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Intergenerational transmission of cognitive control capacity among children at risk for depression

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ABSTRACT

A maternal history of major depressive disorder (MDD) is a well-known risk factor for depression in offspring. However, the mechanism through which familial risk is transmitted remains unclear. Cognitive control alterations are common in MDD, and thus, the current study investigated whether altered control capacity is transmitted intergenerationally, and whether it then contributes to the developmental pathways through which depression is passed from mothers to children. We recruited children (N = 65) ages 4–10-years-old, of which 47.7 % (n = 31) reported a maternal history of MDD, and their biological mother (N = 65). Children performed a child-friendly Go/NoGo task while electroencephalography (EEG) data were recorded, and mothers performed a Flanker task. Children exhibited heightened sensitivity to error versus correct responses, which was characterized by an error-related negativity (ERN), error positivity (Pe) as well as prominent delta and frontal midline theta (FMT) oscillations. Interestingly, worse maternal performance on the Flanker task associated with an increased Go/NoGo error rate and a smaller ERN and Pe in children. However, there was no association between maternal or child control indices with child depression symptoms. Our results suggest a familial influence of cognitive control capacity in mother-child dyads, but it remains unclear whether this confers risk for depressive symptoms in children. Further research is necessary to determine whether alterations in cognitive control over time may influence symptom development in at-risk children.

1. Introduction

Major depressive disorder (MDD) is a leading cause of disability worldwide. Rates of depression increase across childhood, and the peak onset occurs during adolescence (Avenevoli, Swendsen, He, Burstein, & Merikangas, 2015). A parental history of MDD is one of the most robust risk factors for depression among youth (Weissman et al., 2006) and is associated with an earlier onset as well as higher risk for more chronic depressive symptoms in offspring (Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002). However, the mechanism through which familial risk is transmitted remains unclear. Thus, identifying factors that confer risk during a critical developmental period may offer targeted intervention before clinically significant symptoms emerge.

The Cognitive Systems within the Research Domain Criteria (RDoC) offers a useful heuristic to identify intergenerational risk factors, particularly cognitive control capacity, which is a well-known contributor to the development and maintenance of MDD (De Raedt & Koster, 2010; Edwards et al., 2022; Nelson et al., 2018; Pizzagalli, 2011; Wagner, Müller, Helmreich, Huss, & Tadić, 2015). Cognitive control facilitates goal-directed behavior by overcoming automatic or habitual

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thoughts and responses, and compared to healthy individuals, poorer cognitive control performance is common in MDD among youth (Wagner et al., 2015) and adults (Snyder, 2013). Further, Kertz et al. (2016) found that parental report of reduced control in preschoolers predicted increased depressive and anxiety symptoms in children over a 7.5-year period. Similarly, Nelson et al. (2018) expanded this finding and showed that children's poor control task performance at age 5 predicted depression and anxiety symptoms in elementary school. Accordingly, the development of control capacity may be particularly important for regulating maladaptive emotion, mood, and behavior in children at risk for depression (Kertz, Belden, Tillman, & Luby, 2016; Wagner et al., 2015). However, it is not well understood whether control capacity alterations are transmitted intergenerationally, and whether it then contributes to the emergence of depression symptoms passed from mothers to children. To address this question, the current study tested behavioral and electrophysiological indices of cognitive control in sample of children enriched with a maternal history of MDD.

Electrophysiological indices of cognitive control related to performance monitoring have been extensively investigated as neural risk markers of internalizing disorders (Muir, Hedges-Muncy, Clawson, Carbine, & Larson, 2020; Olvet & Hajcak, 2008; Olvet, Klein, & Hajcak, 2010; Weinberg, Kotov, & Proudfit, 2015). Performance monitoring relates to detection of response errors and subsequent adjustment of actions, and it often is probed through event-related brain potentials (ERP) in response to the commission of errors. Specifically, the error-related negativity (ERN) is characterized by a frontocentral negative ERP occurring within 100 ms of error responses (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993). Evidence suggests that the ERN stems from activity within the dorsal anterior cingulate cortex (ACC), which plays a key role in cognitive control regulation. By contrast, correct responses elicit a correct-related negativity (CRN) within the same time window. The error positivity (Pe) follows the ERN and is a positive ERP with a centroparietal distribution and believed to reflect elaborative or conscious processing of errors (Falkenstein et al., 1991). The ERN is associated with worry and negative affect in youth (Meyer, 2022; Torpey et al., 2013) and adults (Hajcak, McDonald, & Simons, 2004; Moser, Moran, & Jendrusina, 2012; Weinberg et al., 2015). Altered ERN has been observed in anxious children and adolescents (Bress, Meyer, & Hajcak, 2015; Meyer et al., 2013) and adults (Clayson, Carbine, & Larson, 2020; Weinberg et al., 2015). However, the depression-ERN link has been mixed. For example, studies have found both increased (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008) and decreased (Weinberg, Klein, & Hajcak, 2012; Weinberg et al., 2016) ERN in depression, while others have found no relationship (Moran, Schroder, Kneip, & Moser, 2017). A recent meta-analysis found a small and heterogeneous association between ERN and depressive symptoms (Clayson et al., 2020), suggesting a complex relationship that may depend on co-occurring symptoms and disorders. Less research has investigated the Pe. There is some indication that aberrant Pe amplitudes may relate to anxiety symptoms (Moser et al., 2012; Weinberg, Olvet, & Hajcak, 2010), but further research on the Pe in the context of internalizing disorders is warranted.

The error-related activity also can be decomposed into neural oscillations known as the frontal midline theta (FMT) and delta power, which increases following commission of errors (Luu, Tucker, & Makeig, 2004; Yordanova, Falkenstein, Hohnsbein, & Kolev, 2004). Evidence suggests that FMT and delta may provide complementary information to ERN (Cavanagh et al., 2017; Munneke, Nap, Schippers, & Cohen, 2015). FMT appears to be functionally related to the ERN (Munneke et al., 2015) and is generally believed to reflect increased implementation of control through the dorsal ACC (Cavanagh & Frank, 2014; Holroyd & Umemoto, 2016). Limited evidence suggests that FMT power to error-related feedback may be associated with altered learning and cognitive control in MDD patients (Cavanagh, Bismark, Frank, & Allen, 2011; Gheza, Bakic, Baeken, De Raedt, & Pourtois, 2019; Muir et al., 2020). By contrast, increased FMT has been reliably associated with anxiety in adults (Cavanagh & Shackman, 2015; Umemoto et al., 2021). Although the functional significance of delta power is not fully understood, FMT and delta power may provide dissociable information (Cohen & Cavanagh, 2011). For example, increased delta to errors has been observed in patients with social anxiety disorder and may reflect motivational salience to errors (Umemoto et al., 2021), but has not been sufficiently investigated in depression. Taken together, although the neurophysiological correlates of cognitive control have been increasingly well-studied, its role in familial transmission during early child development remains unclear.

It is notable that cognitive control, which relies on the functioning of prefrontal cortical regions, including the ACC, has strong genetic influences (Friedman et al., 2008; Thompson et al., 2001). For example, twin studies have shown that control capacity is highly heritable (Chen et al., 2020; Friedman et al., 2008; Thompson et al., 2001). Similarly, there seems to be substantial heritability of ERPs. For example, ERN, CRN, and Pe are reported to be 40–60 % heritable among adolescent twin pairs (Anokhin, Golosheykin, & Heath, 2008). Additionally, Burwell et al. (2016) found that twin pairs were highly similar in ERP measures across different control tasks (e.g., Go/No-Go task, Flanker task), which remained stable over one year. Further, recent studies support mother-child familial transmission of ERN/CRN (Moser, Fisher, Hicks, Zucker, & Durbin, 2018; Suor, Calentino, Granros, & Burkhouse, 2022). Moser and colleagues found a positive correlation in ERN between mothers and their children. Further, Suor et al. (2022) reported that heightened maternal ERN was associated with increased internalizing symptoms (both depression and anxiety) in 9–16 year-olds, however, this relationship was mediated by enhanced child ERN and negative parenting styles, above and beyond maternal internalizing symptoms. As a whole, these findings highlight the importance of investigating cognitive control alterations as a possible intergenerational marker of transmission from mothers to their children that increases risk for MDD in youth.

1.1. Current study

In the current study, we tested the intergenerational transmission of control capacity from mothers to their children, and further, whether alterations in their control ability contributed to children's depression symptom severity. First, we hypothesized that worse maternal control capacity, as measured by Flanker task behavioral performance (i.e., reduced inhibitory control), would associate with worse children's behavioral performance (i.e., poor inhibitory control, resulting in increased error rate) and neurophysiological alterations (i.e., reduced inhibitory control) assessed through the Go/NoGo task. Notably, EEG data were not collected from mothers, thus maternal Flanker behavioral performance was used as an index of their control capacity across all analyses. Second, in line with the prior finding showing that maternal error-related process associated with child internalizing symptoms as mediated by child ERN (Suor et al., 2022), we hypothesized that worse maternal Flanker task behavioral performance would associate with increased child depression symptoms through diminished child control capacity (ERN, Pe, and FMT). Moreover, as anxiety symptoms tend to emerge earlier in development (Avenevoli, Stolar, Li, Dierker, & Ries Merikangas, 2001; Beesdo et al., 2007), we similarly tested whether maternal control capacity would associate with child anxiety symptom severity through reduced child control capacity. Last, the role of delta power in cognitive control is not well understood (Umemoto et al., 2021), and therefore, to support future work, we tested the role of delta power as a correlate of control capacity.

2. Material and methods

2.1. Participants

The current study recruited participants through a larger, ongoing

parent study, which is investigating the intergenerational effect of cognitive control deficits in mother-child dyads using Magnetic Resonance Imaging. We attempted to contact 186 families who had already participated in the larger study. Of those, 88 families (52 %) were willing and eligible to participate. Of these 88 families, 65 families (74 %) responded and participated in the current EEG study. Although all mothers in the current study were recruited regardless of their mental health conditions, the parent study enrolled mothers with a history of MDD. Thus, the final sample included 65 mothers ages 20–48-years-old (*M*=32.35, *SD*=7.49), with 47.7 % ($n = 31$) reporting a history of MDD, and 65 children ages 4–10-years-old (*M*=6.70, *SD*=1.35; 75.38 % right-handed). Children were included if they were ages 4–11-years-old and fluent in English. Exclusion criteria included a history of seizure, neurological disorder, or head injury (loss of consciousness > 5 min). Mother-child dyad sociodemographic information is summarized in Table 1.

2.2. Procedure

This study was approved by the Institutional Review Board at the New York State Psychiatric Institute (Protocol# 7656). All mothers provided written consent and children assented. After consent and assent procedures, clinical interviews were administered to mothers, which assessed lifetime psychiatric disorders for both the mother and the child. Mothers also completed self-report questionnaires about their own and child's current depression and anxiety symptoms. To assess cognitive control capacity, Flanker task (Eriksen & Eriksen, 1974) data were acquired from the mother. Additionally, the child completed a modified Go/No-Go task while EEG data were recorded. Each dyad was compensated $100 for the clinical interview and behavioral tasks, as well as $100 for completing the EEG task.

Table 1
Sociodemographic data for mother-child dyads.

	Mothers (n = 65)	**Children** (n = 65)
Age *M* (*SD*)	32.19 (7.35)	6.70 (1.35)
Biological Sex *n* (%)		
Female	65 (100)	31 (47.70)
Male	—	34 (52.30)
Race *n* (%)		
Black	21 (32.30)	20 (30.80)
White	6 (9.23)	6 (9.23)
Native American	6 (9.23)	4 (6.15)
Asian	—	1 (1.54)
Multi-racial	8 (12.30)	10 (15.40)
Other	24 (36.90)	24 (36.90)
Hispanic *n* (%)	52 (80.00)	52 (80.00)
Family Income *n* (%)		
< $25,000	18 (27.70)	—
$26,000-$50,000	19 (29.20)	—
$51,000-$100,000	9 (13.80)	—
> $100,000	9 (13.84)	—
Not Reported	10 (15.38)	—
Current Medication Status *n* (%)		
Yes	7 (10.77)	—
No	50 (76.92)	—
Not Reported	8 (12.31)	—
Lifetime Psychiatric Disorders *n* (%)		
Lifetime MDD	31 (47.69)	1 (1.54)
Current	9 (13.85)	1 (1.54)
Current with recurrent episodes	8 (12.31)	—
Past	22 (33.85)	—
Past with recurrent episodes	8 (12.31)	—
Bipolar Disorder	3 (4.62)	—
Anxiety Disorder	20 (30.80)	13 (20.00)
ADHD	—	22 (33.80)
Symptom Severity *M* (*SD*)		
Depression Symptoms	17.82 (6.60)	1.42 (1.79)
Anxiety Symptoms	36.21 (10.52)	11.46 (8.14)

Note. MDD=Major Depressive Disorder; ADHD=Attention-Deficit/Hyperactivity Disorder; Depression symptoms=Center for Epidemiologic Studies Depression Scale (CESD) for mothers and the depression subscale of the Revised Children's Anxiety and Depression Scale (RCADS) for children; Anxiety Symptoms= Trait Anxiety Inventory for mothers, and the anxiety subscale of RCADS for children. Seven mothers (10.77 %) reported using medication (Selective Serotonin Reuptake Inhibitor = 3 [Escitalopram (n = 1), Sertraline (n = 1), Citalopram (n = 1)]; Selective Serotonin and Norepinephrine Reuptake Inhibitor = 1 [Duloxetine (n = 1)]; Anxiolytic = 2 [Diazepam (n = 1), Buspirone (n = 1)]; Antipsychotic = 2 [Olanzapine (n = 1), Aripiprazole (n = 1)]; Anticonvulsant = 1 [Lamotrigine (n = 1 to treat bipolar disorder]).

2.3. Clinical characterization

Trained study staff who received 50+ h of clinical training by a licensed clinical psychologist administered a structured diagnostic interview to probe lifetime disorders in mothers using the Mini-International Neuropsychiatric Interview (MINI 7.0.2; Sheehan et al., 1998) and the Mini-International Neuropsychiatric Interview (MINI-Kid 7.0.2; Sheehan et al., 2010) for children. For the MINI-Kid study staff interviewed the mother and child together to assess psychiatric disorders in children. Mothers' current depression symptoms were assessed with the Center for Epidemiologic Studies Depression Scale (CESD; Radloff, 1977), a 20-item inventory assessing depression symptoms over the past 2 weeks. Scores ranged from 0 to 60, with higher scores indicating greater depression severity ($\alpha = 0.91$). Mothers' trait anxiety was assessed with the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983), a 20-item inventory assessing dispositional anxiety severity. Scores ranged from 20 to 80, with higher scores indicating more severe trait anxiety (Trait $\alpha = 0.71$). Child depression and anxiety symptoms were assessed with maternal report on the Revised Children's Anxiety and Depression Scale (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000), a 47-item scale assessing internalizing symptoms. Raw scores ranged from 0 to 30 for the depression subscale and 0–111 for the anxiety subscale with higher scores reflecting greater depression and anxiety symptom severity (Depression subscale $\alpha = 0.78$; Anxiety subscale $\alpha = 0.93$).

2.4. Experimental task

2.4.1. NIH toolbox flanker task

The mothers completed a modified iPad-based Eriksen Flanker task that assessed response inhibition (www.nihtoolbox.org). The NIH toolkit was developed for research purposes to provide scalable access to behavioral tasks that were also generalizable across the life span. It has been validated in adults (Zelazo et al., 2014), showing high internal consistency (Heaton et al., 2014). On each trial, a star-shaped fixation appeared on the screen for between 1000 and 1500 ms. The word "Middle" then replaced the fixation for 1000 ms. Following this, four horizontally aligned flanker arrows were presented for 100 ms, pointing in the same direction with a space in the middle (<< << or >> >>). A central arrow probe then appeared in the middle along with the flankers for 10,000 ms, which aligned (congruent trials; <<<<< or >>>>>) or did not align (incongruent trials; << > << or >> < >>) with the direction of the flankers. Participants were instructed to indicate the direction of the central arrow by pressing the right or left button on the touch screen quickly and accurately (within the response window of 10,000 ms). The two buttons stayed on the screen throughout the trials, and the stimuli disappeared when participants made a response. Following a fixation screen for 800 ms (i.e., inter-trial interval [ITI]), the next trial began.

This task consisted of four practice trials (two congruent and two incongruent trials), and participants had to correctly respond to at least three trials to begin the task. If they did not meet this criterion, they were given two additional practice rounds. Audio performance feedback was given after each practice trial ("*Great job*" or "*The arrow is pointing this way, so you should choose this button*"). The experimental block consisted of 12 congruent trials (60 %) and 8 incongruent trials (40 %)

for a total of 20 trials with no performance feedback.

2.4.2. The Go/NoGo zoo task

Child participants completed a developmentally-appropriate Go/NoGo Task called the Zoo Task (Grammer, Carrasco, Gehring, & Morrison, 2014), in which they were told they would be helping a zookeeper put escaped animals back in the cage. On each trial, a fixation cross appeared on the screen for 300 ms followed by a picture of an animal for 750 ms (Fig. S1). Following a blank ITI screen for 500 ms, the next trial began with the onset of a fixation cross. Children were told to press a button to "catch the animal" (Go trials) but withhold response for friendly orangutans (NoGo trials) using a response box (RB-844 Response Pad, Cedrus, San Pedro, CA). Responses could be made anytime from the onset of the picture to the offset of the ITI screen. Children first practiced the task, which consisted of 9 Go and 3 NoGo trials. The actual task consisted of 8 blocks with 40 trials in each block, with 30 Go trials (75 %) and 10 NoGo trials (25 %) per block. Each block presented 30 unique pictures of animals and 10 orangutans (three different pictures were used with repetition) in a random order. Feedback was given at the end of each block to keep children motivated ("*Try to catch them even faster next time!*" and "*Keep watching out for the orangutan friends!*").

Given that both the Flanker task and Go/NoGo task are suggested paradigms to probe the Cognitive Control Construct of the Cognitive Systems within the RDoC, it is believed that using different control tasks may be beneficial to study the cognitive system more broadly across the dyads. Specifically, we used a developmentally appropriate measure of cognitive control that has been used for children of similar age in prior studies (Grammer et al., 2014; Isbell & Grammer, 2022). For the mothers we used a Flanker task that also has been commonly used to measure cognitive control.

2.5. EEG acquisition and analysis

EEG data were acquired using either a 64-channel or 32-channel[2] ActiCHamp from Brain Products (Brain Products, Munich, Germany), digitized at a 500 Hz sampling rate, and referenced online to FCz. Impedances were kept below 20 kΩ. Analyses were performed offline using Brain Vision Analyzer 2.1 software (Brain Products, Gilching, Germany). For participants with a 64-channel montage, the number of electrodes were reduced to 32, which ensured that the same montage was used for all analyses. Bad channels were visually identified if more than 50 % of the trials included non-ocular artifacts, which were excluded from the subsequent processes and then interpolated. EEG data were bandpass filtered with 0.1–30 Hz. Segments of non-ocular, muscular artifacts affecting more than 50 % of the channels were manually removed, and ocular artifacts (blinks and eye movements) were corrected with independent component analysis. Bad channels were then topographically interpolated (M=0.35, range=0–3 electrodes across participants), and EEG data were re-referenced to the average of all channels.

For ERP analysis, EEG data were segmented into −500 to 800 ms epochs, time-locked to the onset of response. Data were then baseline corrected for each channel by subtracting from each data point the average amplitude during the −500 to −300 ms interval preceding the response. Remaining artifacts were removed using a semiautomatic procedure based on individual channels with the following criteria: (1) a maximally allowed voltage step of 50 μV, (2) a maximum allowed absolute voltage difference of 100 μV, (3) +/−100 μV voltage threshold, and (4) a minimum allowed activity of 0.5 μV within a 100 ms interval. Data were then segmented separately into correct (button-press on Go trials) and incorrect (button-press on NoGo trials) response trials. Trials were averaged separately to create an ERP for correct (i.e., CRN) and incorrect (i.e., ERN) response trials, and ERP amplitudes were extracted at electrode site FCz where it was maximal between 0 and 100 ms. The difference ERN (ΔERN) was then created by subtracting the CRN from the ERN. Similarly, the difference Pe (ΔPe) was created by subtracting Pe on correct versus incorrect response trials pooled across electrode sites POz and Oz 200 and 500 ms post-response. The electrode(s) and the time windows of interest were based on the grand average across all participants for the difference wave ERPs (Luck & Gaspelin, 2017), which was consistent with prior research testing the ΔERN (Danovitch, Fisher, Schroder, Hambrick, & Moser, 2019; Torpey, Hajcak, Kim, Kujawa, & Klein, 2012) and ΔPe (Grammer et al., 2014; Isbell & Grammer, 2022).

For time-frequency analysis, EEG data were segmented into −1500 to 1500 ms epochs, time-locked to the onset of response. Artifact removal used the same criteria and procedure as described for the ERPs, and data were segmented separately into correct and incorrect response trials. The analysis used a continuous wavelet transformation from 1 to 30 Hz with 40 frequency steps on a logarithmic scale with a Morlet parameter of 3.5. Baseline correction was performed using the 200 ms interval from −500 to −300 ms preceding the response, and the resulting power was averaged separately for correct and incorrect response trials. The FMT (4–8 Hz) was extracted as the average power between 50 and 250 ms at electrode site FCz where it reached maximal power. The FMT band had a mean frequency of 6.1 Hz (range=4.1–8.0 Hz). The delta power (1–3 Hz) was extracted as the mean power between 100 and 400 ms, pooled across electrode sites FCz and Cz where it reached maximal power. The delta band had a mean frequency of 1.9 Hz (range=1.0–3.1 Hz). Similar to the ERP analyses, difference scores were also calculated by subtracting the power on the correct response trials from incorrect response trials for both FMT (ΔFMT) and delta (ΔDelta). More positive ΔFMT and ΔDelta power indicated increased power on the incorrect response trials relative to correct response trials.

2.6. Data analysis

Analyses were completed using R Studio version 4.1.2 (R Core Team, 2021). Outliers were winsorized using the DescTools package in R. Specifically, outliers that were lower than the 5 % quantile across participants for a given variable were assigned the value of the 5 % quantile of a given variable. Similarly, outliers that were higher than the 95 % quantile across participants for a given variable were assigned the value of the 95 % quantile of this variable.

2.6.1. Flanker task

Flanker task performance was scored automatically using the NIH toolbox. To be consistent with the standard use with the NIH toolkit, we used the composite score that integrated both accuracy and speed. Briefly, an accuracy score was calculated for each participant by multiplying the number of correct responses by 0.125. This created a score ranging between 0 and 5. For participants who were accurate on 80 % or fewer trials, their performance scores equaled the accuracy score. For participants who performed with above 80 % accuracy, their reaction times (RT) were also calculated and added to the accuracy score. For this calculation, RTs smaller than 100 ms or greater than 3 standard deviations (SDs) were excluded from each participant as outliers. Median RTs were calculated and log-transformed to normalize the

[2] Due to the Covid-19 pandemic, we reduced the number of electrodes from 64 to 32 for 30 participants to decrease the amount of time spent in direct contact with participants. For those with 32 channels, the electrooculogram (EOG) was acquired for 17 participants to capture blinks and eye movements (for the remaining 13 participants EOGs were not collected). For these participants, vertical electrooculography (VEOG) was recorded using a supra- to suborbital bipolar montage surrounding the right eye, and horizontal electrooculography (HEOG) was recorded from electrodes placed on the left and right outer canthi. A ground electrode for the EOGs was also placed on the center of the forehead. For participants with a 64-channel montage (n = 35), EOGs were not recorded. Thus, 4 electrodes (AF3, AF4, AF7, and AF8) were used in lieu of the EOG channels (but excluded from grand-averages and statistical analysis).

distribution of the scores for each participant. The RT score was then transformed to range between 0 and 5 before being added to participants' accuracy scores (for the scoring manual see NIH Toolbox, 2021). The total scores ranged from 0 to 10, with higher scores indicating better performance (i.e., higher accuracy and faster response).

2.6.2. Go/NoGo task

For the Go/NoGo Task, child participants with at least 6 incorrect response trials were included in the analyses. In addition, trials with an RT shorter than 200 ms were excluded. For each participant, the error rate was calculated by dividing the number of total incorrect response trials (responding on the NoGo trials and not responding on Go trials) by the total number of trials.

2.6.3. Electrophysiology

Consistent with prior research (Grammer et al., 2014; Isbell & Grammer, 2022; Lawler et al., 2021), we tested whether the expected task effects were produced in children in the Go/NoGo task (i.e., larger activity on the incorrect response trials relative to the correct response trials), separate paired *t*-tests were first conducted by comparing between the correct and incorrect response trials for ERN/CRN (i.e., ERN more negative than CRN), Pe (i.e., Pe more positive on incorrect relative to correct trials), FMT (i.e., FMT power larger on incorrect relative to correct trials), and delta (i.e., delta power larger on incorrect relative to correct trials). Additionally, we tested whether the error rate would correlate with these electrophysiological markers (i.e., higher the error rate, smaller (less negative) ΔERN and (less positive) ΔPe, ΔFMT, and ΔDelta would be). For this, separate regression models tested whether ERPs (i.e., ΔERN, ΔPe) and oscillations (i.e., ΔFMT, ΔDelta) predicted error rate, controlling for age. Pearson correlations examined whether age was associated with ΔERN, ΔPe, ΔFMT, and ΔDelta. Two children were excluded from analysis, as they failed to meet the minimum number of trials needed for the EEG analysis (i.e., 6 trials) (Olvet & Hajcak, 2008), and one child was excluded due to technical issue, resulting in a final sample of 62 children. Internal consistency of electrophysiological measures was computed by examining the correlation of odd- and even-numbered trials across correct and incorrect response trials with a Spearman-Brown correction.

Two models were estimated. *First*, we tested cognitive control associations within the mother-child dyad. Separate regressions tested whether the maternal Flanker score related to children's behavioral (i.e., error rate) and electrophysiological indices (i.e., ΔERN, ΔPe, ΔFMT, ΔDelta), controlling for age. Flanker data was not available for 5 mothers due to technical issues and scheduling challenges, resulting in 57 dyads for these analyses. *Second*, we tested whether children's control capacity mediated the relationship between maternal control and child symptom severity. Within these models, the mediator included ΔERN, ΔPe, and ΔFMT, and given the heterotypic course of MDD, we tested both child depression and anxiety symptoms separately as the dependent variable. All models controlled for maternal depressive symptoms, and when testing child anxiety symptoms, maternal trait anxiety symptoms were included as a covariate. The COVID-19 pandemic resulted in many challenges for scheduling clinical assessments. Consequently, only 51 dyads were included for these analyses.

As exploratory analysis, we tested the relationship between depression and cognitive control capacity in mothers. First, we conducted simple *t*-tests to test whether maternal Flanker performance differed between mothers with and without a history of MDD. Second, we conducted linear regressions to test whether maternal Flanker performance associated with maternal depression symptom severity. Additional analyses controlling for a maternal history of MDD, other maternal disorders, and child disorders (anxiety, ADHD) are reported in the Supplement.

3. Results

Bivariate correlations across behavioral performance, electrophysiological measures, and depression and anxiety symptoms are shown in Table 2.

3.1. Behavior

Go/NoGo Task. The overall error rate was 22 % (*SD*=0.11, *Range*=8–45 %), including errors of commission (36 %, *SD*=0.16) and omission (17 %, *SD*=0.14).

NIH Toolbox Flanker Task. Results were in line with prior research in adults (e.g., Weintraub et al., 2014), as the mean performance score was 8.12 for mothers (*SD*=0.90, *Range*=6.37–9.33).

3.2. Electrophysiology

Before estimating our primary models, we examined task effects. The internal consistency ranged from poor to excellent across ERPs and time frequency indices: ERN/CRN (α = .82), Pe (α =[0.89, FMT (α = .34), and delta (α =[0.71. Additionally, the ERN was significantly larger (more negative) than the CRN ($t(61) = -8.92$, p <[0.01, and the Pe was larger (more positive) on incorrect trials compared to the correct trials ($t(61) = 13.72$, $p < .01$) (Fig. 1). Similarly, both FMT ($t(61) = 6.00$, $p < .01$) and delta power ($t(61) = 6.36$, $p < .01$) were larger on incorrect relative to correct trials (Fig. 2). Separate multiple regressions, controlling for age, revealed that (among children) increased Go/NoGo error rate significantly associated with smaller ΔERN (β=12.62, *SE*=3.67, $p < .01$), ΔPe (β=−29.56, *SE*=7.67, $p < .01$), and ΔDelta (β=−49.40, *SE*=18.75, $p = .01$), but not ΔFMT (β=−25.80, *SE*=22.09, $p = .25$) (Fig. 3). Pearson correlations revealed that child age correlated positively with ΔFMT power ($r = .29$, p =[0.02, but not with ΔERN, ΔPe, or ΔDelta (*ps*>.41).

Mother-Child Control Capacity. Separate multiple regressions, controlling for children's age, revealed that worse maternal performance on the Flanker task was associated with increased Go/NoGo error rate (β=−−0.04 *SE*==0.02 p =[0.01 as well as smaller ΔERN (β=−1.06, *SE*=.45, p =[0.02 and ΔPe (β=2.90, *SE*=.10, p <[0.01 in children (Fig. 4). No significant association was detected between maternal Flanker performance and ΔFMT (β=3.32, *SE*=2.87, $p = .25$) and ΔDelta (β=3.64, *SE*=2.51, $p = .15$). These results remained unchanged when controlling for a history of maternal MDD, any maternal anxiety disorders, or a total number of any maternal disorders (see Supplement).

Mediation Models. Mediation models tested whether child's control capacity (i.e., ΔERN, ΔPe, and ΔFMT) mediated the association between maternal control capacity (i.e., Flanker performance) and child symptom severity (i.e., depression and anxiety symptoms). When testing depression symptoms as the dependent variable (Fig. 5; Table 3), results indicated that worse maternal Flanker performance significantly associated with smaller child ΔERN (β=−1.01, *SE*=.45, p =[0.03 and ΔPe (β=3.08, *SE*=1.03, $p < .01$), but not ΔFMT (β=2.70, *SE*=2.90, $p = .36$). However, child control capacity was not associated with child depression scores (*ps*>.13), and there was no significant direct association between maternal Flanker performance and child depression scores, controlling for maternal depression and child age (β=0.02 *SE*=0.27 $p = 0.94$).

When testing anxiety symptoms as the dependent variable, results again indicated that worse maternal Flanker performance significantly associated with smaller child ΔERN (β=−1.05, *SE*=.46, p =[0.03 and ΔPe (β=3.18, *SE*=1.05, $p < .01$), but not ΔFMT (β=2.69, *SE*=2.96, $p = .37$). There was a non-significant trend that larger child ΔFMT associated with increased child anxiety scores (β=.12, *SE*==0.06 p = [0.054. However, maternal Flanker performance did not predict child anxiety symptoms directly (β=−.05, *SE*=1.31, p =[0.97. These results remained unchanged when controlling for a history of maternal MDD, any maternal anxiety disorders, or a total number of any maternal

Table 2
Pearson correlations among behavioral and electrophysiological measures and symptoms across mother-child dyads.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Maternal CESD	—									
2. Maternal STAI	0.61***	—								
3. Maternal Flanker	0.12	-0.03	—							
4. Child RCADS-dep	.28	0.24	0.07	—						
5. Child RCADS-anx	0.20	0.26	0.01	0.71***	—					
6. Child Error Rate	0.03	0.05	-0.34*	-0.14	-0.06	—				
7. ΔERN	-0.09	-0.13	-0.30*	0.07	0.03	0.32*	—			
8. ΔPe	-0.13	-0.01	0.35*	-0.21	-0.17	-0.36**	-0.45***	—		
9. ΔFMT	0.24	0.08	0.18	0.10	0.27	-0.23	-0.02	-0.09	—	
10. ΔDelta	-0.09	0.02	0.21	0.01	0.15	-0.33*	-0.10	0.11	0.10	—
11. Child Age	-0.05	-0.19	0.07	0.29*	0.20	-0.23	0.14	-0.17	0.30*	0.04

Note. *** p < .001; ** p < .01; * p < .05
CESD=Center for Epidemiologic Studies Depression Scale; STAI=The State-Trait Anxiety Inventory (Trait);
RCADS-dep=The Revised Child Anxiety and Depression Scale depression subscale;
RCADS-anx=The Revised Child Anxiety and Depression Scale anxiety subscale.

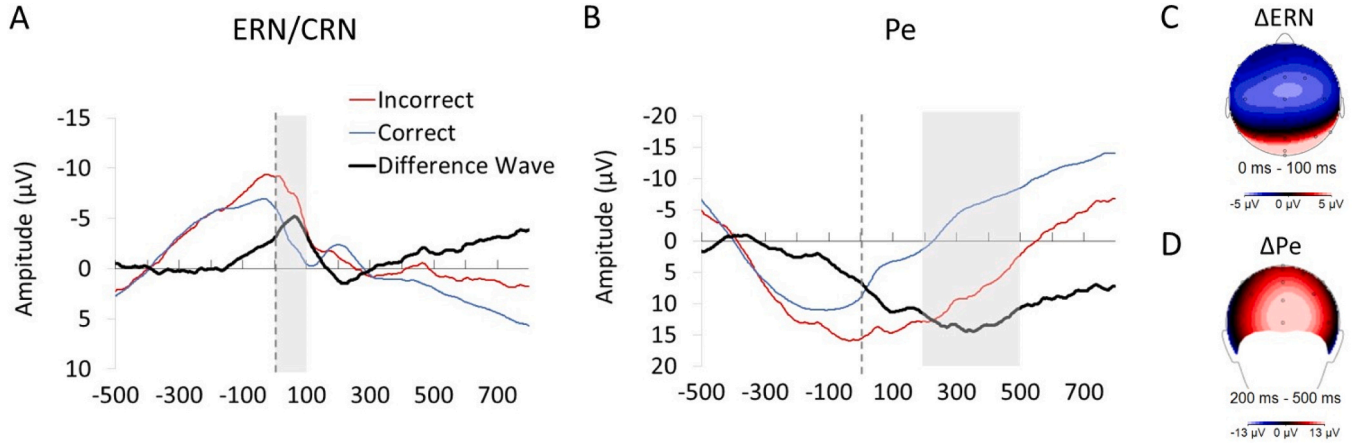

Fig. 1. Event-Related Potentials Elicited by Correct and Incorrect Responses, their Difference Waves, and Associated Scalp Voltage Maps. Difference wave (black line) is calculated by subtracting the ERP on the incorrect response trials (red line) from the ERP on the correct response trials (blue line) separately for (**A**) ERN (ΔERN) and (**B**) Pe (ΔPe). ΔERN is measured at channel FCz, and Pe is assessed at channels POz and Oz. Scalp voltage map of the difference wave in (**C**) ΔERN and (**D**) ΔPe. The window of measurement is highlighted in grey. Response onset occurs at 0 ms (dotted vertical line).

disorders (see Supplement).

Relationship between depression and cognitive control capacity in mothers. Simple *t*-tests revealed that there was no significant difference in maternal Flanker performance between mothers with ($n = 23$, M=8.03, SD=.96) and without ($n = 20$, M=8.32, SD==0.93 a history of MDD, ($t(40.52) = --0.99$ p =[0.33 (there were also no effects of maternal anxiety or any diagnoses, see Supplement). Similarly, linear regressions showed no significant association between maternal Flanker performance and maternal depression symptom severity (β=.02, SE==0.02 p =[0.41, which remained non-significant when controlling for maternal anxiety symptoms (β=.03, SE==0.02 p =[0.22.

4. Discussion

Despite extensive work linking maternal history of MDD with offspring depression, the underlying mechanisms that contribute to this relationship are not well understood. We tested whether cognitive control alterations are transmitted intergenerationally and constitute a risk factor for the familial transmission of depression in child offspring. By probing known electrophysiological indices of control capacity in children age 4–10-years-old while they performed a child-friendly Go/NoGo Task (Grammer et al., 2014), we first demonstrated task effects in the children. That is, they elicited larger (i.e., more negative) ERN and Pe amplitude to error compared to correct responses (Grammer et al., 2014; Isbell & Grammer, 2022), similar to older children (Meyer, 2022; Suor et al., 2022) and adults (Moser et al., 2012; Weinberg et al., 2015). They also elicited larger FMT and delta power to error compared to correct responses, consistent with findings in adults (Cavanagh & Shackman, 2015; Umemoto et al., 2021). Further, poorer child behavioral performance (error rate) associated with reduced ΔERN and ΔPe amplitude and ΔDelta power (but not ΔFMT power). Of importance to the goal of the current study, we found that worse maternal performance on the Flanker task, an index of maternal control capacity, significantly associated with worse error rate and smaller ΔERN and ΔPe on the Go/NoGo task in children. A mediation model, however, revealed no associations between maternal control capacity and child depression symptoms directly or indirectly through child control capacity, or between child control capacity and their depression symptoms.

Our findings are consistent with prior research reporting a strong heritability of cognitive control capacity based on genetic (Friedman et al., 2008; Thompson et al., 2001) and electrophysiological (Moser et al., 2018; Suor et al., 2022) studies. The current study complements this by showing that the intergenerational transmission of control capacity is observable as early as 4–10 years of age and suggests a potential biological influence of cognitive control in mother-child dyads. Although ΔFMT power increased with child age, which may suggest an improved control capacity as children matured (van Noordt, Heffer, & Willoughby, 2022), maternal control capacity did not relate to child ΔFMT or ΔDelta power, contrary to our prediction. One possibility is that maternal EEG markers, rather than the behavioral index of control, may be a more robust measure of control capacity (e.g., Moser et al., 2018; Suor et al., 2022) and potentially, associates better with

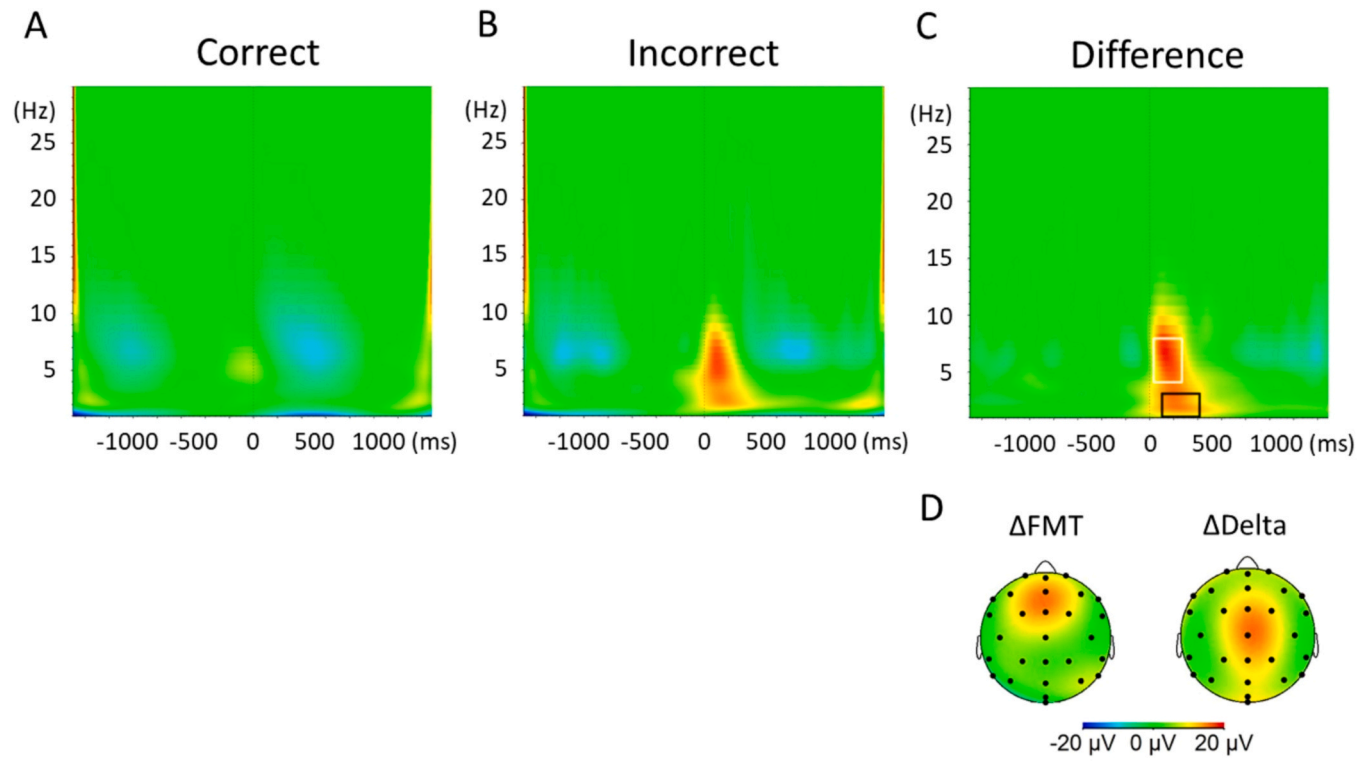

Fig. 2. Time-Frequency Plots for Frontal Midline Theta (FMT) (4–8 Hz) and Delta (1–3 Hz) Power Elicited by Correct and Incorrect Responses, their Differences, and Associated Scalp Maps in Difference scores. The power on the (**A**) correct response trials was subtracted from the power on the (**B**) incorrect response trials to create (**C**) Difference scores (Difference) for FMT (ΔFMT) and delta (ΔDelta). Power is measured at channel FCz for FMT, and at channel FCz and Cz for delta. Heatmaps are shown from the channel FCz for the purpose of visualization. The window of measurement is highlighted for ΔFMT (white square) and ΔDelta (black square). (**D**) Topographical maps for ΔFMT and ΔDelta. Response onset occurs at 0 ms (dotted vertical line).

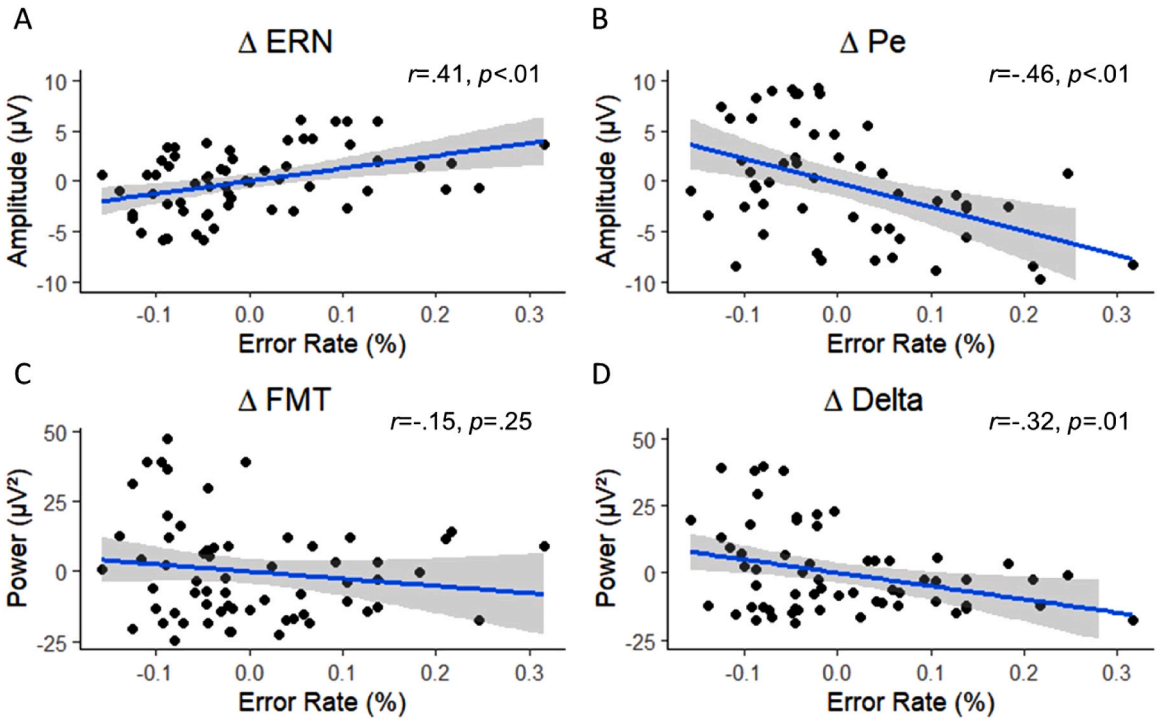

Fig. 3. Scatterplots Depicting the Relationship between Children's Behavioral Performance and electrophysiological markers during the Go/NoGo Task. Error rate (x-axis) is associated with the: (**A**) error-related negativity (ΔERN), (**B**) error positivity (ΔPe), (**C**), frontal midline theta (ΔFMT) power, and (**D**) delta (ΔDelta) power. Error rate depicts a residual score based on the multiple regression analysis that controlled for children's age.

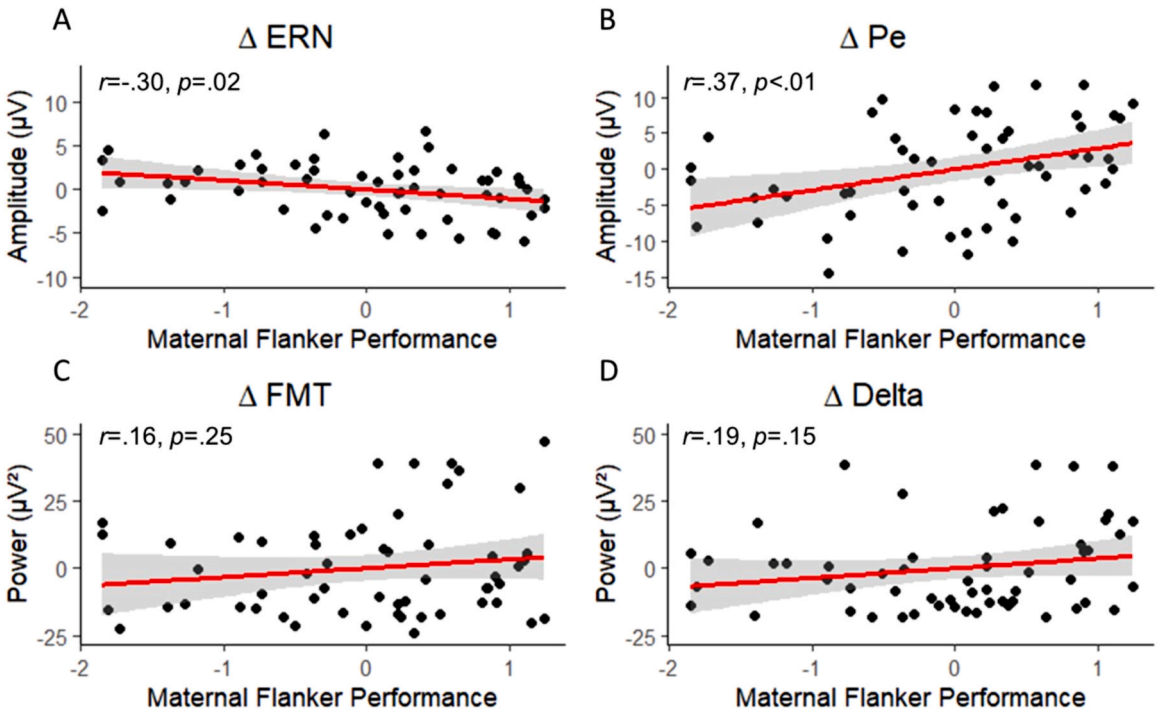

Fig. 4. Scatterplots Depicting the Relationship between Maternal Control Capacity and Children's Control Capacity. Maternal Flanker performance (x-axis) significantly associated with children's electrophysiological markers in difference wave (**A**) error-related negativity (ΔERN) and (**B**) error positivity (ΔPe) but not with difference scores (**C**) frontal midline theta (ΔFMT) power, or (**D**) delta (ΔDelta) power. Maternal Flanker performance shows a residual score based on the multiple regression analysis that controlled for children's age.

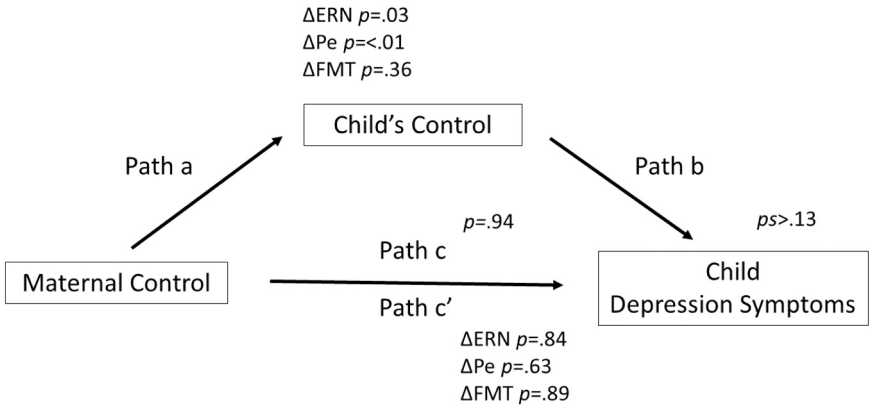

Fig. 5. Maternal control capacity (Maternal Control: Flanker performance) significantly associated with child control capacity (Child's Control: ΔERN and ΔPe, but not ΔFMT) (Path a), but not with child depression symptoms (Path b). Direct (Path c) and Indirect (Path c′) pathways were non-significant.

Table 3
Mediation Model Results Predicting Child Depression Symptoms from Maternal Control Mediated by Child Control.

Path	Predictors	Outcome	*β*	*SE*	*p*
a	Maternal Control	Child Control			
		ΔERN	-1.01	0.45	0.03*
		ΔPe	3.08	1.03	<0.01**
		ΔFMT	2.7	2.9	0.36
b	Child Control	Child Depression Symptoms			
	ΔERN		0.04	0.09	0.64
	ΔPe		-0.05	0.04	0.14
	ΔFMT		0.01	0.01	0.47
c (direct)	Maternal Control	Child Depression Symptoms	0.02	0.27	0.94
c′ (indirect)	Maternal Control	Child Depression Symptoms			
		ΔERN	0.06	0.29	0.84
		ΔPe	0.14	0.3	0.63
		ΔFMT	0.04	0.28	0.89

corresponding oscillatory indices in children. That said, this is an understudied area of research that warrants further research.

Our results suggest a familial influence of cognitive control capacity in mother-child dyads; however, it remains unclear whether this then confers risk for depressive symptoms in children. Although the null results should be interpreted with caution regarding the relationship between maternal and child control capacity and child depression symptoms, it is worth noting that depression symptoms across all children were far below the clinically significant level of depression. Thus, the overall low scores and a limited range of scores may have contributed to the null finding. We also tested whether the maternal and child control capacity may relate to child anxiety symptoms, as these symptoms tend to appear earlier in development (Avenevoli et al., 2001; Beesdo et al., 2007), but we also did not find this relationship. A future study should incorporate a longitudinal design to test how the mother-child control capacity influences symptom development in children who ultimately develop depression.

The null mediation results may also point to the importance of considering other factors in the familial risk for depression (e.g., Gotlib, Goodman, & Humphreys, 2020; Hammen, 2018). For example, poor maternal control and emotion regulation is associated with negative discipline strategies (e.g., a harsh and rigid parenting style, increased reactivity to child emotions), which then adversely impacts children's functioning and internalizing symptoms (Crandall, Deater-Deckard, & Riley, 2015; Goodman, Simon, Shamblaw, & Kim, 2020). For example, Suor et al. (2022) found that negative parenting style mediated the relationship between maternal ERN (assessed through a Flanker task) and child internalizing symptoms above and beyond maternal internalizing symptoms. Thus, it is reasonable to speculate that reduced maternal control capacity may make it difficult to adaptively respond to child emotional and behavioral needs, including performance mistakes, which may in turn make children more (or less) sensitive to their performance errors and dysregulate their emotions. This idea aligns with the view that ERN is not only an established cognitive control index (or performance monitoring) but also a key component of the Sustained Threat Construct withing the RDoC Negative Valence System (e.g., Weinberg et al., 2016) and consistent with evidence that affective dysregulation is a common characteristic of depression (e.g., Bylsma, 2021). Accordingly, reactivity to errors, as assessed by EEG, may be particularly relevant as altered control in the context of psychologically threatening stimuli. Nevertheless, an intergenerational transmission of risk for depression likely is complex and involves multiple biological (e.g., cognitive abilities, sex, race) and environmental (e.g., stressors, socioeconomic status, support system) factors. Elucidating how specific risk factors interact to contribute to the onset of MDD in offspring would advance our standing not only of the underlying mechanisms but also of effective intervention strategies.

4.1. Limitations

There are several limitations to consider when interpreting our results. First, although our sample was very ethnically and racially diverse (Table 1), the sample size was relatively small, and accordingly, future replication is necessary with larger samples, particularly related to the mediation analyses. Second, maternal control capacity was assessed with a brief Flanker task using a NIH Toolbox. Thus, future research may benefit from using more standard assessment of control capacity, which has more trials, which allows for more computationally sophisticated analyses. Relatedly, maternal control capacity was assessed solely with behavioral performance, which is not always related to physiological measures. Accordingly, future research may also benefit from probing electrophysiological processes in mother-child dyads. Third, low overall depression symptom scores among children may have contributed to the null mediation results. Perhaps following participants longitudinally, and incorporating different perspectives (e.g., teacher report) may enable a more comprehensive test of our proposed model. Fourth, several mother and child participants had disorders other than MDD. Future research should investigate the impact of comorbid disorders with a larger sample. Relatedly, interviews were not recorded and thus, no inter-rater concordance was assessed. Last, our study tested a maternal but not paternal perspective. Given the heritability of cognitive control (e.g., Anokhin et al., 2008; Burwell, Malone, & Iacono, 2016; Moser et al., 2018; Suor et al., 2022), it may prove important to understand both the maternal and paternal effect to operationalize risk for control alterations and subsequent vulnerability to depression.

5. Conclusion

Overall, poor maternal control capacity associated with reduced control capacity in child offspring. However, maternal and child control capacity did not predict child depression symptoms. Elucidating how cognitive control alterations underlie the developmental pathway through which depression is passed from mothers to children will help us to better understand which children may be particularly vulnerable to depression. This work may, ultimately, offer insight into effective intervention strategies.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Dr. Auerbach is an unpaid scientific advisor for Ksana Health, and he is a paid scientific advisor for Get Sonar, Inc. No other authors have conflicts of interest to disclose.

Data availability

Data will be made available on request.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.biopsycho.2023.108652.

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