



## The association between parenting and the error-related negativity across childhood and adolescence

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### ABSTRACT

Anxiety is the most common form of psychopathology, and it is often characterized by chronic impairment across the lifespan. Researchers have identified core neural markers that confer risk for anxious outcomes. An increased error-related negativity (ERN) in anxious individuals has been shown to prospectively predict onset of anxiety disorders across development. Hence, it is critical to examine environmental factors that may shape the ERN. In the current study, we use a large sample of 170 female adolescents aged 10–17 to investigate whether the ERN mediates the relationship between parenting style and anxiety diagnostic status. This study replicates previous findings, and it extends previous work by suggesting that this relationship is more robust in young children as compared to adolescents. Interventions targeting the ERN via parenting may be most effective during childhood.

### 1. Introduction

Anxiety disorders are the most common type of psychopathology, frequently beginning in early childhood and resulting in chronic impairment across the lifespan (Beesdo et al., 2009a, 2009b; Kessler et al., 2005). A nationally representative survey of American adolescents using clinical interview methods (i.e., the National Comorbidity Survey – Adolescent Supplement [NCS-A]) showed that nearly a third of American adolescents had experienced some form of DSM-IV anxiety disorder (Merikangas et al., 2010). Anxiety disorders are also associated with high societal costs (Bodden et al., 2008) – children with anxiety disorders have an elevated risk for psychosocial impairments and comorbid disorders such as depression and substance use (Bittner et al., 2007; Strauss et al., 1987; Woodward and Fergusson, 2001). In addition, anxiety disorders are associated with 60 % higher mortality rates, resulting in a life expectancy of eight fewer years than average (Pratt et al., 2016).

A growing body of research has focused on identifying neural biomarkers underlying anxiety disorders (Pine, 2007), which may allow for novel cognitive, behavioral, and pharmacological treatment approaches that result in improved long-term functioning (Mancebo et al., 2014). Consequently, identifying developmental trajectories that lead to anxiety disorders may increase our understanding of the etiopathogenesis of clinical anxiety and aid in the development of prevention and intervention strategies.

Substantial evidence suggests that clinical anxiety is associated with an increased neural response to errors (i.e., larger error-related negativity, ERN; Cavanagh and Shackman, 2014; Meyer, 2016; Meyer et al., 2016). The ERN is thought to index a generic error monitoring system (Falkenstein et al., 1991; Gehring et al., 1993). It is observed as a sharp negative deflection in the event-related potential (ERP) waveform at fronto-central sites, and peaks at approximately 50 ms following the commission of an error (Gehring et al., 1993). Researchers have theorized that between-subject variability in the ERN reflects individual differences in sensitivity to making mistakes (i.e., error sensitivity; e.g., Chong and Meyer, 2018; Meyer, 2017). Anxious individuals are known to be especially sensitive to threat-related cues (Bar-Haim et al., 2007). In this model, errors are perceived as an internally generated threat, to which clinically anxious individuals may be highly attuned (Muris et al., 2000, 2003). Thus, the ERN may be increased in individuals with anxiety disorders because errors activate the threat response system.

Over 50 studies have shown that the ERN is elevated in clinically anxious adults, and the ERN has been proposed as a biomarker for anxiety disorders (Meyer, 2017; Weinberg et al., 2012a, 2012b). There are two meta-analyses on this topic (Cavanagh and Shackman, 2014; Moser et al., 2013). The results from these have estimated the relationship between the ERN and anxiety to have a small-to-medium effect size ( $r = -.25$ ); moreover, this relationship was shown to be moderated by anxiety type (i.e., anxious apprehension versus anxious distress; Moser et al., 2013), wherein the ERN is elevated in disorders

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characterized by anxious apprehension (i.e., cognitive symptoms of anxiety). These disorders include: generalized anxiety disorder (GAD; Weinberg et al., 2012a; Weinberg et al., 2010; Xiao et al., 2011), obsessive-compulsive disorder (OCD; Endrass et al., 2008; Lahat et al., 2014; Weinberg et al., 2015), and social anxiety disorder (Barker et al., 2015; Endrass et al., 2014). In contrast, the ERN is not elevated in disorders characterized by anxious arousal (i.e., acute fear response, e.g., panic disorder, phobia, PTSD; Hajcak et al., 2003; Rabinak et al., 2013).

An elevated ERN has been demonstrated in clinically anxious children as young as 6 years old (Meyer et al., 2013a, 2013b), children with OCD (Hanna et al., 2012), children with subclinical OCD (Santesso et al., 2006), older adolescents with subclinical anxiety (Meyer et al., 2012), children characterized by early temperamental behavioral inhibition (McDermott et al., 2009), and unaffected siblings of children with OCD (Carrasco et al., 2013). Additionally, after controlling for baseline symptoms, an elevated ERN in early childhood predicts the onset of new anxiety disorders later in development (Meyer et al., 2015a), and this finding has been replicated in adolescents (Meyer, Nelson, et al., 2018). Children with larger ERNs at baseline were also more prone to increases in anxiety symptoms in response to environmental stressors (i.e., Hurricane Sandy; Meyer, 2016; Meyer et al., 2016). Thus, the ERN appears to be a correlate of clinical anxiety, as well as a *predictor of risk* for anxiety disorders.<sup>1</sup> While the ERN magnitude appears to be moderately heritable (Anokhin et al., 2008), a substantial amount of variance (about 40–60 %) is unaccounted for by genetic factors, suggesting that environmental influences may play a large role in the development of the ERN. Empirical studies have implicated some of these environmental influences. For instance, punishment for errors on lab-based tasks (e.g., receiving an electrical shock after error commission) increases the magnitude of the ERN, and this effect may last for up to 24 h after punishment ends (Meyer and Gawlowska, 2017; Riesel et al., 2019, 2012). Hence, one crucial environmental factor that may impact the ERN is learning experiences surrounding error commission.

A child's learning environment is largely shaped by their caregivers' behavior. While adults possess internal mechanisms for feedback and regulation to guide behavior, children may more often rely on external sources (e.g., a caregiver) for performance monitoring cues (Bernier et al., 2010; Ghatala, 1986; Skinner et al., 1988; Zimmerman, 1990). Abundant work has shown that harsh, restrictive parenting styles may lead to poor outcomes in children and adolescents. Indeed, a large body of evidence suggests that adolescents growing up with critical or harsh parenting are at increased risk for negative outcomes, such as externalizing behaviors, withdrawn behavior, trait anxiety and clinical anxiety, depression symptoms, depersonalization, interpersonal rejection sensitivity, anger, and poor health (Booth-LaForce et al., 2012; Burnette et al., 2012; Hale et al., 2006; Rowe et al., 2015; Wolfradt et al., 2003). A large meta-analysis also revealed that authoritarian and other restrictive parenting styles were associated with greater internalizing symptoms in children and adolescents (Pinquart, 2017). Research has also demonstrated the importance of overreactive parental discipline in children's anxiety etiology, showing that dysfunctional cognitive styles partially mediated the relationship between punitive parenting and anxiety (Gallagher and Cartwright-Hatton, 2008). In this study, harsh and punitive parenting were more strongly related to elevated anxiety in children than other aspects of parenting style, such as warmth, overprotection, and rejection. Further evidence for the importance of harsh parenting style in the etiology of anxiety comes from treatment literature demonstrating decreased anxiety symptoms in anxiety-disordered offspring of parents who underwent training to decrease harsh and

inconsistent parenting behaviors (Cartwright-Hatton et al., 2005). Harsh and authoritarian parenting styles have also been linked to offspring's increased concern over making mistakes, and this relationship was shown to be even stronger in girls (Kawamura et al., 2002). It is possible that critical, harsh, or overly demanding parents may punish mistakes more frequently in their children, thereby causing children to react more intensely to making mistakes. In other words, parents may shape their children's reactivity to errors over time through their own verbal and non-verbal responses to their children's mistakes. Repeated exposure to overly harsh and critical parenting may condition children to overreact to their mistakes, thereby increasing risk for anxiety disorders.

Given this link, Meyer et al. (2015a, 2015b) have proposed that parenting style may impact the magnitude of the ERN in children. Harsh parenting is characterized by high control and low warmth, and more frequent and intense punishment of children's mistakes (Robinson et al., 2001), often leading to children's excessive concern around making mistakes (Kawamura et al., 2002). And indeed, harsh parenting styles in early childhood have been linked to larger ERNs in offspring (Banica et al., 2019; Brooker and Buss, 2014; Meyer et al., 2019, 2018a, 2018b, 2018c, 2018d). A prospective study by Meyer, Proudfit and colleagues (2015) found that punitive parenting at age 3 predicted child ERN magnitude at age 6. Additionally, Meyer et al. (2019) found that the *presence* of a controlling parent (compared to the presence of an experimenter) increased the ERN in young children – further supporting the importance of parental context in shaping the magnitude of the ERN. Given evidence indicating an association between hostile child rearing practices and anxiety disorders in children (Barrett et al., 2002; Brown and Whiteside, 2008; Bögels and Brechman-Toussaint, 2006; Rapee, 1997), these findings collectively suggest that one mechanism through which parenting may impact anxiety in children is via potentiation of the ERN. In support of this, Meyer et al. (2019, 2015a) found that the relationship between parenting style and child anxiety disorder status was mediated by the ERN.

While several studies have demonstrated a relationship between parenting and the ERN in offspring in early childhood, no study has explored this relationship across development in older children and adolescents. Research on this topic is lacking, and it is important to identify periods of development wherein biomarkers of risk may be particularly sensitive to environmental influence. Indeed, few studies have examined sensitive periods in the development of threat-sensitivity, such as the ERN, in humans. Previous non-human research on critical periods in brain plasticity has mainly focused on the development of perceptual systems (Hensch, 2004; Knudsen, 2004); however, it is likely that there are similar developmental periods of plasticity in affective function, particularly regarding threat sensitivity (Hartley and Lee, 2015). During periods of sensitivity, neural circuits are especially susceptible to environmental input. In an fMRI study looking at frontoamygdala activity during an emotional go/no-go task, children showed fewer false alarms in the presence of their mother as compared to a stranger (Gee et al., 2014). However, this effect of maternal buffering was not present in adolescents; they showed no difference in performance regardless of maternal or stranger presence. In addition, another study showed that children, but not adolescents, benefited from parental support during a laboratory-induced stress task (Hostinar et al., 2015b). Tottenham (2015) has also proposed that parental scaffolding *prior to adolescence* (i.e., when frontoamygdala circuitry is highly plastic) may be most influential in modulating amygdala circuitry and its long-term functioning. Collectively, these studies indicate that parenting may have a larger effect on children, but not adolescents. In the current study, we aimed to examine the association between parenting and the ERN (a neural marker of risk for anxiety disorders) across development. Given that previous work suggests parental input may be most formative early in life (Armsden and Greenberg, 1987; Gee, 2016; Hostinar et al., 2015a; Laible et al., 2000; Tottenham, 2015), we hypothesized that parenting may impact error sensitivity (i.e., the ERN), and thus risk for anxiety disorders, more substantially earlier in development.

<sup>1</sup> It should be noted that the relationship between the ERN and anxiety symptoms may change across development (Ip et al., 2019; Lo et al., 2017; Meyer, 2017; Meyer et al., 2012, 2018). However, because the current study focuses on older children and adolescents, we do not fully review this literature in the current manuscript

In the current study, we wished to replicate previous findings that have linked harsh parenting to the ERN (Brooker and Buss, 2014; Meyer et al., 2015a, 2015b); moreover, we wanted to determine whether the ERN mediates the relationship between harsh parenting and anxiety disorder status (and in particular, anxious apprehension disorders including GAD, OCD, and social phobia) across development. In a large sample of adolescent females between the ages of 10 and 17 years old, we examine the relationship between parenting and the ERN cross-sectionally, and whether this relationship varies by age. We focus on females due to work suggesting that they are more likely than males to experience anxiety disorders (Pine et al., 1998; Wittchen et al., 1998). Furthermore, we examine whether the ERN mediates the relationship between parenting and anxiety disorder status. Given evidence of specificity in the anxiety-ERN relationship, we also examined differential associations with disorders characterized by anxious apprehension (i.e., GAD, OCD, and social phobia) versus anxious arousal (i.e., PTSD, phobias). Based on previous work, we hypothesized that the ERN would be linked to parenting in younger children, but not in older children. Therefore, we expected that the mediation model would be moderated by age.

## 2. Method

### 2.1. Participants

The study included 265 parent-child dyads recruited from the community in Long Island, New York. Children were female adolescents between the ages of 10 and 17 years old ( $M = 14.4$ ,  $SD = 1.8$ ). Parent-reported child race was as follows: 0.8 % American Indian/Alaskan Native, 7.2 % Black/African American, 87.2 % White/Caucasian, and 4.9 % Other.

Further, 88.6 % of parents were biological mothers while 11.4 % were biological fathers. Parent age ranged from 31.0–57.7 years ( $M = 47.9$ ,  $SD = 4.7$ ). Regarding parent education, 0.4 % of parents reported completing some high school; 5.7 % reported high school degree or GED; 29.1 % reported some college or a 2-year degree; 32.1 % completed a 4-year degree, 28.3 % obtained a master's degree, and 4.5 % obtained a doctoral degree. For estimated annual household income, 4.1 % reported making less than \$25,000 per year; 8.3 % between \$25,000–50,000; 11.2 % between \$50,000–75,000; 19.4 % between \$75,000–100,000; 24.4 % between \$100,000–150,000; and 32.6 % reported making more than \$150,000 per year.

Out of the full sample, 197 adolescents had EEG data. EEG data from 23 adolescents were excluded from analyses for the following reasons: did not make enough errors (i.e., less than 7 total errors,  $n = 2$ ; Meyer, Riesel, et al., 2013), accuracy was not substantially better than chance (i.e., less than 65 % accuracy,  $n = 8$ ), or data included too many artifacts ( $n = 5$ ). Of the 174 adolescents with valid EEG data, self-report questionnaires and diagnostic interviewing were available for 170 (4 participants' data were lost due to experimenter error). Thus, 64.15 % ( $N = 170$ ) of the original sample was retained. The excluded adolescents did not differ from the rest of the sample on demographics or any of the key study variables, all  $ps > .05$ . The mean age of participants with usable EEG and self-report data ( $N = 170$ ) was 14.5 years,  $SD = 1.8$ . All parents and child participants consented/assented to participation in the study. The current study was part of a larger NIMH-funded longitudinal study (R01 MH097767) focusing on neural risk markers of depression. Data from this sample have been published in previous works showing that increases in the ERN across development are partially mediated by social anxiety symptoms (Meyer, Carlton, et al., 2018) and that developmental increases in the ERN are linked to puberty (Gorday and Meyer, 2018).

### 2.2. Measures

#### 2.2.1. Self-report

Caregivers completed the Parenting Styles and Dimensions Questionnaire (PSDQ). The PSDQ is a 32-item self-report measure of parenting styles. Response options range from 1 (*never*) to 5 (*always*), and assess three global parenting styles outlined by Baumrind (1971): authoritative, authoritarian, and permissive. Authoritative parenting (high control and warmth) is directive, rational, and issue-oriented. For instance, authoritative parents endorse items such as "I encourage my child to talk about his/her problems," "I emphasize the reasons for rules," and "I allow my child to give input into family rules." Authoritarian parents (high control, low warmth; i.e., harsh parenting) may demand that their children adhere to their rules without question. Examples of items include "I slap my child when he/she misbehaves," "I explode in anger towards my child," and "I use threats as punishments with little or no justification." Permissive parents (low control, high warmth) are non-punitive, accepting, and adopt a *laissez-faire* approach to parenting, endorsing items such as "I spoil my child" and "I give in to my child when he/she causes a commotion about something." Higher scores on a subscale indicate greater endorsement of that parenting style. This measure has been shown to have good internal consistency in both children and adolescents (Olivari et al., 2013).

#### 2.2.2. Task and materials

As part of a larger study, children completed an arrow version of the Flanker task (Eriksen and Eriksen, 1974) while EEG data was recorded. The task was administered using Presentation software (Neurobehavioral Systems, Inc.) to control the presentation and timing of all stimuli. Each trial consisted of five horizontally aligned arrowheads in the middle of the screen presented for 200 ms, with an ITI of 2300–2800 ms that varied randomly. Half of the trials were compatible ("<<<<<<" or ">>>>>>"), and half were incompatible ("<<<<<<" or ">>>>>>"). The order of compatible and incompatible trials was randomly determined. Participants were instructed to respond as quickly and as accurately as possible by clicking the right mouse button if the center arrow was pointing to the right, and the left mouse button if the center arrow was pointing to the left. After receiving instructions and completing 30 practice trials to ensure adequate performance, the participants completed the full task consisting of 11 blocks of 30 trials (330 trials total), wherein the participant initiated each block. Performance feedback was given at the end of each block. Performances of 75 % accuracy or less triggered the message "Please try to be more accurate"; performances of more than 90 % accuracy prompted the message "Please try to respond faster"; otherwise, the message "You're doing a great job" was shown.

#### 2.2.3. Diagnostic interview

Adolescent psychiatric history was assessed with the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children–Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997) by trained interviewers. The K-SADS-PL assesses current and past diagnoses of anxiety disorders as reported by parents and children and demonstrates excellent test-retest reliability (.77–1.00) and interrater agreement (93%–100%; Kaufman et al., 1997).

Amongst adolescents with usable EEG data, 6 had panic disorder; 3 separation anxiety disorder; 25 simple phobia; 11 social phobia; 1 agoraphobia; 20 GAD; 6 OCD; 2 PTSD; and 1 had anxiety disorder not otherwise specified (NOS). For the purposes of analyses, 2 anxiety groups were created: The first group included any participant with a current threshold of any anxiety disorder ( $n = 55$ ). The second group was composed of a subset of the first group – this group only included participants with a current threshold diagnosis of GAD, OCD, or social phobia (i.e., disorders shown to be related to an increased ERN;  $n = 34$ ). In other words, the second group excluded participants who had panic disorder, separation anxiety disorder, simple phobia, agoraphobia,

PTSD, and anxiety disorder NOS.

### 2.3. Procedure

Upon a participant's arrival into the laboratory, a research assistant provided a brief description of the experiment and obtained informed consent (signed consent from the parents and written/verbal assent from children). The duration of the full lab visit lasted approximately 4–5 hours and included tasks such as diagnostic interviewing, self-report measures, and psychophysiological tasks. To measure the ERN, children/adolescents completed a Flanker task as continuous EEG data were collected.

### 2.4. EEG data acquisition and processing

Continuous EEG data at thirty-four electrode sites, placed according to the 10/20 system, and two electrodes on the left and right mastoids were recorded with an elastic cap and the BioSemi ActiveTwo system (BioSemi, Amsterdam, Netherlands). Electrooculogram (EOG) data produced by eye movements and eye blinks were collected using four facial electrodes: two approximately 1 cm outside the outer edge of the right and left eyes (horizontal eye movements), and two approximately 1 cm above and below the right eye (vertical eye movements and blinks). All electrodes were sintered Ag/AgCl electrodes. 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. During data acquisition, all active electrodes were referenced to a common mode sense (CMS) active electrode producing a monopolar (non-differential) channel. EEG was recorded with a low-pass fifth order sinc filter with a half-power cutoff of 204 Hz and digitized at a 24-bit resolution with a sampling rate of 1024 Hz.

For offline analysis, we used Brain Vision Analyzer Version 2.1 (Brain Products, Gilching, Germany). EEG data were referenced to the mean of the left and right mastoids, and band-pass filtered between 0.1 and 40.0 Hz, with a 24 dB/oct roll-off, and corrected for eye blinks and eye movements as per Gratton et al. (1983). Through an automatic procedure, specific intervals were eliminated from individual channels in each trial by detecting and rejecting artifacts using the following criteria: a voltage step of more than 50.0  $\mu\text{V}$  between sample points, a voltage difference of 175.0  $\mu\text{V}$  within a trial, and a maximum voltage difference of less than 0.50  $\mu\text{V}$  within 100-ms intervals.

The EEG was segmented –500 to 800 ms prior to and following response onset for each trial. Response-locked ERPs were averaged separately for correct and error trials, and baseline corrected using the interval from –500 to –300 ms. Peak detection was employed to identify the maximal negative peak from -10 to 100 ms around response onset for error and correct trials separately. The error-related negativity (ERN) and correct-related negativity (CRN) were scored separately as the average voltage in the 100-ms window centered around the negative peak for each individual at mid-line electrode FCz, where error-related brain activity was maximal.<sup>2</sup> We also created residualized difference scores for the ERN by using saved residuals from regressions (i.e., entering the CRN predicting ERN and saving the unstandardized residuals as a measure of the electrocortical activity specific to error trials; see Meyer et al., 2017). Behavioral data were recorded as number of error and correct trials for each individual, as well as mean reaction time

<sup>2</sup> A repeated measures ANOVA comparing difference scores (i.e., error minus correct) at sites FCz, Cz, and Fz suggested that FCz was the site of maximal difference,  $F(2, 346) = 14.96$ ,  $p < .0001$ . ERN scores were significantly more negative at FCz ( $M = -4.76$ ,  $SD = 6.04$ ) as compared to Fz ( $M = -4.10$ ,  $SD = 5.48$ ) and Cz ( $M = -3.43$ ,  $SD = 5.37$ ). It should also be noted that the pattern of results was similar at sites Fz and Cz, such that the relationship between authoritarian parenting and child anxiety diagnostic status was mediated by a larger ERN, particularly for younger children/adolescents.

(RT) across trials.

### 2.5. Statistical analysis

Statistical analyses were conducted using SPSS (Version 23.0) general linear model software. Greenhouse-Geisser correction was applied to  $p$  values with multiple degrees of freedom and repeated-measures comparisons when the assumption of sphericity was violated. Associations between all study variables were examined using Pearson's  $r$  and one-way analyses of variance (ANOVA). A repeated-measures ANOVA was also conducted to examine error-related brain activity by condition (error vs. correct).

A nonparametric bootstrapping approach (MacKinnon et al., 2004) was used to examine whether child age (as a continuous variable) moderated the relationship between parenting style and the ERN (both raw and residualized scores). To assess the specificity of this relationship, we conducted the same regression analyses, controlling for accuracy and RTs during the task. Next, logistic regression analyses were used to examine the relationship between the ERN (raw and residualized scores) and anxiety diagnoses. Then, a bootstrapping test was employed to explore a moderated mediation model, wherein the mediation of the ERN (raw and residualized scores) on the relationship between authoritarian parenting style and anxiety disorders was dependent upon child age. MacKinnon et al. (2002) showed that this approach is more statistically powerful than other tests of mediation (e.g., the Sobel test) in that it does not assume normality of the indirect effect sampling distribution. All moderated mediation analyses were conducted using the SPSS PROCESS macro (Preacher and Hayes, 2004) model 7, which involves a resampling estimate of the indirect effect between independent and dependent variables, an estimated standard error, and 95 % confidence intervals for the population value of the indirect effect. The bootstrapping strategy in this study used 10,000 sample replicates to test direct and indirect effects. An indirect effect was determined as significant at the  $p < .05$  level if its confidence intervals did not overlap with zero. PROCESS estimates regions of significance using the Johnson-Neyman technique and estimates two-way interactions in moderation models using ordinary least squares methods.

## 3. Results

A repeated-measures ANOVA was conducted with error versus correct entered as within-subject variables to compare the magnitude of these two responses. Consistent with previous work, the ERP response was significantly more negative on error trials ( $M = 0.33$ ,  $SD = 7.93$ ) as compared to correct trials ( $M = 5.00$ ,  $SD = 5.65$ ),  $F(1, 173) = 107.69$ ,  $p < .001$ ,  $\eta_p^2 = 0.38$ . Descriptive statistics and correlation analyses amongst the ERN, the CRN, child age, PSDQ subscales are provided in Table 1. Overall, PSDQ scores for authoritarian parenting ranged from 12 to 34, authoritative parenting scores ranged from 32 to 74, and permissive parenting scores ranged from 5 to 22. As shown in Table 1, the only parenting scale that related to the ERN was authoritarian parenting, such that higher authoritarian parenting was associated with a larger (i.e., more negative) ERN. Neither the CRN nor the residualized ERN was correlated to any parenting style,  $ps > .05$  (Table 1).

Regarding sociodemographic information, we found that household income was significantly related to the ERN,  $r = -.17$ ,  $p = .03$  and child age,  $r = .13$ ,  $p = .05$ . In other words, a larger (i.e., more negative) ERN was associated with higher household income. However, household income was not related to any of the parenting subscales, all  $ps > .05$ .

### 3.1. Moderation of child age on the relationship between parenting style and child ERN

Consistent with Meyer et al. (2015a, 2015b), harsh parenting (i.e., higher self-reported authoritarian parenting on the PSDQ) was related to a more negative ERN,  $r = -.17$ ,  $p = .03$ . We used a nonparametric

**Table 1**  
Descriptives and Bivariate Correlations Among Key Study Variables.

|                       | 1     | 2    | 3    | 4    | 5      | 6     | 7 | M     | SD   |
|-----------------------|-------|------|------|------|--------|-------|---|-------|------|
| 1. ERN                | 1     |      |      |      |        |       |   | 0.33  | 7.93 |
| 2. CRN                | .67** | 1    |      |      |        |       |   | 5.00  | 5.65 |
| 3. Residualized ERN   | .75** | .00  | 1    |      |        |       |   | ∅     | 5.92 |
| 4. Child Age          | .05   | .18* | -.09 | 1    |        |       |   | 14.51 | 1.75 |
| 5. PSDQ Authoritarian | -.17* | -.12 | -.12 | -.11 | 1      |       |   | 18.16 | 4.35 |
| 6. PSDQ Authoritative | .10   | .04  | .10  | -.08 | -.28** | 1     |   | 60.23 | 8.47 |
| 7. PSDQ Permissive    | -.09  | -.11 | -.03 | .00  | .50**  | -.11† | 1 | 10.07 | 3.60 |

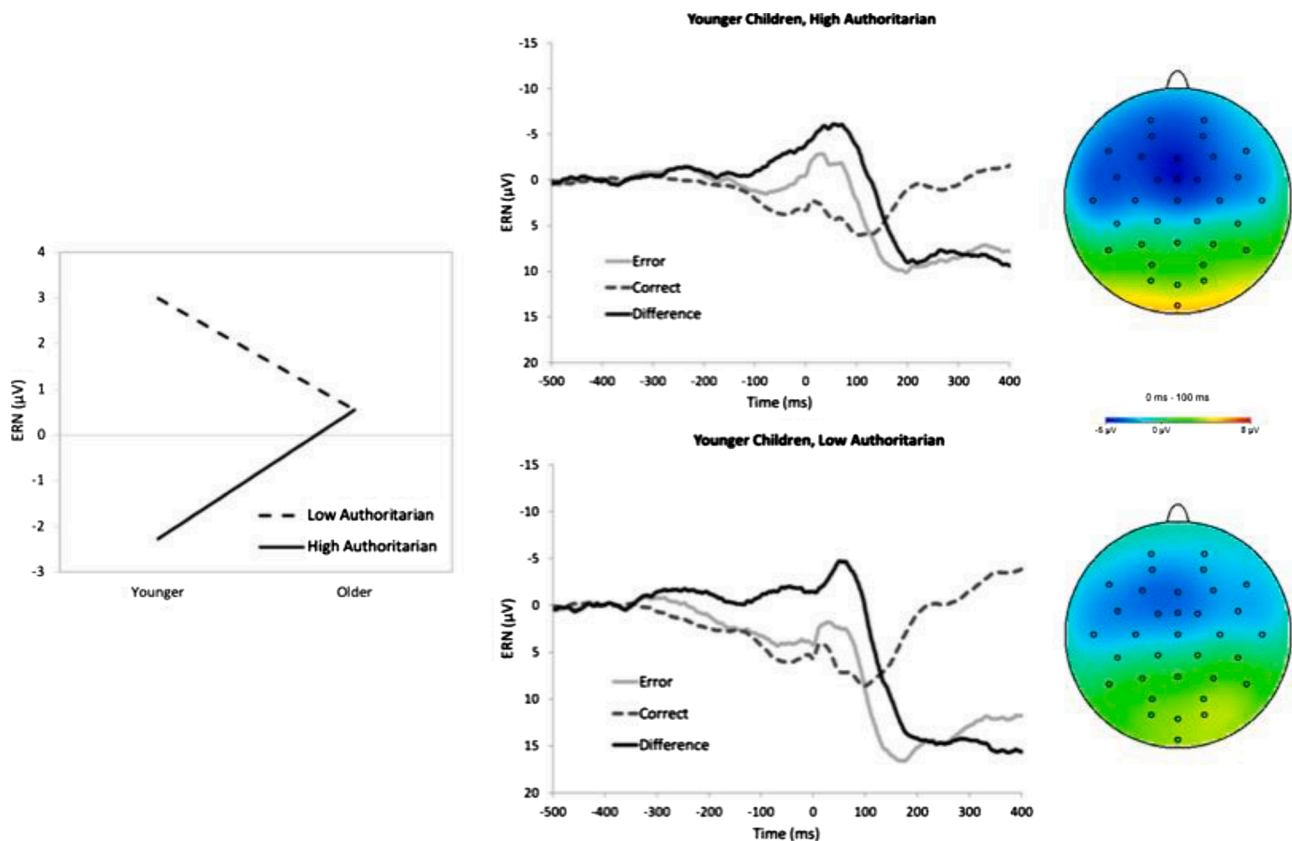
Note. † $p < .09$ ; \* $p < .05$ ; \*\* $p < .001$ ; ERN (in  $\mu V$ ) = error-related negativity; CRN (in  $\mu V$ ) = correct-related negativity; PSDQ = Parenting Styles and Dimensions Questionnaire.

bootstrapping method (MacKinnon et al., 2004) to examine whether child age interacted with parenting style to impact the magnitude of the ERN. Results showed that the main effect of child age was not significant,  $coeff = .02, SE = .35, t = .06, p = .95$ , 95 % confidence interval (CI) [-0.67, .71]. The main effect of authoritarian parenting style was significant,  $coeff = -0.31, SE = .14, t = -2.17, p = .03$ , 95 % CI [-0.59, -.03]. The interaction between child age and authoritarian parenting style explained a significant amount of variance in child ERN magnitude,  $\Delta R^2 = .03, F(1, 168) = 4.49, p = .04$ . Younger children (1 SD below mean age) with parents characterized by authoritarian parenting styles displayed a larger (i.e., more negative) ERN,  $effect = -0.61, SE = .20, t = -3.02, p < .01$ , 95 % CI [-1.01, -0.21] (Fig. 1); this effect was not observed in older children (1 SD above mean age),  $effect = -0.01, SE = .20, t = -0.06, p = .95$ , 95 % CI [-0.41, .38]. By probing regions of significance in the interaction, we found that authoritarian parenting style related to ERN magnitude, only amongst younger children (below 14.62 years,  $p < .05$ ), and not amongst older children (aged 15.00 and higher, all  $ps > .10$ ). For illustrative purposes, waveforms and topographical

headmaps for younger children grouped by high/low authoritarian parenting styles (based on median splits) are depicted in Fig. 1.

We repeated the above analyses with the residualized ERN scores and found that the main effect of authoritarian parenting style predicting residualized ERN scores was marginally significant,  $coeff = -0.19, SE = .11, t = -1.73, p = .09$ , 95 % CI [-0.40, .03]. The main effect of child age on residualized ERN score was not significant,  $coeff = -0.36, SE = .26, t = -1.38, p = .17$ , 95 % CI [-0.88, .16]. Furthermore, the interaction between child age and authoritarian parenting was not significant,  $\Delta R^2 = 0.00, F(1, 168) = .83, p = .36$ .

Given that task performance may be associated with child age, parenting style, or the ERN (or their relationship), we entered accuracy and mean reaction time as additional covariates into the previous model examining the interaction between child age and authoritarian parenting to predict the ERN. Results showed that mean reaction time and accuracy did not predict the ERN,  $coeff = -0.01, SE = .01, t = -0.69, p = .49$ , 95 % CI [-0.03, .01] and  $coeff = -.08, SE = 0.10, t = .81, p = .42$ , 95 % CI [-0.12, .29], respectively. The main effect of child age was not



**Fig. 1.** On the left: simple slopes observed at  $\pm 1$  SD from the mean for child age and PSDQ authoritarian parenting. On the right: response-locked ERP waveforms (i.e., correct, error, and error-minus-correct) for younger children with high (top) and low (bottom) authoritarian parenting, based on a median split. Topographical headmaps depicting differences (in  $\mu V$ ) between error and correct responses are included for both groups, 0–100 milliseconds after the response.

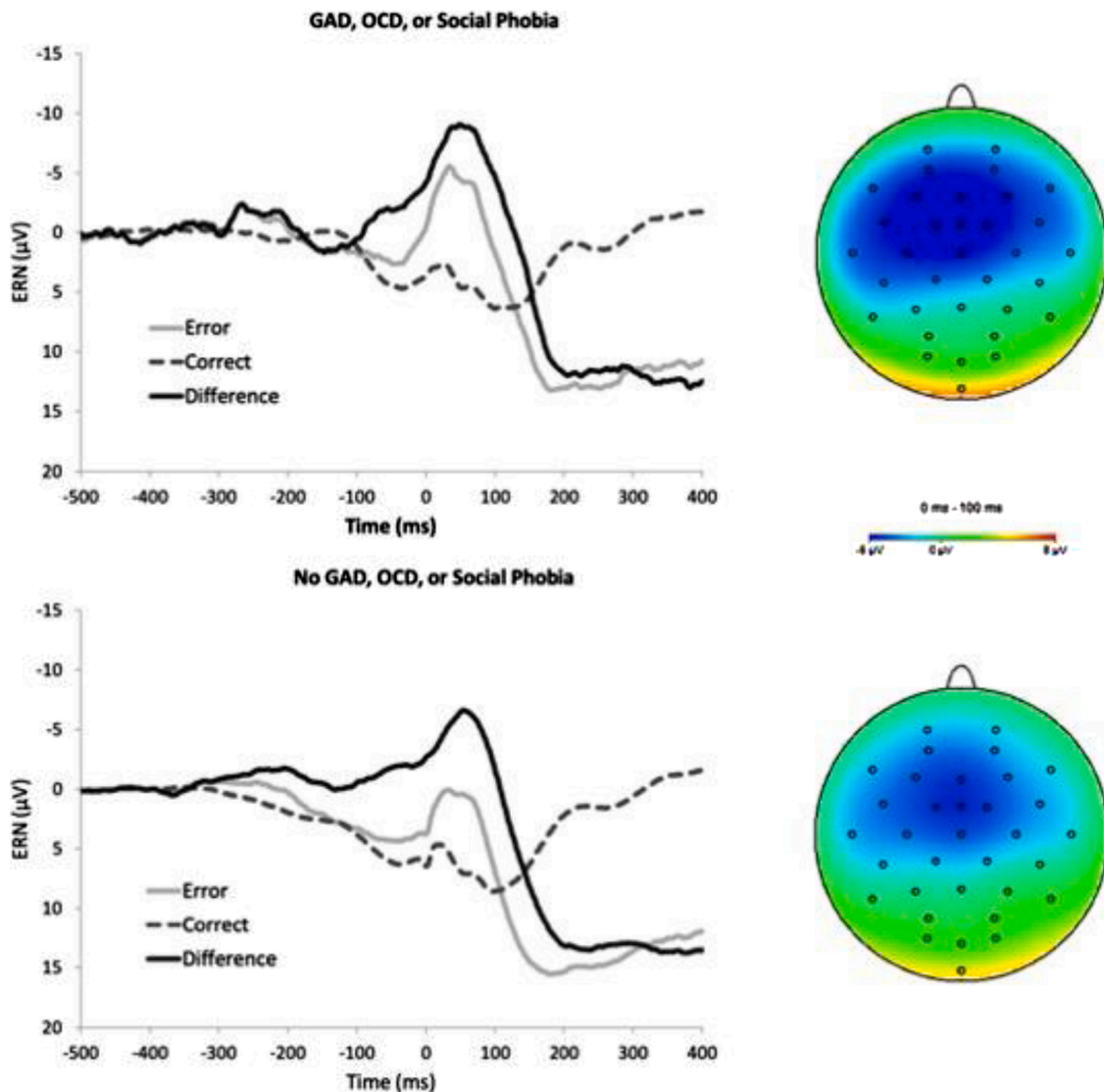


Fig. 2. Response-locked ERP waveforms (i.e., correct, error, and error-minus-correct) for children with (top) and without (bottom) GAD, OCD, or Social Phobia. Topographical headmaps depicting differences (in  $\mu\text{V}$ ) between error and correct responses are included for both groups, 0–100 milliseconds after the response (right).

significant,  $coeff = -0.20$ ,  $SE = .40$ ,  $t = -0.50$ ,  $p = .62$ , 95 % CI [-0.99, .59]. Authoritarian parenting significantly predicted the ERN,  $coeff = -0.31$ ,  $SE = .15$ ,  $t = -2.05$ ,  $p = .04$ , 95 % CI [-0.60, -0.01]. The interaction between child age and authoritarian parenting remained significant even after taking into account the impact of task performance,  $\Delta R^2 = .02$ ,  $F(1, 164) = 4.25$ ,  $p = .04$ .

Controlling for task performance (i.e., mean reaction time and accuracy), we ran the same moderation model investigating the interaction between child age and authoritarian parenting predicting residualized ERN scores. Mean reaction time and accuracy did not significantly predict residualized ERN scores,  $coeff = .01$ ,  $SE = .01$ ,  $t = 1.61$ ,  $p = .11$ , 95 % CI [-0.00, 0.03], and  $coeff = -0.03$ ,  $SE = .08$ ,  $t = -0.33$ ,  $p = .74$ , 95 % CI [-0.18, .13], respectively. The child age X authoritarian parenting interaction was not significant, after taking into account task performance,  $\Delta R^2 = .01$ ,  $F(1, 164) = .89$ ,  $p = .35$ .

Other parenting styles did not interact with child age to predict ERN magnitude. The interaction between child age and authoritative parenting style did not predict ERN magnitude,  $\Delta R^2 = 0.00$ ,  $F(1, 164) = .07$ ,  $p = .79$ , after taking into account task performance. The interaction between child age and permissive parenting style predicted ERN magnitude at a trend level after taking into account task performance,

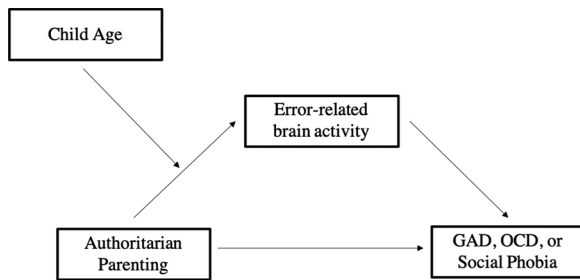
$\Delta R^2 = .02$ ,  $F(1, 164) = 2.74$ ,  $p = .10$ . The pattern of results was similar with residualized ERN scores – i.e., the interaction between child age and authoritative or permissive parenting style did not predict residualized ERN scores,  $\Delta R^2 = 0.00$ ,  $F(1, 164) = 0.00$ ,  $p = .97$ , and  $\Delta R^2 = .01$ ,  $F(1, 164) = 2.32$ ,  $p = .13$ , respectively.

### 3.2. Moderated mediation model predicting anxiety disorders

In Model 1, all anxiety diagnoses were combined (i.e., panic disorder, separation anxiety disorder, simple phobia, social phobia, agoraphobia, adjustment disorder, GAD, OCD, PTSD, and anxiety NOS). The ERN was entered into the equation predicting the presence of any anxiety disorder. Results showed that the ERN predicted diagnosis of any anxiety disorder at a trend level,  $B = -0.05$ ,  $SE = .02$ ,  $OR = 0.96$  (95 % CI [0.91, 1.00]),  $Wald = 3.58$ ,  $p = .06$ .

Next, we entered residualized ERN scores into the logistic regression equation to predict the presence of any anxiety disorder. We found that the residualized ERN score did not significantly predict the diagnosis of any anxiety disorder,  $B = -0.04$ ,  $SE = .03$ ,  $OR = 0.96$ , 95 % CI [0.91, 1.02],  $Wald = 1.43$ ,  $p = .23$ .

In Model 2, only participants with anxiety disorders characterized by



**Fig. 3.** Graphical depiction of the moderated mediation model, wherein the relationship between authoritarian parenting style (i.e., PSDQ Authoritarian subscale) and a diagnosis of GAD, OCD, or Social Phobia (i.e., Anxious Apprehension Disorder, via the K-SADS-PL) is mediated by error-related brain activity (i.e., the ERN).

*anxious apprehension* (i.e., GAD, social phobia, and/or OCD) were coded as a case. This grouping was made due to previous research linking the ERN specifically to disorders characterized by anxious apprehension, in contrast to disorders characterized by anxious arousal (Lahat et al., 2014; Xiao et al., 2011). The ERN significantly predicted diagnostic status of GAD, social phobia, or OCD,  $B = -0.09$ ,  $SE = .03$ ,  $OR = 0.92$  (95 % CI [0.86, 0.97]),  $Wald = 8.66$ ,  $p = .003$ . In other words, children with larger ERNs were more likely to have GAD, OCD, or social phobia (see Fig. 2).

We also ran the same analyses with residualized ERN scores as the independent variable predicting the diagnostic status of anxious apprehension disorders. Residualized ERN scores significantly predicted the diagnostic status of disorders characterized by anxious apprehension,  $B = -0.09$ ,  $SE = 0.04$ ,  $OR = 0.92$  (95 % CI [0.85, 0.99]),  $Wald = 5.39$ ,  $p = .02$ , indicating that children with heightened ERNs were more likely to have GAD, OCD, or social phobia.

We then examined a moderated mediation model (for Model 2) wherein the pathway between authoritarian parenting and child anxiety disorder diagnostic status of GAD, OCD, or social phobia was mediated by the interaction between child age and the ERN (see Fig. 3). The interaction between authoritarian parenting and child age significantly predicted ERN,  $coeff = .18$ ,  $SE = .08$ ,  $t = 2.19$ ,  $p = .03$ , 95 % CI [.02, .33]. Further, as depicted in Table 2, the ERN significantly predicted a diagnosis of GAD, OCD, or social phobia,  $coeff = -0.08$ ,  $SE = .03$ ,  $z = -2.76$ ,  $p = .01$ , 95 % CI [-0.14, -0.02]. The direct path from authoritarian parenting to GAD/OCD/social phobia was not significant,  $coeff = .05$ ,  $SE = .05$ ,  $z = .93$ ,  $p = .35$ , 95 % CI [-0.05, .14]. However, results supported the moderated mediation model,  $index\ of\ moderated\ mediation = -0.01$ ,  $SE = .01$ , 95 % CI [-0.04, -0.00]. The indirect path from Authoritarian parenting to GAD/OCD/social phobia diagnosis via the ERN was

**Table 2**  
Moderated Mediation Model Predicting Anxiety Disorders Characterized by Anxious Apprehension.

|  | <i>coeff</i> | <i>SE</i> | <i>z</i> | <i>p</i> | LLCI  | ULCI   |
|--|--------------|-----------|----------|----------|-------|--------|
| Direct effect on GAD, OCD, or Social Phobia  |              |           |          |          |       |        |
| ERN  | -.08         | .03       | -2.76    | .01      | -.14* | -.02*  |
| Authoritarian parenting  | .05          | .05       | .93      | .35      | -.05  | .14    |
| Conditional indirect effects of authoritarian parenting on GAD, OCD, or Social Phobia by child age |              |           |          |          |       |        |
| Younger children   | .05          | .03       | -        | -        | .01*  | .12*   |
| Older children   | -.002        | .02       | -        | -        | -.04  | .03    |
| Full model: Index of moderated mediation   | -.01         | .01       | -        | -        | -.04* | -.001* |

Note. \* $p < .05$ ; \*\* $p < .001$ ; Younger children: 1 SD below mean; Older children: 1 SD above mean; LLCI = lower level confidence interval, ULCI = upper level confidence interval; GAD, OCD, and Social Phobia = disorders of Anxious Apprehension.

significant, specifically for younger children (i.e., 1 SD below mean age),  $effect = .05$ ,  $SE = .03$ , 95 % CI [.01, .12]. However, the mediation model was not supported for older children (i.e., 1 SD above the mean age),  $effect = -0.00$ ,  $SE = .02$ , 95 % CI [-0.04, .03].<sup>3, 4</sup>

Finally, we also examined the same moderated mediation model using residualized ERN scores as the mediator. The child age X authoritarian parenting interaction did not significantly predict residualized ERN scores,  $coeff = .06$ ,  $SE = .06$ ,  $t = 1.00$ ,  $p = .32$ , 95 % CI [-0.06, .18]. Residualized ERN scores significantly predicted anxious apprehension diagnostic status,  $coeff = -0.09$ ,  $SE = .04$ ,  $z = -2.28$ ,  $p = .02$ , 95 % CI [-0.16, -0.01]. The direct path from authoritarian parenting to GAD/OCD/social phobia was not significant,  $coeff = .06$ ,  $SE = .05$ ,  $z = 1.18$ ,  $p = .24$ , 95 % CI [-0.04, .15]. Results did not support the moderated mediation model,  $index\ of\ moderated\ mediation = -0.01$ ,  $SE = .01$ , 95 % CI [-0.02, .00].

#### 4. Discussion

As expected and consistent with previous research, authoritarian parenting was associated with an elevated ERN in *younger* children and adolescents. Moreover, the ERN was increased in anxious children — and that effect was particularly pronounced for participants with GAD, OCD, or social phobia. A moderated mediation model was significant — wherein the relationship between authoritarian parenting and these anxious diagnoses was mediated by the ERN — but only for *younger* children and adolescents. Findings from the current study replicate and extend previous work by examining how the relationship between parenting and a neural marker of risk for anxiety (i.e., the ERN) differs across development.

Results from the present study indicated that authoritarian/harsh parenting is linked to the ERN in younger children (i.e., early adolescence), but not older children and adolescents. These findings are in line with evidence that fear learning processes may change across development, and may be characterized by differential regions of brain activation (Hartley and Lee, 2015). The transition from childhood to

<sup>3</sup> We also ran the same moderated mediation model for Model 2, entering household income as a covariate. The pattern of results remained the same. The authoritarian parenting X child age interaction significantly predicted ERN,  $coeff = .22$ ,  $t = 2.68$ ,  $p = .01$ . The ERN significantly predicted GAD, OCD, or social phobia diagnostic status,  $coeff = -0.07$ ,  $z = -2.36$ ,  $p = .02$ . The direct pathway between authoritarian parenting and anxious apprehension diagnostic status (i.e., GAD, OCD, or social phobia) was not significant,  $coeff = .06$ ,  $z = 1.20$ ,  $p = .23$ . Moreover, household income entered as a covariate did not significantly predict anxious apprehension diagnostic status,  $coeff = .00$ ,  $z = -0.03$ ,  $p = .98$ . Overall, the moderated mediation model was significant, even after controlling for household income as a covariate,  $index\ of\ moderated\ mediation = -0.02$ , 95% CI [-0.05, -0.001].

<sup>4</sup> Additionally, we examined individual differences of anxiety using a continuous measure as the outcome variable. Given that the ERN is related to anxious apprehension rather than anxious distress, we focused analyses on the SCARED subscales that measure GAD and social anxiety. For the first analysis, we entered parent-reported SCARED GAD subscale scores as the outcome variable. Specifically, child age interacted with authoritarian parenting style to predict the ERN,  $coeff = .17$ ,  $t = 2.12$ ,  $p = .04$ . The ERN predicted SCARED GAD scores at trend level,  $coeff = -0.06$ ,  $t = -1.81$ ,  $p = .07$ . The direct path from authoritarian parenting to SCARED GAD scores was not significant,  $coeff = .07$ ,  $t = 1.27$ ,  $p = .21$ . And, the overall moderated mediation model was not significant,  $index\ of\ moderated\ mediation = -0.01$ ,  $SE = .01$ , 95% CI [-0.03, .00]. Next, we entered SCARED social anxiety subscale scores as the outcome variable. Authoritarian parenting style and child age interacted to predict the ERN,  $coeff = .17$ ,  $t = 2.12$ ,  $p = .04$ . However, the ERN did not significantly predict SCARED social anxiety scores,  $coeff = -0.02$ ,  $t = -0.72$ ,  $p = .47$ . The direct path from authoritarian parenting to SCARED social anxiety scores was not significant,  $coeff = .03$ ,  $t = .49$ ,  $p = .63$ . The overall moderated mediation model was not significant,  $index\ of\ moderated\ mediation = -0.00$ ,  $SE = .01$ , 95% CI [-0.03, .02].

adolescence has itself been conceptualized as a potential developmentally sensitive period (Suleiman and Dahl, 2019). Younger children may be particularly sensitive to inputs from the environment (e.g., parenting behaviors) than older children and adolescents (Tottenham, 2013). The present study demonstrates that harsh or authoritarian parenting styles may be a particularly relevant learning cue to children, as indexed by an elevated ERN and greater risk of meeting diagnostic criteria for an anxious apprehensive anxiety disorder.

Results from the current study align with available evidence suggesting that the ERN-anxiety link is particularly robust for disorders characterized by anxious apprehension rather than acute fear-based or anxious-arousal anxiety disorders (Moser et al., 2005, 2013; Vaidyanathan et al., 2009; Weinberg et al., 2010). This is unsurprising given that our sample consists of older children who may be in developmental stages characterized by more abstract, self-conscious shyness and worry as compared to fears of concrete, external threat commonly seen in young children (Gullone, 2000). This finding is also consistent with animal and human research suggesting disparities between neural circuitry underlying fear (i.e., stimulus-specific defensive reactivity) and anxiety (i.e., chronic generalized defensive reactivity; Davis et al., 1997; Grillon and Davis, 1997; Rosen and Schulkin, 1998; Vaidyanathan et al., 2009). Hence, the extent to which parenting styles relate to the ERN and thus risk for anxiety may be limited to those disorders most associated with *generalized anxiousness*, and not increased stimulus-driven fearfulness (Weinberg et al., 2012).

In addition, some studies have suggested that parental influence on children tends to decline across development, with this change typically occurring in adolescence (Bauman et al., 2001; Biddle et al., 1980; Davison and Jago, 2009; Kandel, 1996). In our sample, we found that this transition may occur on a neural level between the ages of 14–15. Adolescence is a developmental period frequently associated with increasing autonomy and independence from parents (Bauman et al., 2001). Aside from biological changes, increased time dedicated to school activities may also heighten the salience of peer groups and decrease adolescents' involvement with parents (Bauman et al., 2001; Kandel, 1986). Hence, the impact of parenting styles on offspring anxiety may diminish as the child enters adolescence. Given that peer relationships may become more salient as children grow older (Adams et al., 2011; Calhoun et al., 2014), future work might explore the possibility of peer relationships impacting the ERN in later adolescence.

In the present study, we did not find a significant relationship between the ERN and child age. Work by Davies et al. (2004) has suggested that the ERN may increase in non-linear ways, particularly in adolescent females. It is possible that several other factors, such as pubertal hormones (Gorday and Meyer, 2018), may have impacted the age/ERN correlation in our current sample of adolescent females.

Notably, the present study found a significant interaction between authoritarian parenting and child age predicting the ERN, but not for the residualized ERN. This suggests that enhanced error-related brain activity in younger adolescents with authoritarian parents is not specific to errors and may be related to generic response-monitoring. In other words, enhanced error-related brain activity in younger adolescents with authoritarian parents is attributed to both error and correct trials. While the specific construct underlying the CRN has been subject to debate (Coles et al., 2001; Vidal et al., 2003), prior work has found that the ERN and CRN are highly correlated, and both measures have been found to be related to anxious apprehension disorders (Hajcak et al., 2003).

Recent work by Banica et al. (2019) showed that undergraduates who retrospectively endorsed that their parents were high in over-protective/authoritarian parenting styles during their childhood displayed an elevated ERN. While these results may appear to contradict findings from the present study, we highlight methodological differences that may account for the discrepancy in findings. In our study, parents reported on their own parenting styles at the time of the study; in Banica and colleagues' (2019) work, participants retrospectively

reported on parenting styles experienced across their entire childhood. Future studies should investigate whether the relationship between parenting and the ERN differs based on the informant or the timeframe of reporting (current vs. retrospective). Additionally, future work could explore whether combining reports from multiple informants strengthens the relationship between parenting and the ERN.

The present study had several limitations. First, the sample only included girls; therefore, conclusions cannot be drawn regarding the relationship between parenting styles and anxiety disorder outcomes for male or nonbinary adolescents. Moreover, the current sample was predominantly characterized by a relatively high socioeconomic status. Recent work has suggested that the ERN may be related to socioeconomic status (SES; Brooker, 2018). Consistent with previous work in young children (Brooker, 2018; Conejero et al., 2018), we found that higher household income was associated with a larger ERN in adolescents. SES may serve as a proxy for assessing broad developmental contextual factors, such as geographic neighborhood, stress, access to resources, and exposure to hardships (Bradley and Corwyn, 2002); these differences in contextual factors may have important implications for the development of neural systems (Hanson et al., 2013). Additional work is needed to identify the specific mechanisms through which SES may be related to the ERN. Relatedly, results from this study may not be generalizable to low SES or predominantly non-white samples. Future studies expanding the age range and socioeconomic status of the sample can determine whether the present findings are generalizable. In addition, this study utilized a cross-sectional design; hence, we were unable to assess temporal or causal relationships amongst parenting styles, child age, the ERN, and anxiety diagnoses. Future work should employ a longitudinal design, with multiple assessments, to examine causal mediation from parenting to anxiety via the ERN. We used a self-report measure to assess parenting styles; it is possible that parents may have under- or over-reported certain behaviors. Future work should include other means of determining parenting styles, such as observational measures or multiple informants. Additionally, more specific measures of parenting, such as observational or self-report measures of parental reactivity to children's errors, are needed to draw stronger conclusions about the effects of parenting on error sensitivity. Given potential genetic influences on the ERN and anxiety, future work should incorporate measures of parent ERN and anxiety to examine whether genetic factors may account for the relationship amongst parenting styles, child ERN, and clinical anxiety across development.

Despite these limitations, the findings from this study have important implications. Parenting interventions can decrease anxiety in children (Rapee et al., 2010), but interventions targeting maladaptive parenting styles may be most effective from early childhood to pre-adolescence, to the extent that these interventions coincide with decreases in the ERN. After this sensitive period ends, individual child treatment for anxiety disorders may prove more useful. Additionally, future work should examine whether it is possible to reduce the ERN and clinical anxiety via parenting interventions focused on decreasing harsh, punitive behavior. The present study also provides rationale for examining the relative efficacy of parenting interventions for anxiety and anxiety-related phenomena across different stages of development.

Overall, this study replicated multiple previous studies and proposed a model that ties these findings together: 1) anxiety disorders – especially disorders characterized by anxious apprehension – are distinguished by a larger ERN; 2) harsh parenting is associated with an increased ERN in offspring; and 3) this increased ERN appears to mediate the relationship between parenting and anxious outcomes. Importantly, these data further suggest that harsh parenting styles are associated with anxious disorder status via the ERN (Meyer et al., 2015, 2015b), particularly for *young* children prior to age 15.

#### Declaration of Competing Interest

None.



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