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Increased Error-Related Brain Activity in Six-Year-Old Children with Clinical Anxiety

Alexandria Meyer[a,*], **Greg Hajcak**[a], **Dana C. Torpey**[b], **Autumn Kujawa**[a], **Jiyon Kim**[a], **Sara Bufferd**[a], **Gabrielle Carlson**[a], and **Daniel N. Klein**[a]

[a]Department of Psychology, Stony Brook University, Stony Brook, New York

[b]Department of Veterans Affairs San Diego, San Diego, California

Abstract

Anxiety disorders are the most frequently diagnosed form of psychopathology in children and often result in chronic impairment that persists into adulthood. Identifying neurobehavioral correlates of anxiety that appear relatively early in life would inform etiological models of development and allow intervention and prevention strategies to be implemented more effectively. The error-related negativity (ERN), a negative deflection in the event-related potential at fronto-central sites approximately 50 ms following the commission of errors, has been consistently found to be larger among anxious adults. The current study sought to extend these findings to even younger individuals: the ERN was elicited by a Go/NoGo task in 48 six year-old children with a clinical anxiety disorder assessed by diagnostic interview and 48 age-matched controls. In addition to child anxiety disorder, the ERN was examined in relation to maternal history of anxiety disorder, which was previously related to a *smaller* ERN. Anxious children were characterized by a larger (i.e., more negative) ERN and maternal history of anxiety disorder was associated with a smaller ERN. Thus, the relationship between an increased ERN and clinical anxiety is evident by age 6, and this effect appears independent from an opposing influence of maternal anxiety history on the ERN. These findings support the ERN as a promising neurobehavioral marker of anxiety, and implications are discussed.

Anxiety disorders are the most frequently diagnosed form of psychopathology in children and adolescents (Beesdo et al. 2009), and adult anxiety disorders most commonly begin in childhood (Kessler et al. 2005; Last et al. 1996; Beesdo 2010). Prospective-longitudinal studies in youth suggest anxiety disorders are moderately stable over time and predict anxiety and depressive disorders in adolescence and adulthood (Bittner et al. 2007; Pine et al. 1998; Wittchen et al. 2000). For example, one study found that 73% of children diagnosed with a specific phobia met criteria for an anxiety or depressive disorder 10 years later (Emmelkamp and Wittchen 2009). Although specific developmental pathways are not yet fully understood, these studies suggest that anxiety disorders follow developmental trajectories that begin early in life and often result in chronic impairment (Pine 2007). Therefore, it may be important to identify early biomarkers that correspond to normative

[*]Corresponding Author: Department of Psychology, Stony Brook University, Stony Brook, New York, ammeyer3@gmail.com, (631) 632-7843.

versus anxious trajectories of development (Casey et al. 2005). Identifying neural correlates of anxiety that appear relatively early in life may aid in the prediction of subsequent outcomes, and allow intervention and prevention strategies to be implemented more effectively. One promising approach is to examine neural measures in young children that have previously been linked to anxiety disorders in adults (Pine 2007).

A substantial amount of research in adult anxiety disorders has focused on an event-related potential (ERP) index of error processing. Specifically, the error-related negativity (ERN) is a negative deflection occurring at fronto-central sites approximately 50 ms after the commission of an error (Falkenstein et al. 1991; Gehring et al. 1993) and has been hypothesized to reflect the activation of a generic error detection system that becomes active across a range of response and stimulus modalities (Holroyd et al. 1998; Nieuwenhuis et al. 2001). Variation in the ERN is thought to reflect individual differences in performance monitoring and defensive reactivity following mistakes (Hajcak 2012). For example, the ERN is increased in individuals with obsessive-compulsive disorder (OCD) (Endrass et al. 2008; Gehring et al. 2000; Hajcak et al. 2008; Xiao et al. 2011) and generalized anxiety disorder (Weinberg et al. 2010; Weinberg et al. 2012). An enhanced ERN has also been associated with personality traits that characterize anxiety, such as worry (Hajcak et al. 2003), behavioral inhibition (Amodio et al. 2008), high negative affect (Hajcak et al. 2004), and punishment sensitivity (Boksem et al. 2006). Building on these findings, the ERN has been proposed as a neurobehavioral trait (Hajcak 2012) that may be useful in identifying developmental risk trajectories associated with anxiety disorders (Meyer et al. 2012b).

Studies that combine ERP and fMRI (Debener et al. 2005) as well as source localization studies (Dehaene et al. 1994; Mathalon et al. 2003) suggest that the ERN is generated in the anterior cingulate cortex (ACC). Consistent with previous findings on the relationship of the ERN and anxiety, fMRI studies also indicate that anxious individuals have increased error-related ACC activity (Fitzgerald et al. 2005; Paulus et al. 2002; Ursu et al. 2003). Developmental studies suggest that activation of the ACC increases over development (Adleman et al. 2002; Van Bogaert et al. 1998) and that the structure of the ACC matures into early adulthood (Cunningham et al. 2002). Consistent with the increase of volume and activation of the ACC, the magnitude of the ERN has also been shown to increase across development (Davies et al. 2004), reaching adult-like levels in the late teen years. Although the magnitude of the ERN increases with development, it can be reliably elicited in children as young as 4 to 7 years of age (Wiersema et al. 2007; Brooker et al. 2011). In general, the morphology and distribution of the ERN in children is similar to that of adults (Arbel and Donchin 2011).

Because the ERN can be elicited in children and has been identified as a neural correlate of anxiety disorders in adults, it may be fruitful to examine the relationship between anxiety and the ERN in young children. Indeed, existing studies have found an increased ERN within a heterogeneous group of clinically anxious children (Ladouceur et al. 2006), children with obsessive-compulsive disorder (Hajcak et al. 2008; Hanna et al. 2012; Carrasco et al. 2013), children with non-clinical symptoms of obsessive-compulsive disorder (Santesso et al. 2006), adolescents with non-clinical anxiety symptoms (Meyer et al. 2012b), and early behavioral inhibition (McDermott et al. 2009).

If the ERN might be used as a neural correlate of anxious clinical trajectories, it is important to investigate the relationship between the ERN and clinical anxiety disorders in young children as early in development as possible. Thus far, the mean ages of children in clinical studies on ERN and anxiety were 13.3 (Hajcak et al. 2008), 13.9 (Carrasco et al. 2013), 11.42 (Ladouceur et al. 2006), and 13.9 (Hanna et al. 2012) years old. Although some participants in these studies were as young as 8 years old, we sought to extend this work to a homogenously aged and younger sample.

In the current study, we sought to extend previous findings by examining the ERN in young children with clinical levels of anxiety as assessed by diagnostic interviews. ERPs were recorded while 96 children (48 diagnosed with an anxiety disorder and 48 age-matched controls) aged 5 – 7 years performed a simple Go/No-Go task. Given that an increased ERN has previously been associated with clinical anxiety in older children, we hypothesized that we would observe a similar pattern in young children. We also sought to examine how maternal history of anxiety disorder related to the ERN among anxious and non-anxious children. In a previous study, we found that a *smaller* ERN was related to maternal history of an anxiety disorder – results that appear opposite to what one would predict based on extant work on anxiety and the ERN (Torpey et al. 2013). We tentatively hypothesized that maternal history of anxiety and child clinical anxiety would have independent and opposing influences on the ERN.

Method

Participant recruitment

The original sample included 413 children (Torpey et al. 2011). The current study included a subset of the larger study: 96 children, 48 of whom were chosen because they met criteria for an anxiety disorder and 48 of whom were chosen randomly because they did not meet criteria for an anxiety disorder. Originally, potential participants were identified through a commercial mailing list. An initial assessment was completed when the children were approximately 3 years of age; children then returned to the lab three years later around 6 years of age and completed EEG tasks as well as a diagnostic interview, the Preschool Age Psychiatric Assessment, Version 1.4 (Egger et al. 1999).

Diagnostic Interviewing

The Preschool Age Psychiatric Assessment (PAPA) is designed to assess a range of disorders from the Diagnostic and Statistical Manual of Mental Disorders in young children (Egger et al. 1999). DSM-IV diagnoses were derived using algorithms made by the developers of the instrument. Anxiety disorders included specific phobia, separation anxiety disorder, social phobia, generalized anxiety disorder, obsessive-compulsive disorder, and agoraphobia. Panic disorder was assessed, but no participants met criteria. Post-traumatic stress disorder was not assessed due to its rarity at this age. Symptoms occurring within the 3 months prior to the interview are rated to maximize recall. For information on the interview's psychometric properties, see Egger et al. 2006.

Interviews were conducted by M.A. level psychologists with extensive experience with structured diagnostic interviews and training on the PAPA. Interviews were conducted face-to-face with parents. A second diagnostician (an advanced graduate student trained on the interview) rated audiotapes of 35 interviews, oversampling participants with psychopathology. Kappas were: any anxiety disorder (.89), separation anxiety (1.00), specific phobia (.79), agoraphobia (1.00) (neither rater diagnosed other anxiety disorders in this subsample).

Follow-up analyses were conducted to examine the impact of maternal history of anxiety on the ERN and its relationship with child anxiety disorder. Maternal anxiety was assessed by the Structured Clinical Interview for DSM-IV, non-patient version (First et al. 1995) during the age 3 assessment. Interviews were conducted by M.A. level psychologists by telephone. For more information on this interview and diagnoses in the overall sample, see: Torpey et al., 2013. Based on audiotapes of 30 assessments, interrater reliabilities for presence/absence of a lifetime depressive or anxiety disorder were .93 and .91, respectively. In this subset, 41 mothers (43%) had a lifetime history of any anxiety disorder.

Participants

A total of 96 children, 48 (16 female) of whom met diagnostic criteria for an anxiety disorder (ANX) participated in the study along with 48 randomly chosen age-matched controls (CON; 24 female) who did not meet criteria for any anxiety disorder. The mean age of the ANX group was 6.11 (SD = .41) and the mean age of the CON group was 6.06 (SD = .39). Overall 95.8% of the children were Caucasian, 2.1% Asian, 5.2% Hispanic, and 3.1% as other. Among the group of children with an anxiety disorder, 14 met criteria for separation anxiety disorder, 6 for social phobia, 7 for agoraphobia, 22 for specific phobia, 2 for obsessive-compulsive disorder, and 5 for generalized anxiety disorder, with 9 children having more than one anxiety disorder. Within the ANX group, 7 children also met criteria for comorbid major depressive disorder (MDD), 2 for attention deficit/hyperactivity disorder (ADHD), and 6 for oppositional defiant disorder (ODD). In the CON group, 4 of the children met criteria for a MDD, 2 for ADHD, and 2 for ODD. In the ANX group, 24 childre n had mothers with a history of an anxiety disorder and in the CON group, 17 children had mothers with a history of an anxiety disorder. The groups did not differ in maternal history of anxiety, $t(94) = 1.45$, $p = .15$. After a description of the study to the parents and children, written informed consent was obtained and all procedures were approved by the University Institutional Review Board.

EEG Task and materials

A Go/NoGo task, previously described in Torpey et al., 2011 was administered using Presentation software (Neurobehavioral Systems, Inc.). The stimuli consisted of green equilateral triangles in four different orientations. In 60% of the trials, triangles were vertically aligned and point up, 20% were vertically aligned and pointed down, 10% were tilted slightly to the left, and 10% were titled slightly to the right. Children were told to respond to upward-pointing triangles by pressing a button, and to withhold a response to all other triangles.

Psychophysiological Recording

The Active Two system, Biosemi, Amsterdam, Netherlands, was used to acquire data. Thirty-two Ag/AgCl-tipped electrodes arranged according to the American Electroencephalographic Society labeling system (1994) were used with a small amount of electrolyte, Signa Gel; Bio-Medical Instruments Inc., Warren, Michigan, applied to the child's scalp at each electrode position.

Data processing was performed offline with Brain Vision Analyzer, Brain Products, Gilching, Germany. EEG data was re-referenced to the nose, and high and low pass filtered at .1 Hz and 30 Hz, respectively. Segments (i.e., 1500 ms) were extracted from the continuous EEG, beginning 500 ms prior to correct and incorrect responses. ERP data were corrected for eye-movements and blinks using the method developed by Gratton, Coles, and Donchin (Gratton et al. 1983). Artifacts were rejected if any of the following criteria were met: a voltage step of more than 50 microvolts between data points, a voltage difference of 300 microvolts within a single trial, or a voltage difference of less than .5 microvolts within 100 ms intervals. Data were then visually inspected for remaining artifacts. ERP averages were created for each trial type (error and correct) and were baseline corrected by subtracting the average activity the -500 to -300 ms window prior to the response from each data point.

ERP and behavioral results in the full sample have been reported previously (Torpey et al. 2011). In the current study, error-related negativity (ERN) and correct-related negativity (CRN) were scored as the average voltage in the window from 0 ms to 100 ms after the response, at Cz where error-related brain activity was maximal. The ERN can be calculated by averaging the error-trial waveform or by subtracting correct from error (i.e., ΔERN) (Pailing et al. 2002). The ERN on error trials likely includes processes common to error and correct responses. By subtracting correct trial activity from error trials (i.e., the ΔERN), processes common to both correct and error responses are removed, resulting in a measure of neural activity specific to errors. All analyses examined the CRN, ERN, and ΔERN.

Behavioral measures included the number of errors of commission and omission, as well as average reaction times (RTs) on error and correct trials. RTs on correct trials that followed error trials were calculated to evaluate post-error RT slowing. Trials were removed from all analyses if reaction times were faster than 200 ms or slower than 1,300 ms.

All statistical analyses were conducted using SPSS (Version 17.0) General Linear Model Software, with Greenhouse-Geisser correction applied to p values with multiple-df, repeated-measures comparisons when necessitated by violation of the assumption of sphericity. Repeated measures ANOVAs were conducted with response type (i.e., error or correct) as the within-subject variable and diagnostic group (ANX or CON) as the between-subject variable. A follow-up analysis was conducted in which maternal history of anxiety was added as a between-subject variable.

Results

Behavioral data

Accuracy and RT data are presented in Table 1 for the ANX and CON groups. Reaction time varied significantly as a function of Trial Type, $F(1, 94) = 2971.24$, $p < .001$, such that children were faster on error trials, $M = 516.59$, $SD = 95.70$, than correct trials, $M = 628.30$, $SD = 81.16$. The ANX and CON groups did not differ in RT, $F(1, 94) = .51$, $p = .48$, nor did the effect of Trial Type vary by diagnostic status, $F(1, 94) = 2.51$, $p = .12$. Also, the ANX and CON groups made comparable numbers of errors, $t(1, 94) = -.28$, $p = .78$, and had comparable accuracy, $t(1, 94) = .18$, $p = .86$.

Post-error RT data are also presented in Table 1. Overall, children were slower on Go trials that occurred after an error than the average of all Go trials, $F(1, 94) = 6.44$, $p < .05$, but this effect did not vary by group, $F(1, 94) = .35$, $p = .56$.

Error-related brain activity

Figure 1 presents topographic maps for the ANX (right) and CON (left) groups, depicting voltage differences (in μV) across the scalp for ΔERN, from 0 – 100 ms after the response. Grand average response-locked ERPs at Cz are also presented in Figure 1. Average ERN, CRN, and ΔERN values are presented for the CON and ANX groups in Table 1. Overall, the ERP response was more negative following errors than correct responses, $F(1, 94) = 92.19$, $p < .001$; $\eta_p^2 = .50$. There was no overall difference between the ANX and CON groups, $F(1, 94) = .02$, $p = .89$. However, confirming the impression from Figure 1, the difference between the ERN and CRN (i.e., ΔERN) was larger in the ANX group compared to the CON group, $F(1, 94) = 4.97$, $p < .05$; $\eta_p^2 = .05$.[1] There were no group-related differences in ERN, $F(1, 95) = 1.41$, $p = 24$, or CRN, $F(1, 95) = 2.64$, $p = .12$, alone.[2]

Follow-up analysis with maternal anxiety

In the larger group of children from which this sample was drawn, maternal anxiety was previously found to relate to a *smaller* ERN in children (Torpey et al. 2013). To further explore the effects of child anxiety and maternal anxiety history on children's ERN, we performed a follow-up repeated measures ANOVA with response type (i.e., error or correct) as the within-subject variable and diagnostic group (ANX or CON) and maternal anxiety history as between-subject variables. Results suggest that while the three-way interaction between response, diagnostic group, and maternal anxiety was not significant, $F(1, 92) = .04$, $p = .85$, both maternal and child anxiety had significant unique associations with the ΔERN: the ΔERN was larger in the ANX group, $F(1, 92) = 6.47$, $p < .05$; $\eta_p^2 = ..07$, and confirming the impression from Figure 2, significantly *smaller* among children who had

[1] A follow-up Repeated-measures ANOVA suggested that the ERN was larger among children with specific phobia compared to controls, at a trend level, $F(1, 66) = 3.21$, $p = .08$. No other diagnostic group was large enough to perform similar analyses.

[2] The error positivity (Pe) was evaluated on error trials as the average activity at a pooling of Cz, CP1, CP2, and Pz from 200 to 500 ms following response onset. A comparable time window was also evaluated at the same sites on correct trials. Overall, there was a main effect of response type (error vs. correct), $F(1, 94) = 76.35$, $p < .001$, such that incorrect responses, $M = 13.89$, SD = 14.08, were significantly more positive than correct responses, $M = 3.04$, $SD = 4.36$. Overall electrocortical responses in this time window did not vary by group, $F(1, 94) = .25$, $p = .25$. Statistical analyses did not reveal a significant difference between the ANX and CON groups, $F(1, 94) = .65$, $p = .42$.

mothers with a history of anxiety disorder, $F(1, 92) = 4.47$, $p < .05$; $\eta_p^2 = ..05$.[3] A hierarchical multiple regression analyses suggested the variance in the ΔERN accounted for by child anxiety disorder (ANX or CON) alone was 5.0% and after maternal anxiety was added, the variance accounted for increased to a total of 9.4%, a significant increment. As depicted in Figure 3, these results suggest an additive but opposite effect of child anxiety disorder and maternal history of anxiety on children's error-related brain activity.

Discussion

Consistent with our hypothesis and previous research on pediatric anxiety disorders (Hajcak et al. 2008; Ladouceur et al. 2006; Hanna et al. 2012), 6 year-old children with an anxiety disorder were characterized by increased error-related brain activity. Though error-related brain activity was enhanced in the ANX group compared to the CON group, there were no observable behavioral differences, suggesting that increased error processing in the ANX group did not simply reflect differences in accuracy or speed. Considering that the relationship between ERN and clinical anxiety had not yet been characterized in children this uniformly young, and the potential utility of identifying early neurobehavioral biomarkers, this is a novel and important extension of previous work.

These findings are consistent with previous studies suggesting that pediatric anxiety disorders may be related to altered maturational patterns in ACC circuitry (Ladouceur et al. 2006) and greater ACC activation in response to fearful faces (McClure et al. 2007). Given that previous studies suggest the ERN does not reach adult-like magnitude until late adolescence (Davies et al. 2004), it is noteworthy that anxious children as young as 6 years of age have increased ERNs. It is possible that clinically anxious children begin to display adult-like error processing and ACC activity early in the course of illness.

Whereas clinical anxiety was associated with a larger ERN, maternal history of anxiety disorder was associated with a *smaller* ERN in children in both the ANX and CON groups. These opposing effects were additive and did not interact, suggesting two distinct underlying mechanisms. This association of maternal anxiety and an attenuated ERN in children is surprising, given that maternal anxiety is associated with risk for anxiety in children (Last et al. 1991). However, maternal anxiety has also been associated with other psychopathology in children (substance abuse, externalizing disorders, depression, etc.) (Van den Bergh and Marcoen 2004; Van den Bergh et al. 2005; Bijl et al. 2002; Beidel and Turner 1997; Merikangas et al. 1998), some of which have been associated with smaller ERNs (Potts et al. 2006; Hall et al. 2007). Another tentative possibility is that mothers with past or current anxiety are characterized by distinct parenting styles or altered family functioning, which may in turn relate to an attenuated ERN in children.

We previously conducted a study examining the relationship of anxiety symptoms and ERN in a non-clinical sample ages 8-13, finding that a larger ERN was related to anxiety symptoms, but only among older children (11 – 13 year-olds) (Meyer et al. 2012b). Previous

[3]Using children randomly chosen with no diagnoses as a control group, we performed the same analysis. Results were consistent with those reported above, the ΔERN was larger in the ANX group, $F(1, 92) = 7.32$, $p < .01$, and smaller among children with a history of maternal anxiety, $F(1, 92) = 4.74$, $p < .05$.

work on normative anxiety in children suggests that levels of self-conscious shyness and worry about behavioral competence and social evaluation increases across adolescence (Crozier and Burnham 1990; Spence et al. 2001; Vasey et al. 1994). It is possible that younger children with normative levels of anxiety (or maternal history of anxiety) are characterized more by anxious arousal (i.e., fear) and concern with external threat, whereas younger children with clinical levels of anxiety have already begun to monitor more for behavioral competence and are characterized by anxious apprehension (i.e., worry) and adult like error-monitoring. This fits with previous work suggesting that the ERN relates more to anxious apprehension than anxious arousal (Weinberg et al. 2010; Weinberg and Hajcak 2011a; Moser et al. 2012). It is also possible that at a young age, risk status is associated with a reduced ERN and that the ERN may increase along with the development of an anxiety disorder. Prospective studies are needed to explore this; to determine if an increased in ERN is a proximal predictor of anxiety disorder or escalates in concert with the emergence of an anxiety disorder.

Recently, it has been proposed that the ERN may be a useful neurobehavioral risk marker to identify early trajectories of risk associated with anxiety disorders (Hajcak 2012). The ERN has been shown to be larger in pediatric patients with OCD both before and after successful cognitive-behavioral therapy (Hajcak et al. 2008). Additionally, ERN amplitudes are increased in unaffected first-degree relatives of OCD patients (Riesel et al. 2011; Carrasco et al. 2013), significantly heritable (40-60%) (Anokhin et al. 2008), and associated with specific genetic polymorphisms (Meyer et al. 2012a). Furthermore, the ERN has been shown to have excellent test-retest reliability over the course of up to 2 years (Weinberg and Hajcak 2011b). Thus, variability in the ERN is trait-like, and a promising endophenotype for anxiety disorders. In this context, the current findings are important in that they demonstrate the ERN may be able to be utilized earlier in development than previously assumed and suggest different mechanisms relating child anxiety and maternal history of anxiety to abnormal ERN. This is especially important given the chronic and impairing nature of most anxiety disorders.

One limitation to the current study is that the group of children with anxiety disorders was heterogeneous, including children with specific phobia, separation anxiety disorder, social phobia, generalized anxiety disorder, and agoraphobia. Other than specific phobia, the number of children with any single anxiety disorder was small, precluding the ability to examine whether the ERN varied across anxiety disorders. Considering that anxiety disorders differ in underlying neural mechanisms (Etkin and Wager 2007), it is possible that enhanced error processing is associated with some, but not all of these anxiety disorders in youth. Furthermore, it is possible that these children will follow dissimilar developmental pathways, developing psychological disorders that differ from those originally assessed. We are currently following these children longitudinally to investigate this possibility. Further, enhanced or reduced error processing may be associated with other clinical disorders in youth and future research should explore these possibilities with larger clinical groups of young children.

Another notable limitation of the current study is that clinical diagnoses were derived from parent report only. Given that parent and child reports often differ (Achenbach et al. 1987;

Bird et al. 1992; Van Der Toorn et al. 2010) and the possibility that parental report of symptoms may be related to a particular type of parenting which could affect error-processing in children, it is important that future studies investigate the relationship of ERN and child-reported anxiety symptoms as well as parenting style. However, it is also important to note that we would not expect parental reporting bias to account for our findings, given that maternal history of anxiety and child anxiety disorder related to the ERN in opposite directions. Additionally, future studies should examine paternal history of psychopathology in relation to ERN in offspring.

Future studies should also utilize longitudinal designs to explore the relationship of the ERN and clinical anxiety over development in the same individuals. One intriguing possibility is that ERN at an early age may predict follow-up clinical status and functioning. Although tentative, in the context of the current findings, it may be that maternal history of anxiety combined with a *larger* ERN may together prospectively predict risk for clinical anxiety. For example, McDermott (2009) found that among children high in early behavioral inhibition, an enhanced ERN predicted increased risk for anxiety disorder in adolescence. Another area warranting further investigation is the utilization of source localization and imaging techniques (fMRI) to determine whether increased error processing associated with clinical anxiety over development is due to differences in ACC function and structure. Finally, as this is the first study to investigate ERN and clinical anxiety in children as young as 6 years old, future work is needed to replicate these findings. An exciting avenue for future work is investigating the potential that error-related brain activity may prospectively predict specific developmental trajectories of anxiety, potentially leading to improved intervention and prevention strategies.

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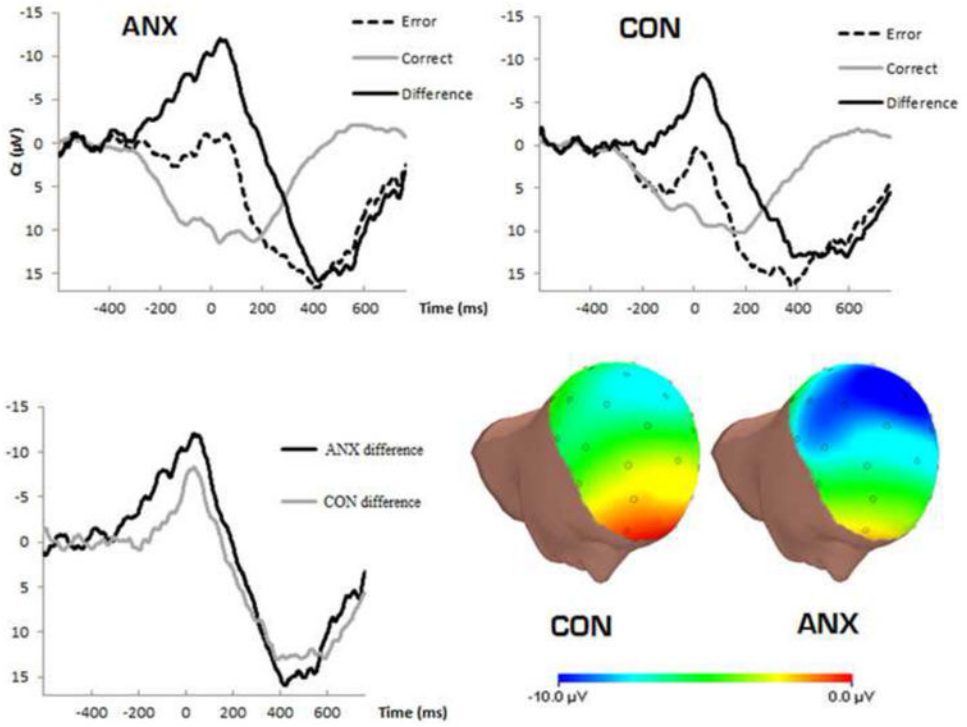

Figure 1.

On the top, response-locked ERP waveforms for correct and error trials, as well as the difference wave, for the group of children with anxiety disorders (ANX: left) and the age-matched healthy controls (CON: right). 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 – 100ms) for the ANX and CON groups.

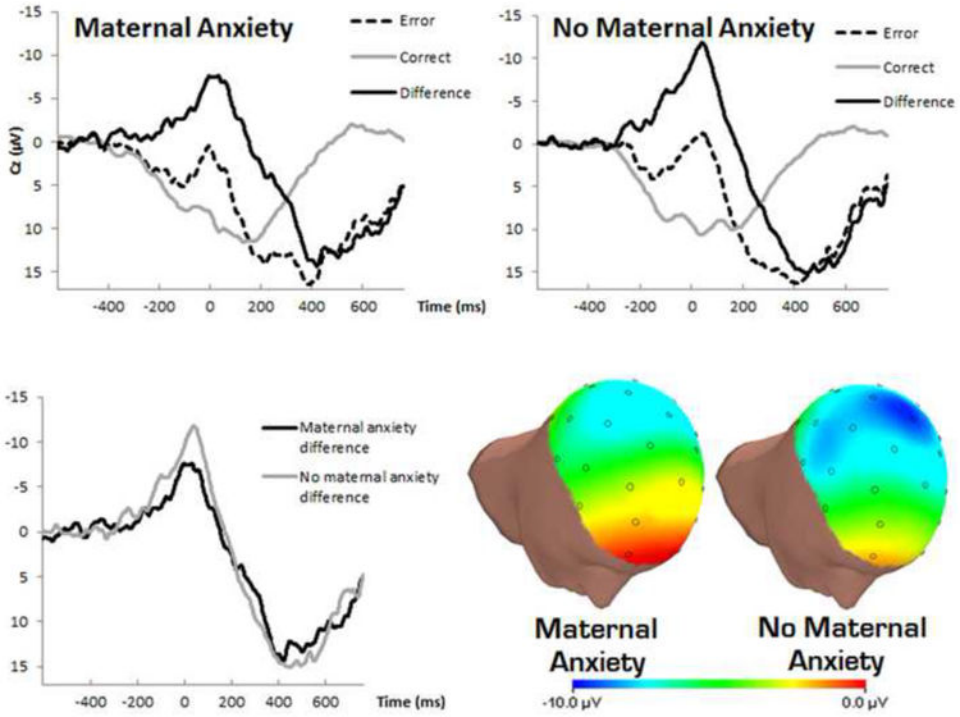

Figure 2.

On the top, response-locked ERP waveforms for correct and error trials, as well as the difference wave, for the group of children with maternal history of anxiety disorder (Maternal Anxiety: left) and the group of children without a Maternal history of anxiety disorder (No Maternal Anxiety: right). On the bottom left, response-locked ERP difference waveforms (error minus correct) for both groups. On the bottom right, topographic maps of activity (error minus correct) in the time-range of the ERN (0-100 ms) for both groups.

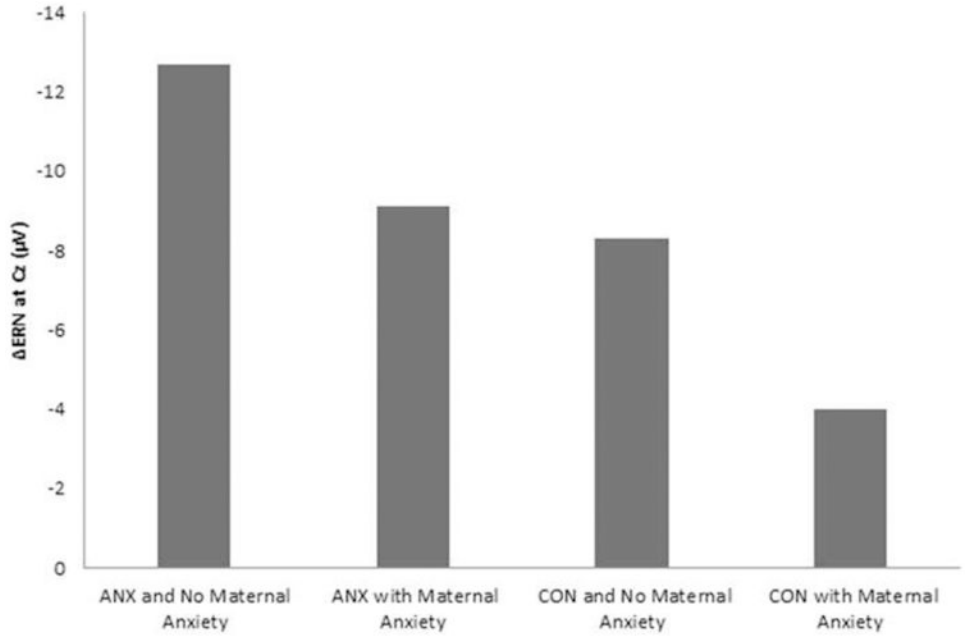

Figure 3.

Magnitude of the ΔERN (error minus correct) at Cz (0-100 ms) for children with an anxiety disorder and no maternal history of anxiety, children with an anxiety disorder and maternal history of anxiety, children without an anxiety disorder and no maternal history of anxiety, and children without an anxiety disorder and no maternal history of anxiety (left to right).

Table 1

Means and standard deviations for reaction times, accuracy, and ERP data for the ANX and CON groups.

	ANX (*N* = 48)	CON (*N* = 48)
Reaction time (ms)		
Error trials	515.67 (101.42)	517.44 (90.58)
Correct trials	617.15 (83.10)	639.68 (78.37)
Post-error trials	634.04 (119.24)	666.87 (132.99)
Accuracy		
No. of errors	26.44 (15.83)	27.28 (13.67)
No. of correct trials	211.92 (17.69)	210.62 (16.18)
% correct	86.73% (9.88)	86.39% (8.84)
ERPs (μV)		
ERN	-.57 (8.11)	1.66 (10.15)
CRN	10.33 (6.75)	8.45 (4.34)
ΔERN	-10.90 (7.70)*	-6.79 (10.17)*

*Indicates $p < .05$ for between-group comparisons.