

# On the relationship between the error-related negativity and anxiety in children and adolescents: From a neural marker to a novel target for intervention

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## Abstract

The current review focuses on our work on the relationship between the error-related negativity (i.e., ERN) and anxiety in children and adolescents. The ERN is an event-related potential (ERP) that appears as a negative deflection in the ERP waveform when individuals make errors and has been found to be increased in anxious individuals. We, and others, have extended this work into developmental populations, finding that the ERN can be measured reliably in children and that the ERN is increased among clinically anxious youth. Furthermore, we have found that the ERN predicts risk for increases in anxiety across development, among healthy and clinically anxious children. We have done work to elucidate what psychological phenomena the increased ERN among anxious children may reflect by creating a self-report measure of error sensitivity (i.e., the Child Error Sensitivity Index) that relates to the ERN. Moreover, we review our work on parenting and the ERN, which suggests that harsh or critical parenting styles may potentiate the ERN in offspring. And, building on these findings, we discuss our recent work to develop novel, computerized intervention strategies to reduce the ERN and thereby risk for anxiety.

## KEYWORDS

anxiety, biomarker, children, development, EEG, ERN, ERP, error-related negativity, neural marker, performance monitoring, risk marker

Our errors are surely not such awfully solemn things. In a world where we are so certain to incur them in spite of all our caution, a certain lightness of heart seems healthier than this excessive nervousness on their behalf.

– William James

## 1 | INTRODUCTION

The ability to detect our errors and adjust our behavior is arguably one of the most important neurobehavioral features in our evolutionary history. Fortunately, we are

able to measure the neural response to errors in the laboratory using electroencephalogram (EEG). The current review will focus primarily on the study of this neural signal—the error-related negativity (i.e., ERN, also called the error negativity, i.e., Ne). The ERN is an event-related potential (ERP) that appears as a negative deflection in the ERP waveform at fronto-central electrode sites when individuals make errors during speeded reaction time tasks in the laboratory (see Figure 1). Since its discovery by two independent research groups in Germany and the United States in the early 1990s (Falkenstein et al., 1991; Gehring et al., 1993), it has been the subject of an extensive amount of research (e.g., collectively, these original studies have been cited approximately 5000 times).

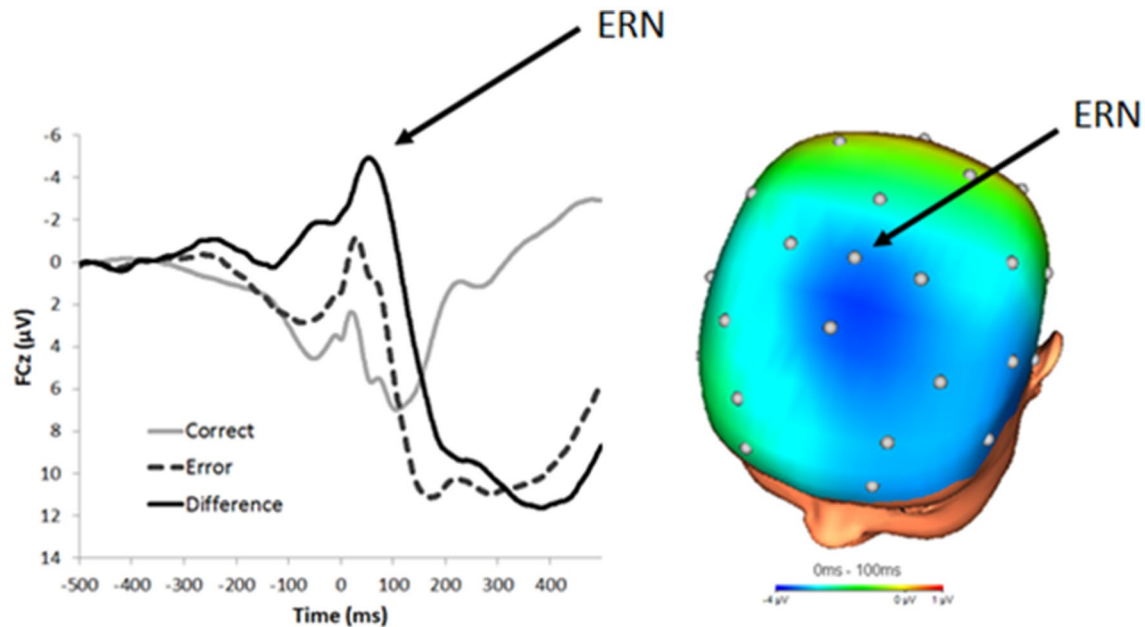


FIGURE 1 Response-locked ERP waveforms at FCz during a flankers task in 150 females between the ages of 8 and 14 years old. On the right, a topographical map depicting the difference between error and correct responses in the time range of the ERN (0–100 ms). Reprinted from Meyer (2017) with permission

Less than a decade after its original discovery, the ERN was found to be increased in individuals with obsessive-compulsive disorder (i.e., OCD, Gehring et al., 2000). In the 20 years that followed that discovery, the link between the ERN and anxiety has been replicated in over 50 studies (Cavanagh & Shackman, 2014; Hajcak, 2012; Meyer, 2016; Moser et al., 2013; Riesel, 2019; Weinberg et al., 2016). The ERN is increased in disorders typically characterized by worry, concern about performance, behavior, or mistakes (i.e., increased error sensitivity); including generalized anxiety disorder (i.e., GAD, Weinberg & Hajcak, 2011; Weinberg, Klein, & Hajcak, 2012; Xiao et al., 2011), OCD (Endrass et al., 2008; Gehring et al., 2000; Riesel, Klawohn, et al., 2019; Weinberg et al., 2015), and social anxiety disorder (Barker et al., 2015, 2018; Endrass et al., 2014; Kujawa et al., 2016). Thus, the ERN has been proposed as a biomarker that indexes individual differences in anxiety (Meyer, 2016).

The ERN is at least partially generated in the anterior cingulate cortex (ACC), a region of the brain where information about pain, threat, and punishment is assimilated to modify behavior (Shackman et al., 2011). We (e.g., my graduate student laboratory and my current students and I) have conceptualized errors as an internally generated type of threat that requires an orienting response and subsequent changes in behavior. Indeed, errors do initiate a range of physiological responses consistent with defensive responding (e.g., pupil dilation, skin conductance response, potentiated startle reflex, corrugator muscle contraction; Weinberg, Riesel, et al., 2012). We

view variability in the ERN to reflect, in part, the degree to which an individual experiences errors as aversive and salient.

## 2 | DEVELOPMENT AND THE ERN

Much of my own work has focused on extending the work on the ERN/anxiety relationship from adults, to child and adolescent populations. While this may seem like a straightforward task, there are a few issues that complicate this endeavor. For one, the ERN appears to change in magnitude across development. Davies and colleagues first discovered this in 2004 (Davies et al., 2004), and this finding has now been replicated over 20 times (for a review, see: Tamnes et al., 2013). In children, the source of the ERN has been localized to the dorsal ACC (Ladouceur et al., 2007; Santesso & Segalowitz, 2008) and DTI studies have found that the cingulum bundle (a white matter tract that underlies the cingulate cortex) matures later than most of the other major tracts (Lebel & Beaulieu, 2011; Lebel et al., 2012). Additionally, an fMRI study including participants between the ages of 8 and 27 years old, found that error-related dorsal ACC activity increases with age (Velanova et al., 2008). Collectively, these studies suggest that error-related neural activity undergoes normative increases across development.

To complicate matters further, not only does the magnitude of the ERN change across development, but many different tasks are used to measure the ERN in child and

adolescent populations. This is, in part, due to the fact that tasks need to be easier, and perhaps more engaging, for younger children. In addition, we happen to use different tasks across research laboratories. Considering the apparent development changes in the ERN, along with the widespread use of various tasks, any investigation of the ERN/anxiety relationship in children must begin by examining the psychometric properties of this neural signal.

We have written broadly about the importance of examining the psychometric properties of ERPs (Hajcak et al., 2017, 2019; Klawohn, Meyer, et al., 2020; Meyer, Bress, et al., 2014). In short, ERPs, or any biomarker, are only valid measures of individual differences if they are psychometrically reliable. This is significant in developmental work insofar as psychometric properties could better account for what appear to be developmental effects. For example, what appear to be developmental increases in the ERN or developmental changes in the ERN/anxiety relationship, may be due to changing internal reliability of the measurement of the ERN (e.g., if, for example, the internal reliability of the ERN was low in younger children, the magnitude of the ERN may appear to be smaller). Research in adults suggests that the ERN is characterized by good psychometric properties (Larson et al., 2010; Meyer, Riesel, et al., 2013; Olvet & Hajcak, 2009).

We, and others, have begun extending this work to children. In one study we examined the reliability and stability of the ERN in children and adolescents initially aged 8–13 years old, over the course of 2 years (Meyer, Bress, et al., 2014). The ERN was characterized by good test–retest reliability ( $r = .63$  for the ERN measured at Cz across 2 years) and good internal consistency (alphas exceeded  $.80$  at both assessments). And further, the ERN elicited by two different tasks (flankers and go/no go) were significantly correlated ( $r = .70$  for the ERN measured at Cz), indicating convergent validity of the ERN. Taken together, these data suggest that the ERN is a reliable and stable measure of error processing in children and adolescents. Future work is needed to examine the psychometric properties of different tasks at different ages and in clinical versus healthy pediatric populations to optimize the ERN as a risk and/or prognostic marker. The internal reliability of a measure is an upper limit in terms of detecting potential relationships with other individual differences (Cronbach & Meehl, 1955). For example, the correlation between the ERN and anxiety cannot exceed the internal reliability (e.g., the correlation between even and odd error trials) of the ERN. If the internal reliability of the ERN is very low, any relationships detected between the ERN and individual differences (e.g., anxiety symptoms) are likely to be the result of Type I error and thus invalid. Considering this, it should be standard practice to report on the psychometric properties of the ERN in any

publication examining the relations between the ERN and any individual differences.

Furthermore, most studies examining error-related brain activity correlate an individual difference variable (e.g., anxiety) with a subtraction-based score calculated by subtracting neural activity on correct trials from neural activity on error trials. This subtraction-based approach is done to isolate neural activity that is specific to error trials. We have done work to show that a regression-based approach may provide an alternative strategy to elucidating the relationships between response monitoring and anxiety (or any individual difference; Meyer, Lerner, et al., 2017). In this study, we show that by using residualized difference scores (i.e., saving the variance from regression analyses wherein the CRN is entered predicting the ERN and vice versa), GAD relates to both a larger ERN and a smaller CRN. Additionally, by probing the interaction between the ERN and CRN, we find that the relationship between GAD and the ERN varies by levels of the CRN. Follow-up analyses suggested that this apparent effect may be due to an increased P300 among individuals with GAD. Taken together, these findings suggest that regression-based approaches to calculating difference scores between conditions in relation to individual differences may be useful.<sup>1</sup>

### 3 | THE ERN AS A CORRELATE OF ANXIETY IN CHILDREN

To date, 10 studies have examined the ERN in clinically anxious child/adolescent populations (Carrasco et al., 2013; Hajcak et al., 2008; Hanna et al., 2012, 2020; Kujawa et al., 2016; Ladouceur et al., 2006, 2018; Meyer, 2017; Meyer et al., 2016, 2019; Meyer, Hajcak, et al., 2013). Consistent with findings in adults, these studies universally find an increased ERN among clinically anxious children. This pattern of results is depicted in Figure 2 wherein the  $\Delta$ ERN is increased in 6-year-old children with anxiety disorders (Meyer, Hajcak, et al., 2013). It should be noted that among these studies, when samples allowed for the examination of specific anxiety disorders, the anxiety disorders characterized by an increased ERN were OCD, social anxiety disorder, and generalized anxiety disorder.

While the relationship between the ERN and clinical anxiety disorders in children appears to be

<sup>1</sup>When discussing the main findings in studies throughout this manuscript, I will refer to the raw ERN as “ERN,” the subtraction-based ERN as “ $\Delta$ ERN,” and the residual-based ERN as “ERN<sub>res</sub>.” In studies that reported significant effects using either the raw ERN or difference-score ERN, I default to using “ERN.”

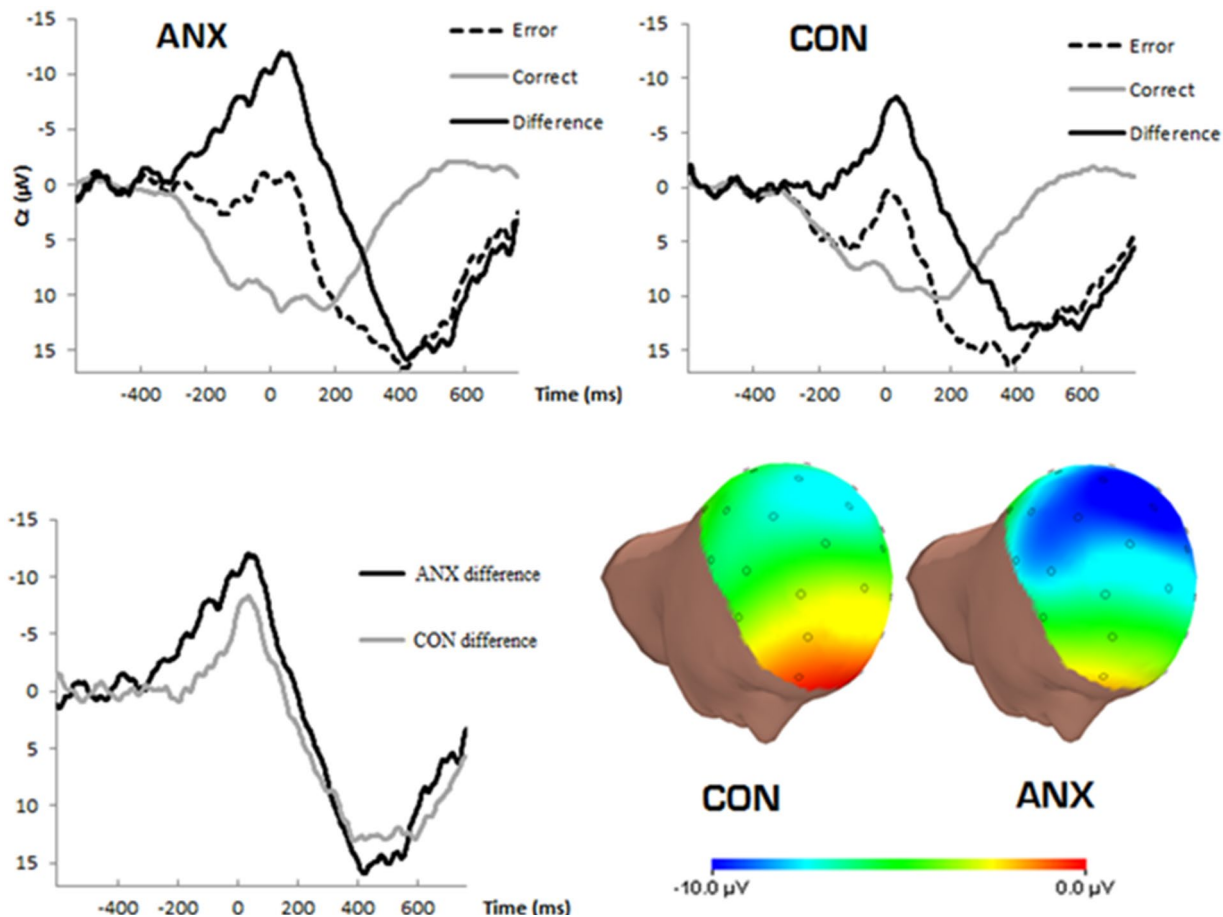


FIGURE 2 On the top, response-locked ERP waveforms for correct and error trials, as well as the difference wave, for 6-year-old children with anxiety disorders (ANX: Left,  $N = 48$ ) and the age-matched healthy controls (CON: Right,  $N = 48$ ). On the bottom left, response-locked ERP difference waveforms (error minus correct) for the ANX and CON groups. On the bottom right, topographic maps of activity (error minus correct) in the time range of the ERN (0–100 ms) for the ANX and CON groups. Reprinted from Meyer, Hajcak, et al. (2013) with permission

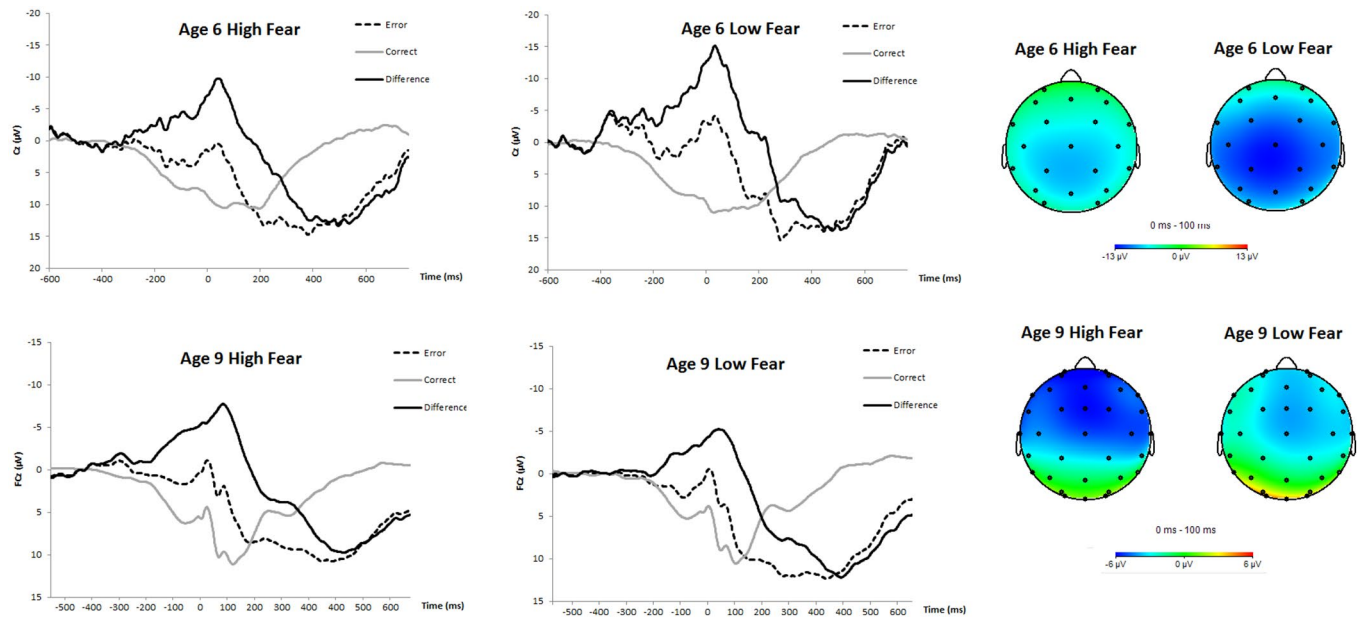
straightforward, things become more complicated when considering findings from studies examining variation in *normative* levels of anxiety. We first observed a change (what we called a “flip”) in the relationship between normative levels of anxiety and the ERN from early to late childhood in a study conducted among 8–13-year-old children/adolescents (Meyer et al., 2012). In this study, we examined the impact of age on the relationship between the ERN and anxiety symptoms. Results suggested that a larger ERN was related to increased anxiety among older children; however, among younger children, the relationship was in the *opposite* direction—a smaller ERN related to increased anxiety symptoms (Meyer et al., 2012). Thus, the relationship between anxiety symptoms and the ERN varied as a function of age.

Subsequent findings have confirmed this general pattern. For example, Torpey et al. (2013) found that 6-year-old children characterized by increased temperamental fearfulness displayed a *blunted* ERN. Similarly,

Moser et al. (2015) found that temperamental fear in 5–6-year-old children related to a blunted ERN, and Lo et al. (2017) found that young children (between the ages of 5 and 8 years old), characterized by increased separation anxiety symptoms, displayed a reduced ERN. Additionally, in a sample of child and adolescent females, we found that the relationship between the  $\Delta$ ERN and anxiety symptoms (checking behavior specifically) changed across development such that among older girls, the ERN was related to anxiety symptoms; however, this relationship was not significant among younger girls (Weinberg et al., 2016). Moreover, Ip et al. (2019) replicated this pattern, finding that age moderated the relationship between the ERN and anxiety. In a sample of children aged 4 to 9 years old, a larger ERN was associated with anxiety in older children; however, among younger children, a *smaller* ERN was associated with increased anxiety.

All of these studies found a similar pattern of results using a between-subject design to examine the moderating





**FIGURE 3** Depicts waveforms for error and correct trials, as well as the difference (error minus correct) for high and low fear groups (based on observational measure at the age 3 assessment) for the age 6 and age 9 EEG assessments. Topographical headmaps are also depicted for these groups wherein activity during correct trials was subtracted from error trials, 0 to 100 ms after response commission. Reprinted from Meyer, Hajcak, et al. (2018) with permission

role of age on the relationship between ERN and anxiety. A prediction that follows from these findings is that we should observe this same pattern *within subjects*—that is, anxious young children characterized by a blunted ERN should undergo developmental changes so that in later childhood, these same children should display an *enhanced* ERN. We tested this hypothesis in a large sample of children ( $N = 271$ ; Meyer, Hajcak, et al., 2018). We had previously reported that, at age 6, children in this sample who were temperamentally fearful were characterized by a *decreased*  $\Delta$ ERN (Torpey et al., 2013). We examined these same children 3 years later, when they were approximately 9 years old. We found that at the age 9 assessment, these same temperamentally fearful children were now characterized by an *enhanced*  $\Delta$ ERN (see Figure 3). That is, we observed this same “flip” in the relationship between anxiety and the  $\Delta$ ERN, longitudinally and within subjects. Another way to state this is that among fearful children, the ERN increased from age 6 to 9, whereas the  $\Delta$ ERN decreased across this same period among children who were low in fear.

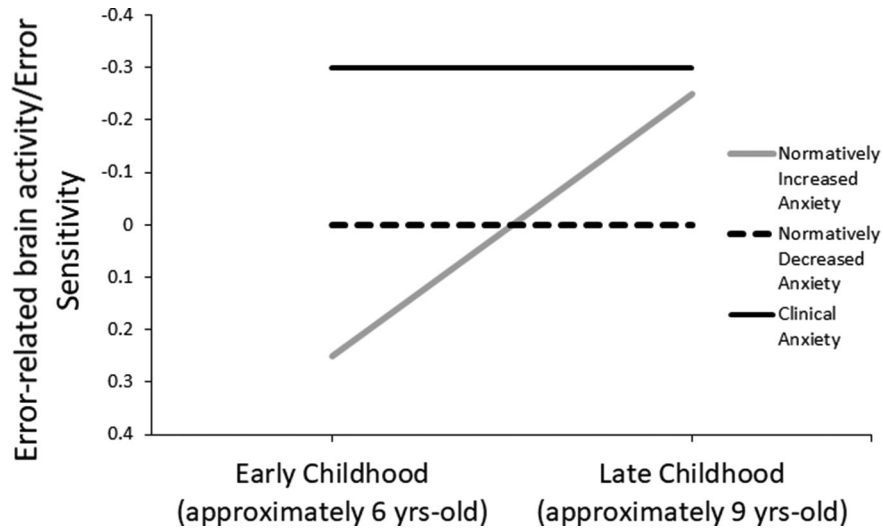
Taken together, the “flip” in the relationship between normative levels of anxiety and the ERN across development has been replicated by multiple research groups and has been found cross-sectionally and longitudinally—that is, in both between- and within-subject designs. Thus, we can be relatively confident that this is a real phenomenon; however, the interpretation of these findings is challenging. One interpretation we have put forth is that this the “flip” in the relationship between normative levels of

anxiety and the ERN may track the changing phenomenology of anxiety across development. We propose that fearful young children may be more focused on external threat (e.g., being alone, separation from their parent, the darkness of the room, interacting with strangers, etc.) and may be less invested in their behavioral performance during the ERN assessment—thus displaying a reduced ERN. Whereas fearful older children may begin to care more about their performance on the laboratory task used to assess the ERN (e.g., they may be more concerned about evaluation of performance by the experimenter or meeting their own standards of performance, etc.)—thus displaying an elevated ERN.

This conceptualization is consistent with developmental work finding that fearfulness in early childhood becomes increasingly associated with self-consciousness in the presence of others across development (Crozier & Burnham, 1990; Jones et al., 2013). As children age, normative anxiety tends to transition from fear of external threat (e.g., monsters, the dark, animals, insects, weather) to self-conscious shyness, worry about behavioral competence, and social evaluation (i.e., internally generated threat; Copeland et al., 2014; Crozier & Burnham, 1990; Gullone, 2000; Spence et al., 2001; Vasey et al., 1994).

## 4 | AN INTERIM SUMMARY

To summarize, the available evidence suggests that *clinical* levels of anxiety are associated with an increased



**FIGURE 4** This figure depicts a theoretical model wherein the relationship between error-related brain activity (i.e., the ERN) and normative levels of anxiety changes during the transition between early and late childhood. A number of studies have found that normative levels of anxiety or fearfulness are related to a *decreased* ERN in young children and an *increased* ERN in older children. This has been found both between and within subjects. However, prior work has also found that clinically anxious children are characterized by an elevated ERN, regardless of developmental stage

ERN, regardless of developmental stage. However, a variety of studies, including both between- and within-subject designs, suggest that *normative* levels of anxiety symptoms or fearfulness may be associated with a blunted ERN in young children and an increased ERN in older children. When, exactly, this “flip” in the relationship between the ERN and normative anxiety occurs is unknown; but based on a large sample of children providing *within-subject* data (Meyer, Hajcak, et al., 2018), we hypothesize that this change occurs between the ages of 6 and 9 years old. To depict this model visually, Figure 4 portrays a model wherein the relationship between the ERN and anxiety “flips” during the transition from early to late childhood (i.e., approximately between the ages of 6 and 9 years old). In this model, as fears related to performance developmentally increase among normatively anxious children, the adult-like ERN/anxiety pattern is observed (i.e., an increased ERN = increased anxiety). However, in this model, children with *clinical* levels of anxiety are characterized by an increased ERN throughout development. We are suggesting that this is because young children with clinical anxiety have already begun to display increased concern over their behavior and performance. Using this framework, children with clinical anxiety do not undergo the normative increase in the ERN, rather, they may have prematurely undergone this change, achieving adult-like levels early in life. Consistent with this proposition, some studies among children with clinical levels of anxiety, do not find a relationship between age and the ERN (Carrasco et al., 2013; Hanna et al., 2012).

It should be noted that one implication of the model depicted in Figure 4 is that, in early childhood, being a child high in anxiety or fearfulness *and being characterized by a relatively large ERN*, should confer the greatest risk for clinical anxiety. In other words, this model suggests that the *interaction between the ERN and early fearfulness/anxiety* should be a superior predictor of clinical anxiety. Indeed, there has been work done to support this notion. A series of studies has suggested that increased behavioral inhibition (i.e., BI, typically measured between 6 months and 7 years old) interacts with the ERN to predict anxiety later in life (Lahat et al., 2014; McDermott et al., 2009; Tang et al., 2020). Indeed, we also previously found that the  $\Delta$ ERN and anxiety symptoms measured in 6-year-old children *interacted to predict new onset clinical anxiety disorders* at age 9, such that among children with increased anxiety symptoms, a large  $\Delta$ ERN predicted new onset anxiety disorders (it should be noted that this interaction was significant at a trend level,  $p = .06$ ; Meyer et al., 2015). Additionally, we have shown that early fearfulness (measured at age 3) interacted with the  $\Delta$ ERN (measured at age 6) to predict stressor-related increases in anxiety symptoms following a hurricane, such children who were high in fearfulness, *and had an increased  $\Delta$ ERN*, experienced the most increases in anxiety (Meyer, Danielson, et al., 2017). Taken together, these findings suggest the possibility that there is a developmental period in early childhood wherein it may be optimal to identify who is at risk for future increases in anxiety—that is, those children who are characterized by high levels of fearfulness *and also an increased  $\Delta$ ERN*. Moreover, this

model suggests that the ERN may have more incremental validity in predicting future increases in anxiety in early childhood, compared to late childhood, adolescence, and adulthood. Future work should investigate this possibility.

## 5 | THE ERN AS A RISK MARKER OF ANXIETY

The work discussed thus far has focused on the increased ERN observed among clinically anxious children and adolescents, as well as the correlation between the ERN and anxiety symptoms across development. However, biomarkers with clinical utility that go beyond detecting current disease state or current symptoms, by predicting who is *at risk* for developing a particular disease or increases in symptoms are of particular interest. Identifying neural markers that not only correlate with anxiety, but that can also predict new onset anxiety disorders across development is critical to furthering our understanding of the underlying mechanisms of anxiety. Moreover, identifying neural markers of risk may also enhance the efficacy of early identification, prevention, and intervention strategies.

We, and others, have examined the potential utility of the ERN as a risk marker in large, longitudinal samples. Results are consistent across all studies: a potentiated ERN relates to increased anxiety prospectively (Filippi et al., 2020; Lahat et al., 2014; McDermott et al., 2009; Meyer, Danielson, et al., 2017; Meyer et al., 2015, 2021; Meyer, Nelson, et al., 2018). One of our studies found that an increased  $\Delta$ ERN predicted the onset of new anxiety disorders in children between the ages of 6 and 9 years old, even while controlling for baseline anxiety symptoms and maternal history of anxiety (Figure 5; Meyer et al., 2015). We have replicated this pattern of results among adolescent females, finding that an increased  $\Delta$ ERN predicted new onset generalized anxiety disorder (GAD) across 18 months among 457 girls between the ages of 13 and 15 years old, even when controlling for baseline symptoms (Meyer, Nelson, et al., 2018).

We, and others, have also examined the extent to which the ERN may interact with other risk markers to predict trajectories of risk for anxiety longitudinally. For example, we examined the interaction between the  $\Delta$ ERN and temperamental fear in predicting *stress-mediated* increases in anxiety symptoms following Hurricane Sandy on Long Island (Meyer, Danielson, et al., 2017). Children who were high in temperamental fear when they were 3 years old and experienced increased hurricane-related stressors when they were 9 years old, were characterized by a subsequent increase in anxiety symptoms—but *only when they were also characterized by an increased  $\Delta$ ERN at the age 6*

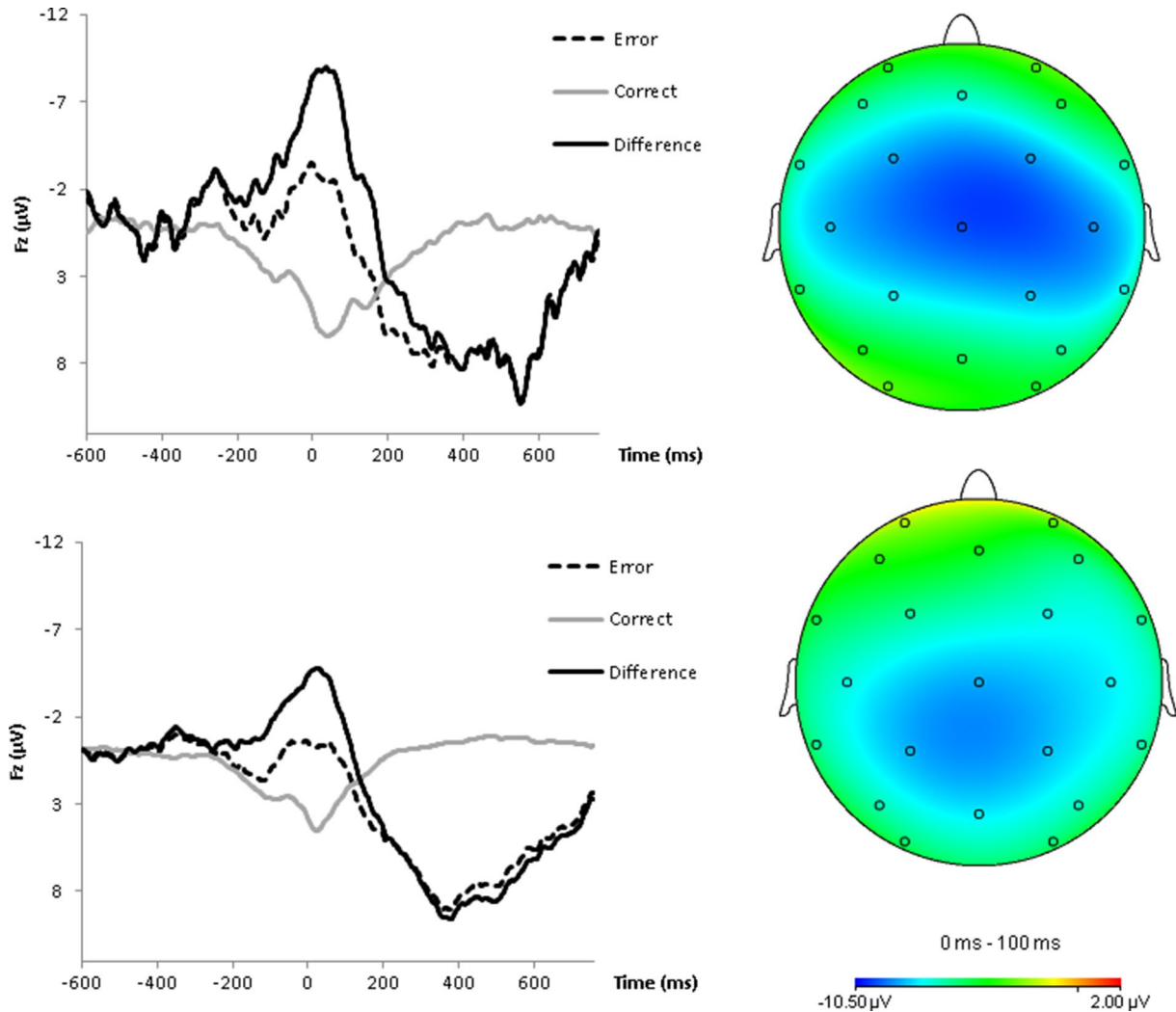
*assessment*. This pattern of results is consistent with other work suggesting that temperament and the ERN interact to predict risk for anxiety (Lahat et al., 2014; McDermott et al., 2009). These findings suggest that the ERN may be a useful marker of risk that can predict longitudinal increases in anxiety, especially when used in conjunction with other risk markers.

Collectively, the studies discussed thus far suggest that the ERN is increased among individuals with a current clinical anxiety disorder, and that the ERN may indicate risk for future increases in anxiety among healthy individuals. However, no previous study had examined whether an increased ERN, among clinically anxious individuals, may also confer risk (i.e., serve as a prognostic indicator). This is important insofar as previous work suggests that traditional cognitive behavioral therapy (CBT) approaches do not impact the ERN, despite decreases in anxiety symptoms (Hajcak et al., 2008; Kujawa et al., 2016; Ladouceur et al., 2018; Riesel et al., 2015). For example, in a large pediatric treatment study, CBT decreased anxiety symptoms but did not impact the ERN (Ladouceur et al., 2018).

To begin to address the question of whether the ERN may be a useful prognostic indicator *within* anxious individuals, we recently conducted a study wherein we examined the extent to which the  $\Delta$ ERN predicted changes in anxiety symptoms across 2 years among children and adolescents with current clinical anxiety disorders (Meyer et al., 2021). Results confirmed that an enhanced  $\Delta$ ERN, among individuals with anxiety disorders, predicted increases in symptoms across time. Additionally, the  $\Delta$ ERN predicted increases in specific domains of anxiety: that is, generalized anxiety, social anxiety, and harm avoidance/perfectionism. The  $\Delta$ ERN did not relate to increases in panic, separation, school avoidance, or physical anxiety symptoms. Moreover, this pattern of results was similar when using two different self-report measures of anxiety (i.e., the Screen for Child Anxiety Related Emotional Disorders and the Multidimensional Anxiety Scale for Children). Thus, these findings suggest that the ERN may be a useful prognostic indicator even among currently anxious individuals.

## 6 | THE ERN IS A TRANSDIAGNOSTIC RISK MARKER—BUT WHAT EXACTLY DOES IT INDEX?

Thus far, I have summarized my (and others') work suggesting that the ERN is increased in anxious individuals and indexes risk for future anxiety. A number of review articles and meta-analyses have been written on this topic (Cavanagh & Shackman, 2014; Hajcak, 2012; Meyer, 2016,



**FIGURE 5** On the left, response-locked ERP waveforms for correct and error trials, as well as the difference waves at the baseline assessment (when children were 6 years old). On the right, topographic maps of activity (error minus correct). Top = children who would develop a new onset anxiety disorder between ages 6 and 9 years old; bottom = children who did not develop a new onset anxiety disorder between ages 6 and 9 years old. Reprinted from Meyer et al. (2015) with permission

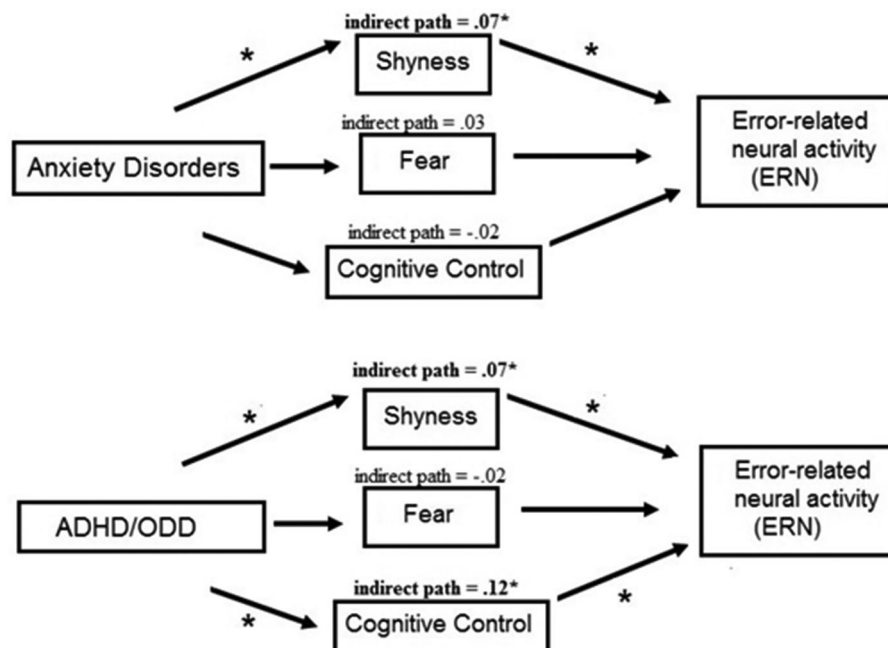
2017; Moser et al., 2013; Riesel, 2019; Weinberg et al., 2016) and there is general consensus among researchers that the ERN is associated with anxiety. However, there has been much debate as to what psychological constructs are indexed by variability in the ERN. More specifically, what psychological phenomena may underlie the association between the ERN and anxiety? Put more simply, why is the ERN increased in anxious individuals?

As I have stated above, the ERN appears to be increased specifically in disorders characterized by increased worry, concern about performance, behavior, or mistakes—including generalized anxiety disorder (i.e., GAD, Weinberg & Hajcak, 2011; Weinberg, Klein, et al., 2012; Xiao et al., 2011), OCD (Endrass et al., 2008; Gehring et al., 2000; Riesel, Klawohn, et al., 2019; Weinberg et al., 2015), and social anxiety disorder (Barker et al., 2015, 2018; Endrass et al., 2014; Kujawa et al., 2016).

Studies wherein we have examined what specific anxiety symptom domains the ERN relates to have found a similar pattern—that is, social anxiety (Meyer, Carlton, et al., 2018), shyness (Meyer & Klein, 2018), and checking symptoms (Weinberg et al., 2016)—all symptom domains that relate to concern over one's own behavior. We also found that among anxious individuals, the  $\Delta$ ERN predicted developmental increases in generalized anxiety, social anxiety, and harm avoidance/perfectionism, but not panic, separation, school avoidance, or physical anxiety symptoms (Meyer et al., 2021). Moreover, we, and others, have found that the ERN relates to perfectionism (Barke et al., 2017; Meyer & Wissemann, 2020; Perrone-McGovern et al., 2017; Schrijvers et al., 2010; Stahl et al., 2015), a set of traits related to hypervigilance toward one's own behavior and mistakes. Taken together, these findings suggest that the ERN may be viewed as a transdiagnostic neural



**FIGURE 6** A conceptual model depicting the indirect pathways between clinical disorders (anxiety disorders and ADHD/ODD) and the error-related negativity (ERN). As can be seen in the model, the indirect pathway from anxiety disorders to shyness to the ERN was significant. Additionally, the indirect pathway from ADHD/ODD to the ERN via shyness and cognitive control was significant. Reprinted from Meyer and Klein (2018) with permission



marker related to anxiety—but more specifically indexing facets of anxiety related to concern about one’s own behavior and/or the extent to which individuals find making mistakes aversive.

Along these same lines, the ERN has also been observed to be *decreased* among individuals with externalizing disorders (Meyer & Hajcak, 2019; Meyer & Klein, 2018; Olvet & Hajcak, 2008). We recently reviewed this body of work and identified 14 studies that found a reduced ERN among individuals with attention deficit hyperactivity disorder (although, it should be noted that 12 studies did not find this association; Meyer & Hajcak, 2019). Although more work is needed investigating the relationship between the ERN and externalizing disorders, and findings are mixed, this general pattern of results could be viewed as consistent with the perspective discussed above—that is, individual differences in the ERN may index concern over mistakes or one’s own behavior. More specifically, it is possible that individuals with ADHD are less concerned with their own behavior compared to individuals without ADHD, and thus, are characterized by a reduced ERN.

There is another perspective that posits that the ERN indexes individual differences in cognitive control (Cavanagh & Frank, 2014; Meyer & Hajcak, 2019; Wessel, 2018). Although work in this area is still emerging, we (and others) have found some evidence to suggest that individual differences in the ERN may index variation in cognitive control processes (Coleman et al., 2017; Larson & Clayson, 2011; Meyer & Klein, 2018; Miller et al., 2012; Weaver et al., 2017). Thus far, this work has suggested that individuals who are characterized by increased cognitive control (e.g., increased working memory, attention, or

inhibitory control) are also characterized by an increased ERN.

Collectively, these findings lead to interesting questions: does the ERN index cognitive control or sensitivity to errors, or both? Is the increased ERN *found among anxious individuals* reflecting differences in cognitive control or error sensitivity? Or both? And, what about the *decreased* ERN found among individuals with externalizing disorders—is this better explained by reduced error sensitivity or deficits in cognitive control? It is also interesting to note that the National Institute of Mental Health (NIMH) includes the ERN in the Research Domain Criteria (RDoC) as both a measure of sustained threat *and* cognitive control (Weinberg et al., 2016).

Despite a substantial amount of discussion of these issues (Moser et al., 2013, 2014; Proudfit et al., 2013), there has been remarkably little *empirical* work done to clarify these possibilities. However, we have begun to examine the possibility that the link between the ERN and anxiety may be explained, in part, by individual variation in concern over mistakes (i.e., error sensitivity). For example, in a large sample of 6-year-old children, we utilized the Children’s Behavior Questionnaire (CBQ; Rothbart et al., 2001) to measure shyness (e.g., concern related to social evaluation and/or social situations) versus fear (e.g., concern related to external threats, like dogs), and used path analyses to examine the extent to which these psychological constructs mediated the relationship between the  $\Delta$ ERN and anxiety disorders (it should be noted, that this study utilized cross-sectional data; Meyer & Klein, 2018). Results from this study suggested that the increased  $\Delta$ ERN observed among anxious children was

explained by increased shyness, but not increased fear (see Figure 6). Thus, an interpretation of these findings is that clinically anxious children exhibited an increased  $\Delta$ ERN *because they were more shy* or concerned about their behavior/performance compared to non-clinically anxious children.

In this same study (Meyer & Klein, 2018), we also examined the extent to which the variance between the ERN and anxiety disorders was explained by differences in cognitive control (i.e., attentional focusing, attentional shifting, and inhibitory control). Consistent with other work, the  $\Delta$ ERN was related to cognitive control (results were significant for all three domains of control), suggesting that children with increased ability to focus and shift their attention, as well as inhibit prepotent responses, were also characterized by increased error-related brain activity. However, cognitive control did not mediate the relationship between anxiety disorders and the  $\Delta$ ERN. Moreover, children with anxiety disorders were not characterized by differences in cognitive control compared to healthy controls. Thus, we can interpret these findings to suggest that differences in cognitive control between anxious and non-anxious individuals do not underlie the differences in the  $\Delta$ ERN that are observed. Rather, it is differences in shyness that better account for the differences in the  $\Delta$ ERN observed between anxious and non-anxious children.

Moreover, in this study (Meyer & Klein, 2018), we also examined the relationship of the  $\Delta$ ERN to ADHD. Consistent with other work (Meyer & Hajcak, 2019), the  $\Delta$ ERN was reduced among children with ADHD. Interestingly, the relationship between the  $\Delta$ ERN and ADHD was mediated by *both cognitive control and shyness*, suggesting that it was because children with ADHD were less shy *and* characterized by deficits in cognitive control that they displayed a blunted  $\Delta$ ERN (Figure 6). Thus, this type of empirical work can begin to answer the question of what psychological constructs may underlie the associations between the  $\Delta$ ERN and various forms of psychopathology.

We have recently begun to investigate the relationship between the ERN and error sensitivity (i.e., fear of making mistakes) more specifically. To do so, we developed a measure for children, the Error Sensitivity Index, a nine-item, self-report measure that includes items such as: I like to do things perfectly; When I make a mistake, I feel anxious; I am afraid of making mistakes in front of other people; When I notice a mistake I made, I feel upset (see Table 1; Chong & Meyer, 2019). In this study, 97 children between the ages of 5 and 7 years old completed the Error Sensitivity Index. The measure demonstrated good internal reliability and good convergent validity with other self-report measures. Additionally, the  $\Delta$ ERN related to error sensitivity,

such that children who reported being more fearful of their own mistakes also displayed an elevated ERN. In this study, we also utilized mediation analyses to investigate the extent to which error sensitivity mediated the relationship between the  $\Delta$ ERN and anxiety symptoms. Using both parent- and child-reported anxiety symptoms, results confirmed that the relationship between the  $\Delta$ ERN and anxiety was mediated by error sensitivity.

While more work is needed to clarify the psychological constructs that underlie the associations between the ERN and various forms of psychopathology, these studies are examples of how we can begin to use empirical investigations to understand these relationships. It will be important for future work to use valid and reliable measurement (e.g., self-report, behavioral and cognitive assessments, observational measures, etc.) to better characterize the psychological components of the relationships between the ERN and anxiety. Doing so may improve intervention and prevention efforts. For example, we may develop novel psychosocial interventions targeting the ERN and its associated psychological constructs to reduce risk for anxiety. Moreover, it will be important for future work to clarify the extent to which these alternate measures (e.g., the Child Error Sensitivity Index) have incremental validity in predicting error-related brain activity, above and beyond current measures of similar constructs.

Moreover, building on these findings, future work should begin to elucidate what mechanisms or processes may underlie the relationship between the ERN and anxiety. For example, it is possible that young children who are characterized by an increased ERN/error sensitivity may engage in more avoidance behaviors (e.g., avoidance of activities they expect to make mistakes in, avoidance of challenging activities, avoidance of socially evaluative experiences, like not raising their hand in class, or general failure avoidance), which may, over time, lead to increased anxiety. Future longitudinal studies in developmental populations are needed to further elucidate these potential mechanisms.

## 7 | FACTORS THAT SHAPE THE ERN

Considering the fact that the ERN is increased in anxious individuals (Meyer, 2016, 2017), predicts risk for future increases in anxiety among healthy and anxious populations (Meyer et al., 2021), and may be elevated early in the course of development, (i.e., before anxiety has become impairing, (Meyer et al., 2015), it is important to better understand what factors contribute to the development of the ERN early in life. While some of the variance in the ERN is heritable, a large portion of the variance appears to

**TABLE 1** Rotated factor loadings in the child error sensitivity index

Item	Rotated factor loadings		
	Factor 1 social concerns	Factor 2 perfectionism	Factor 3 physical reactions
I feel upset when other people do not like something I have done	<b>.78</b>	-.04	-.09
I am afraid of making mistakes in front of other people	<b>.72</b>	.08	.08
When someone notices I did something wrong, I feel upset	<b>.57</b>	.06	.24
If I make a mistake, I always want to fix it	-.12	<b>.97</b>	-.07
I like to do things perfectly	.11	<b>.44</b>	.04
When I make a mistake, I feel anxious	.22	.08	<b>.62</b>
My stomach feels sick when I make a mistake	-.12	-.02	<b>.58</b>
When I make a mistake, I start sweating or blushing	.09	-.07	<b>.51</b>
When I notice a mistake I made, I feel upset	.15	.21	<b>.40</b>
Eigenvalues	3.30	1.32	1.09
% of variance	36.99	14.67	12.11
$\alpha$	.78	.61	.68

*Notes:* This is a nine-item self-report measure indexing error sensitivity. In this study, 97 children between the ages of 5 and 7 years old were administered this self-report measure—which demonstrated good internal reliability and convergent validity. The child error sensitivity index related to the ERN and mediated the relationship between the ERN and child anxiety symptoms. Reprinted from Chong and Meyer (2019) with permission.

Bold values indicates  $p < .05$ .

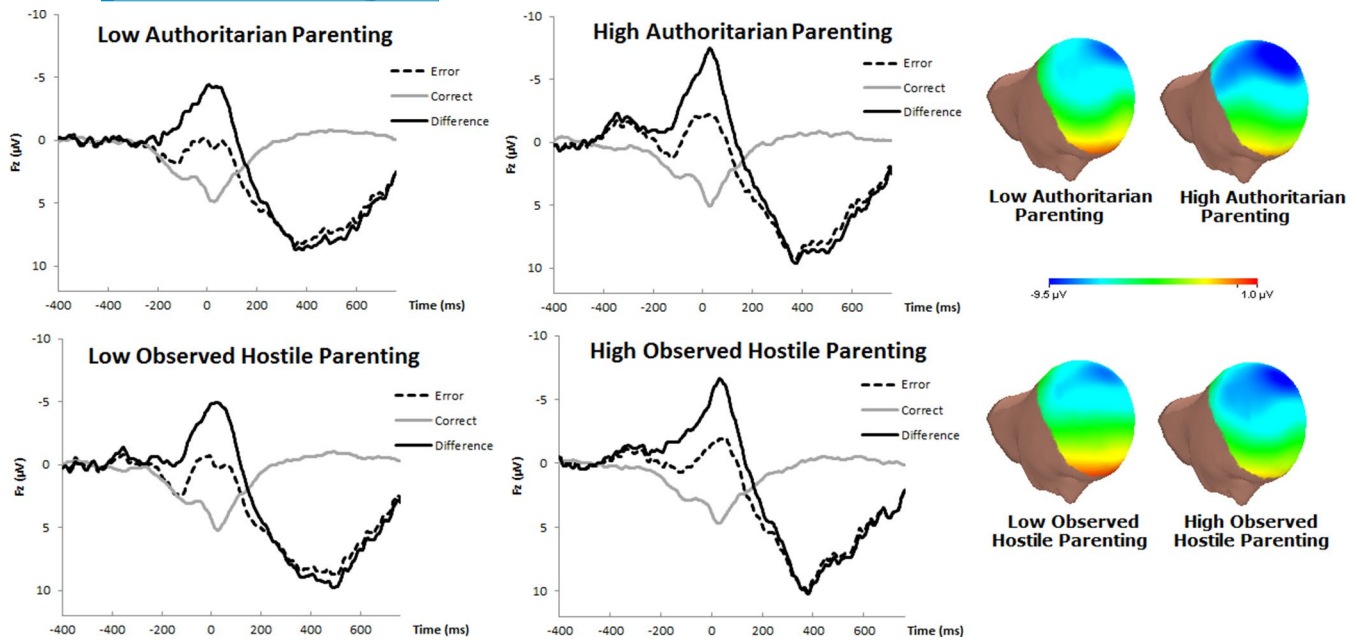
be impacted by environmental factors (40–60%; Anokhin et al., 2008).

Within-subject work in the laboratory indicates that the ERN is sensitive to motivational and environmental factors surrounding performance. For example, the ERN is larger when errors are more significant or costly (Chiu & Deldin, 2007; Ganushchak & Schiller, 2008; Hajcak et al., 2005; Riesel et al., 2012), when performance is being evaluated by someone (Hajcak et al., 2005; Kim et al., 2005), and when accuracy is emphasized over speed (Falkenstein et al., 2000; Gehring et al., 1993).

We, and others, have done work to demonstrate that when individuals are punished for making mistakes during a laboratory-based task, the ERN is potentiated (Meyer & Gawlowska, 2017; Riesel et al., 2012; Riesel, Kathmann, et al., 2019). Riesel et al. (2012) first demonstrated this effect, finding that the ERN was increased during blocks wherein errors were punished. Moreover, this effect persisted even after punishment ended, suggesting that learning about the potential consequences of errors had a lasting impact on the ERN. Riesel and colleagues extended these findings, showing that these effects persisted for up to 24 hr after the initial punishment

experience (Riesel, Kathmann, et al., 2019). We have shown that punishment must be specific to mistakes—that is, the ERN was increased during blocks when participants were sometimes punished for errors, but *not* during blocks when participants were punished randomly (Meyer & Gawlowska, 2017). It is also notable, that in all of these studies, the impact of punishment on the ERN was most pronounced among anxious individuals.

In light of the fact that learning experiences surrounding mistakes and performance appear to impact the ERN in the laboratory, we have begun to extend these findings to naturalistic settings by examining how parenting styles impact the magnitude of the ERN in offspring. Parenting is arguably the most important learning context in early childhood. Critical, controlling, authoritarian, or harsh parents often punish children's mistakes more frequently and more intensely (Robinson et al., 2001), which can result in children experiencing excessive anxiety surrounding their own mistakes (Kawamura et al., 2002). We have proposed that one mechanism that may lead to an increased ERN in children is exposure to a punitive learning environment as a result of critical or harsh parenting styles.



**FIGURE 7** On the left, response-locked ERP waveforms for correct and error trials, as well as the difference wave. On the right, topographic maps of activity (error minus correct). For representation purposes, a median split was performed on both self-reported authoritarian parenting style (top) and observed hostile parenting (bottom). Reprinted from Meyer et al. (2015) with permission

We, and others, have found that harsh or critical parenting styles are linked to an increased ERN in offspring (Banica et al., 2019; Brooker & Buss, 2014; Chong et al., 2020; Meyer et al., 2019; Meyer, Proudfit, et al., 2014; Meyer & Wissemann, 2020). In an early study, we found that both observational measures of parenting (coded in the laboratory, based on recorded interactions) and self-report measures of parenting related to the ERN in 6-year-old children—such that harsh and authoritarian parenting (high control/low warmth) styles were both related to a larger ERN in children (see Figure 7; Meyer, Proudfit, et al., 2014). In this study, we also found that harsh parenting related to increased clinical anxiety, and that the ERN mediated the relationship between parenting and anxiety. We conjectured that parenting may shape children's error processing through environmental conditioning, and thereby risk for anxiety.

Since this original study, we have replicated this finding in adolescents (Chong et al., 2020) and adults (Meyer & Wissemann, 2020). Notably, in both of these follow-up studies, results supported a mediation model wherein the relationship between controlling parenting and anxiety in offspring was mediated by the ERN. These mediation models support the notion that children may be learning about the significance of their errors in the context of their parents' reactions, and this may predispose them to anxiety. Adults primarily rely on their own internal monitoring as feedback to guide their behavior; however, young children rely more on external sources of information (oftentimes their caregiver) for feedback related to

their behavior (Ghatala, 1986; Pressley, Levin, et al., 1984; Pressley, Ross, et al., 1984; Skinner et al., 1988; Stipek & Tannatt, 1984; Zimmerman, 1989, 1990). We propose that parents may be scaffolding children's emerging ability to recognize and respond to their own mistakes, in the form of verbal or non-verbal reactions, or controlling parental behavior.

Thus far, the studies discussed examined the association between parenting and the ERN in children. However, we are proposing a *causal* mechanism, wherein parenting styles are shaping the ERN in children through learning experiences. We are proposing that it is through children's repeated exposure to making mistakes, in the context of their parents' reactions, that the ERN is being impacted. Over time, children may be internalizing their parents' reactions to their mistakes. Thus, parents who are overly harsh or punitive may be conditioning their children to be over-reactive to their own mistakes and thereby more prone to certain types of anxiety.

To further elucidate this causal mechanism, we conducted a study wherein we examined the impact of parental *presence* on the magnitude of the ERN in children (Meyer et al., 2019). We measured the  $\Delta$ ERN in 5–7-year-old children while their parent sat next to them or while an experimenter sat next to them. Results suggested that the  $\Delta$ ERN was increased in children when their parent was present, *but only if that parent was characterized by a controlling parenting style*. Additionally, the  $\Delta$ ERN was increased among children with clinical levels of anxiety and the relationship between controlling parenting style and anxiety



was mediated by the potentiation of the  $\Delta$ ERN when parents were in the room—further supporting the causal mechanism proposed above. This study uses a within- and between-subject design to further support the notion that children learn how to respond to their mistakes in the context of their caregivers, and that this learning process may be relevant to shaping a neural marker and thus, risk for anxiety. We are continuing to examine this causal mechanism in a large, longitudinal study, by administering a parenting intervention that focuses on reducing over-reactivity to children's errors and measuring the ERN in children before and after the intervention.

Although much of our work has focused on how parenting may shape the ERN, we are beginning to explore other factors that may potentially shape the ERN as well. In a recent study, we examine the relationship between interpersonal dependent and non-interpersonal stressful life events and the  $\Delta$ ERN in a sample of child and adolescent females, finding that more frequent interpersonal dependent stressors predict a larger  $\Delta$ ERN (Mehra & Meyer, in press). Previous work found that higher numbers of adverse childhood experiences related to a larger ERN (Lackner et al., 2018; Wu et al., 2021) and one study found that social-evaluative stress during early adolescence was related to an enhanced ERN (Banica et al., 2021). Moreover, two studies have found that the ERN interacts with stressful events to predict changes in anxiety prospectively (Banica et al., 2020; Meyer, Danielson, et al., 2017). Collectively, these studies suggest that the ERN may both be impacted by interpersonal dependent or social evaluative stressors (e.g., failure experiences, peer victimization, humiliation, etc.), as well as interact with stressors to predict future increases in anxiety.

## 8 | TARGETING THE ERN

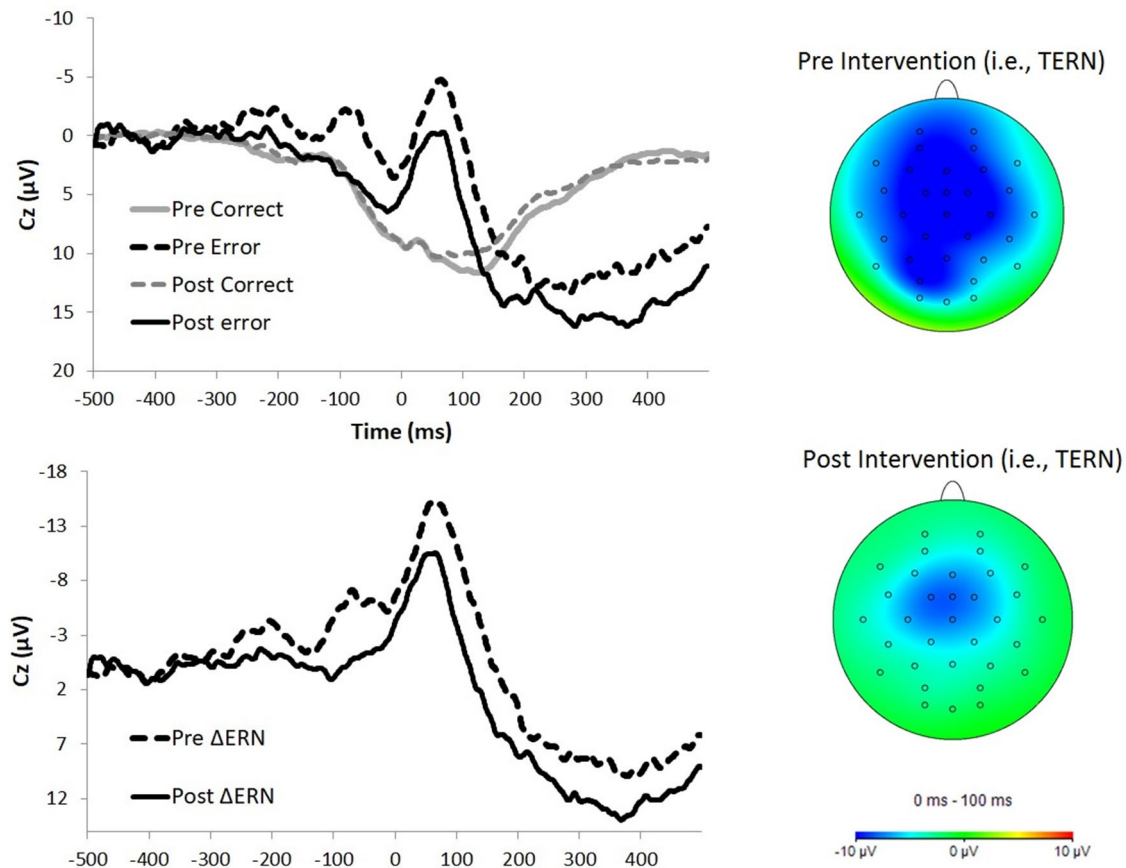
A recent focus of my work has been to examine novel strategies to reduce the ERN. This is especially important in light of the fact that the ERN is elevated early in the course of development, before anxiety symptoms become impairing. There is a large body of theoretical and empirical work suggesting that early intervention and/or prevention methods may be more efficacious than interventions applied after anxiety symptoms have reached clinical levels (Dadds et al., 1997; Mancebo et al., 2014; Pina et al., 2020; Rapee et al., 2005, 2010; Schotanus-Dijkstra et al., 2017; Stoll et al., 2017). Thus, targeting the ERN may be a fruitful strategy to prevent future increases in anxiety.

As discussed previously, the ERN does not appear to be impacted by typical intervention approaches for anxiety disorders. Studies that have investigated the ERN

before and after treatment find that cognitive behavioral approaches (i.e., CBT) do not impact the ERN, despite changes in anxiety symptoms (Hajcak et al., 2008; Kujawa et al., 2016; Ladouceur et al., 2018; Riesel et al., 2015). A recent study suggested that while CBT decreased anxiety symptoms, it did not change either the ERN; importantly, CBT also did not change *worry related to performance* (Ladouceur et al., 2018). This suggests that the ERN (and the extent to which individual find errors aversive, i.e., error sensitivity) remain elevated in anxious individuals, even after treatment. These findings indicate a need for the development of novel treatment strategies that directly target error sensitivity (and thereby the ERN) to complement existing prevention and intervention approaches.

There is some evidence that the ERN can be modified by approaches other than CBT. For example, transcranial direct current stimulation (i.e., tDCS; Reinhart & Woodman, 2014), attention bias modification in healthy adults (Nelson et al., 2015, 2017) as well as in adults and children with OCD (Klawohn, Hajcak, et al., 2020; Tan et al., 2021), and expressive writing (Schroder et al., 2018) have all been shown to impact the ERN in the short term (however, see: Carlson et al., 2021). However, none of these approaches directly target the *psychological* constructs linked to a potentiated ERN (e.g., error sensitivity)—and thereby may not impact anxiety. Moreover, these manipulations may not be ideal for young children (Rajapakse & Kirton, 2013). Additionally, these approaches are not ideal for at-home administration or widespread dissemination—both factors that are important for broad prevention approaches. Considering that an increased ERN *early in life* is a risk factor for anxiety (Meyer, 2017), it is crucial to develop interventions targeting the ERN that could be administered to children.

Building on the work we have done to identify the psychological factors that link the ERN to anxiety, reviewed above, we have begun to test intervention strategies that directly target error sensitivity. In a recent study, we designed a brief, computerized intervention to reduce sensitivity to errors (Meyer et al., 2020). This intervention consisted of a 1-hr computer-based tutorial on perfectionism, over-valuation of the negative consequences of errors, and fears related to the social consequences of making a mistake. The intervention was CBT-based and focuses on concepts such as “everybody makes mistakes,” “making mistakes is how we learn new things,” and “good things come from mistakes.” Eighty undergraduates were randomized to either the intervention or a control condition (a tutorial focused on self-care), and the ERN<sub>resid</sub> was measured before and after the intervention or control condition. Results confirmed that participants in the intervention condition experienced a significant reduction in the ERN<sub>resid</sub>, whereas participants in the control condition



**FIGURE 8** On the left, waveforms at Cz for pre- and post-intervention (“Treating the ERN,” i.e., TERN) for error and correct trials (top) and the  $\Delta$ ERN (error minus correct; bottom). Topographical headmaps (right) depicting neural activity for error minus correct, 0–100 ms after the response, for pre-intervention (top) and post-intervention (bottom). Participants included in this graphical depiction had a relatively large  $\Delta$ ERN (based on a median split) at the pre assessment. Results suggest that TERN reduced the ERN (and  $\Delta$ ERN), especially among individuals with a large  $\Delta$ ERN. Reprinted from Meyer et al. (2019) with permission

showed no change. Furthermore, the impact of the intervention on the  $ERN_{resid}$  was more pronounced among individuals who were characterized by a larger  $ERN_{resid}$  at baseline (Figure 8).

These findings suggest that the ERN can be modified via a targeted, psychosocial intervention. While this is an important first step, future work should examine if these changes in the ERN are lasting. And, perhaps more importantly, we need to examine to what extent modifying the ERN may relate to subsequent reduced risk for anxiety. If, indeed, brief psychosocial interventions can make a meaningful change in the ERN to reduce anxiety, this approach may be beneficially applied to younger children—perhaps especially for those with a relatively large ERN. Along these lines, we are currently conducting a large, longitudinal study in children between the ages of 5 and 7 years old to determine if a computerized, psychosocial intervention targeting child error sensitivity and parenting strategies may impact the ERN and anxiety symptoms. Results from this study

may pave the way for future prevention and intervention approaches.

## 9 | FUTURE DIRECTIONS

In my laboratory, we are focusing on two broad goals going forward: (1) Optimize the ERN as a screening and/or diagnostic tool and (2) develop novel intervention strategies that target the ERN.

I previously discussed the issue of examining the psychometric properties of the ERN—using different tasks, at different ages, and in various healthy and clinical populations. Although we have begun to do this, there is still much work to be done to identify the optimal task to utilize at different ages, to determine how many trials are sufficient, whether or not multiple measurements are useful, etc. We also plan to explore whether it is feasible to measure the ERN in school and clinic settings for screening and diagnostic purposes. Additionally, we have done

some work using ROC curve analyses to examine to what extent the ERN can predict new onset anxiety disorders (Meyer, Nelson, et al., 2018)—finding that an algorithm using baseline anxiety symptoms and the ERN exhibited good positive predictive value (72%) and excellent negative predictive value (94.3%). Future work should examine to what extent the ERN may be a useful in predicting clinical trajectories *at different stages of development*, and in conjunction with other biomarkers. For example, it is possible that the ERN has optimal predictive power when children are younger versus older, and when considered along with other markers (e.g., attention bias, behavioral inhibition, cortisol, other ERPs, etc.). Further work is needed to determine when and how to measure the ERN to optimally utilize it for the purposes of screening and predicting clinical trajectories.

Moreover, while some work has begun to establish norms for the ERN in adults (Imburgio et al., 2020), no work has established norms for the ERN at different developmental stages. Due to the fact that the ERN increases across development (Tamnes et al., 2013) and differs by task (Meyer, Bress, et al., 2014), it is important for future work to establish norms using standardized tasks in individuals across development. The ERN can only be used as a screening tool if we have age-appropriate norms by which to compare individuals. For example, it will be necessary to determine if a 12 years old has an increased ERN, relative to other 12 years old, using a standardized task. Large, longitudinal studies, spanning different stages of development, will be necessary to derive age-specific clinical cut-off scores for the ERN, as well as to determine task optimization in various populations.

Moving beyond the use of the ERN as a screening tool, an intriguing possibility is that the ERN may be a novel *target* for intervention approaches. As discussed above, we developed a brief, computerized intervention to target the ERN in adults (Meyer et al., 2020) and are currently conducting a large, longitudinal study investigating whether a computerized intervention administered to parents and children between the ages of 5 and 7 years old reduces the ERN. This study includes a 6-month follow-up assessment wherein we will evaluate whether changes in the ERN persist, and, importantly, whether changes in the ERN are related to reductions in anxiety symptoms. Another exciting avenue of future investigation is to determine whether even more minimal intervention strategies may impact error sensitivity (and the ERN). For example, text message reminders about the value of learning from mistakes or basic exposure activities (e.g., making mistakes on purpose) may reduce the ERN. Additionally, considering that the ERN remains elevated even after successful CBT therapy (Hajcak et al., 2008; Ladouceur et al., 2018), it may be useful to add targeted intervention strategies to existing

CBT approaches to reduce error sensitivity, and thereby potentially reduce risk for relapse. And, considering that children spend a large portion of their time learning about the value of mistakes in school, another intriguing possibility is to examine whether intervention or prevention strategies administered to teachers may impact children's error sensitivity.

Taken together, work done thus far suggests that error-related brain activity may be a useful risk marker and prognostic indicator, especially when used in conjunction with other assessments (e.g., other neural markers, self-report, etc.). Moreover, one intriguing possibility is that by targeting error-related brain activity early in development, we may be able to alter trajectories of risk for anxiety. If using brief, computerized intervention approaches are able to alter error-related brain activity in a meaningful way (i.e., reduce risk for anxiety), future work should focus on issues related to implementation and dissemination.

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