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RESEARCH ARTICLE

The impact of punishment on error-related brain activity in children

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Abstract

The error-related negativity (ERN) is sensitive to individual differences relating to anxiety and is modulated by manipulations that increase the threat-value of committing errors. In adults, the ERN magnitude is enhanced when errors are followed by punishment, especially among anxious individuals. Punitive parenting is related to an elevated ERN in children; however, the effects of task-based punishment on the ERN in children have yet to be understood. Furthermore, there is a need to assess developmental periods wherein the ERN might be especially prone to modulation by punishment. We examined the impact of punishment on the ERN in a sample of children and assessed whether the impact of punishment on the ERN was moderated by age and anxiety. Punishment potentiated the ERN in children, especially among higher trait-anxious individuals; the punishment potentiation of the ERN was also associated with older age. The interaction between child age and anxiety symptoms did not significantly predict the punishment potentiation of the ERN; however, both child age and anxiety symptoms *uniquely predicted* the punishment potentiation of the ΔERN. Anxious children may be especially prone to punishment-related alterations in error monitoring, and the impact of punishment on the ERN may become more pronounced as children age.

KEYWORDS
anxiety, development, ERN, ERPs, error-related negativity, punishment

1 | INTRODUCTION

The ability to detect errors is vital for learning and adjusting behavior to improve outcomes (Hajcak, 2012; Holroyd & Coles, 2002). The neural response to errors can be measured using electroencephalogram (EEG) and is reflected in the error-related negativity (ERN) event-related potential. The ERN is characterized by a negative deflection at frontocentral electrode sites elicited within 50–100 ms of committing an error (Falkenstein et al., 1991; Gehring et al., 1993). The neural generator of the ERN is believed to be the anterior cingulate cortex (ACC), a brain region related to activation in studies of negative affect, pain, and cognitive control (Hoffmann & Falkenstein, 2010; Holroyd et al., 1998; Shackman et al., 2011). The ERN is thought to reflect a general error detection and monitoring process (Falkenstein et al., 1991), and previous work suggests that the ERN is sensitive to individual differences in error sensitivity and the degree to which errors are perceived as threatening (Chong & Meyer, 2019; Weinberg et al., 2016).

The ERN is associated with individual differences related to error sensitivity, including perfectionism (Barke et al., 2017; Meyer & Wissemann, 2020) and anxiety (Hajcak, 2012; Meyer, 2016; Moser et al., 2013; Riesel, Klawohn, et al., 2019; Weinberg et al., 2016). Of note, these differences are observed in relation to specific subsets of anxiety symptoms, including anxious apprehension (i.e., worry), but not anxious arousal (Moser et al., 2013; Proudfit et al., 2013). Indeed, a meta-analysis demonstrated that the effect size of the relationship between the ERN and anxious apprehension ($r = -0.35$) is about three times greater than the strength of the relationship with the ERN and anxious arousal ($r = -0.09$; Moser et al., 2013). One developmental explanation for this link posits environmental factors such as critical parenting increase the salience of errors and thus increase threat sensitivity,

which is reflected in an elevated ERN amplitude, and some individuals with increased threat sensitivity may employ increased worry as a compensatory strategy (Proudfit et al., 2013). Evidence of an enhanced ERN amplitude in specific anxiety disorders characterized by increased anxious apprehension has been observed among individuals with generalized anxiety disorder (Weinberg & Hajcak, 2011; Weinberg et al., 2012; Xiao et al., 2011), obsessive-compulsive disorder (Endrass et al., 2008; Gehring et al., 2000; Riesel, 2019; Weinberg et al., 2015), and social anxiety disorder (Barker et al., 2015; Endrass et al., 2014; Judah et al., 2015; Kujawa et al., 2016; Umemoto et al., 2021). As such, the ERN appears to be a transdiagnostic biomarker of individual differences in worry-related anxiety disorders and the extent to which one finds their own errors aversive (Meyer, 2016).

The ERN magnitude is also sensitive to environmental influences. Indeed, greater frequency of stressful life events, and interpersonal stressors in particular (specifically those that may relate to failure, social rejection, or negative interpersonal experiences), predict a larger ERN among children and adolescents (Lackner et al., 2018; Mehra & Meyer, 2022). Environmental stressors such as adverse childhood experiences and natural disasters are related to an increased ERN and anxiety (Lackner et al., 2018; Meyer et al., 2017; Wu et al., 2021). Moreover, an elevated ERN moderated the relationship between interpersonal stress and anxiety symptoms 6 months later (Banica et al., 2020), such that among adults with a larger ERN at baseline, increased instances of interpersonal stress over 1 year were related to increased anxiety symptoms. These results suggest that aversive (e.g., punitive, stressful) life experiences often relate to a potentiation of the ERN and may contribute to increased risk for anxiety.

The ERN has also been shown to be modulated by experimental manipulations that increase the threat-value of committing an error, suggesting that the magnitude of the ERN is related to the motivational salience, or perceived consequence of committing an error (Hajcak, 2012). Indeed, the ERN magnitude can be experimentally enhanced by incentivizing correct responses with monetary rewards (Ganushchak & Schiller, 2008; Hajcak et al., 2005), emphasizing accuracy over speed (Gehring et al., 1993), and emphasizing that performance is being evaluated (Hajcak et al., 2005). These findings demonstrate that environmental modulation of the perceived threat-value of errors has an impact on the ERN.

Of note, recent research has also shown that punishment during speeded response tasks can modulate the ERN amplitude by increasing the salience of errors. Three studies to date have demonstrated an elevated ERN when errors are followed by punishments, especially among anxious individuals (Meyer & Gawlowska, 2017; Riesel et al., 2012; 2019). Riesel et al. (2012) and Riesel, Kathmann, et al. (2019) found that when errors were punished with an aversive loud tone, higher trait-anxious individuals demonstrated an increased punishment-related ERN. This effect persisted even in blocks when errors were no longer punished (i.e., the extinction phase; Riesel et al., 2012). A follow-up study demonstrated that punishment-related modulation of the ERN in individuals with high trait anxiety persisted 24 h later, suggesting that punishment can have a lasting effect on the ERN in high anxious individuals (Riesel, Kathmann, et al., 2019).

We replicated these effects with a similar task design, which was modified in two ways: first, we delivered an electric shock as an aversive unconditioned stimulus following commission of errors instead of a loud aversive tone, and second, we examined whether the punishment-related potentiation of the ERN was related to punishment in general (i.e., when shocks were delivered randomly), or if this effect was specific to errors (i.e., shocks were only delivered following commission of errors; Meyer & Gawlowska, 2017). We replicated the above findings (Riesel et al., 2012; Riesel, Kathmann, et al., 2019), such that punishment potentiated the ERN among college-aged individuals with elevated trait anxiety (Meyer & Gawlowska, 2017). This difference in ERN magnitude between individuals high in trait anxiety versus those low in trait anxiety only appeared when punishment was related to errors (Meyer & Gawlowska, 2017). These findings support the hypothesis that the association between anxiety and punishment-related modulation of ERN is related to the threat-value of errors, such that anxious individuals may be more prone to these punishment-related alterations in error monitoring (Meyer, 2017).

As anxiety disorders commonly begin in childhood or adolescence and persist into adulthood (Beesdo et al., 2009), it is important to characterize markers of risk for the development of anxiety in children to inform early intervention and prevention approaches. The ERN has been examined in children and is characterized by developmental alterations and associations with anxiety. Generally, the magnitude of the ERN is smaller in young children and increases with age (Davies et al., 2004; Tamnes et al., 2013; Wiersema et al., 2007). Similar to findings in adults, the ERN is also related to individual differences in children. A larger ERN is related to obsessive-compulsive disorder (Carrasco et al., 2013; Hajcak et al., 2008; Hanna et al., 2012) and anxiety disorders in children and adolescents (Hanna et al., 2020; Kujawa et al., 2016; Ladouceur et al., 2006, 2018; Meyer, 2017; Meyer et al., 2013). Furthermore, a larger ERN predicts increased risk for the development of anxiety in later developmental stages (Filippi et al., 2020; Lahat et al., 2014; McDermott et al., 2009; Meyer et al., 2018; Meyer, Hajcak, et al., 2015) and predicts increases in anxiety symptoms over a 2-year period among clinically anxious adolescents (Meyer et al., 2021). Thus, the ERN appears to be a marker of risk for anxiety in children.

To elucidate developmental pathways of anxiety, there is a pressing need to explicate environmental factors that modulate the ERN in children. Several studies have extended findings from adults suggesting that the ERN in children is modulated by environmental factors. For example, the ERN was increased in children ages 7–11 when performance was evaluated by an observer (Kim et al., 2005). Moreover, the ERN was increased in children ages 5–7 in the presence of a controlling parent, compared to the presence of an experimenter (Meyer et al., 2019). Furthermore, the ERN is elevated in children with heightened anxiety symptoms, and the relationship between controlling parenting and anxiety was shown to be mediated by the potentiating effect of the presence of a controlling parent on the ERN (Meyer et al., 2019). This line of research highlights an important environmental factor (i.e., parenting style) that shapes the ERN. Indeed, punitive parenting style has been linked to an elevated ERN in children and adolescents

(Chong et al., 2020), and punitive parenting moderated the relationship between fearfulness at age 2 and increased ERN at age 4 (Brooker & Buss, 2014). Punitive parenting style also has been shown to predict increased ERN magnitude in children 3 years later, which in turn mediated the relationship between punitive parenting and child anxiety (Meyer, Proudfit, et al., 2015). Additionally, adults and adolescents who experienced harsh or punitive parenting in childhood are also characterized by a larger ERN (Banica et al., 2020; Meyer & Wissemann, 2020). Taken together, these findings suggest that children may learn to associate a greater threat-value to their own mistakes through environmental factors including punitive parenting style, which appears to contribute to the development of anxiety.

While there is evidence of a relationship between environmental factors (e.g., punitive parenting style) and an elevated ERN in children, further research is needed to understand the mechanisms of the direct effects of punishment on error monitoring in real-time, in children. Indeed, no study to date has tested the effects of experimentally modulating the ERN by punishing error commission during a laboratory task in children. Furthermore, there is a need to address the question of whether there are developmental periods during which the ERN might be especially prone to modulation by punishment, as well as how the impact of punishment on the ERN relates to anxiety in children (Meyer & Gawlowska, 2017). The present study sought to replicate and extend findings on the effects of task-based punishment on the ERN and its relationship with anxiety to a child sample between the ages of 7 and 11 years old. The present study aimed to examine (1) the impact of punishment on the ERN in children and (2) whether the impact of punishment on the ERN is moderated by age and anxiety symptoms. Based on findings in adults, we predicted that punishment would potentiate the ERN (i.e., the ERN would be larger in the punishment condition compared to the no-punishment condition). In addition, we hypothesized that the impact of punishment on the ERN would be larger among trait-anxious children. Considering the lack of previous work examining the impact of punishment on the ERN in children, we had no a priori hypotheses regarding the impact of age on the punishment potentiation of the ERN; however, we did conduct exploratory analyses to examine this possibility.

2 | MATERIALS AND METHODS

2.1 | Participants

Participants included 84 children recruited from the community through recruitment events and flyers posted in local businesses, libraries, and schools. The children were between the ages of 7 and 11 years; 34% female, 62% male; 11% Hispanic, 86% not Hispanic or Latino; 64% White or Caucasian, 22% Black or African American, 3% Asian, 1% Native Hawaiian or Pacific Islander, and 7% other. The estimated annual family income for participants was as follows: 2% less than $10,000, 13% between $10,000 and $25,000, 8% between $25,000 and $40,000, 33% between $40,000 and $75,000, and 42% greater than $75,000.

Children were excluded if they did not complete the EEG portion of the study visit ($n = 5$), did not have at least six error trials in both conditions ($n = 15$), or had significant noise or poor-quality EEG data ($n = 19$). Therefore, the final sample ($n = 45$) had valid EEG data for both the punishment and no-punishment conditions. Excluded children did not differ on any demographic or main study variables (e.g., age or anxiety symptoms), all $ps > .40$. All participants provided informed consent and study procedures were approved by the institutional review board at Florida State University.

2.2 | Self-report measure

Children completed the Screen for Child Anxiety Related Emotional Disorders (SCARED) questionnaire (Birmaher et al., 1997). The SCARED broadly assesses symptoms of anxiety, including panic, general anxiety, separation anxiety, social anxiety, and school avoidance. The SCARED contains 41 items and participants rate each item as either 0 (not true or hardly ever true), 1 (sometimes true), or 2 (true or often true). In the current study, we focus on the child-report of total anxiety symptoms. The Cronbach's alpha for total anxiety symptoms reported via the SCARED was $\alpha = 0.90$.

2.3 | EEG task

An EEG was recorded while participants completed an arrowhead version of the flanker task on a computer. During each trial, five arrowheads were presented in the center of the screen. Arrowheads were equiprobably either compatible (> > > > > or < < < < <) or incompatible (< < > < < or > > < > >). This task was modified to include both punishment and no-punishment conditions. The punishment condition consisted of blocks of trials during which errors were punished and the no-punishment condition consisted of blocks of trials during which errors were never punished. Prior to starting the task, participants were told that they would see arrows on the screen that were either blue or green and that their job was to click the mouse button depending on the direction of the center arrow. Participants were told they would sometimes hear a "peep" noise (i.e., the punishment) after making a mistake, which had been played for them earlier in the study visit. They were instructed to pay attention to whether they heard the "peep" noise during blocks with green or blue arrows and told they would be asked after the task about the color of the arrows during which they sometimes heard the peep noise following mistakes. The conditions (control vs. punishment) varied block-wise in an alternating way and there were six blocks of each type of condition with 15 trials per block, such that there were 90 total trials during the punishment conditions and 90 total trials during the no-punishment conditions (i.e., total of 180 trials). During the punishment condition, 50% of errors were punished. Prior to the experiment, participants completed a practice block containing 10 trials with no punishments.

All trials began with the presentation of a central fixation cross, with an intertrial interval between 900 and 1500 ms. Flanker

stimuli were presented for 500 ms. During the punishment condition, an aversive loud sound (i.e., the "peep" [100db; 3500 Hz]) was presented via speakers for 1 s following 50% of errors. This sound was presented 1 s after error commission to prevent interference with the ERP response. Following task completion, participants were queried about the color arrows during which they sometimes heard the "peep" tone after they made mistakes. Most participants responded correctly (n = 35, 84% of the sample with useable EEG and self-report data). Moreover, accuracy on this item did not relate to any main study variables (e.g., error-related brain activity, child anxiety, etc.), all $ps > .05$. Additionally, performance-based feedback was presented at the end of each block to encourage both fast and accurate performance. If performance accuracy was below 90%, participants were instructed to respond more accurately. If performance accuracy was above 95%, participants were instructed to respond more quickly. If performance accuracy was between 90% and 95%, participants were told they were "doing a great job."

2.4 | Psychophysiological recording and data analysis

We collected continuous EEG recordings using an elastic cap and the ActiveTwo system (BioSemi). Thirty-four electrode sites were used, along with two electrodes on the right and left mastoids. We measured horizontal and vertical eye movements using four facial electrodes. The EEG signal was preamplified at the electrode to improve the signal-to-noise ratio and amplified with a gain of 1 by an ActiveTwo system. The data were digitized at a 24-bit resolution with a sampling rate of 1024 Hz, using a low-pass, fifth-order sinc filter with a half-power cut-off of 204.8 Hz.

Offline, all data were processed using Brain Vision Analyzer Version 2.1 (Brain Products, Gilching, Germany). EEG data were re-referenced to the average of the left and right mastoids and band-pass filtered between 0.1 and 30 Hz. Ocular corrections were conducted as per Gratton et al. (1983). Artifact detection and rejection was conducted using an automatic procedure: voltage steps greater than 50.0 μV between sample points, voltage differences of 300.0 μV within a trial, and voltage differences of less than 0.50 μV within 100 ms intervals were rejected from channels in each trial. The EEG data were segmented for each trial, from 500 ms before the response to 1000 ms after the response. Correct and incorrect responses were averaged separately for each condition (punishment vs. no-punishment) from −50 to 100 ms after the response to obtain the correct-related negativity (i.e., CRN) and the error-related negativity (i.e., ERN). Analyses focused on the FCz electrode, where error-related brain activity was maximal. In addition to the automatic artifact detection procedure described above, we also examined the relationship between odd and even ERN trials for each condition. Participants who were characterized by a large discrepancy between odd and even ERN trials (i.e., more than 3 SDs above or below the mean) for either condition (i.e., punishment or no-punishment) were subject to visual inspection and excluded from analyses if EEG data were characterized by artifacts leading to poor quality data (n = 19). After artifact rejection and exclusion of participants with poor quality data, internal reliability of the ERN was as follows: for the punishment condition, Spearman-brown split-half reliability = 0.69 and for the no-punishment condition, Spearman-brown split-half reliability = 0.61.

2.5 | Statistical analysis

We conducted statistical analyses using SPSS (Version 26). We used paired-samples t-tests to examine whether the number of errors differed between conditions (punishment vs. no-punishment). Repeated-measures analyses of variances (ANOVAs) were used to examine potential differences in reaction times (i.e., RTs) as a function of response type (error vs. correct) and condition (punishment vs. no-punishment). We used the Pearson correlation coefficient to examine potential relationships between child anxiety, age, accuracy, and RTs. To examine the potential impact of condition, child age, and child anxiety on error-related brain activity, we conducted a repeated-measures ANOVA wherein the ΔERN during each condition (punishment vs. no-punishment) was entered as a within-subjects factor, and child age and total anxiety symptoms (i.e., the SCARED) were entered as between-subject covariates. To decompose interactions, follow-up analyses were conducted wherein we subtracted the ΔERN during the no-punishment condition from the ΔERN during the punishment condition. (i.e., ΔERN punishment−ΔERN no-punishment: the "punishment potentiation of the ΔERN"). This term indicated the extent to which error-related brain activity was increased when participants were potentially punished for making mistakes compared to the condition wherein they received no punishment. We examined the Pearson correlations between the punishment potentiation of the ΔERN, child age, and anxiety. As exploratory analysis, we conducted a regression wherein child age, child anxiety symptoms, and their interaction were entered predicting the punishment potentiation of the ΔERN.

3 | RESULTS

3.1 | Behavioral data

The number of errors committed did not differ by condition, $t(44) = 0.91$, $p = .37$; $M = 33.67$, SD = 19.52 during the punishment condition; $M = 32.13$, SD = 19.52 during the no-punishment condition. And, while reaction time (RT) was overall faster during error trials compared to correct trials, $F(1, 43) = 70.67$, $p < .001$, neither the interaction of condition by response, $F(1, 43) = 0.06$, $p = .81$, nor the main effect of condition, $F(1, 43) = 0.06$, $p = .80$, significantly predicted RTs, suggesting that RTs did not vary as a function of punishment condition. RT descriptives were as follows: punishment condition, error, $M = 494.33$, SD = 109.23; correct, $M = 573.07$, SD = 97.33; and no-punishment condition, error, $M = 491.63$, SD = 110.67; correct, $M = 572.59$, SD = 91.77.

While total anxiety symptoms (i.e., the SCARED) did not relate to task accuracy or RT, both *p*s > .09, child age related to both accuracy and RTs. Older children made fewer errors during the punishment condition, $r(45) = -0.35$, $p < .05$, and were faster during all trial types and in both conditions, all *p*s < .05. However, in a repeated-measures ANOVA, neither the interaction between condition and age, nor the 3-way interaction between condition, age, and response significantly predicted RTs, all *p*s > .20, suggesting that the impact of age on RT did not differ by trial type or condition.

3.2 | Error-related brain activity

Overall, the ERP response was more negative on error trials compared to correct trials, $F(1, 44) = 4.27$, $p < .05$. To isolate error-related brain activity, all remaining analyses focused on the ΔERN (error minus correct). To examine the impact of condition, child age, and child anxiety, we conducted a repeated-measures ANOVA wherein the ΔERN during each condition (punishment vs. no-punishment) was entered as a within-subjects factor, and child age and total anxiety symptoms were entered as between-subject covariates. The two-way interaction between condition (i.e., punishment vs. no-punishment) and age was significant, $F(1, 42) = 4.59$, $p < .05$. Additionally, the two-way interaction between condition (punishment vs. no-punishment) and total anxiety symptoms was also marginally significant, $F(1, 42) = 3.97$, $p = .05$. Moreover, the main effect of condition (punishment vs. no-punishment) was significant, $F(1, 42) = 6.93$, $p < .05$. It should be noted that the pattern of results remained consistent if accuracy, RTs, and child gender were entered as covariates in the model, that is, both two-way interactions (condition by anxiety symptoms and condition by age) remained significant at a trend-level, all *p*s < .06.[1]

To decompose the two-way interaction between condition (punishment versus no-punishment) and age, we subtracted the ΔERN during the no-punishment condition from the ΔERN during the punishment condition (i.e., ΔERN punishment−ΔERN no-punishment: the "punishment potentiation of the ΔERN"). This term indicated the extent to which error-related brain activity was increased when participants were potentially punished for making mistakes compared to the condition wherein they received no punishment. This difference score was correlated with child age, at a trend level, $r(45) = -0.28$, $p = .06$, such that older children were characterized by a larger increase in error-related brain activity during the punishment condition compared to the no-punishment condition (see scatterplot: Figure 1). Figure 2 depicts waveforms for error, correct, and the difference (error minus correct) for both conditions (punishment and no-punishment). For the purposes of visual display, we conducted a median split on child age and waveforms for each condition are displayed for relatively older and younger participants. As shown in the figure, the impact of punishment on the ΔERN among older children was similar to the pattern observed in adults, that is, the ΔERN was larger when errors were potentially punished.

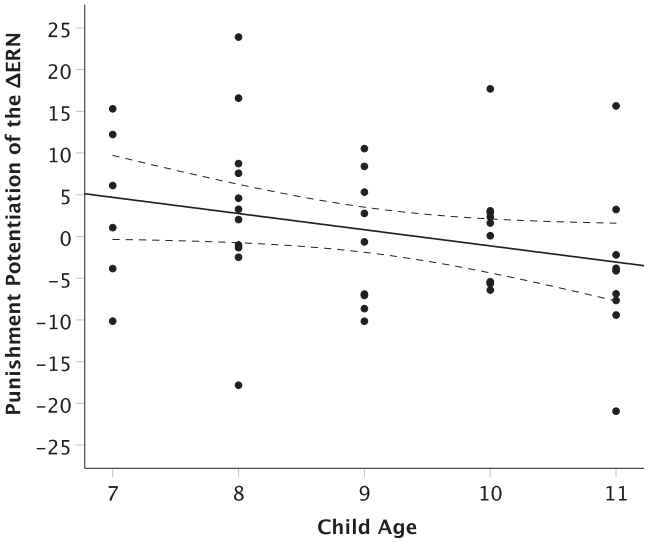

FIGURE 1 Scatterplot depicting the relationship between child age (in years; on the x-axis) and the extent to which punishment potentiated the ΔERN (i.e., ΔERN = error minus correct; in μV, on the y-axis). Older children were characterized by increased error-related brain activity during the punishment condition compared to the no-punishment condition.

Similarly, to decompose the two-way interaction between condition (punishment versus no-punishment) and child total anxiety symptoms, we used the same measure—punishment potentiation of the ΔERN (ΔERN punishment minus ΔERN no punishment)—to indicate the extent to which error-related brain activity was larger in the punishment condition. This difference score was correlated with child total anxiety symptoms, at a trend level, $r(45) = -0.26$, $p = .08$, such that more anxious children were characterized by a larger increase in error-related brain activity during the punishment condition compared to the no-punishment condition (see scatterplot: Figure 3). For the purposes of visual display, in Figure 4, we conducted a median split on total child anxiety symptoms, and waveforms for each condition are displayed for relatively more and less anxious children. As shown in the figure, the impact of punishment on the ΔERN amongst anxious children was similar to the pattern observed in adults, that is, the ΔERN observed among anxious individuals was larger when errors were potentially punished compared to the no-punishment condition.

As an exploratory analysis, we examined whether (1) the relationships between child age and anxiety symptoms and the punishment potentiation of the ΔERN were independent effects and (2) whether child age and anxiety symptoms interact to predict the punishment potentiation of the ΔERN. To do so, we conducted a regression wherein child age, child anxiety symptoms, and their interaction term (child

[1] It should be noted that the pattern of results also remained consistent when the repeated-measures ANOVA was run with response (error vs. correct) as a within-subject factor as well, that is, the three-way interactions between response × condition × age and response × condition × anxiety were both significant, both *p*s ≤ .05. Additionally, in previous work examining the impact of punishment on the ERN, statistical models were run by conducting a median split on anxiety symptoms. To maintain statistical power in the current study, we used both anxiety and age as continuous variables in all analyses. However, it should be noted that when we ran the repeated-measures ANOVA with both age and anxiety symptoms as median-split variables, the interaction between anxiety × age × condition was significant, $F(1, 41) = 8.63$, $p < .01$.

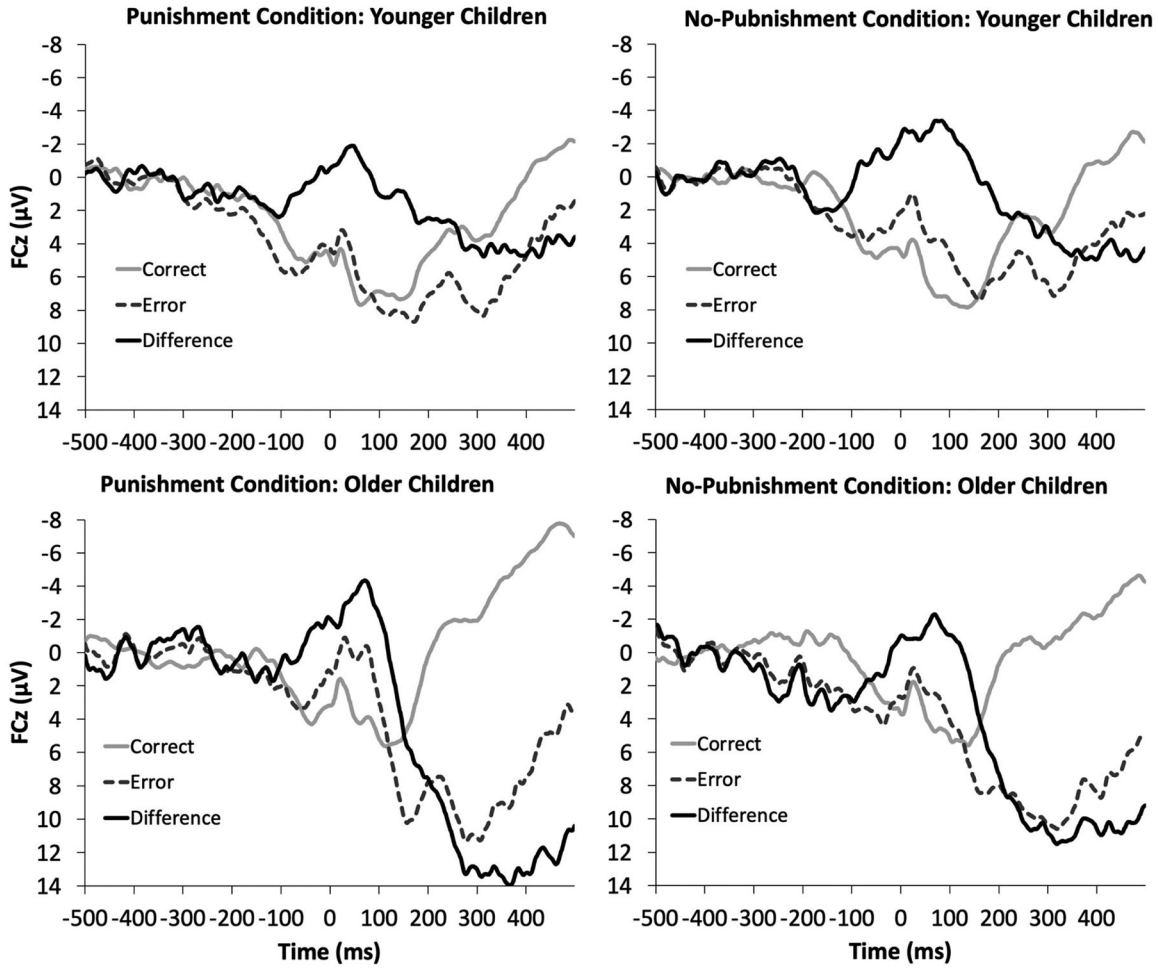

FIGURE 2 Error (dotted line), correct (gray line), and difference (error minus correct; black line) waveforms for the punishment and no-punishment conditions. For the purposes of visual display, we conducted a median split on child age, and waveforms for each condition are displayed for relatively older and younger participants. The impact of punishment on the ΔERN among older children was similar to the pattern observed in adults, that is, the ΔERN was larger when errors were potentially punished.

age by child anxiety symptoms) were entered predicting the punishment potentiation of the ΔERN. Results suggested that the interaction between child age and child anxiety symptoms did not significantly predict the punishment potentiation of the ΔERN, $F(1, 41) = 0.03$, $p = .86$. However, when we removed the interaction term from the equation, the overall model was significant, $F(2, 44) = 3.99$, $p < .05$, and both child age and child anxiety symptoms were significant, unique, predictors of the punishment potentiation of the ΔERN, $t = -2.14$, $\beta = -0.30$, $p < .05$, and $t = -1.99$, $\beta = -0.28$, $p = .05$, respectively. Together, these variables accounted for 16% of the variance in the punishment potentiation of the ΔERN ($R^2 = 0.16$).

4 | DISCUSSION

The present study replicated previous work on the effect of task-based punishment on the ERN and its relationship with anxiety, extending these results to a child sample. In line with our hypotheses and previous findings in adults (Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel, Kathmann, et al., 2019), punishment potentiated the ERN in children, especially among higher trait-anxious individuals. Additionally, the punishment potentiation of the ERN was also associated with child age at a trend level, such that older children had a larger increase in error-related brain activity during the punishment condition compared to the no-punishment condition. These results provide support for the hypothesis that, similar to adults, anxious children may be especially prone to punishment-related alterations in error monitoring and that the impact of punishment on the ERN may be more pronounced as children age.

Our findings are consistent with previous evidence demonstrating that environmental modulation of the perceived threat-value of errors during laboratory tasks potentiates the ERN (Ganushchak & Schiller, 2008; Gehring et al., 1993; Hajcak et al., 2005; Kim et al., 2005). In particular, the current results in children replicate findings among adults in which the punishment potentiation of the ERN was observed among high trait-anxious individuals (Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel, Kathmann, et al., 2019), suggesting that individuals with high levels of trait anxiety may be especially sensitive to punishment

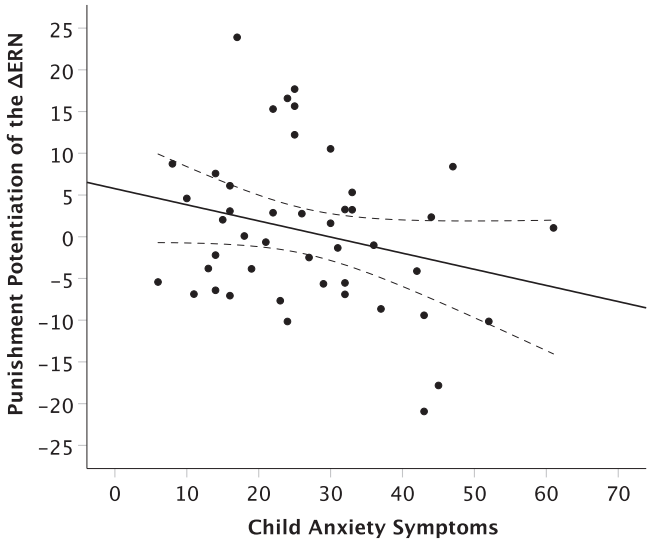

FIGURE 3 Scatterplot depicting the relationship between total child anxiety symptoms (from the Screen for Child Anxiety Related Emotional Disorders [SCARED]; on the x-axis) and the extent to which punishment potentiated the ΔERN (i.e., ΔERN = error minus correct; in μV, on the y-axis). Anxious children were characterized by increased error related brain activity during the punishment condition compared to the no-punishment condition.

of their errors. Based on these previous findings, it appears likely that among anxious individuals, punishment may potentiate the ERN by increasing the threat-value of errors.

While a single laboratory session of errors being punished is unlikely to result in lasting sequelae, the present results are also consistent with studies showing that more distal, naturalistic environmental factors relate to the ERN, such as life stressors and parenting styles. Indeed, greater frequency of stressful life events, and interpersonal stressors in particular (specifically those that may relate to failure, social rejection, or negative interpersonal experiences), predict a larger ERN among children and adolescents (Lackner et al., 2018; Mehra & Meyer, 2022). Additionally, controlling or punitive parenting has been linked to an elevated ERN in children (Brooker & Buss, 2014; Meyer et al., 2019) and punitive parenting prospectively predicted an elevated ERN, wherein the ERN magnitude mediated the relationship between punitive parenting and child anxiety (Meyer, Proudfit, et al., 2015). These findings emphasize punitive parenting as an important environmental factor in shaping the ERN, which may lead children to learn to assign a greater threat-value to their own mistakes, thus contributing to the later development of anxiety. Taken together, high trait-anxious children may be particularly prone to the impact of environmental punishment experiences, thus increasing their ERN, which puts them at risk for further increases in anxiety.

To investigate potential developmental periods of vulnerability to punishment potentiation of the ERN, we conducted exploratory analyses to assess relationships among age, punishment, and ΔERN amplitude. Results indicated that the punishment potentiation of the ERN was associated with child age, such that older children had a larger increase in error-related brain activity during the punishment condition compared to the no-punishment condition. This impact of punishment on the ΔERN among older children is consistent with the pattern observed in adults (Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel, Kathmann, et al., 2019), wherein the ΔERN is larger when errors are potentially punished. However, this finding suggests that older children may be more prone to task-based punishment modulation of the ERN than younger children, which differs from recent findings in which younger children exhibited a larger ERN in relation to punitive parenting and life stressors (Chong et al., 2020; Mehra & Meyer, 2022). These prior studies provide evidence that early childhood may be a developmental period that is particularly sensitive to the impact of the environment on the ERN. However, the current results reflect the possibility that task-based modulation of the threat-value of errors in the laboratory is a less potent environmental factor among younger children compared to older children, whereas more distal or chronic environmental factors (e.g., parenting) may be more impactful among younger children.

One potential reason why younger children might be less sensitive to the effects of task-based punishment on the ERN may relate to developmental maturation of the anterior cingulate cortex (ACC) and other prefrontal regions supporting the error monitoring system (Tamnes et al., 2013). Indeed, the magnitude of the ERN has been shown to increase across development (Davies et al., 2004) which suggests the continuing maturation of the neural systems underlying error monitoring. Furthermore, the neural source of the ERN has been shown to be generated in the ACC in children (Ladouceur et al., 2007), and neuroimaging research has shown that error related dorsal ACC activity increases with age (Velanova et al., 2008). Given these delays in the maturation of brain regions involved in error monitoring, it is possible that younger children may have reduced capacity to integrate punishment following errors as a signal to modify their behavior. In other words, task-based punishment following errors may or may not be experienced as aversive, but still may not signal enhanced error salience to young children, potentially in part due to the neurodevelopmental trajectory of the ACC. Additionally, the Flankers task may be too complex for some younger children to understand, thereby limiting the ability for the potential modulation of the ERN by the delivery of punishment following errors. More work is needed to further assess the impact of task-based punishment on the ERN in young children, potentially using a simplified Flankers task (e.g., using more age-appropriate/engaging stimuli or only including a punishment condition rather than comparing punishment and no-punishment conditions).

Given that the punishment potentiation of the ERN was enhanced among higher trait-anxious individuals as well as older children, we conducted exploratory analyses to assess the possibility that child age and anxiety symptoms may interact to predict the punishment potentiation of the ΔERN. The results suggested that the interaction between child age and child anxiety symptoms did not significantly predict the punishment potentiation of the ΔERN. However, both child age and child anxiety symptoms *uniquely predicted* the punishment potentiation of the ΔERN. This pattern of results suggests that older age and higher trait anxiety both related, independently, to elevated error-related brain activity during the punishment condition. Therefore, the

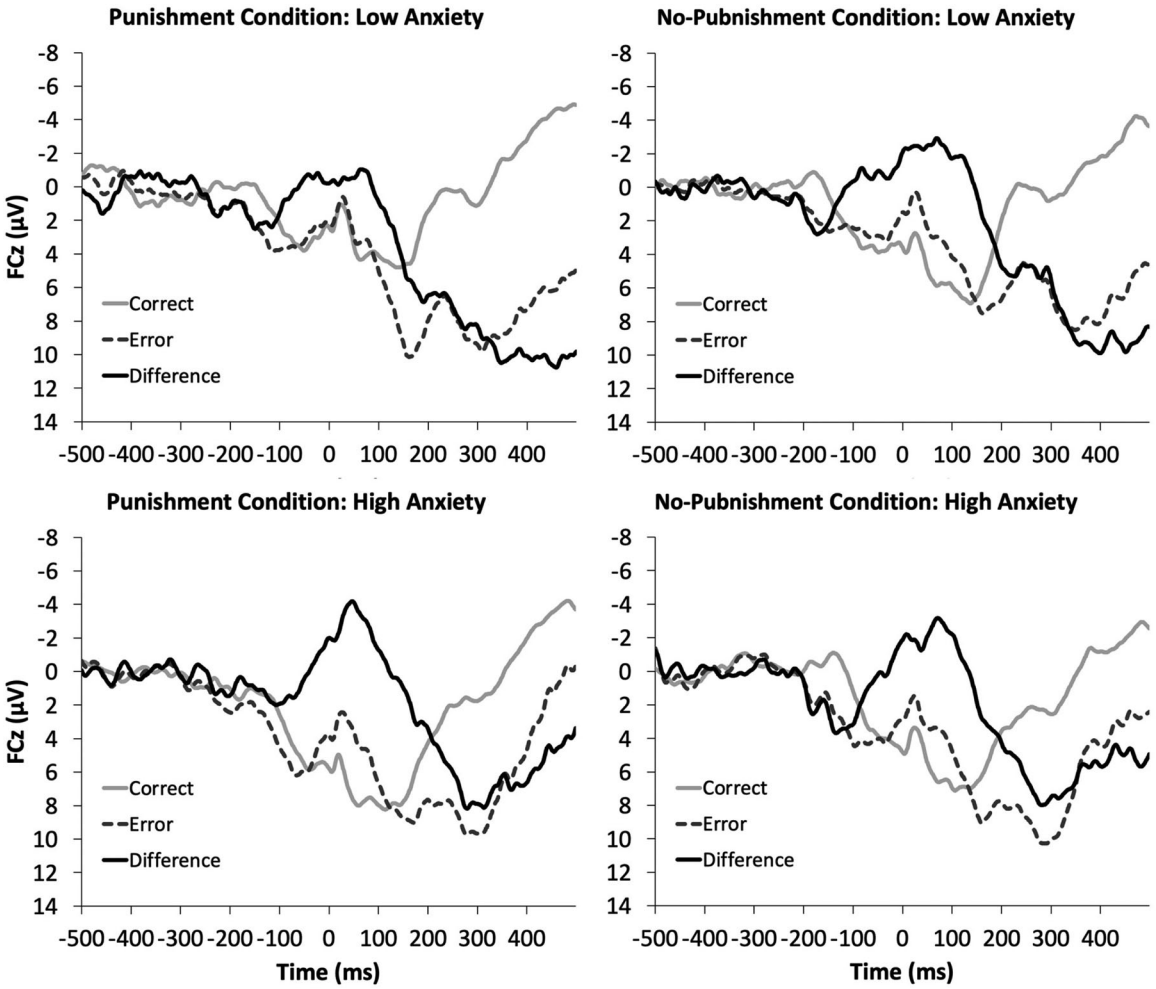

FIGURE 4 Error (dotted line), correct (gray line), and difference (error minus correct; black line) waveforms for the punishment and no-punishment conditions. For the purposes of visual display, we conducted a median split on total child anxiety symptoms and waveforms for each condition are displayed for children characterized by high and low anxiety. The impact of punishment on the ΔERN among anxious children was similar to the pattern observed in adults, that is, the ΔERN was larger among anxious individuals when errors were potentially punished.

effect of age was not attributable to older children being more anxious, or vice-versa. These results also suggest that the impact of age on the punishment potentiation of the ERN did not vary by anxiety level.

One limitation of the current study was a substantial loss of EEG data. Approximately 23% of the EEG data were lost due to substantial noise or low data quality. It is possible that the Flankers task may have been too difficult and thus less engaging for young children, as previous studies with children around 7–11 years old have tended to use Go/No-go tasks, which appear to have a lower proportion of cases excluded due to noise or poor data quality (e.g., 3% data loss; Chong & Meyer, 2019). However, one recent study using a standard Flankers task among children ages 8–12 yielded a slightly lower loss of data across two EEG sessions (approximately 16%; Lin et al., 2021), so it is plausible that the loss of data in the current study may also be related to the addition of the punishment condition. Future studies should assess the potential utility of optimizing the Flankers or adapting Go/No-go tasks with punishment manipulations for use with young children.

5 | CONCLUSION

Despite the limitations discussed above, the current study contributes to our understanding of markers of risk for the development of anxiety in children. The current study was the first to assess the direct effects of experimentally modulating the ERN by providing punishment following error commission during a laboratory task in a child sample. In line with prior studies in adults (Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel, Kathmann, et al., 2019), punishing errors potentiated the ERN among higher trait-anxious children. Furthermore, the punishment potentiation of the ERN was marginally larger among older children, although future research is needed to further understand the developmental trajectory of punishment-related modulation of the ERN. These results have important implications for understanding the mechanisms of how individual differences in the ERN relate to the effects of learning-related punishment feedback (e.g., punitive parenting) on error sensitivity in children. Previous findings have suggested

that harsh or critical parenting may enhance children's sensitivity to errors and risk for anxiety (Meyer et al., 2015). The observed punishment potentiation of the ERN among anxious and older children is important for understanding the mechanisms of harsh parenting and environmental adversity on the neurodevelopment of anxiety in children, which may have implications for improving early intervention and prevention approaches.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data will be made available upon request.

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