HIGH LEVELS OF INTERNALIZING SYMPTOMOLOGY IN EARLY CHILDHOOD AND THEIR RELATION TO ERROR MONITORING IN MIDDLE CHILDHOOD

by

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ABSTRACT

While extensive research has been conducted on the relationship between anxiety disorders and executive function, little is known about the association between internalizing symptoms and executive functioning in childhood, and even less is known about the effect of early internalizing symptoms on early and later error monitoring (ERN), a specific facet of executive functioning. The present study utilized EEG to examine whether children with high levels of internalizing symptoms at age 4 would evidence larger ERN amplitudes to error commission relative to children with low levels of internalizing symptoms. Participants included 22 high-risk children who completed the Erikson Flanker Task while EEG data were recorded. Early childhood internalizing symptoms were assessed via parent report on the CBCL. Linear regression analyses revealed no association between internalizing symptoms at age 4 and ERN amplitude at age 8. Results suggest that, in the present sample, internalizing at age 4 was unrelated to error monitoring at age 8.

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Chapter 1

INTRODUCTION

Internalizing disorders are a class of mental health problems that affect individuals at all stages of life and can have serious implications for multiple realms of development, including cognitive capabilities (Weems, Berman, Silverman & Saavedra, 2001). The use of electroencephalography (EEG) has allowed researchers to examine differences in specific types of cognitive capabilities, including neural response to error commission, between individuals with and without high levels of internalizing symptoms. Using EEG, it is therefore possible to achieve a greater understanding of the relationship between early psychopathology and later executive function than could be obtained by means of behavioral measures alone. The present study investigates the relationship between internalizing symptomatology in early childhood and error monitoring, a particular facet of executive function, in middle childhood in order to gain a better understanding of the developmental consequences of early psychopathology.

As noted, EEG provides one method of studying executive function. This technology allows researchers to examine brain activity related to different cognitive capabilities, as well as how that brain activity is affected by early psychopathology. One such cognitive capability that has been extensively studied in the field of EEG and psychopathology research is error monitoring (Hajcak, Moser, Yeung, & Simons, 2005; Iannaccone, et al., 2015). Error monitoring occurs both consciously and subconsciously after an individual makes a mistake. Individuals who assign more

motivational significance to a task are prone to engage in more error monitoring and tend to have large neural responses to perceived errors (Hajack et al., 2005). It is crucial to examine error monitoring as an indicator of executive function in the context of early psychopathology, as individuals who exhibit overregulated executive function are susceptible to feelings of stress and overstimulation (Blair, 2016).

Event Related Potentials (ERPs) are neural activity associated with specific events, such as an error (Luck, 2014). Studies of error monitoring typically focus on one particular ERP: the error-related negativity (ERN). The ERN is a negative deflection that begins immediately after a mistake is made and peaks approximately 50-100 ms after the error commission. Larger ERN amplitudes have been associated with greater sensitivity to error commission and are thought to reflect a generally more vigilant internal monitoring system (Hajcak et al, 2005). The ERN is therefore a particularly useful tool for investigating how early internalizing symptoms impact later error monitoring and, more broadly, executive functioning.

The relationship between anxiety and the ERN in adults is well established. Research on anxiety disorders has consistently found that adults with generalized anxiety disorder (GAD) and obsessive-compulsive disorder (OCD) have larger ERNs than do control subjects (Endrass, Klawohn, Schuster, & Kathmann, 2008; Gehring, Himle, & Nisenson, 2000; Weinberg, Klein, & Hajcak, 2012; Weinberg, Kotov, & Proudfit, 2015). While fewer studies have been conducted investigating ERNs in children, preliminary research suggests that a similar relationship exists in childhood. For example, a study conducted by Hajcak, Franklin, Foa, and Simons (2008) investigated the ERN in a pediatric sample of OCD patients ranging in age from 8 to 16. The pediatric patients had ERNs of larger amplitude both before and after

successful treatment than healthy controls. In a similar study, Ladouceur, Dahl, Birmaher, Axelson, and Ryan (2006) assessed the relationship between the ERN and clinical levels of anxiety. Children ranging in age from 8 to 14 years receiving outpatient treatment for GAD, Separation Anxiety Disorder, Social Phobia, and Specific Phobias evidenced an ERN of larger amplitude than did a group of age-matched controls. However, it is important to note that each of these studies took a largely cross-sectional approach to examining the relationship between anxiety and the ERN, and none considered the developmental effects of early anxiety on later error monitoring.

While the relationship between anxiety and the ERN has been well established, the relationship between depression and ERN amplitude remains unclear. A substantial body of research on adults suggests that individuals displaying depressive symptomatology have larger ERNs than do controls, but controversy still remains. Though many researchers have found increased ERN amplitudes in depressed subjects relative to controls (Chiu & Deldin, 2007; Holmes and Pizzagalli, 2010; Olvet & Hajcak, 2008), Schirijvers et al. (2008) reported no difference in ERN amplitude between individuals with major depressive disorder (MDD) and a group of matched control subjects. Additional work has failed to find an enhanced ERN in groups of individuals with comorbid anxiety and depression (Weinberg, Klein, & Hajcak, 2012; Weinberg, Kotov, & Proudfit, 2015).

Research focusing on the ERN and depression in children has been particularly sparse and inconsistent (Holler, Kavanaugh, & Cook, 2013). A study by Bress, Meyer, and Hajcak (2015) examined 25 children between the ages of 11 and 13 who, along with their parents, completed the Screen for Child Anxiety Related Emotional

Disorders (SCARED) and the Children's Depressive Inventory (CDI), two measures designed to assess current levels of anxiety and depressive symptoms. Findings suggested that, in late childhood, increased ERN amplitude is uniquely related to current anxiety levels (i.e., not moderated by depressive symptoms). Other researchers, however, have reported contradictory results. Ladouceur and colleagues (2012), for example, found that depressed children aged 7 to 17 demonstrated significantly smaller ERNs than control participants. Additionally, the ERN amplitude of children with MDD did not increase linearly with age. Furthermore, Ladouceur and colleagues (2012) noted that ERNs in children with depression were unaffected by the presence of comorbid anxiety. Again, work on the relationship between depression and error monitoring in children has been largely cross-sectional in nature and has failed to examine the longitudinal association between early depression and later ERN amplitudes.

Research examining internalizing symptoms more broadly in children has also been limited. Moadab, Gilbert, Dishion, and Tucker's (2010) study of a group of 9-13 year olds suggests that children who demonstrate more internalizing symptoms have larger ERN amplitudes than children with fewer internalizing symptoms. However, it is worth noting that only 3 of their 75 participants met criteria for an internalizing disorder. Henderson and colleagues (2015) also found enlarged ERNs in a sample of high functioning autistic children aging 8 to 16 years who reported elevated levels of internalizing symptoms.

As noted, each of the aforementioned studies of internalizing symptoms and error monitoring focus on concurrent internalizing problems and ERN amplitudes;

knowledge of the association between early psychopathology and later executive functioning is limited. The present study seeks to utilize longitudinal measures to expand upon the relationship between internalizing symptomology in early childhood and error monitoring, an index of executive function, in middle childhood. Drawing from Moadab and colleagues' (2010) findings, this study seeks to examine the relationship between internalizing symptoms in early childhood and error monitoring in middle childhood. Based on the documented relationship between internalizing disorders and concurrent executive functioning, it is hypothesized that children with high levels of internalizing at age 4 will demonstrate larger ERN amplitudes at age 8 relative to children with low levels of early childhood internalizing symptoms.

Chapter 2

METHODS

2.1 Participants

The participants for this study were recruited from a larger longitudinal study assessing the effect of an early parenting intervention on parent-child attachment quality. The sample used in the present study consisted of parent-child dyads originally referred to Child Protective Services (CPS) before the age of 24 months due to suspected risk of maltreatment, including abuse and neglect. Children were first assessed regularly in both home and laboratory settings from infancy until approximately 4 years old, after which they were brought back to complete a variety of developmental assessments at age 8. Seventy-seven parent-child dyads completed the initial assessments from infancy until approximately age 4 (m = 49.3 months), and 43 children completed the assessment at age 8 (m = 8.4). Twenty-one children were excluded from data analysis due to excess artifacts (n = 11), being unable to complete the task (n = 1), for committing insufficient errors to elicit a reliable ERN (n = 2 with fewer than 6 errors; Olvet & Hajcak, 2009), and for exceeding a 60-40 ratio of correct trials to incorrect trials (n = 7). The remaining sample (n = 22) was comprised of 14 females and 8 males. 63.6% of the sample identified as African American, 18.2% as Hispanic, 9.1% Caucasian, and 9.1% Biracial.

2.2 Measures

Internalizing. The Child Behavior Checklist (CBCL) was administered to assess internalizing symptomatology. The CBCL is a parent report measure used to assess children's behavioral, social and emotional problems (de la Osa, Granero, Trepat, Domenech, & Ezpeleta, 2016). When participants were approximately 4 years old, parents rated their child's functioning across a number of domains, including anxious/depressive symptomatology, emotional reactivity, and sleep problems on a 3-point Likert scale of agreement. Only the Anxious/Depressed subscale of the CBCL is of relevance to the present study and was therefore the only subscale considered in analyses.

2.3 Procedure

Upon arriving at the lab, participants received a brief introduction to the electrophysiological recording equipment. They were then fitted with an electo-cap and brought into a small room to begin the Erikson Flanker task. During this task, participants were shown sets of 5 arrows that were either congruent (<<<<< or >>>>>) or incongruent (<<><< or >><>>) and instructed to press either a right or left button in accordance with the direction of the middle arrow. An experimenter first explained this task using hand-held, paper stimuli in order to increase children's ease of learning the task. Before beginning the computerized task, children's understanding of the task was assessed via hand-held paper trials. The computerized version of the task was then presented on a computer screen using Presentation Software. Children engaged in two additional practice blocks on the computer. Each practice block lasted until the child completed 10 correct trials. During the first block of the computer practice, an experimenter reiterated the directions and offered encouragement as

needed. After each child had successfully completed the practice blocks, the experimenter left the room and the child completed 6 blocks of 50 trials each. Fifty percent of the blocks contained congruent arrows and 50% incongruent arrows in a randomized order. Arrows were presented at intervals for 200 ms with a fixation cross preceding each trial for an interval of 900-1100 ms. Between blocks, an experimenter offered the child a short break and two stickers for each block successfully completed. After the task, children received an additional prize. Children also completed two additional computerized tasks as part of this experimental protocol, though these tasks are not of relevance to the present study.

2.4 Psychophysiological Recording and Data Reduction

EEG data were collected using an Electro-Cap containing 32 Ag/AgCl sintered electrodes (Electro-Cap International). Data were recorded with a right mastoid (M2) reference and a forehead ground cite (AFz). Continuous EEG data were digitized at 1024 Hz. Impedances for all electrodes were below 20 KΩ for each participant. Advanced Source Analysis (ASA) software was used to process the data. The data were re-referenced offline using the average mastoid reference. Data were corrected for eye blinks, and band-pass filtered from 0.2 to 30 Hz.

Trials during which the child failed to respond within 800 ms following the presentation of the arrow stimulus were rejected. Next, jump artifact correction was accomplished using *z*-transformations of the epoched data. *Z*-values exceeding 20 were assumed to contain jump artifacts and were thus excluded from processing. To reject muscle artifacts, the data were band-pass filtered using a Butterworth digital filter from 110 to 140 Hz. *Z*-transformations were performed and averaged across

sensors. Epochs with *Z*-values greater than 4 were determined to contain excessive muscle artifacts and were omitted. Trials containing excessive artifacts (exceeding a threshold of $\pm$ 75 μV) were also removed from the data.

Starting from a 600 ms baseline to 1000 ms post stimulus onset, epochs were obtained for each trial. Using the average timecourse from a baseline period of 600 to 400 ms pre-response, post-response activity was subtracted from the baseline sample average to obtain a mean of 0. Based off the Woody filtering process, in order to correct for inter-trial latency instability, individual trials were correlated with a template centered upon each participant's raw average ERP between 0-300 ms post-response. The regions of interest (ROIs) for the ERN were determined based on previous literature (Moser, Hajcak, & Simons, 2005) and then verified via visual examination of the headplots. The ERN was quantified as the mean amplitude between -50-100 ms post-response at Cz. SPSS (Version 21) was utilized to perform linear regressions.

Chapter 3

RESULTS

3.1 Behavioral Results

A series of repeated measures analyses of variance (ANOVAs) was conducted to examine the associations between trial type and reaction times (RTs) and accuracy. Results indicated a main effect of trial type on reaction time, with participants responding faster on incorrect than correct trials, $F(1,18) = 62.194$, $p = .001$. Additionally, participants responded faster on congruent than incongruent trials, $F(1,18) = 6.439$, $p = .021$. Participants showed greater accuracy on congruent than incongruent trials, $F(1,18) = 28.424$, $p = .001$. However, results suggest that participants did not slow in their response time after committing an error, as there was no significant difference in reaction times in trials immediately following correct versus incorrect trials, $F(1,18) = 0.024$, $p = .878$.

Bivariate correlations were conducted to examine the association between CBCL scores and these variables. Results indicated that there was no correlation between CBCL internalizing scores and reaction time for correct trials, $r(20) = -.099$, $p = .660$, incorrect trials, $r(20) = .033$, $p = .884$, congruent trials, $r(20) = -.086$, $p = .703$, or incongruent trials, $r(20) = -.072$, $p = .749$. There was no correlation between CBCL scores and accuracy on congruent, $r(20) = .051$, $p = .821$, or incongruent trials, $r(20) = -.115$, $p = .609$. Additionally, there was no correlation between reaction times on trials following a correct trial, $r(20) = -.063$, $p = .781$, or following an incorrect trial, $r(20) = -.153$, $p = .496$.

See Table 1 for means and standard deviations of internalizing scores, accuracy, and reaction time.

3.2 ERP Results

Figure 1 offers an illustration of the average ERP waveform at Cz for correct and error trials. This waveform is characterized by a sharp negative deflection followed by a slower positive deflection.

A repeated measures ANOVA was performed to compare mean ERN and Pe amplitudes for correct and incorrect trials. Results indicated that the ERN was larger on incorrect relative to correct trials, $F(1,21) = 43.375$, $p < .001$, and that the Pe was more positive for incorrect relative to correct trials, $F(1,21) = 32.984$, $p < .001$. See Table 2 for means and standard deviations for ERN and Pe amplitudes by trial type.

Bivariate correlations were preformed to examine the relationship between internalizing in early childhood and error monitoring in middle childhood. Results indicated that there is no significant relationship between participants' CBCL scores at age 4 and their mean ERN amplitudes at age 8, $r(20) = -.207$, $p = .356$, nor their mean Pe amplitudes at age 8, $r(20) = -.032$, $p = .887$.

Chapter 4

DISCUSSION

Despite considerable research on adults and the effects of psychopathology on later executive function, little is known the relationship between early internalizing symptoms and later error monitoring in children. The current study sought to investigate whether internalizing symptoms at age 4 predicted error monitoring at age 8, as indexed by mean ERN and Pe amplitudes.

Behavioral data suggest that the participants understood and were appropriately engaged in the task. Consistent with research on adults, the participants in the current study had faster reaction times during incorrect than correct trials, responded faster to congruent than incongruent trials, and responded with greater accuracy on congruent than incongruent trials (Hajcak, McDonald, & Simons, 2003; Van't Ent, 2002). However, the participants did not engage in typical post-error slowing. In adult samples, the literature suggests that after committing an error, individuals demonstrate increased reaction times on the immediately succeeding trial. Rather than a mere regression towards the mean, post-error slowing is thought to be a compensatory behavior used to diminish the likelihood of committing additional mistakes (Hajcak, McDonald, & Simons, 2003; Hajcak & Simons, 2008). As executive function does not fully develop until late adolescence, it is possible that the participants in the current study were unable to implement post-error reaction time adjustments because of their still immature executive functioning capabilities (Carriedo, Corral, Montoro, Herrero, & Rucian, 2016).

Results indicated that there was no association between internalizing symptoms in early childhood and ERN amplitude or Pe amplitude in middle childhood. These null findings did not support the study's hypothesis that children with high levels of internalizing at age 4 would demonstrate larger ERN amplitudes at age 8 than children with low levels of internalizing symptoms. These null results may be due to the measurement of internalizing as a combination of anxiety and depression, as the literature on the ERN and depression, anxiety, and comorbid depression and anxiety is so inconsistent.

As noted, the relationship between internalizing symptoms and the ERN is unclear. Studies suggest that depression and anxiety may affect error monitoring in different ways. While the literature has consistently shown a direct correlation between anxiety levels and ERN amplitudes, studies examining GAD and MDD concurrently have failed to find this association in samples with comorbid MDD and GAD (Endrass et al., 2008; Gehring et al., 2000; Hajcak et al., 2008; Ladouceur et al., 2006; Weinberg et al, 2012; Weinberg et al., 2015). Additional studies have found ERNs of reduced amplitude in adults with depression compared to healthy controls (Rushsow et. al., 2004; Rushsow et al., 2006). Taken together, these results may suggest that anxiety and depression may affect the ERN in opposing directions and are better measured and studied separately.

The present study had a number of strengths, including its longitudinal nature. By utilizing longitudinal methods, the present study was able to ask causal questions about the association between early internalizing and later executive function. This study also benefited from the novelty of the question posed, as the majority of prior work has not examined the relationship between early internalizing symptoms on later

error monitoring. The present study also contributes to the relatively small body of research of ERPs in children.

However, this study is not without limitations. Statistical power was limited by the small sample size, which may have contributed to the null findings of this study. Additionally, while the use of parent-report measures can offer insight into child functioning, it often is only minimally correlated with child self-report and teacher report measures, especially for internalizing symptoms (De Los Reyes, 2011; Shemmassian & Lee, 2016). Furthermore, future studies would benefit from investigating these constructs in a clinical sample, as the majority of the current sample reported CBCL scores that fell below the clinical threshold. Not only would this alleviate the shortcomings of parent-report measures, but it would also allow for comparisons between a sample consisting of clearly defined anxious/depressed groups and control groups. Additionally, as there is evidence suggesting that the ERN may not be fully developed until mid to late adolescence, the lack of significance in the current study could be due to insufficiently developed ERNs (Ladouceur, Slifka, Dahl, Birmaher, & Axelson, 2012; van Meel, Heslenfeld, Rommelse, Oosteriaan, & Sergeant, 2012). By examining the ERNs of older children, future studies may also be able to reduce the number of subjects dropped from analyses due to an insufficient correct to incorrect response ratio. Investigating children approaching adolescence could alleviate noise associated with the task, as adolescents may be better equipped to understand and complete the flanker activity appropriately.

While the results of the current study were not significant, the relationship between the ERN and internalizing disorders warrants further exploration. The relationship between the ERN and depression is still highly contested. More research

must be completed to fully understand if and how individuals with anxiety and depressive disorders differ from nonclinical individuals in their error monitoring in order to better understand how early psychopathology, specifically internalizing disorders, may affect cognitive capabilities throughout the lifespan.

REFERENCES

Blair C. (2016). Developmental science and executive function. *Current Directions in Psychological Science*, 25(1), 3-7. doi: 10.1177/0963721415622634.

Bress, J.N., Meyer A., Hajcak G. (2015). Differentiating anxiety and depression in children and adolescents: Evidence from event-related brain potentials. *Journal of Clinical Child and Adolescent Psychology*, 44(2), 238-249. doi: http://dx.doi.org.udel.idm.oclc.org/10.1080/15374416.2013.814544

Chiu P. H. & Deldin P.J. (2007). Neural evidence for enhanced error detection in major depressive disorder. *The American Journal of Psychiatry*, 164(4), 608-616. Retrieved from: http://search.proquest.com.udel.idm.oclc.org/docview/220473220?accountid=10457

Carriedo, N., Corral A., Montoro P.R., Herrero L., Rucian M. (2016). Development of the updating executive function: From 7 year olds to young adults. *Developmental Psychology* 52(4), 666-678. doi:10.1037/dev0000091

De la Osa N., Granero R., Trepat E., Domenech J.M., Ezpeleta L. (2016). The discriminative capacity of CBCL/1/1/2-5-DSM5 scales to identify disruptive and internalizing disorders in preschool children. *European Child & Adolescent Psychiatry*, 25(1), 17-23. doi: 10.1007/s00787-015-0694-4

De Los Reyes A. (2011). Introduction to the Special Section: More Than Measurement Error: Discovering Meaning Behind Informant Discrepancies in Clinical Assessments of Children and Adolescents. *Journal of Clinical Child & Adolescent Psychology,* 40(1), 1-9. doi: 10.1080/15374416.2011.533405

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. doi: 10.1016/j.neuropsychologia.2007.12.001

Gehring W.J., Himle J. & Nisenson. (2000). Action-monitoring dysfunction in obsessive-compulsive disorder. *Psychological Science*, 11(1), 1-6. doi: 10.1111/1467-9280.00206

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). To err is automatic: Error-related brain potentials, ANS activity, and post-error compensatory behavior. Psychophysiology, 40(6), 895-903. doi: 10.1111/1469-8986.00107

Hajcak G., Moser J.S., Yeung N., & Simons R.F. (2005). On the ERN and the significance of errors. *Psychophysiology*, 42(2), 151-160. doi:10.1111/j.1469-8986.2005.00270.x

Hajcak G. & Simons R.F. (2008). Oops!..I did it again: An ERP and behavioral study of double-errors. *Brain and Cognition*, 68(1), 15-21. doi: 10.1016/j.bandc.2008.02.118

Henderson H.A., Ono K.E., McMahon C.M., Schwartz C. B., Usher L.V., & Mundy P.C. (2015). The costs and benefits of self-monitoring for higher functioning children and adolescents with autism. *Journal of Autism and Developmental Disorder*s, 45(2), 548-559. doi:10.1007/s10803-013-1968-7

Holler K., Kavanaugh B., & Cook N.E. (2013). Executive Functioning in Adolescent Depressive Disorders. *Journal of child and family studies*, 23(8), 1315-1324. doi:10.1007/s10826-013-9789-z

Holmes A.J. & Pizzagalli D.A. (2010) Effects of task-relevant incentives on the electrophysiological correlates of error processing in major depressive disorder. *Cognitive, Affective, & Behavioral Neuroscience*, 10(1), 119-128. doi:*10.3758/CABN.10.1.119*

Iannaccone, R., Hauser, T. U., Staempfli, P., Walitza, S., Brandeis, D., & Brem, S. (2015). Conflict monitoring and error processing: New insights from simultaneous EEG–fMRI. *NeuroImage, 105*, 395-407. doi:http://dx.doi.org.udel.idm.oclc.org/10.1016/j.neuroimage .2014.10.028

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 and Psychiatry*, 47(10), 1073-1082. doi:10.1111/j.1469-7610.2006.01654.x

Ladouceur C.D., Slifka J.S., Dahl R.E., Birmaher B., & Axelson D.A. (2012). Altered error-related brain activity in youth with major depression. *Developmental Cognitive Neuroscience*, 2(3), 351-362. doi: http://dx.doi.org.udel.idm.oclc.org/10.1016/ j.dcn.2012.01.005

Luck, S.J. (2014). *An introduction to the event related potential technique* (2nd ed.). Cambridge, MA: The MIT Press.

Moadab I., Gilbert T., Dishion T.J., & Tucker D.M. (2010). Frontolimbic activity in a frustrating task: covariate patterns of coping and individual differences in externalizing and internalizing symptoms. *Development and Psychopathology*, 22(2), 391-404. doi:10.1017/S0954579410000131

Moser J.S., Hajcak G., & Simons R.F. (2005). The effects of fear on performance monitoring and attentional allocation. *Psychopsychiatry*, 42(3), 261-268. doi: 10.1111/j.1469-8986.2005.00290.x

Olsen S.L., Bates J.E., Sandy J.M., Schilling E.M. (2002). Early developmental precursors of impulsivity and inattentive behavior: from infancy to middle childhood. *The Journal of Child Psychology and Psychiatry*, 43(4), 435-447. doi: 10.1111/1469-7610.00035

Olvet D.M. & Hajcak G. (2008). The error-related negativity (ERN) and psychopathology: Toward an endophenotype. Clinical Psychology Review, 28(8), 1343-1354. doi:10.1016/j.cpr.2008.07.003

Olvet D.M. & Hajcak G. (2009). The stability of error-related brain activity with increasing trials. *Psychophysiology*, 46(5), 957-961. doi: 10.1111/j.1469-8986.2009.00848.x

Rushsow M., Herrnberger B., Beschoner P., Gron G., Spitzer, M., & Kiefer M. (2006). Error processing in major depressive disorder: Evidence from event-related potentials. *Journal of Psychiatric Research*, 40(1), 37-46. doi:10.1016/j.jpsychires.2005.02.002

Ruchsow M., Hermberger C. W., Wiesend C., Gron G, Spitzer M., & Kiefer M. (2004). The effect of erroneous response monitoring in patients with major depressive disorder: A study with event related potentials. *Psychophysiology*, 41(6), 833-840. doi: 10.1111/j.1469-8986.2004.00237.x

Schirijvers D., de Bruijn, E.R.A., Maas Y., Grave C.D., Sabbe B.G.C., & Hulstijn. (2008). Action monitoring in major depressive disorder with psychomotor retardation. *Cortex*, 44(5), 569-579. doi:10.1016/j.cortex.2007.08.014

Shemmassian S.K. & Lee S.S. (2016). Predictive utility of four methods of incorporating parent and teacher symptom ratings of ADHD for longitudinal outcomes. Journal of Clinical Child & Adolescent Psychology, 45(2), 176-187. doi: 10.1080/15374416.2014.971457

Thorell, L.B. (2007). Do delay aversion and executive function deficits make distinct contributions to the functional impact of ADHD symptoms? A study of early academic skill deficits. *Journal of Child Psychology and Psychiatry, 48*(11), 1061-1070.

van Meel C.S., Heslenfeld D.J., Rommelse N.N., Oosteriaan J, & Sergeant J. (2012). Developmental trajectories of neural mechanisms supporting conflict and error processing in middle childhood. *Developmental Neuropsychology*, 37(4), 358-378. doi: 10.1080/87565641.2011.653062

Van't Ent, D. (2002). Perceptual and motor contributions to performance and ERP components after incorrect motor activation in a flanker reaction task. *Clinical Neurophysiology*, 113(2), 270-283. doi: 10.1016/S1388-2457(01)00730-1

Weems, C. F., Berman, S. L., Silverman, W. K. and Saavedra, L. M. (2001). Cognitive errors in youth with anxiety disorders: the linkages between negative cognitive errors and anxious symptoms. *Cognitive Therapy and Research*, *25*, 559–575.

Weinberg A., Klein D.N., & Hajcak G. (2012). Increased error-related brain activity distinguishes generalized anxiety disorder with and without comorbid major depressive disorder. *Journal of Abnormal Psychology*, 121(4), 885-896. doi: 10.1037/a0028270

Weinberg A., Kotov R., & Proudfit G.H. (2015). Neural indicators of error processing in generalized anxiety disorder, obsessive-compulsive disorder, and major depressive disorder. *Journal of Abnormal Psychology*, 124(1), 172-185. doi: 10.1037/abn0000019

Woody, C. D. (1967). Characterization of an adaptive filter for the analysis of variable latency neuroelectric signals. *Medical and Biological Engineering, 5*(6), 539-554

Zelazo P.D., Anderson J.E., Richler J., Wallner-Allen K, Beaumont J.L. & Weintraub S. (2013). II. National toolbox cognition battery (CB): Measuring executive function and attention NIH toolbox cognitive battery (CB). *Monographs of the Society for Research in Child Developmen*t, 78(4), 16-33. doi:10.1111/mono.12032

Table 1 Means and Standard Deviations for CBCL Internalizing Scores, Accuracy, and Reaction Time

	M	*SD*
CBCL Internalizing	51.955	2.968
Overall Accuracy (%)	0.834	0.110
Accuracy Congruent	0.807	0.157
Accuracy Incongruent	0.657	0.193
Overall RT (ms)	370.540	91.977
RT Correct Trials	374,330	90.201
RT Incorrect Trials	270.968	91.191
RT Congruent Trials	328.032	77.337
RT Incongruent Trials	352.564	73.574
RT Following Correct Trial	370.628	117.867
RT Following Incorrect Trial	370.595	83.557

Table 2 Means and Standard Deviations for the ERN and Pe by Trial Type

	Correct		Incorrect	
	M	*SD*	*M*	*SD*
Mean ERN amplitude (μV)	12.696	13.299	-0.852	8.585
Mean Pe amplitude (μV)	-0.071	9.400	14.303	11.125

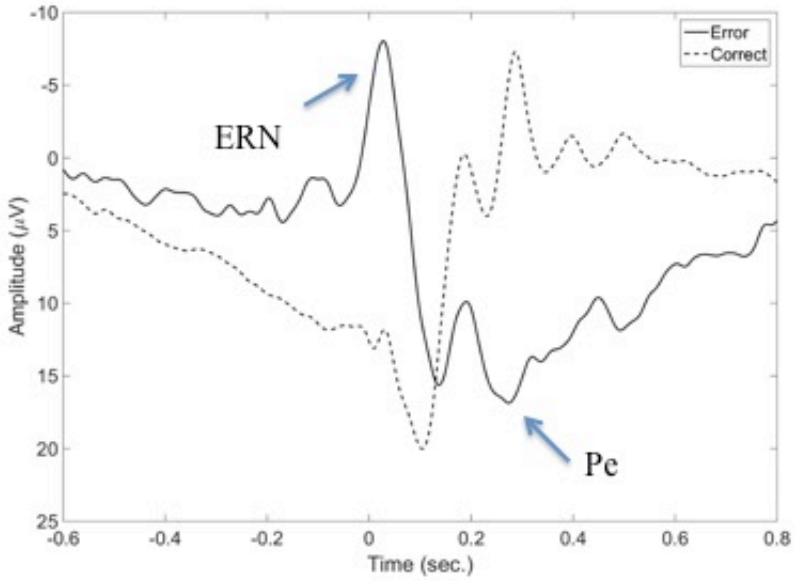

Figure 1 ERP waveforms for correct and error trials at Cz