Developmental Cognitive Neuroscience 2 (2012) 152–161

Contents lists available at SciVerse ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: http://www.elsevier.com/locate/dcn

The development of the error-related negativity (ERN) and its relationship with anxiety: Evidence from 8 to 13 year-olds

Alexandria Meyer, Anna Weinberg, Daniel N. Klein, Greg Hajcak*

Department of Psychology, Stony Brook University, Stony Brook, NY 11794-2500, USA

ARTICLE INFO

Article history:
Received 5 May 2011
Received in revised form
12 September 2011
Accepted 19 September 2011

Keywords:
ERN
Error-related negativity
Anxiety
Children
Adolescents
Development

ABSTRACT

Because anxiety disorders appear to follow developmental trajectories that begin early in development, it may be useful to examine the neurodevelopmental correlates of specific cognitive processes that have been linked to anxiety. For instance, the error-related negativity (ERN) is a negative deflection in the event-related potential that is maximal approximately 50 ms following the commission of errors at fronto-central electrode sites, and has consistently been found to be more negative among anxious adults. Much less, however, is known about anxiety and the ERN in children—especially when this relationship develops. We recorded event-related potentials (ERPs) while 55 children aged 8–13 performed an arrow version of the flankers task. Parents and children both reported on children's anxiety. Results suggest that the relationship between the ERN and anxiety changes as a function of age. Among older children, a larger (i.e., more negative) ERN was significantly related to increased anxiety based on parent report. Although the relationship was less robust, the relationship between ERN and anxiety was opposite among younger children. These results are discussed in terms of existing work on anxiety and the ERN, and the need for longitudinal and developmental studies on the relationship between ERN and anxiety.

Clinical anxiety appears to follow a developmental pathway beginning early in life. For instance, research suggests that infants who react negatively to novel stimuli tend to become toddlers who avoid new social experiences (Fox et al., 2005). Longitudinal studies imply continuity between adolescent and adult anxiety disorders (Birmaher et al., 1997a,b) and early infant and childhood behavioral inhibition has been shown to predict the onset of clinically significant anxiety later in adolescence (Biederman et al., 2001; Chronis-Tuscano et al., 2009; Gladstone et al., 2005; McDermott et al., 2009). Indeed, certain components of anxiety themselves may follow developmental transitions, such as fearful shyness expanding to include self-conscious shyness (Crozier and Burnham, 1990). Furthermore, shyness in middle childhood has been suggested to become increasingly associated with inhibition in the presence of others (Cheek et al., 1985) and with above normal self-concern (Buss, 1980). Although specific developmental pathways are not fully understood, anxiety disorders appear to follow developmental trajectories that begin relatively early in development (Pine, 2007).

Neural and cognitive development proceeds rapidly throughout middle childhood and into adolescence, and it may be possible to identify developmental changes in neural activity that relate to normative versus anxious trajectories of development (Casey et al., 2005). In the context of this developmental cognitive neuroscience perspective, it may be fruitful to measure the neurodevelopment of specific cognitive processes that have been

* Corresponding author. Tel.: +1 631 632 6272; fax: +1 631 632 7876.
E-mail address: greg.hajcak@stonybrook.edu (G. Hajcak).

1878-9293/$ – see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.dcn.2011.09.005

linked to anxiety disorders in adulthood (Pine, 2007). This approach could help identify developmental periods when measures of neural activity previously studied in adults begin to relate to individual differences in anxiety in children.

A growing body of research in adult anxiety disorders has focused on neural correlates of error detection reflected in the event-related potential (ERP). In particular, the error-related negativity (ERN) is an increased negative deflection occurring approximately 50 ms after the commission of errors compared to correct responses in speeded reaction time tasks (Falkenstein et al., 1991; Gehring et al., 1993; Hajcak et al., 2005). The ERN reflects the activation of a generic error detection system that is evident across various stimulus and response modalities (Gehring et al., 1993; van Veen and Carter, 2002).

Consistent with the view that anxiety may be associated with hyperactive error monitoring (McDermott et al., 2009; Olvet and Hajcak, 2008), individuals with anxiety disorders are characterized by increased ERNs (Endrass et al., 2008; Gehring et al., 2000; Hajcak et al., 2007; Weinberg et al., 2010). Along similar lines, an increased ERN has been reported in relation to high trait anxiety and high levels of worry (Hajcak et al., 2003; Olvet and Hajcak, 2009a), but not to increased state levels of anxiety (Moser et al., 2005).

Based on multiple studies that utilize source localization techniques (Dehaene et al., 1994; Mathalon et al., 2003; van Veen and Carter, 2002), as well as work that combines ERP and fMRI (Debener et al., 2005), it is likely that the ERN is generated in the anterior cingulate cortex (ACC). Consistent with work on anxiety and the ERN, fMRI studies also suggest increased error-related ACC activity among anxious individuals (Fitzgerald et al., 2005; Paulus et al., 2002; Ursu et al., 2003).

One fMRI study found that adolescents with generalized anxiety disorder showed greater activation in a network including the amygdala, ventral prefrontal cortex, and ACC in response to fearful faces (McClure et al., 2007). Similar to research findings in adults, some studies have reported increased ERNs among anxious children. The ERN is larger among children with obsessive compulsive disorder (Hajcak et al., 2008), children with non-clinical symptoms of obsessive-compulsive disorder (Santesso et al., 2006), and within a heterogeneous group of clinically anxious children (Ladouceur et al., 2006). Consistent with the possibility that the ERN may relate to developmental processes of risk that emerge across development, one study found that behavioral inhibition (BI) assessed in early childhood predicted a larger ERN in adolescence (McDermott et al., 2009). Moreover, ERN moderated the relationship between BI and the development of anxiety in adolescence: disorders were most common among those children who were high in BI and had a larger ERN. These data suggest that increased error-related brain activity may help delineate anxious versus non-anxious trajectories across development. However, existing studies have focused on somewhat older children in their evaluation of the ERN and its relationship with anxiety (i.e., participants are often early adolescents), and no study to date has examined the relationship between ERN and anxiety as a function of age.

Neurodevelopmental studies suggest that the ACC matures into early adulthood (Cunningham et al., 2002) and that activation of the ACC increases over the course of development (Adleman et al., 2002; Van Bogaert et al., 1998). One neuroimaging study of 5–16 year old individuals found a significant correlation between volume of the right ACC and performance on a go/no-go task (Casey et al., 1997a,b). This same study found a significant correlation between age and volume of the right ACC, but not size of cerebrum. Consistent with these data, the ERN may not reach adult-like levels until the late teen years (Davies et al., 2004). Davies et al. (2004) found that the amplitude of the ERN increased with age in a sample of 7–25 year-olds, with a significant age by gender interaction, which they suggested might reflect associations between pubertal onset and increases in the ERN. Another study found that ERN amplitude related to performance measures on a flankers task in adults but not in adolescents, suggesting that the relationship between ERN and behavioral measures emerges developmentally (Ladouceur et al., 2007). However, the ERN *can* be elicited among much younger children: one study found a robust ERN in children as young as 5–7 years old (Torpey et al., 2009). In light of maturational changes that impact both the ACC and ERN, it may be important to examine the developmental relationship between ERN and anxiety as children transition from middle childhood to early adolescence—especially because this period marks a transition into a higher-risk period for anxiety and mood disorders (Costello et al., 2005).

In addition to the ERN, the error positivity (Pe) is another component associated with response monitoring. The Pe appears within 200–500 ms following an error response, and appears to be independent of the ERN (Falkenstein et al., 2000; Overbeek et al., 2005; Santesso et al., 2006). There is evidence that the Pe is affected by awareness of errors (Nieuwenhuis et al., 2001), and may reflect a P300-like orienting response to errors (Ridderinkhof et al., 2009). The Pe has not been consistently associated with anxiety disorders in adults or children, however (Endrass et al., 2008; Hajcak et al., 2008; Ladouceur et al., 2006; McDermott et al., 2009; Ruchsow et al., 2005). Additionally, studies have suggested that the Pe is more invariant across development than the ERN, with Pe amplitudes in childhood matching those of adults (Davies et al., 2004; Wiersema et al., 2007).

In the current study, ERPs were recorded while 55 children aged 8–13 performed an arrow version of the flankers task (Eriksen and Eriksen, 1974). Both children and their parents reported on children's anxiety so that the relationship between anxiety and the ERN and Pe could be examined, as well as the impact of age on the association between anxiety and ERP measures. Based on previous work, we predicted that the ERN would increase with age in this sample, and that the relationship between ERN and anxiety would be moderated by age, such that the correlation between ERN and anxiety would be larger among older children. We did not expect the Pe to vary with age; in light of inconsistent findings on the Pe, we had no *a priori* hypotheses regarding its relationship with anxiety, or the moderating role of age on this association.

1. Method

1.1. Participant recruitment and screening

Subsequent to approval by the Stony Brook University Institutional Review Board, participants were recruited via a commercial mailing list targeting families with children between 8 and 13 years of age in Stony Brook and the surrounding community. Letters, followed by phone calls, went out to approximately 800 families from the mailing list. A total of 70 participants (30 female) between the ages of 8 and 13 participated in the study. Four participants were excluded from analysis due to poor quality recordings. Additionally, participants who committed errors on more than 25% of trials (i.e., 85 or more errors) and participants who committed fewer than 6 errors (Olvet and Hajcak, 2009b) were excluded from the final sample (11 subjects excluded in total). The final sample consisted of 55 participants (24 female). Behavioral data for one participant was lost as a result of experimenter error; therefore, behavioral results are based on 54 subjects (24 female). Assent was obtained from child participants and informed consent was obtained from their parent prior to the experiment. Participants received $45.00 for their participation in the study.

The participants ranged from 8 to 13 years old and the mean age of the final sample was 10.95 (SD = 1.48); 89.1% of the sample was Caucasian, 1.8% was African-American, 1.8% was Asian, and 7.3% identified as Other.

1.2. Self-report

Two versions of the Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher et al., 1997a,b) were administered: one to the participants (Child-SCARED), and one to the parent who accompanied the child to the laboratory (Parent-SCARED). Both versions of the SCARED broadly assess symptoms of anxiety as they manifest in children, including symptoms of panic, general anxiety, separation anxiety, social phobia, and school phobia (Birmaher et al., 1997a,b). Each version consisted of a 38-item scale on which the participant can answer between 0 ('not true or hardly ever true') to 2 ('true or often true'); 1 corresponded to 'sometimes true'. The maximum score for each version is 76. Both versions also included 5 subscale scores: Panic/Somatic, General Anxiety, Separation Anxiety, Social Phobia, and School Phobia. Children filled out questionnaires either immediately before or after the EEG session; parents filled out measures during their child's EEG session.

1.3. Task and materials

An arrow version of the flanker task (Eriksen and Eriksen, 1974) was administered on a Pentium D class computer, using Presentation software (Neurobehavioral Systems, Inc., Albany, CA, USA) to control the presentation and timing of all stimuli. Each stimulus was displayed on a 19 in (48.3 cm) monitor. On each trial, five horizontally aligned arrowheads were presented. Half of all trials were compatible ("<<<<<" or ">>>>>") and half were incompatible ("<<><<" or ">><>>"); the order of compatible and incompatible trials was random. Each set of arrowheads occupied approximately 1.3° of visual angle vertically and 9.2° horizontally. All stimuli were presented for 200 ms followed by an ITI that varied randomly from 2300 to 2800 ms.

1.4. Procedure

After a brief description of the experiment, EEG electrodes were attached and the subject was given detailed task instructions. All participants performed multiple tasks during the experiment. The order of the tasks was counterbalanced across subjects and the results of other tasks will be reported elsewhere (see, e.g., Bress et al., in press). Participants were seated at a viewing distance of approximately 24 in (61 cm) and were instructed to press the right mouse button if the center arrow was facing to the right and to press the left mouse button if the center arrow was facing to the left. Participants performed a practice block containing 30 trials during which they were instructed to be both as accurate and fast as possible. The actual task consisted of 11 blocks of 30 trials (330 trials total) with each block initiated by the participant. To encourage both fast and accurate responding, participants received feedback based on their performance at the end of each block. If performance was 75% correct or lower, the message "Please try to be more accurate" was displayed; performance above 90% correct was followed by "Please try to respond faster"; otherwise, the message "You're doing a great job" was displayed.

1.5. Psychophysiological recording, data reduction and analysis

Continuous EEG recordings were collected using an elastic cap and the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands). Thirty-four electrode sites were used, based on the 10/20 system, as well as two electrodes on the right and left mastoids. Electrooculogram (EOG) generated from eye movements and eyeblinks was recorded using four facial electrodes: horizontal eye movements were measured via two electrodes located approximately 1 cm outside the outer edge of the right and left eyes. Vertical eye movements and blinks were measured via two electrodes placed approximately 1 cm above and below the right eye. The EEG signal was pre-amplified at the electrode to improve the signal-to-noise ratio and amplified with a gain of one by a BioSemi ActiveTwo system (BioSemi, Amsterdam). The data were digitized at 24 bit resolution with a sampling rate of 1024 Hz using a low-pass fifth order sinc filter with a half-power cutoff of 204.8 Hz. Each active electrode was measured online with respect to a common mode sense (CMS) active electrode producing a monopolar (non-differential) channel. Offline, all data were referenced to the average of the left and right mastoids, and band-pass filtered between 0.1 and 30 Hz; eye-blink and ocular corrections were conducted per Gratton et al. (1983).

A semi-automatic procedure was employed to detect and reject artifacts. The criteria applied were a voltage step of more than 50.0 μV between sample points, a voltage difference of 300.0 μV within a trial, and a maximum

voltage difference of less than .50 μV within 100 ms intervals. These intervals were rejected from individual channels in each trial. Visual inspection of the data was then conducted to detect and reject any remaining artifacts.

The EEG was segmented for each trial beginning 300 ms before response onset and continuing for 1300 ms (i.e., 1000 ms following the response); a 200 ms window from −300 to −100 ms before the response onset served as the baseline. Correct and error trials were averaged separately. For each subject, the most negative peak in a time window from 50 ms prior to response onset to 100 ms following the response was detected on error trials; the ERN was then quantified as the average activity in the 50 ms around this peak (i.e., 25 ms on either side of the peak) on error trials at a pooling of fronto-central sites (Fz, FCz, Cz, FC1, and FC2) where error-related brain activity was maximal. In addition, the correct response negativity (CRN) was evaluated in the same time window and pooling of electrodes on correct trials. Finally, 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 400 ms following response onset. A comparable time window was also evaluated at the same sites on correct trials.

Behavioral measures included both the number of error trials for each subject, as well as accuracy expressed as a percentage of all valid trials. Average reaction times (RTs) on error and correct trials were also calculated separately, as were RTs on correct trials that followed correct and errors trials to evaluate post-error RT slowing. Trials were removed from all analyses if reaction times were faster than 200 ms or slower than 1300 ms.

In examinations of the full sample, statistical analyses were conducted using SPSS (Version 17.0) General Linear Model software, with Greenhouse–Geisser correction applied to p values associated with multiple-df, repeated-measures comparisons when necessitated by violation of the assumption of sphericity.

The Pearson correlation coefficient (r) was also used to examine associations between anxiety, behavioral and ERP measures. Because parent- and child-reported anxiety were only moderately related, $r(54) = .35$, $p < .01$, behavioral and ERP measures were related to both child- and parent-reported anxiety separately. Hierarchical regression analyses were used to examine interaction effects.

2. Results

2.1. Self-report

Overall, the mean score for the Child-SCARED was 18.00, SD = 10.17. The mean score for the Parent-SCARED was 10.00, SD = 7.14. The scores on each scale did not deviate from a normal distribution (Parent-SCARED/Child-SCARED kurtosis and skewness were .31/2.10 and .79/.89, respectively). In the Child-SCARED, the subscales were highly correlated with one another (r's ranged from .55 to .82, all significant at $p < .001$).[1] In the Parent-SCARED, the subscales were also highly correlated (r's ranged from .49 to .78, all significant at $p < .001$).[2] Additionally, children tended to skip more questions than their parents, $t(1, 54) = 2.36$, $p < .05$.

2.2. Behavioral data

Overall, participants committed an average of 40.28 (SD = 18.46) errors, and were correct on 86.17% (SD = 7.29) of trials. Age was unrelated to accuracy, $r(53) = -.17$, $p = .21$. Consistent with previous work, participants were faster on error, $M = 371$ ms, SD = 66, than correct trials, $M = 517$ ms, SD = 105; $F(1,53) = 148.60$, $p < .001$; $\eta_p^2 = .74$. Older children were characterized by faster reaction times on both error, $r(53) = -.34$, $p < .05$ and correct, $r(53) = -.56$, $p < .01$, trials. Participants were slower to generate a correct response on trials that occurred after an error, $M = 525$ ms, SD = 107, than after a correct response, $M = 479$ ms, SD = 109; $F(1,53) = 36.74$, $p < .001$; $\eta_p^2 = .41$; moreover, the degree of post-error slowing (i.e., post-error RT minus post-correct RT) *decreased* with age, $r(53) = -.30$, $p < .03$, and *increased* as a function of anxiety symptoms on the Child-SCARED, $r(53) = .36$, $p < .01$. Accuracy was not related to Parent-SCARED or Child-SCARED, $r(53) = -.10$, $p = .47$; $r(53) = -.02$, $p = .87$, respectively. Despite post-error increases in RT, accuracy was comparable after correct, $M = 87.67\%$, SD = 6.32, and incorrect responses, $M = 86.45\%$, SD = 10.87, $t(1,53) = .907$, $p > .40$. Parent's report of child anxiety was moderately correlated with post-error accuracy, such that increasing anxiety predicted worse post-error accuracy, $r(53) = .26$, $p < .06$.

Correlational analyses were conducted to examine possible speed-accuracy trade-off effects. Partial correlations (controlling for age) were conducted between the percentage of errors and incorrect RTs, $r(53) = .11$, $p < .43$, and correct RT, $r(53) = .466$, $p < .001$. These results indicate that faster RTs were associated with reduced accuracy.

Overall then, errors were faster than correct trials, and there was evidence for post-error RT slowing, although accuracy was not improved following errors. Increasing age was associated with faster RTs and reduced post-error RT slowing. Moreover, post-error RT slowing was larger and post-error mistakes were more frequent among more anxious children.

2.3. Error-related brain activity

Fig. 1 (top; right) presents a topographic map depicting voltage differences (in μV) across the scalp for error minus correct responses in the time window of the ERN. Grand average response-locked ERPs at the pooling of sites (i.e., Fz, FCz, Cz, FC1, and FC2) where the error minus correct difference was maximal are also presented in Fig. 1 (top; left). Confirming the impression from Fig. 1, the ERN was

[1] Means and standard deviations for the Child-SCARED subscales: Somatic Panic, $M = 1.29$, SD = .27, General Anxiety, $M = 1.49$, SD = .40, Separation Anxiety, $M = 1.55$, SD = .39, Social Phobia, $M = 1.95$, SD = .54, School Phobia, $M = 1.30$, SD = .34.

[2] Means and standard deviations for the Parent-SCARED subscales: Somatic Panic, $M = 1.10$, SD = .17, General Anxiety, $M = 1.40$, SD = .33, Separation Anxiety, $M = 1.25$, SD = .27, Social Phobia, $M = 1.61$, SD = .53, School Phobia, $M = 1.17$, SD = .27.

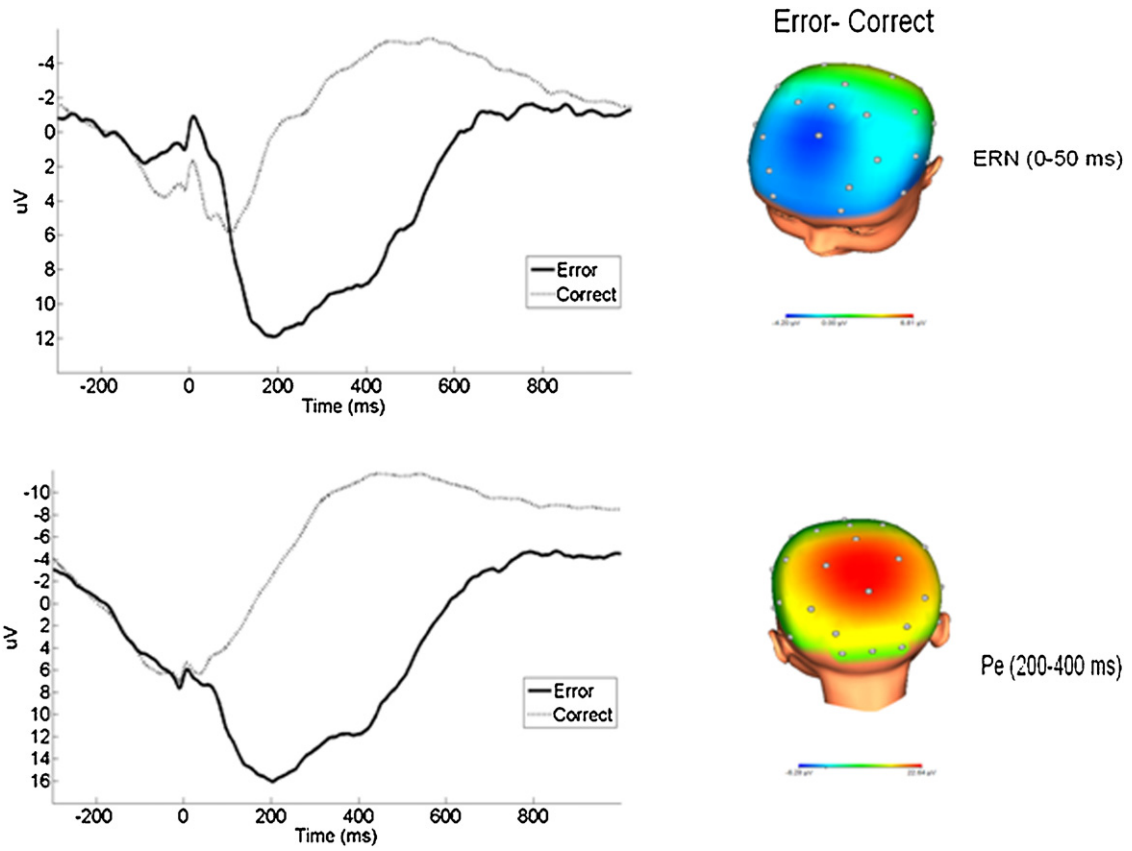

Fig. 1. Response-locked ERP waveforms at Fz, FCz, Cz, FC1, and FC2 (top left), where the ERN was maximal. Below this are response-locked ERP waveforms at Cz, CP1, CP2 and Pz (bottom left), where the Pe is maximal. To the right are topographic maps depicting differences (in μV) between error and correct responses in the time range of the ERN (0–50 ms; top) and the Pe (200–400 ms; bottom).

significantly more negative, $M = -0.90$ μV, SD = 5.69, than the CRN, $M = 2.81$ μV, SD = 4.78; $F(1,54) = 25.16$, $p < .001$; $\eta_p^2 = 32$.

A similar topographic map used to depict voltage difference (in μV) in the time window of the Pe is presented in Fig. 1 (bottom; right), along with grand average response-locked ERPs at the pooling of sites (i.e., Cz, CP1, CP2 and Pz) where the difference between error and correct trials was maximal in the time range of the Pe (bottom; left). As suggested by Fig. 1, the Pe was significantly more positive following error trials, $M = 14.18$ μV, SD = 8.18, than following correct trials, $M = 8.81$, SD = 5.37; $F(1,54) = 13.32$, $p < .001$; $\eta_p^2 = 21$. Pe and ERN were uncorrelated ($p > .65$).

2.4. Correlations between ERPs and anxiety, and the moderating effect of age

In the full sample, none of the measures of anxiety (including the subscale scores) correlated with any ERP measures. Age was unrelated to the CRN and activity in the time-range of the Pe (on both error and correct trials); however, older children were characterized by a marginally larger (i.e., more negative) ERN, $r(53) = -.24$, $p < .10$.

A series of hierarchical regression analyses were next conducted according to the procedures outlined by Aiken and West (1991) to examine the potential moderating effect of age on the relationship between anxiety and the ERN. Both child and parental report of child anxiety were included in these analyses. Age and both measures of anxiety were included in the model as independent variables, as were the two cross-products of age and each measure of anxiety (both centered). As indicated in Table 1, though child report of anxiety was not significantly associated with the magnitude of the ERN, age did have a moderating effect on the relationship between Parent-SCARED and the ERN.[3] The interaction between age and Parent-SCARED was significant, as indicated by the product term having a significant unique effect, $t(49) = 2.07$, $p < .05$, effect size (partial r) = .28. Fig. 2 (top) illustrates this pattern by showing the regression lines (based on the overall regression equation) for Parent-SCARED predicting ERN at one SD above and below the mean age of the sample. Among older children (i.e., one standard deviation above the mean age; 12.43 years), greater parental report of anxiety predicted a larger (i.e., more negative) ERN, $r = -.35$, $\beta = -.53$, $t = 2.69$, $p < .01$, whereas in the younger children (i.e., one standard deviation below the mean age; 9.47 years) the relationship

[3] The same analyses were run using the factors of the Parent and Child SCARED. Of the factors, it appears that age has a moderating effect on the relationship between Factor 5 (School Phobia) and both ERN and Pe.

Table 1
Results of hierarchical multiple regression examining the moderating effect of age on the relationship between child anxiety and the magnitude of the ERN.

Variables entered	ERN		Pe on error trials	
	β	t	β	t
Step one				
Child's age	−.25	1.79†	.22	1.57
Child self-report of anxiety	−.05	.31	−.06	.39
Parental report of child anxiety	.04	.26	.08	.55
Step two				
Child's age	−.29	2.10*	.17	1.27
Child self-report of anxiety	−.03	.21	.10	.68
Parental report of child anxiety	−.01	.04	−.10	.71
Age × Child self-report of anxiety	.03	.19	.14	.99
Age × Parental report of child anxiety	−.30	2.07*	−.31	2.13*

Note: for the ERN analyses, R^2 for step 1 = .06; R^2 for step 2 = .14; for the Pe analyses, R^2 for step 1 = .22; R^2 for step 2 = .36.
* $p < .05$.
** $p < .01$.
*** $p < .001$.
† $p < .10$.

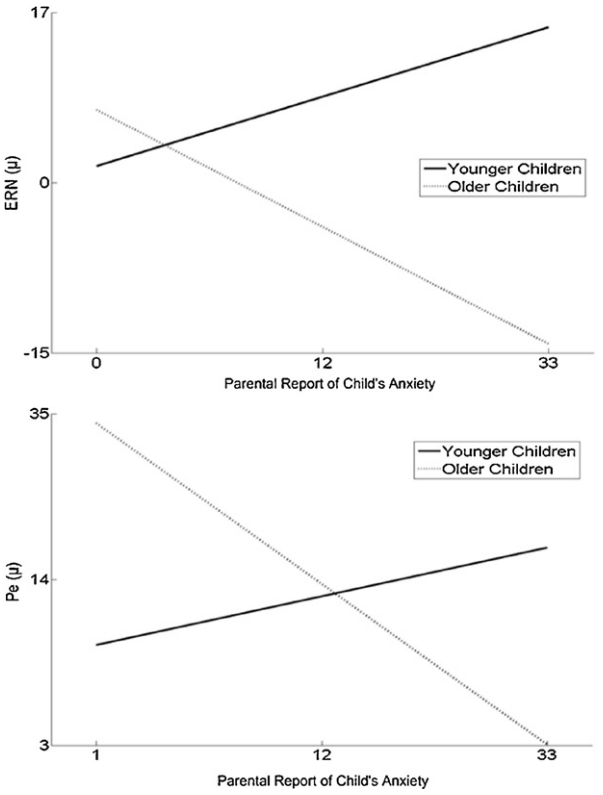

Fig. 2. The results of moderation analyses for the ERN (top) and Pe (bottom). Among older children a larger (i.e., more negative) ERN was associated with greater parental report of child anxiety (top); the opposite is true for younger children. A smaller (i.e., less positive) Pe was associated with greater parental report of child anxiety (bottom); again, this effect is reversed in younger children.

was positive and did not reach significance, $r = .23$, $\beta = .35$, $t = 1.67$, $p = .09$.[4] At the mean age (10.95 years), there was no significant relationship between the magnitude of the ERN and parental report of anxiety $r = -.06$, $\beta = -.09$, $t = .44$, $p > .25$. Analyses conducted with CRN activity did not reveal any significant relationships (all p's > .4).

For presentation purposes, a median split was conducted on the Parent-SCARED scores to create high- and low-anxiety groups among older (11–13 years old) and younger (8–10 years old) children. Fig. 3 (right) presents topographic maps depicting voltage differences (in μV) across the scalp for error minus correct responses in the time window of the ERN in each of these four groups. In addition, grand average response-locked ERPs for each group are also presented in Fig. 3 (left).

The same analyses were repeated with the Pe on error trials; as noted in Table 1, the relationship between Parent-SCARED and the Pe also appeared to be moderated by the age of the child, such that a smaller Pe was associated with greater anxiety, but only among older children, $r = -.45$, $\beta = -.65$, $t = 3.29$, $p < .01$. There was not a significant effect of anxiety on the Pe for younger children $r = .14$, $\beta = .27$, $t = 1.00$, $p > .10$; see Fig. 2, bottom. Similarly, at the mean age, there was not a significant relationship between the magnitude of the Pe and parental report of anxiety $r = -.06$, $\beta = -.19$, $t = 1.15$, $p > .10$.

3. Discussion

The results of the current study suggest that the relationship between the ERN and anxiety is moderated by age: only for older children in the current study, a larger (i.e., more negative) ERN was related to increased parent reports of child anxiety. Although the effect was not as robust in younger children, the observed relationship was opposite in direction: increasing parent-report of child anxiety was related to smaller (i.e., less negative) ERN.

Across the entire sample, ERN tended to be larger among older children at a trend level. These findings are broadly consistent with previous findings that the ERN fluctuates throughout development and begins to substantially increase around age 12 (Davies et al., 2004). Event-related fMRI data also suggests that the ACC does not have a

[4] The same pattern of results was found when congruent and incongruent trials were analyzed separately, though the Ns were reduced to 37 and 51, respectively, and the effects failed to reach significance.

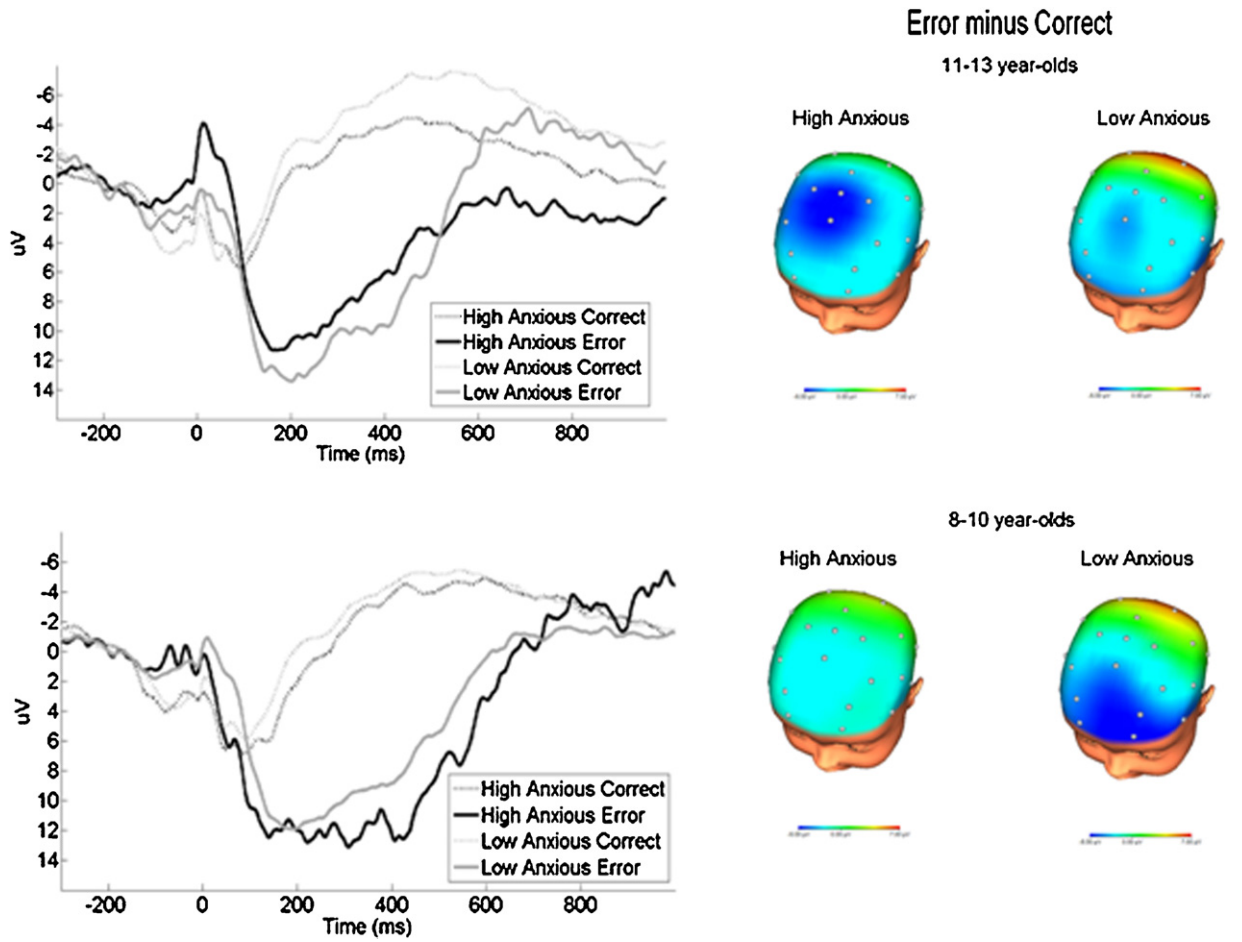

Fig. 3. Response-locked ERP waveforms for participants high and low in anxiety based on parental report, among older (top) and younger (bottom) participants separately. Response-locked ERP waveforms (left) and topographic maps of activity in the time-range of the ERN (0–50 ms; right) for each of the four groups.

mature activation pattern until late adolescence (Crone et al., 2008). Participants in the current study may have been too young overall to detect stronger robust age-related changes in the ERN that have been reported around age 13 (Segalowitz et al., 2010).

Age was robustly related to RT measures in the current study: both RT and post-error RT slowing decreased among older children. Age was unrelated to performance accuracy, suggesting that as children got older, they were faster to respond and slowed less after errors but did not commit more errors (i.e., they performed better on the task). Child anxiety was related to both increased post-error RT slowing, and worse post-error accuracy. Thus, anxious children tended to slow down more following errors, but also perform worse after making a mistake. Post-error slowing is thought to be a behavioral adjustment to improve performance (Rabbitt, 1966) that results from the recruitment of cognitive control processes (Botvinick et al., 2001). The fact that anxious children slowed more after errors but also performed worse suggests that efficiency of ACC and prefrontal network functioning following an error might be compromised among anxious children. This is consistent with the assertion of attentional control theory that anxiety is associated with decreased processing efficiency, especially under stress (Eysenck et al., 2007).

In the current study, the ERN related to anxiety among 11–13 year olds—results that are consistent with existing data (Hajcak et al., 2008; Ladouceur et al., 2006; Santesso et al., 2006) which have generally assessed somewhat older children (e.g., mean ages of 13.3, 10.2, 11.42, respectively). Because this relationship was not found among younger children, our data suggest that the relationship between increased error-related brain activity and anxiety may emerge in early adolescence. It has previously been suggested that pediatric anxiety disorders are 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 evidence indicates that the ERN does not reach adult-like levels until late adolescence (16–18 years old), it may be that there is a subset of anxious younger adolescents who begin to display adult-like ERNs and excessive error-related ACC activity. Excessive ACC activity may only begin to emerge as the ACC establishes some baseline of functional connectivity and adult-like structure.

It is also important to note that among younger children (i.e., 8–10 year olds), we found a weaker relationship between the magnitude of the ERN and anxiety that was in the *opposite* direction, such that higher child anxiety was related to a smaller (less negative) ERN. Collectively, these data indicate that the nature of the relationship between ERN and anxiety may fundamentally change across development—an unexpected finding that certainly warrants further study. One possible explanation for the changing relationship between anxiety and the ERN may be the differential development of the rostral and dorsal ACC. Specifically, one study found that the rostral ACC activity during response monitoring varied as a function of age, whereas dorsal ACC activation was only evident among adults (Velanova et al., 2008). Thus, the ratio of dorsal to rostral ACC activity during response monitoring may increase across development. Considering that some studies have related anxiety to hypoactive rostral ACC activity in particular (Adleman et al., 2002; Cunningham et al., 2002), it is possible then that anxiety is related to hypoactive rostral ACC activity in both adults and children—and to increased dorsal ACC activity with increasing developmental changes. Future studies examining the relationship of childhood and adult anxiety to rostral and dorsal ACC activity in response to errors are needed to further investigate this possibility.

Alternatively, it is possible that the observed developmental change in the relationship between ERN and anxiety could relate to the changing phenomenology of anxiety across development. In particular, it is possible that younger children are more focused on external threat, whereas adolescents begin to monitor more for internal signals of danger. Work by Crozier suggests that children around age 5 display fearful shyness that expands to include self-conscious shyness by age 10 (Crozier and Burnham, 1990). In line with this, worry about behavioral competence and social evaluation increases with age (Spence et al., 2001; Vasey and Crnic, 1994). Given the strong relationship between the ERN and generalized anxiety disorder (Weinberg et al., 2010) and pathological worry (Hajcak et al., 2003), it is possible that ERN relates to more 'cognitive' forms of anxiety such as anxious apprehension, which may develop later than anxious arousal, or fear (Nitschke et al., 2001). Along these lines then, future research might examine the relationship between dimensions of anxiety and the ERN in a developmental context. It is possible that anxious arousal (i.e., fear) in younger children is related to smaller ERNs and anxious apprehension (i.e., worry) in older children is related to larger ERNs.

It is important to note that task difficulty could have played a role in the age group differences we observed in the ERN. A previous study found that ERN amplitudes were comparable between a group of adolescents and adults during a simple task, but adults showed larger ERNs during a more complex task (Hogan et al., 2005). Future studies might relate the ERN to developmental changes utilizing multiple tasks that vary in difficulty.

Consistent with previous findings (Davies et al., 2004), we found no relationship between Pe amplitude and age. However, we did find that that age moderated the relationship between Pe and parent report of anxiety, such that among older children, higher levels of parent reported anxiety were related to a smaller Pe. This fits with evidence in some studies that adults with high negative affect or anxiety have decreased Pe amplitudes, possibly due to do reduced error awareness (Gehring et al., 2000; Hajcak et al., 2004; Hajcak and Simons, 2002). Thus, we found that in older children, the relationship between both Pe and ERN with anxiety was similar to that of adults. This is a novel finding in children and warrants further investigation.

It should also be noted that associations between anxiety and ERN/Pe in both the younger children and older children were found only when using parent report of anxiety. No significant associations were found using child reports. One possible reason for this discrepancy may be that children tended to skip more questions on the SCARED than their parents. Future work using both self-report and interview-based measures over multiple assessment points should be able to clarify whether an increased ERN/Pe is more related to parent or child reports of children's anxiety.

One other limitation that merits discussion is the possibility that other psychological conditions could have influenced the results. Specifically, ADHD has been implicated in impaired error monitoring (Crone et al., 2008; Segalowitz and Dywan, 2009). It may be useful in future studies to examine the relationship between ERN/Pe and anxiety in children who have also been evaluated for other psychological conditions, and to utilize other measures of anxiety.

Further work in larger samples is necessary to better understand the developmental trajectory of the relation between ERN/Pe and anxiety. In particular, it will be important to track the development of the ERN/Pe and its relationship to anxiety within individuals across development, using longitudinal experimental designs. An intriguing possibility is that anxious young children with smaller ERNs may develop into anxious adolescents with larger ERNs. Insofar as the ERN has been proposed as a viable endophenotype for anxiety disorders (Olvet and Hajcak, 2008), the developmental relationship between anxiety and ERN will be important to characterize using longitudinal study designs.

References

Adleman, N.E., Menon, V., Blasey, C.M., White, C.D., Warsofsky, I.S., Glover, G.H., et al., 2002. A developmental fMRI study of the stroop color-word task. NeuroImage 16 (1), 61–75.

Aiken, L.S., West, S.G., 1991. Multiple regression: Testing and interpreting interactions. Sage, Newbury Park, CA.

Biederman, J., Hirshfeld-Becker, D.R., Rosenbaum, J.F., Herot, C., Friedman, D., Snidman, N., et al., 2001. Further evidence of association between behavioral inhibition and social anxiety in children. The American Journal of Psychiatry 158 (10), 1673–1679.

Birmaher, B., Khetarpal, S., Brent, D., Cully, M., Balach, L., Kaufman, J., et al., 1997a. The screen for child anxiety related emotional disorders (SCARED): scale construction and psychometric characteristics. Journal of the American Academy of Child and Adolescent Psychiatry 36 (4), 545–553.

Birmaher, B., Khetarpal, S., Brent, D., Cully, M., Balach, L., Kaufman, J., et al., 1997b. The Screen for Child Anxiety Related Emotional Disorders (SCARED): scale construction and psychometric characteristics. Journal of the American Academy of Child & Adolescent Psychiatry 36 (4), 545–553.

Botvinick, M.M., Braver, T.S., Barch, D.M., Carter, C.S., Cohen, J.D., 2001. Conflict monitoring and cognitive control. Psychological Review 108 (3), 624–652.
Bress, J., Smith, E., Foti, D., Hajcak, G., Depressive children show reduced electrocortical activity to monetary rewards. Biological Psychology, in press.
Buss, A.H., 1980. Self-consciousness and Social Anxiety. Freeman, San Francisco.
Casey, B.J., Tottenham, N., Liston, C., Durston, S., 2005. Imaging the developing brain: what have we learned about cognitive development? Trends in Cognitive Sciences 9 (3), 104–110.
Casey, B.J., Trainor, R., Giedd, J., Vauss, Y., Vaituzis, C.K., Hamburger, S., et al., 1997a. The role of the anterior cingulate in automatic and controlled processes: a developmental neuroanatomical study. Developmental Psychobiology 30 (1), 61–69.
Casey, B.J., Trainor, R.J., Orendi, J.L., Schubert, A.B., Nystrom, L.E., Giedd, J.N., et al., 1997b. A developmental functional MRI study of prefrontal activation during performance of a Go-No-Go task. Journal of Cognitive Neuroscience 9 (6), 835 [Article].
Cheek, J.M., Carpentieri, A.M., Smith, G.M., Rierdan, J., Koff, E., 1985. Shyness: Perspectives on Theory and Research. Plenum, New York.
Chronis-Tuscano, A., Degnan, K.A., Pine, D.S., Perez-Edgar, K., Henderson, H.A., Diaz, Y., et al., 2009. Stable early maternal report of behavioral inhibition predicts lifetime social anxiety disorder in adolescence. Journal of the American Academy of Child & Adolescent Psychiatry 48 (9), 928–935.
Costello, E.J., Egger, H.L., Angold, A., 2005. The developmental epidemiology of anxiety disorders: phenomenology, prevalence, and comorbidity. Child and Adolescent Psychiatric Clinics of North America 14 (4), 631–648.
Crone, E., Zanolie, K., Van Leijenhorst, L., Westenberg, P., Rombouts, S., 2008. Neural mechanisms supporting flexible performance adjustment during development. Cognitive, Affective, & Behavioral Neuroscience 8 (2), 165–177.
Crozier, W.R., Burnham, M., 1990. Age-related differences in children's understanding of shyness. British Journal of Developmental Psychology 8 (2), 179–185 [Article].
Cunningham, M.G., Bhattacharyya, S., Benes, F.M., 2002. Amygdalocortical sprouting continues into early adulthood: implications for the development of normal and abnormal function during adolescence. The Journal of Comparative Neurology 453 (2), 116–130.
Davies, P.L., Segalowitz, S.J., Gavin, W.J., 2004. Development of response-monitoring ERPs in 7- to 25-year-olds. Developmental Neuropsychology 25 (3), 355–376.
Debener, S., Ullsperger, M., Siegel, M., Fiehler, K., von Cramon, D.Y., Engel, A.K., 2005. Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. The Journal of Neuroscience 25 (50), 11730–11737.
Dehaene, S., Posner, M.I., Don, M.T., 1994. Localization of a neural system for error detection and compensation. Psychological Science 5 (5), 303–305.
Endrass, T., Klawohn, J., Schuster, F., Kathmann, N., 2008. Overactive performance monitoring in obsessive-compulsive disorder: ERP evidence from correct and erroneous reactions. Neuropsychologia 46 (7), 1877–1887.
Eriksen, B., Eriksen, C., 1974. Effects of noise letters upon the identification of a target letter in a nonsearch task. Attention, Perception, & Psychophysics 16 (1), 143–149.
Eysenck, M.W., Derakshan, N., Santos, R., Calvo, M.G., 2007. Anxiety and cognitive performance: attentional control theory. Emotion 7 (2), 336–353.
Falkenstein, M., Hohnsbein, J., Hoormann, J., Blanke, L., 1991. Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. Electroencephalography and Clinical Neurophysiology 78 (6), 447–455.
Falkenstein, M., Hoormann, J., Christ, S., Hohnsbein, J., 2000. ERP components on reaction errors and their functional significance: a tutorial. Biological Psychology 51 (2–3), 87–107.
Fitzgerald, K.D., Welsh, R.C., Gehring, W.J., Abelson, J.L., Himle, J.A., Liberzon, I., et al., 2005. Error-related hyperactivity of the anterior cingulate cortex in obsessive-compulsive disorder. Biological Psychiatry 57 (3), 287–294.
Fox, N.A., Nichols, K.E., Henderson, H.A., Rubin, K., Schmidt, L., Hamer, D., et al., 2005. Evidence for a gene–environment interaction in predicting behavioral inhibition in middle childhood. Psychological Science 16 (12), 921–926.
Gehring, W.J., Goss, B., Coles, M.G.H., Meyer, D.E., Donchin, E., 1993. A neural system for error detection and compensation. Psychological Science 4 (6), 385–390.
Gehring, W.J., Himle, J., Nisenson, L.G., 2000. Action-monitoring dysfunction in obsessive-compulsive disorder. Psychological Science 11 (1), 1–6.
Gladstone, G.L., Parker, G.B., Mitchell, P.B., Wilhelm, K.A., Malhi, G.S., 2005. Relationship between self-reported childhood behavioral inhibition and lifetime anxiety disorders in a clinical sample. Depression and Anxiety 22 (3), 103–113.
Gratton, G., Coles, M.G.H., Donchin, E., 1983. A new method for off-line removal of ocular artifact. Electroencephalography and Clinical Neurophysiology 55 (4), 468–484.
Hajcak, G., Franklin, M.E., Foa, E.B., Simons, R.F., 2008. Increased error-related brain activity in pediatric obsessive-compulsive disorder before and after treatment. The American Journal of Psychiatry 165 (1), 116–123.
Hajcak, G., McDonald, N., Simons, R.F., 2003. Anxiety and error-related brain activity. Biological Psychology 64 (1–2), 77–90.
Hajcak, G., McDonald, N., Simons, R.F., 2004. Error-related psychophysiology and negative affect. Brain and Cognition 56 (2), 189–197.
Hajcak, G., Moser, J.S., Holroyd, C.B., Simons, R.F., 2007. It's worse than you thought: the feedback negativity and violations of reward prediction in gambling tasks. Psychophysiology 44 (6), 905–912.
Hajcak, G., Moser, J.S., Yeung, N., Simons, R.F., 2005. On the ERN and the significance of errors. Psychophysiology 42 (2), 151–160.
Hajcak, G., Simons, R.F., 2002. Error-related brain activity in obsessive-compulsive undergraduates. Psychiatry Research 110 (1), 63–72.
Hogan, A.M., Vargha-Khadem, F., Kirkham, F.J., Baldeweg, T., 2005. Maturation of action monitoring from adolescence to adulthood: an ERP study. Developmental Science 8 (6), 525–534.
Ladouceur, C.D., Dahl, R.E., Birmaher, B., Axelson, D.A., Ryan, N.D., 2006. Increased error-related negativity (ERN) in childhood anxiety disorders: ERP and source localization. Journal of Child Psychology & Psychiatry 47 (10), 1073–1082.
Ladouceur, C.D., Dahl, R.E., Carter, C.S., 2007. Development of action monitoring through adolescence into adulthood: ERP and source localization. Developmental Science 10 (6), 874–891.
Mathalon, D.H., Whitfield, S.L., Ford, J.M., 2003. Anatomy of an error: ERP and fMRI. Biological Psychology 64 (1–2), 119–141.
McClure, E.B., Monk, C.S., Nelson, E.E., Parrish, J.M., Adler, A., Blair, R.J.R., et al., 2007. Abnormal attention modulation of fear circuit function in pediatric generalized anxiety disorder. Archives of General Psychiatry 64 (1), 97–106.
McDermott, J.M., Perez-Edgar, K., Henderson, H.A., Chronis-Tuscano, A., Pine, D.S., Fox, N.A., 2009. A history of childhood behavioral inhibition and enhanced response monitoring in adolescence are linked to clinical anxiety. Biological Psychiatry 65 (5), 445–448.
Moser, J.S., Hajcak, G., Simons, R.F., 2005. The effects of fear on performance monitoring and attentional allocation. Psychophysiology 42 (3), 261–268.
Nieuwenhuis, S., Ridderinkhof, K.R., Blom, J., Band, G.P., Kok, A., 2001. Error-related brain potentials are differentially related to awareness of response errors: evidence from an antisaccade task. Psychophysiology 38 (5), 752–760.
Nitschke, J.B., Heller, W., Imig, J.C., McDonald, R.P., Miller, G.A., 2001. Distinguishing dimensions of anxiety and depression. Cognitive Therapy and Research 25 (1), 1–22.
Olvet, D., Hajcak, G., 2009a. The effect of trial-to-trial feedback on the error-related negativity and its relationship with anxiety. Cognitive, Affective, & Behavioral Neuroscience 9 (4), 427–433.
Olvet, D., Hajcak, G., 2009b. The stability of error-related brain activity with increasing trials. Psychophysiology 46 (5), 957–961.
Olvet, D.M., Hajcak, G., 2008. The error-related negativity (ERN) and psychopathology: toward an endophenotype. Clinical Psychology Review 28 (8), 1343–1354.
Overbeek, T.J.M., Nieuwenhuis, S., Ridderinkhof, K.R., 2005. Dissociable components of error processing: on the functional significance of the Pe vis-à-vis the ERN/Ne. Journal of Psychophysiology 19 (4), 319–329.
Paulus, M.P., Hozack, N., Frank, L., Brown, G.G., 2002. Error rate and outcome predictability affect neural activation in prefrontal cortex and anterior cingulate during decision-making. NeuroImage 15 (4), 836–846.
Pine, D.S., 2007. Research review: a neuroscience framework for pediatric anxiety disorders. Journal of Child Psychology & Psychiatry 48 (7), 631–648.
Rabbitt, P.M., 1966. Errors and error correction in choice-response tasks. Journal of Experimental Psychology 71 (2), 264–272.

Ridderinkhof, K.R., Ramautar, J.R., Wijnen, J.G., 2009. To P(E) or not to P(E): a P3-like ERP component reflecting the processing of response errors. Psychophysiology 46 (3), 531–538.

Ruchsow, M., Grön, G., Reuter, K., Spitzer, M., Hermle, L., Kiefer, M., 2005. Error-related brain activity in patients with obsessive-compulsive disorder and in healthy controls. Journal of Psychophysiology 19 (4), 298–304.

Santesso, D.L., Segalowitz, S.J., Schmidt, L.A., 2006. Error-related electrocortical responses are enhanced in children with obsessive-compulsive behaviors. Developmental Neuropsychology 29 (3), 431–445.

Segalowitz, S.J., Dywan, J., 2009. Individual differences and developmental change in the ERN response: implications for models of ACC function. Psychological Research 73 (6), 857–870.

Segalowitz, S.J., Santesso, D.L., Murphy, T.I., Homan, D., Chantziantoniou, D.K., Khan, S., 2010. Retest reliability of medial frontal negativities during performance monitoring. Psychophysiology 47 (2), 260–270.

Spence, S.H., Rapee, R., McDonald, C., Ingram, M., 2001. The structure of anxiety symptoms among preschoolers. Behaviour Research and Therapy 39 (11), 1293–1316.

Torpey, D.C., Hajcak, G., Klein, D.N., 2009. An examination of error-related brain activity and its modulation by error value in young children. Developmental Neuropsychology 34 (6), 749–761.

Ursu, S., Stenger, V.A., Shear, M.K., Jones, M.R., Cameron, S.C., 2003. Overactive action monitoring in obsessive-compulsive disorder: evidence from functional magnetic resonance imaging. Psychological Science 14 (4), 347–353.

Van Bogaert, P., Wikler, D., Damhaut, P., Szliwowski, H.B., Goldman, S., 1998. Regional changes in glucose metabolism during brain development from the age of 6 years. NeuroImage 8 (1), 62–68.

van Veen, V., Carter, C.S., 2002. The anterior cingulate as a conflict monitor: fMRI and ERP studies. Physiology & Behavior 77 (4–5), 477–482.

Vasey, M.W., Crnic, K.A., 1994. Worry childhood: a developmental perspective. Cognitive Therapy & Research 18 (6), 529–549 [Article].

Velanova, K., Wheeler, M.E., Luna, B., 2008. Maturational changes in anterior cingulate and frontoparietal recruitment support the development of error processing and inhibitory control. Cerebral Cortex 18 (11), 2505–2522.

Weinberg, A., Olvet, D.M., Hajcak, G., 2010. Increased error-related brain activity in generalized anxiety disorder. Biological Psychology 85 (3), 472–480.

Wiersema, J.R., van der Meere, J.J., Roeyers, H., 2007. Developmental changes in error monitoring: an event-related potential study. Neuropsychologia 45 (8), 1649–1657.