



The Presence of a Controlling Parent Is Related to an Increase in the Error-Related Negativity in 5–7 Year-Old Children

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Abstract

Anxiety disorders often begin early in life and there is substantial interest in identifying neural markers that characterize developmental trajectories that result in anxiety. The error-related negativity (ERN) is elicited when people make errors on lab-based reaction-time tasks, is increased in anxious children, and can predict the onset of anxiety across development. In light of this, there is an increasing interest in identifying environmental factors that may shape the ERN in children. Previous work suggests that controlling parenting styles may relate to the ERN in offspring. However, no study had yet examined the specific mechanism whereby parenting style may impact the ERN in children. We propose that it may be children's repeated exposure to making mistakes *in the context of their parents' reactions* (i.e., *verbal or non-verbal reactions, displays of parental control, etc.*) that may lead to an increased ERN. We test this novel hypothesis by measuring the ERN in 94 children between the ages of 5–7 years old, while their parent observes them and then while an experimenter observes them complete a Go-No/Go task. Results suggest that the presence of parents characterized by high control potentiates the ERN in their children. Moreover, the relationship between controlling parenting styles and child anxiety disorder status was mediated by the parent presence potentiation of the ERN. These findings are important and novel insofar as they highlight the impact of an environmental factor (i.e., parenting) in shaping a neural marker of risk for anxiety in children (i.e., the ERN).

Keywords Error-related negativity · ERN · Anxiety · Children · Parenting

Introduction

Anxiety disorders are the most common form of psychopathology and are often associated with substantial lifelong impairment (Kessler et al. 2005). Prospective work has demonstrated that anxiety most often begins early in life and persists into adulthood (Beesdo et al. 2010; Bittner et al. 2007; Copeland et al. 2014; Pine 2007). Additionally, anxiety in children is associated with significant psychosocial impairment (Langley et al. 2004; Strauss et al. 1987) and confers risk for depression, alcohol use, suicidality, and psychiatric hospitalization across the lifespan (Bittner et al. 2007; Costello et al. 2005; Ferdinand and Verhulst 1995; Pine et al. 1998). In light of these findings, it is critical to elucidate early developmental trajectories leading to anxiety disorders. Increasingly, research on the etiopathogenesis

of anxiety has focused on the development of core neural systems (Pine 2007). Identifying neural markers that manifest early in development and relate to the onset of anxiety may increase our ability to implement preventative strategies. Considering evidence that treatment earlier in the course of development results in better long-term functioning (Mancebo et al. 2014), identification of early risk markers is particularly important. Crucially, identifying *modifiable environmental factors* that impact neural markers of risk may provide novel treatment approaches.

A substantial amount of research has focused on an event-related potential (ERP) related to error-monitoring. The error-related negativity (ERN) is elicited when people make mistakes on lab-based reaction-time tasks and appears as a negative deflection in the waveform occurring approximately 50 ms after error commission at fronto-central electrode sites. The ERN is thought to reflect the activation of a generic error monitoring system (Falkenstein et al. 1991; Gehring et al. 1993). Errors are motivationally-salient, internal events that threaten an individual's safety – often requiring immediate attention and corrective action. Indeed, errors do prompt a cascade of physiological changes: skin conductance, heart rate

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deceleration, potentiated startle reflexes, pupil dilation – all which suggest a defensive motivational response in preparation for action (for a review, see: Weinberg et al. 2012a, b). Consistent with this, source localization and fMRI studies suggest the ERN is generated in the anterior cingulate cortex (ACC) – a region of the brain that integrates information about pain, threat, and punishment to change future behavior (Shackman et al. 2011).

Thus, the ERN has been proposed as a biomarker for anxiety disorders (Hajcak 2012; Weinberg et al. 2012a, b). Consistent with this notion, the ERN has been shown to be elevated in anxious adults in over 50 studies to date (Meyer 2017b). An increased ERN has been found in individuals with obsessive-compulsive disorder (Endrass et al. 2008; Gehring et al. 2000; Xiao et al. 2011), generalized anxiety disorder (Weinberg et al. 2012a, b, 2015; Xiao et al. 2011), and social anxiety disorder (Barker et al. 2015; Endrass et al. 2014). Consistent with findings in adults, the ERN is increased in children with anxiety disorders as well (Meyer 2017a). Children with anxiety disorders as young as 6 years old have been shown to have an increased ERN (Meyer et al. 2013). Moreover, we have also found evidence that an increased ERN early in development (5–7 years old) predicts the onset of new anxiety disorders later in development, even when controlling for baseline anxiety symptoms (Meyer et al. 2015). We have recently replicated this finding in adolescents (Meyer et al. 2018), and have shown that children with an elevated ERN are particularly prone to environmentally-induced increases in anxiety symptoms in a large sample of children who experienced Hurricane Sandy (Meyer 2017a, b). In light of these findings, the ERN has been proposed as a neural marker that may be useful in characterizing developmental trajectories associated with risk for anxiety.

Given the fact that the ERN is elevated early in the course of development, before anxiety symptoms become impairing, it is critical to identify factors that may shape the ERN early in life. Although the ERN appears to be moderately heritable (Anokhin et al. 2008), a large portion of variance is better accounted for by environmental factors (40–60%). Work in the lab indicates that the ERN is increased when errors are punished and that this effect persists after punishment ends (Meyer and Gawlowska 2017; Riesel et al. 2012). Thus, learning-related experiences surrounding error commission appear to impact the ERN.

Parenting is one of the most important elements in the *early* childhood learning environment. Controlling parents tend to punish children's mistakes more intensely and more frequently (Robinson et al. 2001) – often resulting in children's excessive concern related to making mistakes (Kawamura et al. 2002). We have proposed that that one mechanism underlying an increased ERN in children may be exposure to controlling parenting styles. In a previous study, 295 parent and child dyads came into the lab when children were 3 years-old and

participated in a structured observational measure of parenting, and parents completed a self-report measure of parenting style. At the follow-up assessment, when children were 6 years-old, the ERN was measured. Results suggested that both observational and self-report measures of authoritarian parenting (high control, low warmth) predicted a larger ERN in offspring (Meyer et al. 2014a, b). A similar pattern of results has also been found in even younger children (i.e., toddlers; Brooker and Buss 2014). Thus, in light of these findings, parenting may play an important role in shaping the magnitude of the ERN in children.

In early childhood, *presence* of the primary caregiver is known to play a potent role in the development of threat-sensitivity in offspring among rodents (Levine 2001), non-human primates (Bayart et al. 1990), and children (Conner et al. 2012; Gee et al. 2014; Gunnar and Donzella 2002; Tottenham 2012). Adults rely primarily on internal sources of feedback and regulation to guide their behavior. However, children have yet to fully develop these internal mechanisms and rely more on external sources (oftentimes the caregiver) for performance monitoring cues (Ghatala 1986; Pressley et al. 1984a, b; Skinner et al. 1988; Stipek and Tannatt 1984; Zimmerman 1989, 1990). Important to the current investigation, parents may scaffold children's emerging ability to recognize and respond to errors. This may take the form of verbal or non-verbal reactions to children's mistakes, or displays of parental control in the context of children's behavior. In light of previous findings linking controlling parenting styles to an increased ERN in offspring, we hypothesize that the mechanism through which the ERN is potentiated in offspring is through *children's repeated exposure to making mistakes in the context of their parents' reactions (i.e., verbal or non-verbal reactions, displays of parental control, etc.)*. Over time, we hypothesize, that children may internalize their parent's reactivity to their mistakes. Thereby, parents who are overly punitive or controlling may be conditioning their children to over-respond to their own mistakes, thus placing them at greater risk for developing anxiety.

In the current investigation, we aimed to replicate and extend previous findings regarding the relationship of parenting styles to the ERN in young children. Additionally, to examine the impact of *parental presence* on the magnitude of the ERN in children, we measured the ERN in 5–7 year-old children while they completed a go/no-go task in two conditions: 1.) while their parent was sitting next to them and 2.) while an experimenter was sitting next to them. Some previous work suggests that the ERN may be increased when an observer is present (Barker et al. 2015; Kim et al. 2005) – and that this potentiation of the ERN may be a meaningful indicator of individual differences. For example, the ERN is increased when individuals' performance on a task is being critically evaluated (Hajcak et al. 2005) and social contexts increase the ERN particularly amongst socially anxious individuals

(Barker et al. 2015), suggesting that social-emotional factors related to being observed impact the ERN (perhaps by making errors more salient).

In the current study, we wished to build on these findings to examine to what extent *the presence of the caregiver* would potentiate the ERN in young children. Additionally, we wished to investigate whether this potentiation would differ by parenting style. The current investigation aims to characterize a more specific mechanism whereby parents may *shape a neural marker of risk for anxiety by being in their child's presence*. We hypothesized that parents characterized by controlling parenting styles would have a greater impact on the magnitude of the ERN in offspring (i.e., the ERN would be increased in children when controlling parents were present). And, we hypothesized that the ERN measured when the experimenter was in the room would not relate to controlling parenting styles. We had no *a priori* hypotheses regarding the relationship of the other parenting styles included (i.e., acceptance and firmness) and the ERN, and included these in the current study to examine the specificity of the relationship between parental control and the ERN. Additionally, we focus on *young* children considering that the impact of parenting on children is generally greater earlier in development (Gee 2016). Based on previous work suggesting that the ERN mediates the relationship between parenting styles and anxiety disorders in children (Meyer et al. 2014a, b), we wished to examine to what extent the ERN measured *when parents were present* would mediate the relationship between parenting style and anxiety disorders in children. We hypothesized that the ERN measured when parents were present, but not the ERN measured when an experimenter was present, would mediate the relationship between controlling parenting styles and anxiety disorders in children. In other words, the extent to which parental presence increased the ERN (i.e., the parent presence ERN minus the experimenter presence ERN) would mediate the relationship between controlling parenting styles and anxiety disorders.

Method

Participants

The overall study included 97 children between the ages of five and seven years old, who were recruited from the Tallahassee community. Families were recruited via recruitment events in the community and fliers that were distributed to local businesses, libraries, and schools. Children and families were eligible for participation if they did not have a significant developmental disorder and had at least one parent fluent in English. Of these children, 79 had complete data for the go/no-go task for both the parent and experimenter conditions. Reasons for missing go/no-go data include: child

refusal ($N=5$), too much movement during EEG recording ($N=2$), child did not make responses during the go/no-go task ($N=1$), computer or experimenter error ($N=2$), child quit during the task ($N=4$), could not get good signal due to child's hair ($N=1$). Additionally, children were only included in analyses if they made at least 6 errors per condition (Meyer et al. 2014a, b; Olvet and Hajcak 2009) and had at least 6 usable EEG trials for each response type (error and correct), as well as condition (parent and experimenter), leading to the exclusion of 3 children.¹ Children excluded from the current study did not differ on any demographic or main study variables, all $ps > .10$.

A total of 36 female and 43 male children participated in the study (total $N=79$). The average age of the child was 5.78 years-old, $SD = .77$. Overall, 7% of the sample identified as Hispanic or Latino; 6% as Asian, 18% as Black, 67% as White, and 7% as Other. Parents were on average 37.27 years-old, $SD = 6.50$. Most parents who participated in the study were female, 88%. Regarding social economic status, 3% of parents reported "some high school or a high school diploma", 29% reported "some college or a 2-year degree", 29% reported obtaining a college degree, and 37% reported obtaining a graduate degree. Additionally, for estimated annual family income, 3% reported making less than \$10,000 per year, 7% reported making between \$10,000–25,000 per year, 9% reported making between \$25,000–40,000, 37% reported making between \$40,000–75,000, and 43% reported making more than \$75,000 per year.

Protocol

During the lab visit, when families arrived in the laboratory, parents and children were consented by a research coordinator. The assessment consisted of a variety of behavioral and psychophysiological measures, as well as the go/no-go task described below. During the lab visit, parents also completed self-report measures, including the CRPBI (Children's Report of Parental Behavior Inventory), as well as a diagnostic clinical interview regarding their child. This research project was approved by the Florida State University Institutional Review Board.

Self-Report: Parental Behavior Inventory, CRPBI

The current study focuses on the self-report measure evaluating parenting styles. Parents completed an adapted version of the Children's Report of Parental Behavior Inventory (i.e., CRPBI; Schludermann and Schludermann 1970). This

¹ Overall, the mean number of useable error trials for each condition was: parent condition = 27.03, $SD = 16.49$, experimenter condition = 23.75, $SD = 19.13$. 90% of children made 10 or more errors during the parent condition and 85% of children made 10 or more errors during the experimenter condition.

version of the CRPBI has been adapted for parents and consists of the shortened version (i.e., 30 items; Schludermann and Schludermann 1988). This measure evaluates parenting style along three dimensions: acceptance (e.g., enjoys doing things with child), control (e.g., I often tell my child how to behave), and firmness (e.g., I am strict with my child). This measure has demonstrated good psychometric properties - alphas between .77 and .90 (McClure et al. 2001; Schludermann and Schludermann 1970; Schludermann and Schludermann 1988). In the current study, the subscales obtained acceptable internal consistency (acceptance: $\alpha = .76$, control: $\alpha = .74$, firmness: $\alpha = .67$).

Clinical Interview: K-SADS-PL

The Schedule for Affective Disorders and Schizophrenia for School-Age Children: Present and Lifetime Version (K-SADS-PL; Kaufman et al. 1997) was administered to parents regarding their children's current and past psychopathology. The interviews were administered by a Ph.D. level clinician and clinically trained research interviewers who were trained and supervised by the Ph.D. level clinician. All interviews were recorded. The K-SADS is designed to assess a range or psychopathology in children and lifetime, as well as current, diagnoses were derived from the parent report on the child. The K-SADS demonstrates excellent test-retest reliability and interrater agreement (Kaufman et al. 1997). All diagnoses were reviewed in case conferences led by an experienced clinical psychologist.

In the current study, we focused on current subthreshold and threshold anxiety disorders. Subthreshold disorders were defined as children who displayed at least 1 threshold symptom of a disorder in combination with at least 1 other subthreshold symptom and significant impairment related to the disorder. Of children with usable EEG and self-report data, 28 had a current subthreshold or threshold anxiety disorder. Of these children, 1 met criteria for current subthreshold panic disorder, 10 met criteria for current subthreshold separation anxiety disorder and 2 met for current threshold separation anxiety disorder, 2 met criteria for subthreshold current simple phobia and 8 met for current threshold simple phobia, 1 met criteria for current subthreshold social phobia and 2 met for current threshold social phobia, 13 children met criteria for current subthreshold generalized anxiety disorder and 3 met criteria for current threshold generalized anxiety disorder, 6 met criteria for current subthreshold OCD, 1 met criteria for current subthreshold anxiety disorder not otherwise specified (NOS), and 5 met criteria for current threshold anxiety disorder NOS. Based on 30 audio-recorded interviews that were scored by a second rater, interrater reliability for subthreshold and threshold anxiety disorders was good ($\kappa = .81$).

Tasks and Materials

EEG was recorded from children as they completed an age-appropriate go/no-go task. Children completed the go/no-go task while their parent sat next to them and then in another condition, while an experimenter sat next to them. When parents participated in the parent presence condition, they were instructed to sit next to their child and watch their child complete the game. During the experimenter condition, the research assistant sat in the same position the parent sat in and watched children complete the go/no-go task. The parent and experimenter condition were counterbalanced across participants. Children were instructed that they needed to "shoot" aliens (i.e., press the mouse button) as soon as they saw them appear on the screen and "save" astronauts (i.e., refrain from pressing the mouse button) when they appeared on the screen. Stimuli consisted of an image of an alien or an astronaut that appeared on the screen for 500 ms, with an ITI of 1000 to 2000 ms. After receiving instructions and completing 5 practice trials, children completed 200 trials in each condition (400 trials total).

Psychophysiological Recording and Data Analysis

Continuous EEG recordings were collected using an elastic cap and the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands). Thirty-four electrode sites were used, as well as two electrodes on the left and right mastoids. Electrooculogram (EOG) generated from eye movements and eye blinks was recorded using four facial electrodes: horizontal eye movements were measured via two electrodes located approximately 1 cm outside the outer edge of the right and left eyes. Vertical eye movements and blinks were measured via two electrodes approximately 1 cm above and below the right eye. The EEG signal was preamplified at the electrode to improve the signal-to-noise ratio and amplified with a gain of one by a BioSemi ActiveTwo system. The data was 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 cutoff of 204.8 Hz. Each active electrode was measured online with respect to a common mode sense (CMS) active electrode producing a monopolar (non-differential) channel. Offline, all data was referenced to the average of the left and right mastoids, and band-pass filtered between 0.1 and 30 Hz; eye-blink and ocular corrections were conducted per Gratton et al. (1983). A semi-automatic procedure was employed to detect and reject artifacts. The criteria applied was a voltage step of more than 50.0 μV between sample points, a voltage difference of 300.0 μV within a trial, and a maximum voltage difference of less than .50 μV within 100 ms intervals. These intervals were rejected from individual channels in each trial. Visual inspection of the data were then conducted to detect and reject any remaining artifacts.

The EEG data were segmented for each trial beginning 500 ms before the response and continuing for 1000 ms after the response. The response-locked ERPs were averaged separately for each trial type (e.g., correct and incorrect responses), and baseline correction was performed using the interval from -500 to -300 ms. For each individual, a difference score was calculated (error minus correct) at mid-line electrode Cz, where error-related brain activity was maximal. Peak detection was used to identify the most negative peak of the difference wave from -50 to 100 ms around the response and the average activity 50 ms around the identified peak for each individual was exported to derive the ERN. Behavioral measures included the number of error and correct trials for each subject, as well as average reaction times (RTs) in each condition.

Statistical analyses were conducted using SPSS (Version 17.0) General Linear Model software, with Greenhouse-Geisser correction applied to p values associated with multiple-df, repeated-measures comparisons when necessitated by the violation of the assumption of sphericity. A repeated-measures ANOVA was utilized to examine error-related brain activity by condition (experimenter vs. parent) and potential interactions with parenting styles (acceptance, control, firmness). Additionally, we utilized a regression-based method of calculating the difference in error-related brain activity between conditions (experimenter vs. parent) based on recent work suggesting this approach may provide a superior measure of within-subject variance (Meyer et al. 2017a, b, c). The Pearson correlation coefficient (r) was used to conduct follow-up analyses examining associations between error-related brain activity and parenting styles. Additionally, we conduct regression analyses controlling for child age, gender, RTs and accuracy during the task to examine the specificity of the relationships between error-related brain activity and parenting styles. And then, as an exploratory analysis, we conducted analyses with all of the items on the CRPBI and error-related brain activity to further examine specific aspects of parenting that may relate to child ERNs.

To examine a mediation model wherein the relationship between parenting style and child anxiety disorders was mediated by the parent-potential of the ERN, we utilized a nonparametric bootstrapping approach (MacKinnon et al. 2004). This approach has been shown to be more statistically powerful than other tests of mediation (MacKinnon et al. 2002). We used an SPSS macro (Preacher and Hayes 2004), which provides a bootstrap estimate of the indirect effect between the independent and dependent variable, an estimated standard error, and 95% confidence intervals for the population value of the indirect effect. When confidence intervals for the indirect effect do not include zero, this indicates a significant indirect effect at the $p < .05$ level. Direct and indirect effects were tested using 5000 bootstrap samples.

Results

Parenting Style

Overall, the average score on the CRPBI acceptance scale was 28.35, $SD = 1.90$. The average score on the CRPBI control scale was 14.43, $SD = 2.29$, and the average score on the CRPBI firm scale was 22.00, $SD = 2.66$. None of the CRPBI scales related to parent or child age, nor did they relate to parent or child gender, all $ps > .10$.

Error-Related Brain Activity

To examine the extent to which parenting style may have impacted the magnitude of ERN when parents were in the room versus an experimenter, we performed a repeated-measures ANOVA with condition entered as a within-subject variable (parent vs. experimenter) and the CRPBI parental scales (control, acceptance, and firmness) entered as covariates. While neither parental acceptance nor firmness interacted with condition, both $ps > .10$, results suggested that the interaction between condition (parent vs. experimenter) and CRPBI parental control was significant, $F(1, 29) = 4.23$, $p < .05$, $\eta^2 = .06$.

To deconstruct this interaction, we created difference scores between the two conditions (parent vs. experimenter) using the saved residuals from regressions (i.e., we conducted a regression wherein the experimenter ERN was entered predicting the parent ERN and the unstandardized residuals are saved as a measure of the difference between conditions that is specific to the parent condition and then vice versa). This approach has previously been shown to be beneficial (Meyer 2017a, b). We then conducted correlations with between parenting styles on the CRPBI and the ERN measured during the parent and experimenter condition, as well as the difference scores between the conditions (see Table 1). As can be seen in Table 1, children of parents characterized by an increased controlling parenting style displayed an increased (i.e., more negative) ERN during the parent condition. This can be seen in the significant correlation between CRPBI control and the ERN during the parent condition, as well as the significant correlation between CRPBI control and the ERN difference score (the difference between the parent and experimenter condition). In other words, children with controlling parents displayed an increased ERN during the parent condition, and *the extent to which parental presence potentiated the ERN in children related to parental control* (as reflected by the residualized difference score). We have depicted the correlation between the residualized difference score of the parent condition ERN and the CRPBI controlling scale in Fig. 1. Additionally, for illustration purposes, in Fig. 2, we present waveforms (error, correct, and the difference – error minus correct) and topographical headmaps (error minus correct) during the parent condition for high and low CRPBI control

Table 1 Correlations between the three CRPBI subscales measuring parenting style (control, acceptance, and firmness) and the ERN in the parent condition, the ERN in the experimenter condition, the residualized ERN difference score for the parent and experimenter condition

	CRPBI. Parental Control	CRPBI. Parental Acceptance	CRPBI. Parental Firmness
ERN parent condition	-.31**	.01	-.05
ERN experimenter condition	.13	-.06	-.01
ERN difference score parent condition	-.29**	.05	-.07
ERN difference score experimenter condition	.03	-.11	.06

* $p < .05$, ** $p < .01$

groups based on a median-split. As can be seen in the figure, children characterized by parents high in control display an increased ERN during the parent condition compared to children characterized by parents low in control.

To further examine specificity, we conducted a simultaneous regression wherein we entered all three CRPBI parent scales (acceptance, control, and firmness) predicting the residualized difference score for the ERN in the parent condition. Results suggested that while neither acceptance nor firmness related to the ERN parent condition residualized score, parental control was a significant predictor, $B = -.32$, $t = -2.65$, $p < .01$. In other words, even when controlling for the impact of the other parental subscales, only parental control predicted the extent to which parental presence potentiated the ERN in children.

Considering that behavior during the task (accuracy or reaction time; RT), child age, and child gender may relate to the ERN or parenting style (or their relationship), we conducted a simultaneous regression wherein we entered CRPBI parental control, as well as accuracy during both conditions (parent and experimenter), RT during both conditions (parent and experimenter), as a well as child age and gender into a regression predicting the residualized difference score for the ERN in the parent condition. Results suggested that the only two

significant predictors were CRPBI parental control and RT during the parent condition, $B = -.34$, $t = -2.87$, $p < .01$, and $B = .64$, $t = 3.26$, $p < .05$, respectively. The positive associated between RT during the parent condition and the residualized difference score for the ERN in the parent condition suggest that the extent to which children slowed down (i.e., had larger/longer reaction times) during the parent condition related to less potentiation of the ERN during the parent condition. Additionally, the association between parental control and the residualized difference score for the ERN in the parent condition remained significant even after controlling for behavior during the task, child age, and child gender.

Child Anxiety Disorders

Overall, 28 children in the study met criteria for at least one subthreshold or threshold anxiety disorder. As can be seen in Fig. 3, the ERN during the parent condition was larger (i.e., more negative) in children with anxiety disorders, $M = -18.84$, $SD = 10.03$, compared to healthy children, $M = -12.03$, $SD = 10.62$, $F(1, 71) = 6.70$, $p < .01$, $\eta^2 = .09$. However, the ERN measured during the experimenter condition did not differ between anxious, $M = -17.94$, $SD = 11.85$,

Fig. 1 A scatterplot depicting the relationship between controlling parenting style (as measured by the CRPBI) and the residualized difference score for the parent vs. experimenter condition

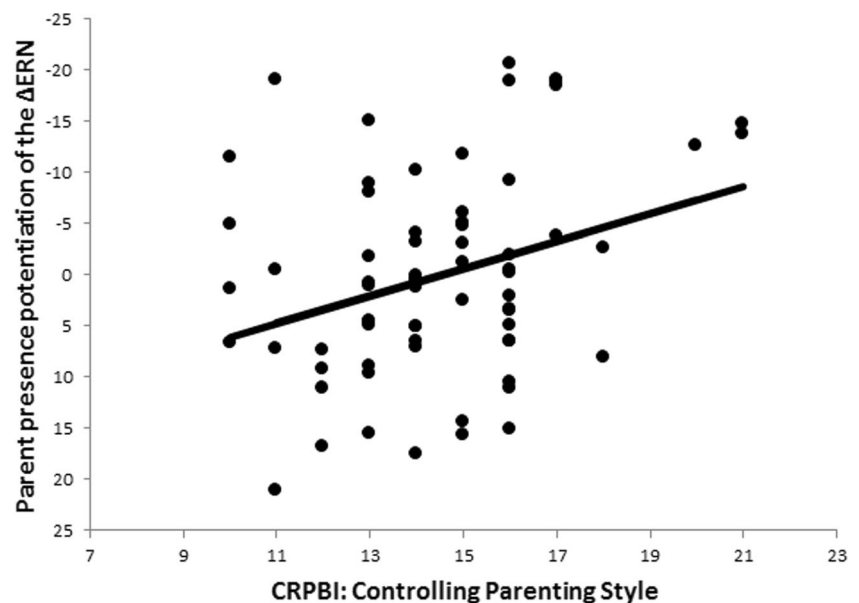
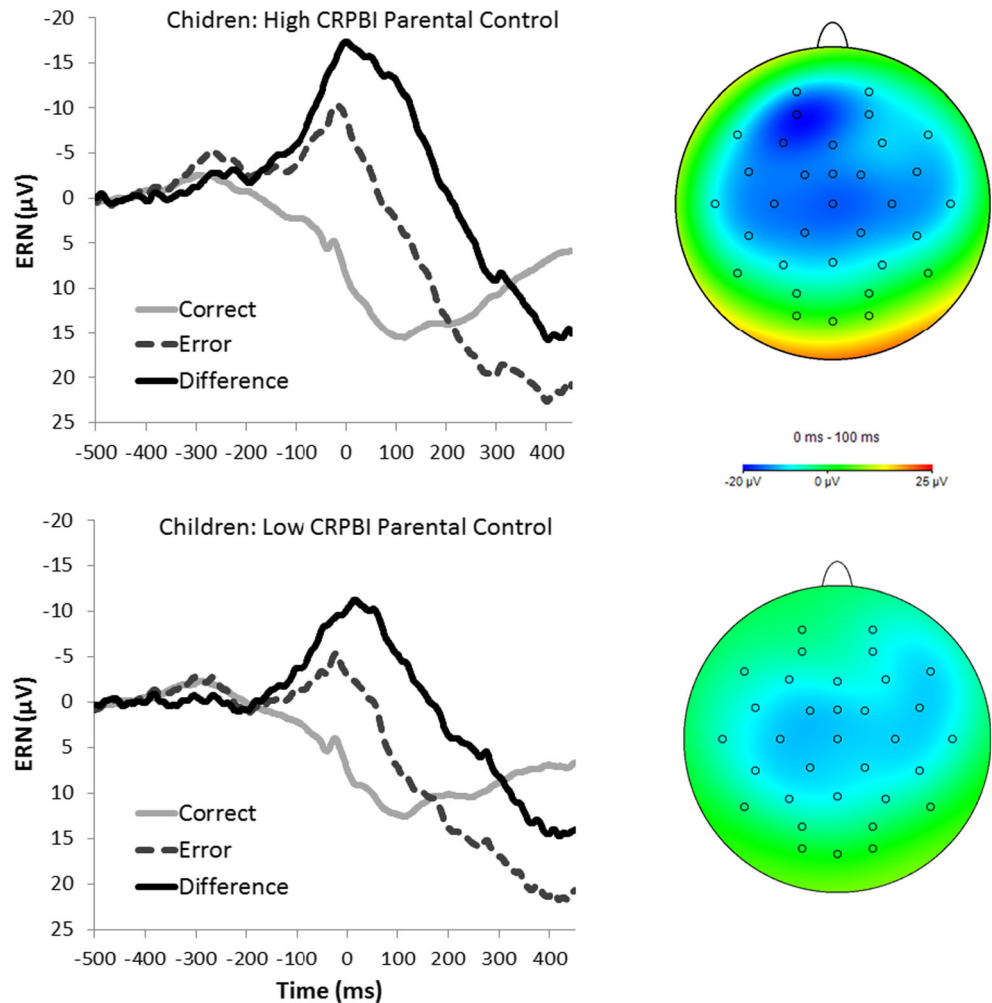


Fig. 2 For the purposes of graphical depiction, the sample was divided based on a median-split on parental control (measured by the CRPBI). The waveforms (error, correct, and error minus correct) for the parent present condition are depicted on the left. On the right, topographical headmaps are depicted for 0–100 ms after the response (error minus correct)



and non-anxious children, $M = -16.73$, $SD = 11.83$, $F(1, 71) = .18$, $p = .68$. Additionally, the residualized difference score ERN in the parent condition (the extent to which parental presence increased the ERN relative to the experimenter condition) was increased in anxious, $M = -3.97$, $SD = 8.30$, compared to non-anxious children, $M = 1.86$, $SD = 10.22$, $F(1, 71) = 5.70$, $p < .05$, $\eta^2 = .08$. However, the residualized ERN in the experimenter condition did not differ between anxious, $M = .11$, $SD = 10.32$, and non-anxious children, $M = -.05$, $SD = 10.90$, $F(1, 71) = .00$, $p = .95$.

We previously reported a mediation model where the ERN mediated the relationship between authoritarian parenting (high control, low warmth) and child anxiety disorder status (Meyer et al. 2014a, b). In the current study, we examine a similar mediation model wherein the relationship between controlling parenting style and child anxiety disorders (sub-threshold and threshold) is mediated by the parent potentiation of the ERN (i.e., the residualized difference score ERN in the parent condition; see Fig. 4). In this model, parental control related to the parent potentiated ERN, $coeff = -1.33$, $se = .48$, $t = -2.75$, $p < .01$, $95\%CI [-2.29 \text{ to } -.37]$. And, the parent potentiated ERN related to child anxiety disorder status,

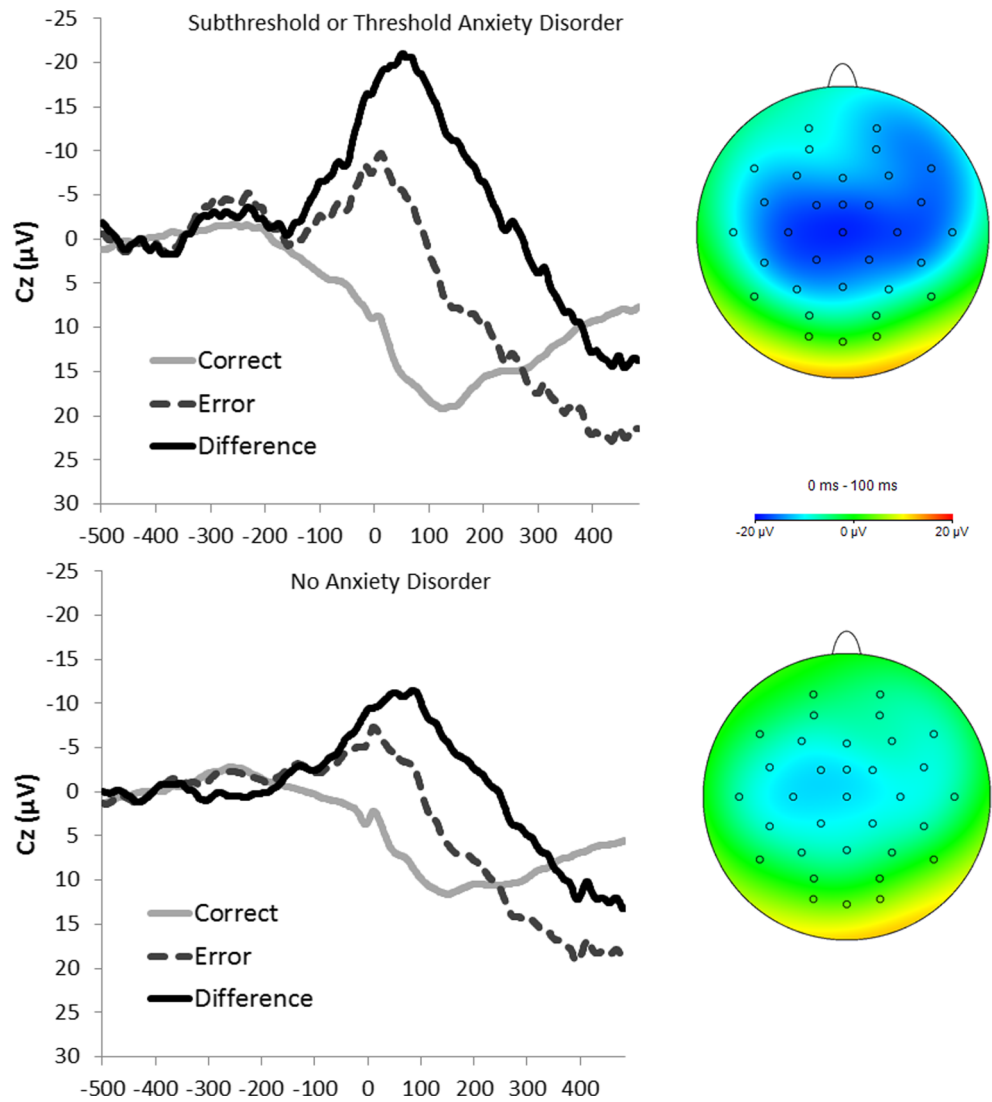
$coeff = -.06$, $se = .03$, $z = -2.03$, $p < .05$, $95\%CI \{-.12 \text{ to } -.01\}$. While the direct path between parental control and child anxiety disorder status did not reach significance, $coeff = .08$, $se = .11$, $z = .69$, $p = .49$, $95\%CI [-.15 \text{ to } .31]$, there was a significant indirect effect of controlling parenting on childhood anxiety disorders, mediated through the parent potentiated ERN, $effect = .08$, $se = .06$, $95\%CI [.01 \text{ to } .22]$.^{2,3}

We also examined a mediation model wherein the relationship between controlling parenting style and child anxiety disorders status was mediated by the experimenter potentiation of the ERN (i.e., the residualized difference

² The pattern of results was the same if we used the ERN in the parent condition or the parent potentiated ERN (i.e., the residualized difference score). Overall, results were consistent with the parent condition ERN mediating the association between controlling parenting and child anxiety disorder status, $effect = .08$, $se = .06$, $95\%CI [.01 \text{ to } .24]$.

³ If we conducted the mediation model entering both the ERN in the parent condition and the ERN in the experimenter condition as simultaneous mediators, the pattern of results remained the same – i.e., the ERN measured during the parent present condition mediated the relationship between parental control and child anxiety disorders, $effect = .08$, $se = .06$, $95\%CI [.08 \text{ to } .21]$, while the ERN measured during the experimenter condition did not, $effect = .00$, $se = .02$, $95\%CI [-.03 \text{ to } .03]$.

Fig. 3 Waveforms and headmaps are presented for children with an anxiety disorder (threshold or subthreshold; top) and for children without an anxiety disorder (bottom). The waveforms (error, correct, and error minus correct) for the parent present condition are depicted on the left. On the right, topographical headmaps are depicted for 0–100 ms after the response (error minus correct)

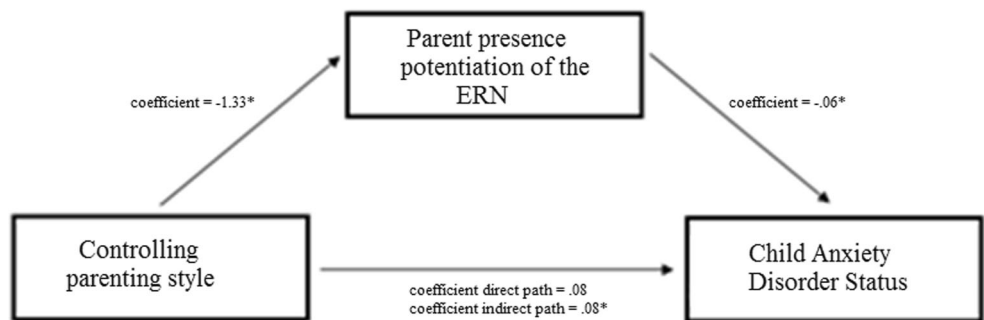


score ERN in the experimenter condition). The indirect path from controlling parenting style to child anxiety disorders status via the experimenter potentiation of the ERN failed to reach significance, $effect = -.00$, $se = .02$, $95\%CI [-.07 \text{ to } .03]$. Thus, the extent to which the experimenter potentiated the ERN did not mediate the relationship between parenting and child anxiety.

Discussion

Consistent with previous work indicating that controlling parenting styles impact the ERN in offspring (Brooker and Buss 2014; Meyer 2017b; Meyer et al. 2014a, b), results from the current investigation suggest that the presence of parents characterized by high control potentiates the ERN in their children.

Fig. 4 Figure depicting a mediation model wherein the relationship between controlling parenting and child anxiety disorder status is mediated by the parent-potentiation of the ERN



Furthermore, the *extent to which parents potentiate the ERN in offspring* compared to an experimenter, also relates to controlling parenting styles. Additionally, the parent potentiation of the ERN mediated the association between controlling parenting styles and anxiety disorder status, suggesting that the parent-potentiated ERN may be one mechanism through which parenting impacts child anxiety. Moreover, these findings are important and novel insofar as they highlight the impact of an environmental factor (i.e., parenting) in shaping a neural marker of risk for anxiety in children (i.e., the ERN).

Previous work suggests that parenting styles relate to the ERN in children. However, the mechanism by which parenting style may impact the ERN in offspring is not yet well understood. Results from the current study suggest a more specific mechanism whereby the *context of a controlling parent* may shape a neural marker of risk for anxiety in children. Young children have yet to fully develop internal mechanisms of performance monitoring and thus may rely on the primary caregiver for performance monitoring cues. Parents may scaffold children's emerging ability to recognize and respond to errors by displaying verbal or non-verbal reactions to children's mistakes or by displaying controlling behavior in the context of children's performance. Results from the current investigation suggest that children's repeated exposure to making mistakes in the context of a controlling parent may be one mechanism through which the ERN is shaped.

The current investigation focused on children early in childhood (between the ages of 5 and 7 years-old). We chose to do so because the impact of parenting on children is generally greater earlier in development (Gee 2016). While we expect parenting and thus, parental presence, to have the greatest impact early in childhood, future work could examine the relationship between parenting and the ERN across a larger age-range. It is possible that there are sensitivity periods in development wherein the ERN is more vulnerable to the impact of controlling parenting styles, and thus, may provide unique opportunities for intervention to prevent the onset of psychopathology. Future works should also consider the impact of various other environmental factors on the ERN in children (e.g., socioeconomic status, stressors, adversity, peer relationships, nutrition/health, etc.). For example, it is possible that later in development, peer criticality may become more impactful on the ERN than parental behavior. It is also possible that specific types of stressors related to performance (e.g., failing an important test, making a mistake during a sports game), may impact the ERN.

Results from the current study suggest that presence of controlling parents may impact a neural marker of risk for anxiety (i.e., the ERN). While previous work has demonstrated that an increased ERN in childhood relates to risk for subsequent anxiety disorders (McDermott et al. 2009;

Meyer et al. 2015; Meyer et al. 2018), it is possible that the parent-potentiated ERN may be a superior predictor of risk for anxiety, compared to the ERN measured while children are alone. We would expect the parent-potentiated ERN to capture variance unique to sensitivity to making mistakes related to external evaluation – which may be a better indicator of risk. Future work should examine the extent to which the parent-potentiated ERN may predict new onset anxiety disorders across development.

Findings from the current investigation suggest that the ERN was uniquely potentiated by parents characterized by high control. However, parenting styles related to acceptance and firmness were unrelated to the potentiation of the ERN. Moreover, it should be noted that the internal reliability of the firmness scale was low to moderate (.67). The CRPBI factor of control is thought to index “covert, psychological methods of controlling the child's activities and behaviors that would not permit the child to develop as an individual apart from the parent” (Schaefer 1965). Indeed, there is some evidence that this type of control may be particularly problematic and predicts internalizing problems across development (Barber 1996; Pettit et al. 2001). While previous work on the relationship between the ERN and parenting found relationships with observed hostility and self-report authoritarian style (i.e., high control, low warmth; Meyer, Proudfit, et al., 2014), the current study focuses specifically on parental control. Moreover, much work has focused on the relationship between parental over-protection and child anxiety (Bögels and Brechman-Toussaint 2006; Bögels and Melick 2004; Moore et al. 2004) – a construct that was not measured in the current study. Future work should aim to identify a more specific model of parenting behaviors and attitudes that may impact the ERN, and thus risk for anxiety, in offspring. Doing so may pave the way for novel parent-based intervention strategies. For example, brief interventions may be utilized to target this specific aspect of parenting behavior in the context of children with an elevated ERN.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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