at a later timepoint. EEG and children's psychopathology were assessed concurrently for most children (75%), while children's psychopathology was assessed two (18%) or three (7%) years later for some children. Four-year-old children had EEG data but did not have data on parent reports of internalizing and externalizing behaviors. Participants with and without data on internalizing and externalizing behaviors did not differ in any indicators of prenatal SES and prenatal maternal psychopathology nor by children's race and ethnicity. The only difference was in children's sex ratio. Participants with data on internalizing and externalizing behaviors had a higher percentage (56%) of females than participants without data on internalizing and externalizing behaviors (44%), $\chi^2(1) = 7.18$, $p = .007$.

Procedure

All mothers provided informed consent, and children, ages eight and older, provided assent before data collection. During the prenatal period, mothers self-reported their depression and anxiety symptoms as well as demographic information, including SES (i.e., income, education, and information about insurance). When children were 4–11 years old, they came to the laboratory with their primary caregivers for health (e.g., anthropometrics and spirometry) and cognitive assessments, including the Go/No-Go task called the "Zoo Game," which was administered while acquiring EEG. E-Prime 2.0.10 (Psychology Software Tools, Pittsburgh, PA) was used to present the task. The code and stimuli for the task are publicly available (see https://github.com/ChildDevLab/Tasks). Children sat approximately 70 cm in front of the presentation computer during task administration. The Zoo Game commenced after children completed a 3-minute baseline and a three-stimulus auditory oddball paradigm (Morales et al., 2022) as part of a separate protocol. Primary caregivers reported children's

internalizing and externalizing problems while children completed the EEG and/or other assessments. At the end of the visit, families were compensated, and children were provided a small toy. All tasks and procedures were approved by the Avera Institutional Review Board.

EEG data acquisition

EEG data were obtained through a 64-channel HydroCel Geodesic Sensor Net, sampled at 500 Hz and with a gain of 12 dB via EGI software (Net Station Version 5.4; Electrical Geodesics, Inc., Eugene, OR). We removed four face channels (E61–E64) of the nets because they were not used to collect EEG data but other psychophysiological data (e.g., heart rate). Before data collection, the research staff checked impedance values for all EEG channels and confirmed them to be below 50 kΩ.

Zoo game (Go/No-Go task)

ERP components and event-related delta/theta power were collected through the Zoo Game. As previously described (Morales et al., 2022), the Zoo Game is a computer-based Go/No-go task developed by Fox and colleagues (He et al., 2010; Morales et al., 2020; Troller-Renfree et al., 2019) based on the design of Durston et al. (2002). In the task, children were asked to help a zookeeper catch animals that escaped from cages in a zoo. They were told to catch all animals except the orangutans because the orangutans were the zookeeper's assistant, helping to catch other animals. Children who were four years old were presented with only one picture of an orangutan, while children who were older than four years were presented with three different pictures of orangutans. In Go trials, after children saw pictures of animals that were not orangutans, they needed to press a button to catch them. In No-go trials, after children saw pictures of orangutans, they needed to withhold their response without pressing any button. On each trial, an animal stimulus was presented on the screen for 750 ms, followed by a blank screen for 500 ms or until the child pressed the button (whichever occurred first). The intertrial interval was 500–1000 ms. Children first practiced the task until the experimenter thought they understood the task procedures. After the practice trials, children completed up to 320 trials, including eight blocks with 40 trials in each block. The task had 75% Go trials and 25% No-go trials. For both Go and No-go trials, children needed to finish at least half of the trials and were accurate in 60% or higher rate of the trials to be included in subsequent analyses. The average numbers of Go trials were 118.03, 140.06, 158.58, 190.58, and 204.72 for 4-, 5-, 7-, 9-, and 11-year-old children, respectively. The average numbers of No-go trials were 32.26, 36.02, 39.25, 49.13, and 55.93 for 4-, 5-, 7-, 9-, and 11-year-old children, respectively. The number of trials were related with measures of SES and EEG (see supplementary materials; Table S1). After controlling for age, number of trials were related to children's theta power and externalizing problems ($rs = .08 - .26$, $ps < .05$). Importantly, a sensitivity analysis showed that controlling for the number of trials showed the same pattern of results, including a significant mediation (see supplementary materials; Table S5).

EEG preprocessing

The preprocessing of the data has been previously described in Morales et al. (2022). In brief, we conducted the EEG preprocessing using the EEGLAB toolbox (Delorme & Makeig, 2004) with custom MATLAB scripts (The MathWorks, Natick, MA) using the Maryland Analysis of Developmental EEG pipeline (Debnath

et al., 2020). Continuous EEG data were high-pass filtered at 0.3 Hz and low-pass filtered at 49 Hz. We identified and removed bad channels using the EEGLAB plug-in FASTER (Nolan et al., 2010). To remove ocular and other stereotyped artifacts, we utilized independent component analysis (ICA). Artifactual independent components were removed from the original dataset using the Adjusted-ADJUST algorithm (Leach et al., 2020; Mognon et al., 2011). The data were then segmented into three-second epochs for two additional steps of artifact rejection. First, to capture the presence of residual ocular activity which was kept after ICA, we rejected any epochs in which ocular channel (E1, E5, E10, and E17) voltages exceeded $\pm$ 150 μV. Second, we interpolated non-ocular channels that exceeded $\pm$ 125 μV at the epoch level. However, if over 10% of the channels (not considering globally rejected channels) exceeded $\pm$ 125 μV, we rejected the epoch instead. Then, we interpolated any remaining missing channels using the spherical spline method (Perrin et al., 1989). The data was then referenced to the average reference. All epochs were time-locked to the presentation of the stimuli (i.e., animal) using only correct trials. Moreover, we excluded trials with anticipatory responses (<150 ms).

ERPs

ERPs for each child were averaged separately for each of the two conditions (Go and No-go). We baseline corrected the average voltage in the −200 to 0 ms pre-stimulus period, using mean amplitude measures for each child and each condition. The time windows and electrode clusters for each ERP component (N2 and P3) were selected based on previous publications with developmental populations (John et al., 2019; Sullivan et al., 2022) and visual inspection. For the N2, we focused on the frontocentral electrode cluster (FCz; E4, E7, and E54) 250–500 ms after the stimulus. For the P3, we focused on the parietal electrode cluster (E33, E34, E36, and E38) 400–700 ms after the stimulus. A figure of the electrode clusters is shown in the supplementary materials (Figure S2).

Time-frequency (TF) power

We computed TF power in each epoch of interest using custom MATLAB scripts (Morales & Bowers, 2022), adapted from Cohen (2014). First, we applied a surface Laplacian filter to the epoched data to mitigate volume conduction over the scalp by filtering out spatially broad features of the data (Cohen, 2014), so that both spatial and functional specificity of brain activity were improved (Tenke & Kayser, 2012). Each epoch was convolved with Morlet wavelets, which estimated spectral power in the frequency range of $1 - 30$ Hz (in 60 steps spaced logarithmically). To optimize the TF resolution and balance the tradeoff between frequency and time resolution, we set three wavelet cycles at the lowest frequency (1 Hz) and gradually increased the number of wavelet cycles to 10 for the highest frequency (30 Hz). We calculated power separately for correct trials in each of the Go and No-go conditions for all channels. For each condition, we normalized power using a (dB) transform (dB power = 10 $\times$ log 10[power/baseline]). In the transform, the baseline was the average power for the condition from -300 to -100 ms before the stimulus was presented (Delorme & Makeig, 2004). Finally, to facilitate data manipulation, we downsampled data to 50 Hz following TF decomposition.

ITPS

We computed ITPS using custom MATLAB scripts (Morales & Bowers, 2022), adapted from Cohen (2014). ITPS measures the consistency of phase oscillations across trials for each frequency and each timepoint. ITPS values range from zero, which indicates completely random phase alignment at a certain time point, to one, which indicates perfect phase alignment at a certain time point. We computed ITPS by first taking the phase angle difference across trials, and then averaging the phase angle differences. When we estimated ITPS, we used a subsampling procedure to eliminate biases associated with having different numbers of trials per condition (Cohen, 2014). We computed ITPS using 10 trials randomly selected per condition. This subsampling procedure was performed 50 times to make sure that all the data were used, and then all subsamples were averaged to calculate the final ITPS. ITPS was baseline corrected per condition based on − 300 to − 100 ms before stimulus onset. This process created ITPS surfaces per condition with the same dimensions as the TF measures for each electrode for each participant. Finally, to facilitate data manipulation, we downsampled data to 50 Hz following TF decomposition and computation of ITPS.

TF regions of interest selection

We selected the frequencies and time windows for the regions of interest (ROIs) a priori based on previous literature on developmental populations (Buzzell et al., 2020) and confirmed with visual inspection. In line with previous literature on young children (3–6 years; e.g., Canen & Brooker, 2017; DuPuis et al., 2015), we defined the theta frequency band to be 4–8 Hz and delta to be 1–4 Hz with overlapping boundary. We focused on delta and theta power and ITPS over the frontocentral electrodes (FCz; E4, E7, and E54). To determine the time windows of interest unbiased for condition effects, we averaged each measure over all conditions throughout the interval and created time windows around the peak while ensuring most of the peak were captured. For theta and delta power, we selected from 200 to 700 ms (theta) and from 250 to 1000ms (delta) post stimuli. For theta and delta ITPS, we selected from 0 to 500 ms (theta) and from 0 to 750 ms (delta) post stimuli.

Prenatal maternal depressive symptoms

Mothers reported their depressive symptoms during the prenatal period using the Edinburgh Postnatal Depression Scale (EPDS; Cox et al., 1987), which has 10 items rated on a 4-point scale (1= *not at all/almost never* to 4 = *as much as I always could/most of the time*). Example items include "I have felt sad or miserable" and "I have been so unhappy that I have been crying." EPDS has been validated for mothers during the pregnancy with high test-retest reliability and high concurrent validity (Bergink et al., 2011). The 10 items were first standardized as z-scores and then averaged to create a composite score of prenatal maternal depression. For mothers who completed the EPDS more than one time during pregnancy, we took the average across all their reports in pregnancy. This measure also showed high internal consistency (Cronbach's $\alpha = .82$) with the sample of the current study.

Prenatal maternal anxiety

Mothers reported their trait and state anxiety symptoms during the prenatal period using the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1971). It differentiates between "state anxiety" which refers to the temporary condition (e.g., "I am presently worrying over possible misfortunes.") and "trait anxiety" which refers to the more general and long-standing quality (e.g., "I am included to taking things hard."). Each of the two conditions has

20 items rated on a 4-point scale (1= *not at all/almost never* to 4 = *very much so/almost always*). STAI has been validated for mothers during the pregnancy with high construct and content validity (Gunning et al., 2010). It also showed high internal consistency (Cronbach's αs = .90 and .88, for state anxiety and trait anxiety, respectively) with the sample of the current study.

Child behavioral problems

Mothers reported children's internalizing and externalizing problems using the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997). SDQ contains 25 items to form 4 difficulties subscales–conduct problems (e.g., "often has temper tantrums"), hyperactivity/inattention (e.g., "constantly fidgeting"), emotional problems (e.g., "many fears, easily scared"), and peer problems (e.g., "rather solitary, prefers to play alone"), and a prosocial subscale (e.g., "kind to younger children"). Parents rated statements on a 4-point scale (0 = *not true*, 1 = *somewhat true*, or 2 = *certainly true*). SDQ has been validated for parent reports on school-aged children with high construct, concurrent, discriminate, and predictive validity (Stone et al., 2010). In the current study, we used the four difficulties subscales to measure children's behavioral problems. We combined emotional and peer problems to create the internalizing problem subscale and combined conduct problems and hyperactivity/inattention to create the externalizing problem subscale. The two types of behavioral problems showed adequate internal consistency (Cronbach's αs = .67 and .84, for internalizing and externalizing problems, respectively) with the sample of the current study.

Prenatal socioeconomic status (SES)

For SES, we included mothers' reported household income, education level, and insurance type (i.e., public or private insurance) during the prenatal period. We included information on insurance as a measure of SES because accessing to health care is an important aspect of SES (Adler & Newman, 2002). Moreover, insurance information has been found to be strongly correlated with other SES measures, including income and education level (Casey et al., 2018), as was the case in our sample.

Analytic approach

First, in order to examine differences in all ERPs and TF measures between Go and No-Go conditions, we used *t*-tests to examine the main effect of conditions (Go vs. No-Go). If the effect of interest was significant, indicating that the ERP or TF measures successfully captured a correlate of inhibitory control, we then conducted correlations with other study variables. Second, we examined the partial correlations between prenatal predictors (maternal psychopathology and SES), neurophysiological measures, and children's psychopathology, while controlling for age. In order to examine the mediating role of ERPs and TF measures in the prediction from prenatal SES and maternal psychopathology to children's internalizing and externalizing problems, we first examined if there were significant relations from at least one prenatal predictor to an EEG-based mediator and from the same mediator to at least one outcome. Third, after identifying potential mediators based on the partial correlations, we performed a structural equation model (SEM) using the *lavaan* package in R (Rosseel, 2012) to examine potential mediation effects using the bootstrap method to define the confidence intervals for mediation effects using 10,000 bootstrap samples (Mackinnon et al., 2004). Because we did not have specific hypotheses regarding individual

components of SES or types of maternal psychopathology, we created latent measures of prenatal SES (education level, income, and insurance) and psychopathology (state and trait anxiety and depression). This model utilized full information maximum likelihood estimation to account for missing data and MLR estimator to deal with non-normality and non-independence of the variables. For evaluating model fit, we followed Hu and Bentler's (1999) guidelines.

Results

Descriptive analyses and differences between Go and No-Go conditions

Descriptive statistics and partial correlations controlling for age among all the study and control variables are presented in Table 1. Correlations among all study variables, including age, can be found in the supplementary materials. As expected, as age increased, accuracy in the Go ($r = .61$) and No-Go ($r = .29$) conditions increased, while reaction time in both conditions decreased (Go, $r = -.77$, No-Go, $r = -.69$). In addition, behavioral indices of inhibitory control were related with indices of SES and children's externalizing behaviors. Higher SES predicted higher accuracy and slower reaction times, and higher accuracy and slower reaction times predicted lower externalizing behaviors (Table S1). However, behavioral indices were not related to either SES or children's psychopathology after controlling for child age and sex.

Differences between Go and No-Go conditions

ERP amplitudes
As shown in Figure 1, in contrast to the hypothesis, we found a more positive amplitude deflection between 250 and 500 ms in frontocentral electrodes in the No-go condition ($M = -9.72$), compared to the Go condition ($M = -11.46$), $t(559) = 13.74$, $p < .001$, $d = .58$. Because of this, we did not explore further the role of the N2 in its relations with prenatal predictors or children's psychopathology. As expected, we observed a clear P3 component, indexed by a more positive amplitude deflection between 400 and 700 ms in parietal electrodes in the No-Go condition ($M = 24.35$), compared to the Go condition ($M = 22.57$), $t(559) = 9.45$, $p < .001$, $d = .40$.

TF power
As shown in Figure 2, between 250 and 750 ms in frontocentral electrodes, we observed a higher theta power in the No-Go condition ($M = 1.23$), compared to the Go condition ($M = .76$), $t(559) = 12.27$, $p < .001$, $d = .52$. Between 250 and 1000ms in frontocentral electrodes, we observed a higher delta power in the No-Go condition ($M = .79$), compared to the Go condition ($M = .50$), $t(559) = 6.74$, $p < .001$, $d = .29$.

TF ITPS
As shown in Figure 3, between 250 and 750 ms in frontocentral electrodes, we observed a higher theta ITPS in the No-Go condition ($M = .05$), compared to the Go condition ($M = .04$), $t(559) = 4.60$, $p < .001$, $d = .19$. Between 250 and 1000ms in frontocentral electrodes, we observed a higher delta ITPS in the No-Go condition ($M = .11$), compared to the Go condition ($M = .08$), $t(559) = 11.04$, $p < .001$, $d = .47$.

Primary analyses

Based on the correlations shown in Table 1, theta power was the only potential mediator that was related to at least one of the

Table 1. Means, standard deviations, and partial correlations of all study variables and covariates controlling for age

Variable	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. Child Sex	.48	.50																		
2. Maternal Education	.03	.98	−.04																	
3. Household Income	.02	.98	−.03	.55**																
4. Health Insurance	.71	.46	−.01	.53**	.54**															
5. Depression	.00	.98	.01	−.08	−.22**	−.14**														
6. Trait Anxiety	.01	1.01	−.05	−.10*	−.22**	−.16**	.72**													
7. State Anxiety	.00	1.00	−.07	−.06	−.19**	−.15**	.55**	.66**												
8. N2 Amplitude	1.56	2.69	.05	.15**	−.01	.08	−.07	−.07	−.04											
9. P3 Amplitude	1.78	4.46	.07	−.04	.01	−.06	.01	−.00	−.07	−.31**										
10. Theta Power	.47	.90	.13**	.08	.13**	.06	.00	−.02	−.05	.07	−.02									
11. Delta Power	.29	1.02	.07	−.06	.01	−.02	−.02	−.03	−.06	.03	.07	.38**								
12. Theta ITPS	.02	1.00	−.07	−.06	−.06	.01	.00	.03	.04	−.00	−.04	.05	.03							
13. Delta ITPS	.00	1.01	−.06	−.00	−.01	−.01	−.07	−.06	−.03	−.11**	.11*	.04	.25**	.17**						
14. N2 Latency	.00	1.01	.06	−.12*	−.08	−.18**	−.01	.03	−.03	−.19**	.18**	.00	−.05	.03	−.05					
15. P3 Latency	.01	1.00	−.03	.02	.03	.00	.03	.03	−.04	.03	.14**	.04	−.06	−.12**	−.07	.03				
16. Theta Latency	.00	1.00	.01	−.05	−.03	.04	.02	.01	−.05	−.03	.04	.16**	.15**	−.09*	.00	.07	.20**			
17. Delta Latency	.00	1.00	.04	−.02	.00	.00	−.02	−.01	−.04	.08	−.01	.09	.27**	.08*	−.09*	.03	−.02	.18**		
18. External	4.27	3.31	.23**	−.10	−.24**	−.13*	.25**	.21**	.17**	−.11*	−.04	−.12*	−.01	−.01	−.01	.04	.00	−.03	−.02	
19. Internal	2.86	2.58	.00	−.16**	−.28**	−.19**	.24**	.22**	.17**	−.01	−.07	−.08	.01	.05	−.03	−.03	.08	−.02	−.09	.43**

Note. M = mean; SD = standard deviation; External = children's externalizing problems; Internal = children's internalizing problems; For Child Sex, 0 = female, 1 = male; For Health Insurance, 0 = public assistance, 1 = commercial health insurance. Maternal education, household income, maternal depression, maternal trait anxiety, maternal state anxiety, theta ITPS, delta ITPS, and all latency measures were standardized by their means and standard deviations.
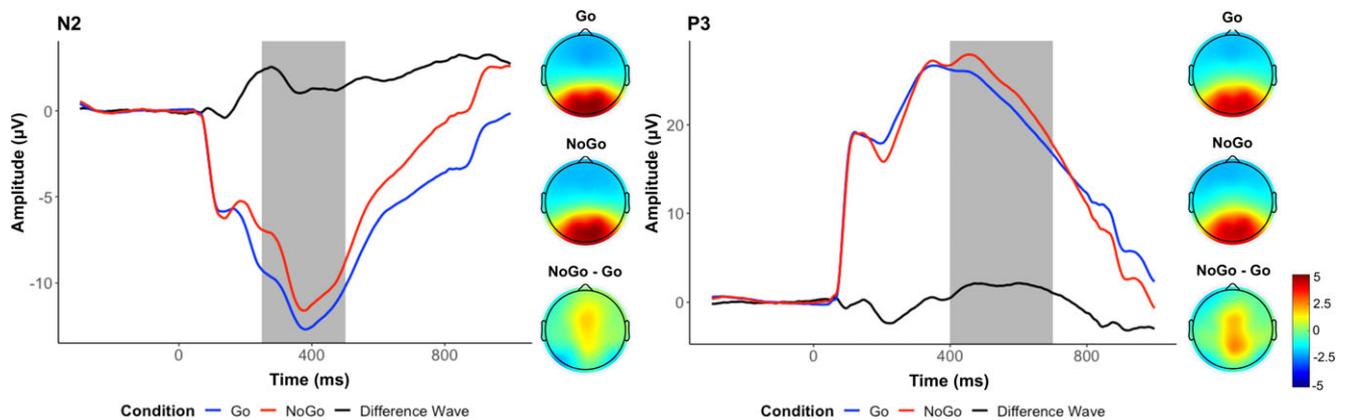*p < .05. **p < .01.

Figure 1. Event-related potential measures: N2 and P3. Average waveforms across all participants for the N2 (left) and the P3 (right), and topographs for each condition and their difference during the selected time window (shaded area).
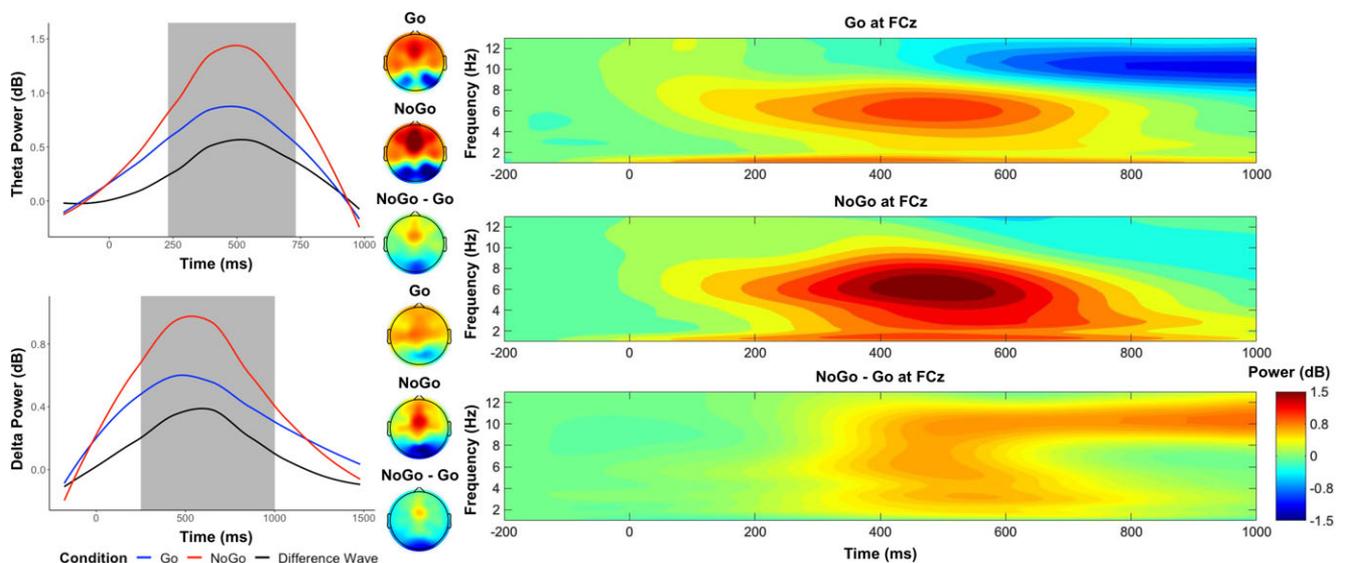

Figure 2. Time–frequency dynamics of theta and delta power of Go and No-go trials at the FCz cluster. Plots show time–frequency power for each condition for theta (top left panel) and delta (bottom left panel), and topographs for each condition and their difference during the selected time window (shaded area). The right panels display the time-frequency surfaces of power by condition for frontocentral cluster.

prenatal predictors (i.e., prenatal SES and maternal psychopathology) and at least one of children's behavioral problems (i.e., internalizing and externalizing behaviors). Thus, we only examined the mediating role of theta power in the relationships between prenatal SES and children's externalizing problems because both prenatal SES and children's externalizing problems were correlated with theta power. Theta power as the mediator was represented by the difference score between the Go and No-go trials. In the SEM model, SES, child gender, and child age were specified as predictors to predict the mediator and externalizing problems, which was specified as the outcome to be predicted by both the predictors and the mediator. Specifically, maternal SES was a latent variable created by the three aspects of SES: education level, income, and insurance type because the scores on the three subscales were moderately and positively correlated. The standardized factor loadings for education level, income, and insurance type were .71, .78, and .72, respectively ($ps < .001$). The model demonstrated good fit for the data, $\chi^2(9) = 19.44$, $p = .02$; CFI = .98; TLI = .94; SRMR = .03; RMSEA = .05; 95% CI [.02,

.08]. The model indicated that maternal SES was positively related to theta power. Theta power and maternal SES were negatively related to children's externalizing problems. Further, we found support for a significant mediation, and the mediation estimated effect was −.069, such that theta power significantly mediated the relation between maternal SES and children's externalizing problems with 95% CIs [−.196, −.005] after 10,000 times of bootstrapping. Significant results are summarized in Figure 4, and all other results (nonsignificant results and results of control variables) are displayed in Table S2 of supplementary materials.

Sensitivity analyses were conducted as some participants in our current sample (four-year-old children) did not have parental assessments of internalizing and externalizing behaviors. Results of the models with and without four-year-old children yielded the same results; thus, we decided to include four-year-old children in models to increase the overall sample size. In addition, because not all children had psychopathology data collected at the time of their EEG measures, we conducted an analysis that included children with only concurrent measures. As shown in the supplementary
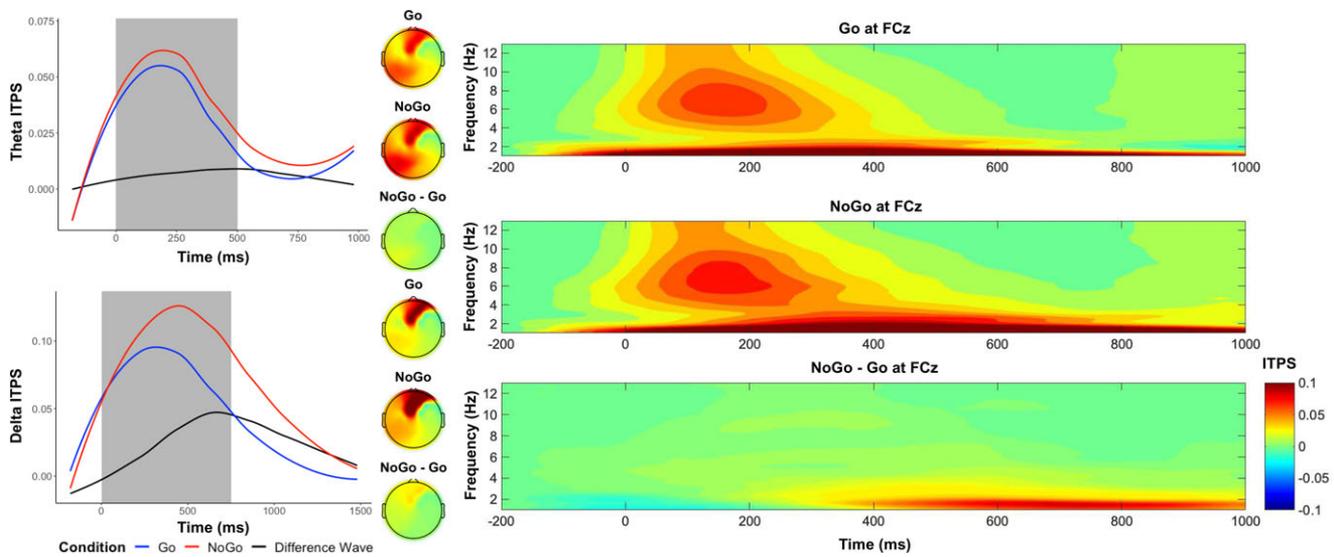
Figure 3. Time–frequency dynamics of theta and delta intertial phase synchrony (ITPS) of Go and No-go trials at the FCz cluster. Plots show time–frequency ITPS for each condition for theta (top left panel) and delta (bottom left panel), and topographs for each condition and their difference during the selected time window (shaded area). The right panels display the time-frequency surfaces of ITPS by condition for frontocentral cluster.
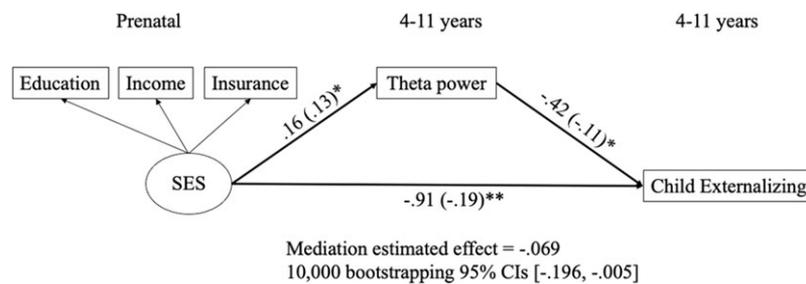

Figure 4. Relations of prenatal SES, theta power, and children's externalizing behaviors. All predictions were controlled for child sex and child age. Unstandarized and standardized (in parentheses) estimates were presented. See Table S2 for results of nonsignificant results and results of control variables. *p < .05. **p < .01.

materials (Table S4), results from this sensitivity analysis revealed a similar pattern of results. Although the effect sizes were similar, the relation between theta and externalizing was only marginally significant (p = .08), likely due to the reduction in sample size. Finally, a sensitivity analysis was conducted while controlling for the number of trials, finding an analogous pattern of results. Although the relation between SES and theta power was marginally significant (p = .06), the effect size was similar, and the indirect effects of the mediator were still significant leading to the same conclusions (see supplementary materials; Table S5).

Discussion

In the current study, we examined if ERPs and time-frequency measures of inhibitory control were significant mediators in the relation between prenatal risk factors and children's psychopathology. We found that children's theta power mediated the negative relation between prenatal SES and children's externalizing problems. Our findings support the DOHaD framework by highlighting the role of prenatal risk factors on the development of children's neurophysiological correlates of inhibitory control and psychopathology. Moreover, consistent with previous findings (Buzzell et al., 2020), the results further support the role of

mediofrontal theta power as a developmental pathway linking early risk factors to later children's psychopathology.

In support of the use of neurophysiological measures of inhibitory control, most of the ERP and time-frequency measures, including P3 amplitudes, theta and delta power, theta and delta consistency across trials, and latency to peak of all ERPs and time-frequency measures, were found to represent children's inhibitory control as indexed by a larger magnitude in the inhibition condition (No-go trials) than in the activation condition (Go trials). It suggests that P3 measures and event-related theta and delta measures could be used to measure children's inhibitory control from preschool years to early adolescence. The only exception was N2 amplitude, exhibiting a more negative magnitude in the activation condition (Go trials) than the inhibition condition (No-go trials). Similarly, Hosch et al. (2024) and Sullivan et al. (2022) both found a more negative magnitude of N2 amplitudes in the activation condition (Go trials) than in the inhibition condition (No-go trials) in children whose average age was around 5 years. In a systematic review and meta-analysis of children's No-go N2 component, Hoyniak (2017) found several studies that reported more negative magnitudes of N2 amplitudes in the activation condition (Go trials) than the inhibition condition (No-go trials) in children (e.g., Algarín et al., 2013; Trinkl et al., 2015). One possibility for these inconsistent findings in the

literature is that the N2 changes in morphology, making it difficult to know if the same component is being captured across ages or if the changes in amplitude represent different components. Similarly, it is possible that earlier components (e.g., P2) or later components (e.g., P3) overlap with the N2 differently at different ages. In addition, N2 of the No-go condition could be more variable in children, making it smaller in averaged data. This may be especially salient for child-friendly Go/No-go tasks, which use more engaging stimuli to keep children's attention instead of more controlled stimuli (e.g., simple Xs and Os commonly used with adults). This highlights one of the challenges of ERPs, which rely on correctly identifying the components. In contrast, time-frequency analyses represent the EEG data as oscillations, providing a more direct representation of the neurophysiological mechanisms without having to correctly identify changing or overlapping ERP components (Morales & Bowers, 2022). Similarly, in this study, we observed theta and delta power and consistency effects between conditions, while the N2 effects were less clear. Moreover, individual differences in theta power were related to prenatal risk and children's psychopathology.

When examining potential developmental pathways, children's theta power mediated the negative relation between prenatal SES and children's externalizing problem. Specifically, prenatal SES negatively and longitudinally predicted children's theta power, which also had a negative and concurrent relation with their externalizing problems. Similarly, Lecheile et al. (2020) found SES predicted children's later effortful control, which includes inhibitory control, and was measured by parent report survey and behavioral tasks. In addition, theta power evoked in response to errors and resting theta power have both been related to measures of SES (Bernier et al., 2016; Conejero et al., 2016). Thus, the finding in the current study supports the emerging EEG literature linking SES and children's self-regulation, as indexed by theta power. The present study was the first to identify the mediating role of event-related theta power in the longitudinal negative relation between prenatal SES and children's externalizing behavior. Furthermore, our findings support reports that reduced mediofrontal theta may be an endophenotype for externalizing disorders for adolescents and young adults (Gilmore et al., 2010; Kamarajan et al., 2015). Similarly, our findings extend Buzzell et al. (2020), which found that theta power acted as a mediator in the positive relation between neglect in institutional rearing and adolescents' psychopathology. The prediction from prenatal SES to theta power supported the DOHaD framework that prenatal factors could predict children's developmental outcomes (Doyle & Cicchetti, 2018). In addition, because SES is stable and is related to parenting starting from early childhood (Azad et al., 2014), the effects of prenatal SES on inhibitory control could also be partly mediated by social interactions with parents (Hackman et al., 2015). Social interactions with caregivers have been associated with increases in theta power in both children (Wass et al., 2018) and rodent pups (Courtiol et al., 2018; Sarro et al., 2014), and are thought to be one of the mechanisms shaping theta development. Thus, it is possible that parent-child interactions lead to increases in theta power, which are, in turn, protective for psychopathology. Future studies should examine this potential developmental pathway, as well as investigate the type of parenting behaviors that lead to increases in theta power during inhibitory control.

The mediation model findings showed that prenatal SES was an important predictor of children's brain development related to inhibitory control, convergent with previous findings of a positive relation between SES and self-regulation (Blair & Raver, 2012;

Lecheile et al., 2020). In addition, although not as frequently implemented as surveys and behavioral measures, electrophysiological measures of inhibitory control were also related to children's psychopathology. Interestingly, behavioral indices of inhibitory control did not mediate the relation between early risk factors and children's psychopathology in the current study. Because theta power was the only electrophysiological assessment of inhibitory control that mediated the prediction from early risk factors to children's psychopathology, the finding highlights the importance of time-frequency measures of inhibitory control. In the current study, we found a longitudinal association of prenatal SES with children's theta power, but not with theta consistency across trials. These results showed the uniqueness of different time-frequency measures and the importance of differentiating the distinct aspects of the EEG signal (strength and consistency). Another strength of time-frequency analyses pertains to the convergence of findings across species (Narayanan et al., 2013; Tsujimoto et al., 2006; Womelsdorf et al., 2010). In line with our current findings, both rodents and humans who experienced early neglect, an extreme type of deprivation, exhibited reductions in mediofrontal theta power during the juvenile period (Buzzell et al., 2020; Reincke & Hanganu-Opatz, 2017). Although the mediation role of inhibitory control in the developmental pathway from early risk factors to children's psychopathology depended on the measure of inhibitory control, we still found evidence to support the importance of neural measures of inhibitory control.

In contrast to SES, prenatal maternal psychopathology did not predict children's inhibitory control. Moreover, children's inhibitory control did not mediate the positive associations of maternal psychopathology with children's internalizing and externalizing behaviors. One explanation could be that we did not examine symptoms of maternal psychopathology on a clinical sample, so the level of maternal psychopathology was relatively low with low variance so that it was not predictable to children's inhibitory control. In addition, instead of inhibitory control, other types of children's characteristics, like emotion regulation and temperament, that were not examined in the study could be mediators of the developmental pathways between prenatal risk factors to children's psychopathology.

Limitations and future directions

The current study has several limitations. First, 4-year-old participants did not have data on internalizing and externalizing problems. That is, in the mediation models, data on early risk factors and electrophysiological correlates of inhibitory control were available for the 4-year-old group, but not for internalizing and externalizing problems. Second, the electrophysiological measures of inhibitory control and children's internalizing and externalizing problems were not collected concurrently for all participants. Future longitudinal studies should measure children's internalizing and externalizing problems and inhibitory control both concurrently and longitudinally to better address the directionality. Third, we examined the role of prenatal SES and maternal psychopathology on children's inhibitory control in early/middle childhood without considering the effects of other related factors in the postnatal environment (e.g., postnatal SES and maternal psychopathology). Although SES and maternal psychopathology are relatively stable from the prenatal period to the postnatal period, changes in these risk factors from pregnancy to early childhood have been found to be differentially related to child executive function outcomes (Morales et al., 2024; Park et al., 2018). Fourth, because maternal depression and anxiety are both

internalizing behaviors, we did not examine the predictions of prenatal maternal externalizing behaviors, which could be a focus for future research. Fifth, our study only examined prenatal risk factors. Future studies should examine and control for the effects of risk factors during the postnatal period to better understand prenatal mechanisms. Sixth, the internal consistency was low for internalizing behaviors, potentially due to relatively low levels of children's internalizing behavior in the sample. Further, based on the findings from the mediation models in the current study, future research could explore the mediating role of electrophysiological measures of inhibitory control with other important behavioral measures of inhibitory control (e.g., Lecheile et al., 2020; Spinrad et al., 2012), and other EEG paradigms (e.g., oddball).

Conclusion

The current study utilized ERPs and time-frequency measures of inhibitory control to examine if these measures could mediate the relation between prenatal risk factors and children's psychopathology. Children's theta power mediated the negative association between prenatal SES and children's externalizing problems, suggesting the importance of incorporating time-frequency measures of inhibitory control. These findings confirm that prenatal SES and maternal psychopathology play important roles in children's brain development and are related to inhibitory control and children's psychopathology. The negative relation between children's inhibitory control and their psychopathology was supported through electrophysiological measures of inhibitory control. Thus, interventions that improve families' SES and reduce maternal psychopathology during the prenatal period could help improve children's self-regulation skills and psychological well-being.

References

Adam, N., Blaye, A., Gulbinaite, R., Delorme, A., & Farrer, C. (2020). The role of midfrontal theta oscillations across the development of cognitive control in preschoolers and school-age children. *Developmental Science*, 23(5), e12936. https://doi.org/10.1111/desc.12936

Adler, N. E., & Newman, K. (2002). Socioeconomic disparities in health: Pathways and policies. *Health Affairs*, 21(2), 60–76. https://doi.org/10.1377/hlthaff.21.2.60

Algarín, C., Nelson, C. A., Peirano, P., Westerlund, A., Reyes, S., & Lozoff, B. (2013). Iron-deficiency anemia in infancy and poorer cognitive inhibitory control at age 10 years. *Developmental Medicine & Child Neurology*, 55(5), 453–458. https://doi.org/10.1111/dmcn.12118

Andreu, C. I., García-Rubio, C., Melcón, M., Schonert-Reichl, K. A., & Albert, J. (2023). The effectiveness of a school mindfulness-based intervention on the neural correlates of inhibitory control in children at risk: A randomized control trial. *Developmental Science*, 26(6), e13403. https://doi.org/10.1111/desc.13403

Azad, G., Blacher, J., & Marcoulides, G. (2014). Longitudinal models of socio-economic status: Impact on positive parenting behaviors. *International Journal of Behavioral Development*, 38(6), 509–517. https://doi.org/10.1177/0165025414532172

Bergink, V., Kooistra, L., Lambregtse-van den Berg, M. P., Wijnen, H., Bunevicius, R., van Baar, A., & Pop, V. (2011). Validation of the edinburgh depression scale during pregnancy. *Journal of Psychosomatic Research*, 70(4), 385–389. https://doi.org/10.1016/j.jpsychores.2010.07.008

Bernier, A., Calkins, S. D., & Bell, M. A. (2016). Longitudinal associations between the quality of mother-infant interactions and brain development across infancy. *Child Development*, 87(4), 1159–1174. https://doi.org/10.1111/cdev.12518

Blair, C., & Raver, C. C. (2012). Child development in the context of adversity: Experiential canalization of brain and behavior. *American Psychologist*, 67(4), 309–318. https://doi.org/10.1037/a0027493

Bowers, M. E., Buzzell, G. A., Bernat, E. M., Fox, N. A., & Barker, T. V. (2018). Time-frequency approaches to investigating changes in feedback processing during childhood and adolescence. *Psychophysiology*, 55(10), e13208. https://doi.org/10.1111/psyp.13208

Brody, G. H., Murry, V. M., Kim, S., & Brown, A. C. (2002). Longitudinal pathways to competence and psychological adjustment among African American children living in rural single-parent households. *Child Development*, 73(5), 1505–1516. https://doi.org/10.1111/1467-8624.00486

Bruin, K. J., & Wijers, A. A. (2002). Inhibition, response mode, and stimulus probability: A comparative event-related potential study. *Clinical Neurophysiology*, 113(7), 1172–1182. https://doi.org/10.1016/S1388-2457(02)00141-4

Buss, C., Davis, E. P., Hobel, C. J., & Sandman, C. (2011). Maternal pregnancy-specific anxiety is associated with child executive function at 6-9 years age. *Stress-the International Journal On The Biology of Stress*, 14(6), 665–676. https://doi.org/10.3109/10253890.2011.623250

Buzsaki, G., & Draguhn, A. (2004). Neuronal oscillations in cortical networks. *Science*, 304(5679), 1926–1929. https://doi.org/10.1126/science.1099745

Buzzell, G. A., Troller-Renfree, S. V., Wade, M., Debnath, R., Morales, S., Bowers, M. E., Zeanah, C. H., Nelson, C. A., & Fox, N. A. (2020). Adolescent cognitive control and mediofrontal theta oscillations are disrupted by neglect: Associations with transdiagnostic risk for psychopathology in a randomized controlled trial. *Developmental Cognitive Neuroscience*, 43, 100777. https://doi.org/10.1016/j.dcn.2020.100777

Canen, M. J., & Brooker, R. J. (2017). ERN, theta power, and risk for anxiety problems in preschoolers. *Biological Psychology*, 123, 103–110. https://doi.org/10.1016/j.biopsycho.2016.12.003

Casey, J. A., Pollak, J., Glymour, M. M., Mayeda, E. R., Hirsch, A. G., & Schwartz, B. S. (2018). Measures of SES for electronic health record-based research. *American Journal of Preventive Medicine*, 54(3), 430–439. https://doi.org/10.1016/j.amepre.2017.10.004

Cavanagh, J. F., & Frank, M. J. (2014). Frontal theta as a mechanism for cognitive control. *Trends in Cognitive Sciences*, 18(8), 414–421. https://doi.org/10.1016/j.tics.2014.04.012

Cohen, M. X. (2014). *Analyzing neural time series data: Theory and practice*. MIT Press.

Conejero, Á., Guerra, S., Abundis-Gutiérrez, A., & Rueda, M. R. (2016). Frontal theta activation associated with error detection in toddlers: Influence of familial socioeconomic status. *Developmental Science*, 21(1), e12494. https://doi.org/10.1111/desc.12494

Conejero, A., Rico-Picó, J., Moyano, S., Hoyo, Á., & Rueda, M. R. (2023). Predicting behavioral and brain markers of inhibitory control at preschool age from early measures of executive attention. *Frontiers in Psychology*, 14, 983361. https://doi.org/10.3389/fpsyg.2023.983361

Connell, A. M., Danzo, S., Magee, K., & Uhlman, R. (2019). Children's appraisals of maternal depression and responses to emotional faces in early-adolescence: An event related potential (ERP) study. *Journal of Affective Disorders*, 250, 241–248. https://doi.org/10.1016/j.jad.2019.03.038

Courtiol, E., Wilson, D. A., Shah, R., Sullivan, R. M., & Teixeira, C. M. (2018). Maternal regulation of pups' cortical activity: Role of serotonergic signaling. *eNeuro*, 5(4), 1–12. https://doi.org/10.1523/ENEURO.0093-18.2018

Cox, J. L., Holden, J. M., & Sagovsky, R. (1987). Detection of postnatal depression: Development of the 10-item edinburgh postnatal depression scale. *British Journal of Psychiatry*, 150(6), 782–786. https://doi.org/10.1192/bjp.150.6.782

Debnath, R., Buzzell, G. A., Morales, S., Bowers, M. E., Leach, S. C., & Fox, N. A. (2020). The Maryland analysis of developmental EEG (MADE) pipeline. *Psychophysiology*, 57(6), e13580. https://doi.org/10.1111/psyp.13580

Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, 134(1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009

Doyle, C., & Cicchetti, D. (2018). Future directions in prenatal stress research: Challenges and opportunities related to advancing our understanding of prenatal developmental origins of risk for psychopathology. *Development and Psychopathology*, 30(3), 721–724. https://doi.org/10.1017/S095457941800069X

Dukes, K. A., Burd, L., Elliott, A. J., Fifer, W. P., Folkerth, R. D., Hankins, G. D. V., Hereld, D., Hoffman, H. J., Myers, M. M., Odendaal, H. J., Signore, C., Sullivan, L. M., Willinger, M., Wright, C., Kinney, H. C., & PASS Research Network (2014). The safe passage study: Design, methods, recruitment, and follow-up approach. *Paediatric and Perinatal Epidemiology*, 28(5), 455–465. https://doi.org/10.1111/ppe.12136

DuPuis, D., Ram, N., Willner, C. J., Karalunas, S., Segalowitz, S. J., & Gatzke-Kopp, L. M. (2015). Implications of ongoing neural development for the measurement of the error-related negativity in childhood. *Developmental Science*, 18(3), 452–468. https://doi.org/10.1111/desc.12229

Durston, S., Thomas, K. M., Yang, Y., Ulug, A. M., Zimmerman, R. D., & Casey, B. J. (2002). A neural basis for the development of inhibitory control. *Developmental Science*, 5(4), F9–F16. https://doi.org/10.1111/1467-7687.00235

Eimer, M. (1993). Effects of attention and stimulus probability on ERPs in a go/Nogo task. *Biological Psychology*, 35(2), 123–138. https://doi.org/10.1016/0301-0511(93)90009-W

Evans, G. W., & Kim, P. (2013). Childhood poverty, chronic stress, self-regulation, and coping. *Child Development Perspectives*, 7(1), 43–48. https://doi.org/10.1111/cdep.12013

Geeraerts, S. B., Endendijk, J. J., Deković, M., Huijding, J., Deater-Deckard, K., & Mesman, J. (2021). Inhibitory control across the preschool years: Developmental changes and associations with parenting. *Child Development*, 92(1), 335–350. https://doi.org/10.1111/cdev.13426

Gillman, M. W., & Blaisdell, C. J. (2018). Environmental influences on child health outcomes, a research program of the NIH. *Current Opinion in Pediatrics*, 30(2), 260–262. https://doi.org/10.1097/MOP.0000000000000600

Gilmore, C. S., Malone, S. M., & Iacono, W. G. (2010). Brain electrophysiological endophenotypes for externalizing psychopathology: A multivariate approach. *Behavior Genetics*, 40(2), 186–200. https://doi.org/10.1007/s10519-010-9343-3

Goodman, R. (1997). The strengths and difficulties questionnaire: A research note. *Journal of Child Psychology and Psychiatry*, 38(5), 581–586. https://doi.org/10.1111/j.1469-7610.1997.tb01545.x

Goodman, S. H., Rouse, M. H., Connell, A. M., Broth, M. R., Hall, C. M., & Heyward, D. (2011). Maternal depression and child psychopathology: A meta-analytic review. *Clinical Child and Family Psychology Review*, 14(1), 1–27. https://doi.org/10.1007/s10567-010-0080-1

Gunning, M. D., Denison, F. C., Stockley, C. J., Ho, S. P., Sandhu, H. K., & Reynolds, R. M. (2010). Assessing maternal anxiety in pregnancy with the state-trait anxiety inventory (STAI): Issues of validity, location and participation. *Journal of Reproductive and Infant Psychology*, 28(3), 266–273. https://doi.org/10.1080/02646830903487300

Hackman, D. A., Gallop, R., Evans, G. W., & Farah, M. J. (2015). Socioeconomic status and executive function: Developmental trajectories and mediation. *Developmental Science*, 18(5), 686–702. https://doi.org/10.1111/desc.12246

He, J., Degnan, K. A., McDermott, J. M., Henderson, H. A., Hane, A. A., Xu, Q., & Fox, N. A. (2010). Anger and approach motivation in infancy: Relations to early childhood inhibitory control and behavior problems. *Infancy*, 15(3), 246–269. https://doi.org/10.1111/j.1532-7078.2009.00017.x

Hernandez, A., Sania, A., Bowers, M. E., Leach, S. C., McSweeney, M., Yoder, L., Fifer, W., Elliott, A. J., Shuffrey, L., Rauh, V., Him, D. A., Fox, N. A., & Morales, S. (2024). Examining the impact of prenatal maternal internalizing symptoms and socioeconomic status on children's frontal alpha asymmetry and psychopathology. *Developmental Psychobiology*, 66(3), e22476. https://doi.org/10.1002/dev.22476

Hill, S. Y., Shen, S., Locke, J., Steinhauer, S. R., Konicky, C., Lowers, L., & Connolly, J. (1999). Developmental delay in P300 production in children at high risk for developing alcohol-related disorders. *Biological Psychiatry*, 46(7), 970–981.

Hoff, E., Laursen, B., & Tardiff, T. (2002). Socioeconomic status and parenting. In M. H. Bornstein (Ed.), *Handbook of parenting* (pp. 231–252). Erlbaum.

Hosch, A., Swanson, B., Harris, J. L., Oleson, J. J., Hazeltine, E., & Petersen, I. T. (2024). Explaining brain-behavior relations: Inhibitory control as an intermediate phenotype between the N2 ERP and the externalizing spectrum in childhood. *Research On Child and Adolescent Psychopathology*, 52(4), 505–520. https://doi.org/10.1007/s10802-023-01162-w

Hoyniak, C. (2017). Changes in the NoGo N2 event-related potential component across childhood: A systematic review and meta-analysis. *Developmental Neuropsychology*, 42(1), 1–24. https://doi.org/10.1080/87565641.2016.1247162

Hoyniak, C. P., & Petersen, I. T. (2019). A meta-analytic evaluation of the N2 component as an endophenotype of response inhibition and externalizing psychopathology in childhood. *Neuroscience & Biobehavioral Reviews*, 103, 200–215. https://doi.org/10.1016/j.neubiorev.2019.06.011

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, 6(1), 1–55. https://doi.org/10.1080/10705519909540118

Huster, R. J., Enriquez-Geppert, S., Lavallee, C. F., Falkenstein, M., & Herrmann, C. S. (2013). Electroencephalography of response inhibition tasks: Functional networks and cognitive contributions. *International Journal of Psychophysiology*, 87(3), 217–233. https://doi.org/10.1016/j.ijpsycho.2012.08.001

Jodo, E., & Kayama, Y. (1992). Relation of a negative ERP component to response inhibition in a go/No-go task. *Electroencephalography and Clinical Neurophysiology*, 82(6), 477–482. https://doi.org/10.1016/0013-4694(92)90054-L

John, A. M., Finch, K., & Tarullo, A. R. (2019). Socioeconomic status and neural processing of a go/no-go task in preschoolers: An assessment of the P3b. *Developmental Cognitive Neuroscience*, 38, 100677. https://doi.org/10.1016/j.dcn.2019.100677

Johnstone, S. J., Pleffer, C. B., Barry, R. J., Clarke, A. R., & Smith, J. L. (2005). Development of inhibitory processing during the go/NoGo task: A behavioral and event-related potential study of children and adults. *Journal of Psychophysiology*, 19(1), 11–23. https://doi.org/10.1027/0269-8803.19.1.11

Kamarajan, C., Pandey, A. K., Chorlian, D. B., Manz, N., Stimus, A. T., Anokhin, A. P., Bauer, L. O., Kuperman, S., Kramer, J., Bucholz, K. K., Schuckit, M. A., Hesselbrock, V. M., Porjesz, B., & Abulseoud, O. A. (2015). Deficient event-related theta oscillations in individuals at risk for alcoholism: A study of reward processing and impulsivity features. *PloS One*, 10(11), e0142659. https://doi.org/10.1371/journal.pone.0142659

Kishiyama, M. M., Boyce, W. T., Jimenez, A. M., Perry, L. M., & Knight, R. T. (2009). Socioeconomic disparities affect prefrontal function in children. *Journal of Cognitive Neuroscience*, 21(6), 1106–1115. https://doi.org/10.1162/jocn.2009.21101

Kopp, B., Mattler, U., Goertz, R., & Rist, F. (1996). N2, P3 and the lateralized readiness potential in a nogo task involving selective response priming. *Electroencephalography and Clinical Neurophysiology*, 99(1), 19–27. https://doi.org/10.1016/0921-884X(96)95617-9

Lamm, C., White, L. K., McDermott, J. M., & Fox, N. A. (2012). Neural activation underlying cognitive control in the context of neutral and affectively charged pictures in children. *Brain and Cognition*, 79(3), 181–187. https://doi.org/10.1016/j.bandc.2012.02.013

Leach, S. C., Morales, S., Bowers, M. E., Buzzell, G. A., Debnath, R., Beall, D., & Fox, N. A. (2020). Adjusting ADJUST: Optimizing the ADJUST algorithm for pediatric data using geodesic nets. *Psychophysiology*, 57(8), e13566. https://doi.org/10.1111/psyp.13566

Lecheile, B. M., Spinrad, T. L., Xu, X., Lopez, J., & Eisenberg, N. (2020). Longitudinal relations among household chaos, SES, and effortful control in the prediction of language skills in early childhood. *Developmental Psychology*, 56(4), 727–738. https://doi.org/10.1037/dev0000896

Lewis, A. J., Austin, E., Knapp, R., Vaiano, T., & Galbally, M. (2015). Perinatal maternal mental health, fetal programming and child development. *Healthcare*, *3*(4), 1212–1227. https://doi.org/10.3390/healthcare3041212

Lipszyc, J., & Schachar, R. (2010). Inhibitory control and psychopathology: A meta-analysis of studies using the stop signal task. *Journal of the International Neuropsychological Society*, *16*(6), 1064–1076. https://doi.org/10.1017/S1355617710000895

Lo, S. L. (2018). A meta-analytic review of the event-related potentials (ERN and N2) in childhood and adolescence: Providing a developmental perspective on the conflict monitoring theory. *Developmental Review*, *48*, 82–112. https://doi.org/10.1016/j.dr.2018.03.005

Luijten, M., Machielsen, M., Veltman, D., Hester, R., de Haan, L., & Franken, I. (2014). Systematic review of ERP and fMRI studies investigating inhibitory control and error processing in people. *Journal of Psychiatry & Neuroscience*, *39*(3), 149–169. https://doi.org/10.1503/jpn.130052

MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavioral Research*, *39*(1), 99–128. https://doi.org/10.1207/s15327906mbr3901_4

Meyer, M., Endedijk, H. M., Van Ede, F., & Hunnius, S. (2019). Theta oscillations in 4-year-olds are sensitive to task engagement and task demands. *Scientific Reports*, *9*(1), 1–11. https://doi.org/10.1038/s41598-019-42615-x

Mognon, A., Jovicich, J., Bruzzone, L., & Buiatti, M. (2011). ADJUST: An automatic EEG artifact detector based on the joint use of spatial and temporal features. *Psychophysiology*, *48*(2), 229–240. https://doi.org/10.1111/j.1469-8986.2010.01061.x

Monk, C., Lugo-Candelas, C., & Trumpff, C. (2019). Prenatal developmental origins of future psychopathology: Mechanisms and pathways. *Annual Review of Clinical Psychology*, *15*(1), 317–344. https://doi.org/10.1146/annurev-clinpsy-050718-095539

Morales, S., & Bowers, M. E. (2022). Time-frequency analysis methods and their application in developmental EEG data. *Developmental Cognitive Neuroscience*, *54*, 101067. https://doi.org/10.1016/j.dcn.2022.101067

Morales, S., Bowers, M. E., Leach, S. C., Buzzell, G. A., Fifer, W., Elliott, A. J., & Fox, N. A. (2023). Time-frequency dynamics of error monitoring in childhood: An EEG study. *Developmental Psychobiology*, *64*(3), e22215. https://doi.org/10.1002/dev.22215

Morales, S., Bowers, M. E., Leach, S. C., Buzzell, G. A., McSweeney, M., Yoder, L., Fifer, W., Elliott, A. J., & Fox, N. A. (2022). Development of auditory change-detection and attentional capture, and their relation to inhibitory control. *Psychophysiology*, *60*(4), e14211. https://doi.org/10.1111/psyp.14211

Morales, S., Bowers, M. E., Shuffrey, L., Ziegler, K., Troller-Renfree, S., Hernandez, A., Leach, S. C., McGrath, M., Ola, C., Leve, L. D., Nozadi, S. S., Swingler, M. M., Lai, J.-S., Schweitzer, J. B., Fifer, W., Camargo, C. A., Khurana Hershey, G. K., Shapiro, A. L. B., Keating, D. P., Hartert, T. V., Deoni, S., Ferrara, A., & Elliott, A. J. (2024). Maternal education prospectively predicts child neurocognitive function: An environmental influences on child health outcomes study. *Developmental Psychology*. Advance online publication. https://doi.org/10.1037/dev0001642

Morales, S., Miller, N. V., Troller-Renfree, S. V., White, L. K., Degnan, K. A., Henderson, H. A., & Fox, N. A. (2020). Attention bias to reward predicts behavioral problems and moderates early risk to externalizing and attention problems. *Development and Psychopathology*, *32*(2), 397–409. https://doi.org/10.1017/S0954579419000166

Narayanan, N. S., Cavanagh, J. F., Frank, M. J., & Laubach, M. (2013). Common medial frontal mechanisms of adaptive control in humans and rodents. *Nature Neuroscience*, *16*(12), 1888–1895. https://doi.org/10.1038/nn.3549

NICHD Early Child Care Research Network, (1999). Chronicity of maternal depressive symptoms, maternal sensitivity, and child functioning at 36 months. NICHD Early Child Care Research Network. *Developmental Psychology*, *35*, 1297–1310. https://doi.org/10.1037/0012-1649.35.5.1297

Nigbur, R., Ivanova, G., & Stürmer, B. (2011). Theta power as a marker for cognitive interference. *Clinical Neurophysiology*, *122*(11), 2185–2194. https://doi.org/10.1016/j.clinph.2011.03.030

Nolan, H., Whelan, R., & Reilly, R. B. (2010). FASTER: Fully automated statistical EEG artifact rejection. *Journal of Neuroscience Methods*, *192*(1), 152–162. https://doi.org/10.1016/j.jneumeth.2010.07.015

Olson, S. L., Tardif, T. Z., Miller, A., Felt, B., Grabell, A. S., Kessler, D., Wang, L., Karasawa, M., & Hirabayashi, H. (2011). Inhibitory control and harsh discipline as predictors of externalizing problems in young children: A comparative study of US, Chinese, and Japanese preschoolers. *Journal of Abnormal Child Psychology*, *39*(8), 1163–1175. https://doi.org/10.1007/s10802-011-9531-5

Papenberg, G., Hämmerer, D., Müller, V., Lindenberger, U., & Li, S.-C. (2013). Lower theta inter-trial phase coherence during performance monitoring is related to higher reaction time variability: A lifespan study. *NeuroImage*, *83*, 912–920. https://doi.org/10.1016/j.neuroimage.2013.07.032

Park, M., Brain, U., Grunau, R. E., Diamond, A., & Oberlander, T. F. (2018). Maternal depression trajectories from pregnancy to 3 years postpartum are associated with children's behavior and executive functions at 3 and 6 years. *Archives of Women's Mental Health*, *21*(3), 353–363. https://doi.org/10.1007/s00737-017-0803-0

Perrin, F., Pernier, J., Bertrand, O., & Echallier, J. F. (1989). Spherical splines for scalp potential and current density mapping. *Electroencephalography and Clinical Neurophysiology*, *72*(2), 184–187. https://doi.org/10.1016/0013-4694(89)90180-6

Petersen, I. T., Hoyniak, C. P., Bates, J. E., Staples, A. D., & Molfese, D. L. (2018). A longitudinal, within-person investigation of the association between the P3 ERP component and externalizing behavior problems in young children. *Journal of Child Psychology and Psychiatry*, *59*(10), 1044–1051. https://doi.org/10.1111/jcpp.12975

Peverill, M., Dirks, M. A., Narvaja, T., Herts, K. L., Comer, J. S., & McLaughlin, K. A. (2021). Socioeconomic status and child psychopathology in the United States: A meta-analysis of population-based studies. *Clinical Psychology Review*, *83*, 101933. https://doi.org/10.1016/j.cpr.2020.101933

Pierce, L. J., Thompson, B. L., Gharib, A., Schlueter, L., Reilly, E., Valdes, V., Roberts, S., Conroy, K., Levitt, P., & Nelson, C. A. (2019). Association of perceived maternal stress during the perinatal period with electroencephalography patterns in 2-month-old infants. *JAMA Pediatrics*, *173*(6), 561–570. https://doi.org/10.1001/jamapediatrics.2019.0492

Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology*, *118*(10), 2128–2148. https://doi.org/10.1016/j.clinph.2007.04.019

Prada, L., Barceló, F., Herrmann, C. S., & Escera, C. (2014). EEG delta oscillations index inhibitory control of contextual novelty to both irrelevant distracters and relevant task-switch cues: P300-delta response indexes contextual novelty. *Psychophysiology*, *51*(7), 658–672. https://doi.org/10.1111/psyp.12210

Reincke, S. A. J., & Hanganu-Opatz, I. L. (2017). Early-life stress impairs recognition memory and perturbs the functional maturation of prefrontal-hippocampal-perirhinal networks. *Scientific Reports*, *7*(1), 1–16.

Riggins, T., & Scott, L. S. (2020). P300 development from infancy to adolescence. *Psychophysiology*, *57*(7), e13346. https://doi.org/10.1111/psyp.13346

Rosseel, Y. (2012). Lavaan: An *R* package for structural equation modeling. *Journal of Statistical Software*, *48*(2), 1–36. https://doi.org/10.18637/jss.v048.i02

Sarro, E. C., Wilson, D. A., & Sullivan, R. M. (2014). Maternal regulation of infant brain state. *Current Biology*, *24*(14), 1664–1669. https://doi.org/10.1016/j.cub.2014.06.017

Schmiedt-Fehr, C., & Basar-Eroglu, C. (2011). Event-related delta and theta brain oscillations reflect age-related changes in both a general and a specific neuronal inhibitory mechanism. *Clinical Neurophysiology*, *122*(6), 1156–1167. https://doi.org/10.1016/j.clinph.2010.10.045

Shackman, A. J., & Gee, D. G. (2023). Maternal perinatal stress associated with offspring negative emotionality, but the underlying mechanisms remain elusive. *American Journal of Psychiatry*, *180*(10), 708–711. https://doi.org/10.1176/appi.ajp.20230630

Smith, J. L., Smith, E. A., Provost, A. L., & Heathcote, A. (2010). Sequence effects support the conflict theory of N2 and P3 in the go/NoGo task. *International Journal of Psychophysiology*, *75*(3), 217–226. https://doi.org/10.1016/j.ijpsycho.2009.11.002

Spielberger, C. D., Gonzalez-Reigosa, F., Martinez-Urrutia, A., Natalicio, L. F., & Natalicio, D. S. (1971). The state-trait anxiety inventory. *Revista Interamericana De Psicologia/Interamerican Journal of Psychology*, *5*(3 & 4), 145–158. https://doi.org/10.30849/rip/ijp.v5i3%20&%204.620.

Spinrad, T. L., Eisenberg, N., Silva, K. M., Eggum, N. D., Reiser, M., Edwards, A., Iyer, R., Kupfer, A. S., Hofer, C., Smith, C. L., Hayashi, A., & Gaertner, B. M. (2012). Longitudinal relations among maternal behaviors, effortful control and young children's committed compliance. *Developmental Psychology*, *48*(2), 552–566. https://doi.org/10.1037/a0025898

Stone, L. L., Otten, R., Engels, R. C. M. E., Vermulst, A. A., & Janssens, J. M. A. M. (2010). Psychometric properties of the parent and teacher versions of the strengths and difficulties questionnaire for 4- to 12-year-olds: A review. *Clinical Child and Family Psychology Review*, *13*(3), 254–274. https://doi.org/10.1007/s10567-010-0071-2

Sullivan, E. F., Xie, W., Conte, S., Richards, J. E., Shama, T., Haque, R., Petri, W. A., & Nelson, C. A. (2022). Neural correlates of inhibitory control and associations with cognitive outcomes in Bangladeshi children exposed to early adversities. *Developmental Science*, *25*(5), e13245. https://doi.org/10.1111/desc.13245

Tenke, C. E., & Kayser, J. (2012). Generator localization by current source density (CSD): Implications of volume conduction and field closure at intracranial and scalp resolutions. *Clinical Neurophysiology*, *123*(12), 2328–2345. https://doi.org/10.1016/j.clinph.2012.06.005

Tien, J., Lewis, G. D., & Liu, J. (2020). Prenatal risk factors for internalizing and externalizing problems in childhood. *World Journal of Pediatrics*, *16*(4), 341–355. https://doi.org/10.1007/s12519-019-00319-2

Trinkl, M., Greimel, E., Bartling, J., Grünewald, B., Schulte-Körne, G., & Grossheinrich, N. (2015). Right-lateralization of N2-amplitudes in depressive adolescents: An emotional go/no-go study. *Journal of Child Psychology and Psychiatry*, *56*(1), 76–86. https://doi.org/10.1111/jcpp.12282

Troller-Renfree, S. V., Buzzell, G. A., Bowers, M. E., Salo, V. C., Forman-Alberti, A., Smith, E., Papp, L. J., McDermott, J. M., Pine, D. S., Henderson, H. A., & Fox, N. A. (2019). Development of inhibitory control during childhood and its relations to early temperament and later social anxiety: Unique insights provided by latent growth modeling and signal detection theory. *Journal of Child Psychology and Psychiatry*, *60*(6), 622–629. https://doi.org/10.1111/jcpp.13025

Tsujimoto, T., Shimazu, H., & Isomura, Y. (2006). Direct recording of theta oscillations in primate prefrontal and anterior cingulate cortices. *Journal of Neurophysiology*, *95*(5), 2987–3000. https://doi.org/10.1152/jn.00730.2005

Warnock, F. F., Craig, K. D., Bakeman, R., Castral, T., & Mirlashari, J. (2016). The relationship of prenatal maternal depression or anxiety to maternal caregiving behavior and infant behavior self-regulation during infant heel lance: An ethological time-based study of behavior. *BMC Pregnancy and Childbirth*, *16*(1), 1–12. https://doi.org/10.1186/s12884-016-1050-5

Wass, S. V., Noreika, V., Georgieva, S., Clackson, K., Brightman, L., Nutbrown, R., Covarrubias, L. S., Leong, V., & Menon, V. (2018). Parental neural responsivity to infants' visual attention: How mature brains influence immature brains during social interaction. *PLoS Biology*, *16*(12), e2006328. https://doi.org/10.1371/journal.pbio.2006328

Watson, A. J., & Bell, M. A. (2013). Individual differences in inhibitory control skills at three years of age. *Developmental Neuropsychology*, *38*(1), 1–21. https://doi.org/10.1080/87565641.2012.718818

Womelsdorf, T., Johnston, K., Vinck, M., & Everling, S. (2010). Theta-activity in anterior cingulate cortex predicts task rules and their adjustments following errors. *Proceedings of The National Academy of Sciences of The United States of America*, *107*(11), 5248–5253. https://doi.org/10.1073/pnas.0906194107

Yavuz, H. M., Dys, S., & Malti, T. (2022). Parental discipline, child inhibitory control and prosocial behaviors: The indirect relations via child sympathy. *Journal of Child and Family Studies*, *31*(5), 1276–1289. https://doi.org/10.1007/s10826-021-02224-7