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Abstract of the Dissertation

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The error-related negativity (ERN) is an event-related brain potential that is believed to originate in the anterior cingulate cortex (ACC) and is observed in adults and older children when errors are committed. Evidence is mixed regarding whether or not young children reliably demonstrate an ERN. A number of studies suggest that the amplitude of the ERN is modulated by individual differences, including psychopathology and temperament. The correct-response negativity (CRN) is also a negative-deflection that appears on correct trials and is similar to the ERN in terms of scalp topography and morphology. Less is known about its function and whether individual differences influence the amplitude of the CRN. The error positivity (Pe) is a large positive deflection that follows the ERN and appears to have a more posterior origin. Less is known about the function. The current study employed a Go/No-Go paradigm to characterize these response-monitoring ERP components in a community sample of 328 5- to 7- year-old children and confirmed that an ERN can be reliably elicited in a young population. Additionally, associations between these ERP components, parental psychopathology, and temperament were examined. Maternal history of an anxiety disorder was associated with a less negative ERN in the young offspring, as were child negative emotionality and child fear. These results may provide additional evidence of the existence of two overlapping neural mechanisms associated with response monitoring, one of which is localized in the rostral ACC, which is developed as early as 5 – 7 years-old, and the other of which is localized is the dorsal ACC and is not yet developed in early childhood. Maternal history of depression was associated with a less negative CRN, suggesting decreased response monitoring in young children at-risk for depression. There were no associations between the CRN and child temperament, nor were there associations between the Pe and parental psychopathology or between the Pe and child temperament.

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I. Introduction

The Error-Related Negativity

Response monitoring involves the ability to detect errors and subsequently adjust behavior (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). Studies that have examined the response-locked event-related potential (ERP) have identified a component now referred to as the Error-related Negativity (ERN; Gehring, Coles, Meyer, & Donchin, 1990) or Negativity Error (NE; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991) associated with error detection. The ERN is a negative deflection with a fronto-central maximum that peaks approximately 50 ms following an erroneous response (Davies, Segalowitz, & Gavin, 2004; Falkenstein et al., 1991; Falkenstein et al., 2000; Gehring et al., 1990; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Hajcak, Moser, Yeung, & Simons, 2005; Hogan, Vargha-Khadem, Kirkham, and Baldeweg, 2005; Holroyd & Coles, 2002; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; Wiersema, van der Meere, & Roeyers, 2007).

As is discussed by Pailing and colleagues (2002), the ERN can be calculated by either subtracting the correct-trial waveform from the error-trial waveform or by averaging only the error-trial waveform. The primary advantage of using the subtraction method is that it removes components of the waveform that are the same for both correct and error responses and results in a more pure reflection of the negativity associated with error detection. However, the primary disadvantage to using the difference wave is that it is not possible to determine whether relationships with other variables are due to associations with correct responses or to associations with errors.

ERP studies using source localization techniques suggest that the ERN is generated in the medial frontal cortex, specifically the anterior cingulate cortex (ACC; Dehaene, Posner, & Tucker, 1994; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Mathewson, Dywan, & Segalowitz, 2005; van Veen & Carter, 2002). Work using functional magnetic resonance imaging (fMRI) also supports the involvement of the ACC in error-detection (Kiehl, Liddle, & Hopfinger, 2000; Mathalon, Whitfield, & Ford, 2003; Menon, Adleman, White, Glover, & Reiss, 2001). Finally, intracerebral studies by Brázdil and colleagues have provided further evidence that the ERN is generated in the

ACC (Brázdil, Roman, Daniel, & Rektor, 2005; Brázdil, Roman, Falkenstein, Daniel, Jurák, & Rektor, 2002).

Preliminary data suggested that the ERN is not reliably elicited before age 12 (Davies et al., 2004). However, Santesso, Segalowitz, and Schmidt (2006) reported an ERN in 10-year-old children and Wiersema et al. (2007) showed that the ERN could be elicited in children as young as 7-8 years-old, although the amplitude was smaller for children compared to adults in both studies. Further, Kim, Iwaki, Imashioya, Uno, and Fugita (2007) demonstrated that, although the amplitude of the ERN elicited in 7-8 year-olds was smaller than that found in 9-11 year-old children, neither group significantly differed from young adults on ERN amplitude. Recently, our lab demonstrated the ERN in children as young as 5-7 years old (Torpey, Hajcak, and Klein, 2009).

The possibility that the ERN might be smaller in younger compared to older participants is consistent with the structural and functional developmental trajectory of the ACC: the ACC and its connections to the prefrontal cortex continue to develop through adolescence (Cunningham, Bharracharyya, & Benes, 2002). Additionally, activation of the ACC increases from childhood into young adulthood (Adleman, Menon, Blasey, White, Warsofsky, Glover et al., 2002; Van Bogaert, Wikler, Damhaut, Szliwowski, & Goldman, 1998). Thus, it is possible that the later maturation of the ACC may be associated with differences in the ERN over development.

Alternatively, the disparate findings in children could be related to the difficulty of the paradigms used across these studies. Results obtained by Hogan et al. (2005) suggest that task complexity influences the amplitude of the ERN in youth. They found that adolescents, but not adults, demonstrated a smaller ERN when the task was more complex. In fact, the ERNs elicited during the complex version of their task (Hogan et al., 2005) are similar to those shown for 7 year-olds in the Davies et al. paper (2004). Importantly, Davies et al. (2004), who demonstrated that there was substantial variability in the ERN elicited by children under 12 years old, used a flanker paradigm, whereas Wiersema et al. (2007), Kim et al. (2007), and Torpey et al. (2009), who obtained reliable ERNs in young children, used simpler Go/No-Go designs. Because there is limited data examining the ERN in this population, one of the aims of the present study is to more thoroughly characterize this component in young children.

Although original conceptualizations of the ERN emphasized its role in conflict and error detection, findings that this component is moderated by affective and motivational influences suggest a more complex function. Specifically, a number of studies have found that manipulations of the motivational value of the errors themselves are associated with changes in the amplitude of the ERN in adults (Hajcak et al., 2005). This relationship is less clear in children. Although there was no association found between the ERN and error value in children between the ages of 5- and 7-years (Torpey et al., 2009), Kim, Iwaki, Uno, and Fugita (2005) reported larger ERNs in slightly older children when they were being observed by a friend compared to when they were completing the task alone. Together, these data suggest that the ERN is sensitive to the significance of the error committed and may be associated with a motivationally salient evaluation of errors in adults, but more data is needed to clarify this relationship in children.

The ERN and Psychopathology

Evidence that the ERN is influenced by affective and motivational factors suggests that it may also be associated with psychopathology. Indeed, there appear to be meaningful relationships between the amplitude of the ERN and specific psychological disorders. As is discussed below, an enhanced ERN has been demonstrated in individuals with anxiety disorders, who are likely to evaluate their performance negatively and react strongly to committing errors. In contrast, an ERN of reduced amplitude has been elicited in individuals with externalizing disorders, who are likely to react less strongly to committing errors (e.g., Hall, Bernat, & Patrick, 2007; Stieben, Lewis, Granic, Zelazo, Segalowitz, & Pepler, 2007). Further, a diminished ERN has also been demonstrated in patients with schizophrenia (for a review, see Morris, Yee, & Nuechterlein, 2006).

Anxiety disorders. Several studies have reported an enhanced ERN in individuals with anxiety disorders, a population in which error commission would be likely associated with increased negative evaluation of performance compared to controls. For example, the amplitude of the ERN is larger in adults with both subclinical levels of obsessive-compulsive symptoms (Hajcak & Simons, 2002; Gründler, Cavanagh, Figueroa, Frank, & Allen, 2009, although this study only demonstrated an enhanced ERN

in individuals with higher levels of obsessive-compulsive symptoms when a standard speeded response paradigm was used and not when a probabilistic learning task was used) and those with obsessive-compulsive disorder (OCD; Gehring, Himle, & Nisenson, 2000; Johannes, Wieringa, Nager, Rada, Dengler, Emrich et al., 2001; Ruchsow, Grön, Reuter, Spitzer, Hermle, & Kiefer, 2005; although see Nieuwenhuis, Nielen, Mol, Hajcak, & Veltman, 2005) compared to controls. This relationship is not specific to OCD, with studies demonstrating an enhanced ERN in adults who were higher in trait anxiety (Olvet & Hajcak, 2009) and who endorse high levels of worry (Hajcak, McDonald, & Simons, 2003). The amplitude of the ERN is not affected, however, by state anxiety, which suggests that it is the overvaluation of errors that contributes to the findings in trait anxious individuals and not increased arousal levels due to fear (Moser, Hajcak, & Simons, 2005)

This association between the ERN and anxiety has also been demonstrated in children. Specifically, the amplitude of the ERN has been found to be larger in a non-clinical population of children with high levels of obsessive-compulsive symptoms (Santesso et al., 2006) and in children with OCD (Hajcak, Franklin, Foa, & Simons, 2008). Ladouceur, Dahl, Birmaher, Axelson, and Ryan (2006) obtained similar results in a sample of clinically anxious children, suggesting that this relationship is not specific to OCD in children either.

Depression. Far fewer studies have investigated the association between the ERN and depression and the results have been equivocal. Chiu and Deldin (2007) and Holmes and Pizzagalli (2008) both found an enhanced ERN in individuals with major depressive disorder. However, Ruchsow and colleagues found a decreased ERN in depressed patients compared to controls, but only in error trials following error trials (Ruchsow, Herrnberger, Wiesend, Grön, Spitzer, & Kiefer, 2004). Compton, Lin, Vargas, Carp, Fineman, and Quandt (2008) found no association between the ERN amplitude and depression scores, but did find an enhanced ERN was associated with more behavioral slowdown following errors in individuals with high levels of depression, than in non-depressed controls. Behavioral slowdown or post-error slowing refers to significant reaction time slowing on trials that follow errors (e.g., Hajcak, McDonald, & Simons, 2004). This slowing after errors is thought to be a compensatory mechanism to increase

performance following error commission (Gehring & Fencsik, 2001). The findings by Compton et al. (2008) suggest that this mechanism may be enhanced in depressed individuals.

These conflicting results could be due to a number of factors. First, the severity of depression differed widely across these studies. It is possible that there are complex associations between the ERN and depression that vary with severity or course of the disorder. Second, there is a high rate of co-occurrence between depression and anxiety (Fava, Abraham, Alpert, Nierenberg, Pava, & Rosenbaum, 1996; Fava, Rankin, Wright, Alpert, Nierenberg, & Pava et al., 2000; Zimmerman, Chelminski, & McDermut, 2002; Zimmerman, McDermut, & Mattia, 2000), raising the possibility that the findings are influenced by comorbid anxiety. Indeed, most of these studies included individuals with both depressive and anxiety disorders, but did not attempt to distinguish their influence or examine their joint effects on ERN amplitude (Chiu & Deldin, 2007; Compton et al., 2008; Holmes & Pizzagalli, 2008). In fact, Holmes and Pizzagalli (2008) did not directly examine the impact of anxiety on the ERN at all. Chiu and Deldin (2007) did not find a relationship between the ERN and anxious arousal or between the ERN and general anxiety. Compton et al. (2008) found a similar pattern of results when they entered worry scores as the predictor as they did when they used depression symptoms.

These results suggest that a more thorough investigation of the association between the ERN and depression and/ or anxiety is necessary. In order to further clarify both the nature of these associations and the potential function of the ERN, it is important to study a population that is at risk for depression and/ or anxiety, but that has not yet experienced clinically significant levels of these disorders. High risk studies are necessary in order to examine whether an enhanced ERN is associated with increased risk for internalizing disorders, rather than being a concomitant or consequence of these conditions.

The best-established risk factor for depression and anxiety is having a parent with these disorders (e.g., Beidel & Turner, 1997; Goodman & Gotlib, 1999). There appears to be only one previous study that has examined associations between parental psychopathology and the ERN. Fein and Chang (2008) found an inverse relationship between the size of the ERN demonstrated by alcoholics and the proportion of their

relatives with alcohol problems. These findings are consistent with evidence of a reduced ERN in persons with externalizing disorders and support the hypothesis that the ERN is a risk marker for some forms of psychopathology. Further, this study suggests the potential utility of examining the impact of other forms of familial psychopathology on probands' ERN. In particular, the growing evidence that it is possible to measure the ERN in young children suggests that the ERN might provide a window onto risk factors that precede the development of psychiatric disorders, which could facilitate earlier identification of at-risk children, leading to earlier interventions. Additionally, studying these associations in young children who are not disordered decreases the chance that findings of associations between the ERN and internalizing disorders are part of the emergence of psychopathology itself and instead suggests the possibility that the ERN itself is a risk marker. Hence, the current study will examine associations between the ERN and parental psychopathology in young children with depressed and/or anxious parents.

The ERN and Temperament

Several lines of evidence suggest that the ERN may be a trait-like variable. Olvet and Hajcak (2009) demonstrated the temporal stability of the ERN. Additionally, an enhanced ERN was elicited in children with anxiety disorders both before and after treatment (Hajcak et al., 2008; LaDouceur, Dahl, Birmaher, Axelson, & Ryan, 2007). Also, Moser et al. (2005) found that ERN amplitude did not change in individuals with a specific phobia before and during a fear induction task. Further, a number of studies have also found associations between the ERN and individual differences in temperament/personality. For example, as discussed below, data suggest that individuals who are high in negative emotionality (NE) demonstrate an enhanced ERN. Other studies have demonstrated an increased ERN amplitude in both adults and children who are highly socialized (Dikman & Allen, 2000; Santesso, Segalowitz, & Schmidt, 2005), which appears to be a similar construct to Conscientiousness. Further, Pailing and Segalowitz (2004) manipulated the size of the monetary reward and found that, although there was no main effect for error value, the amplitude of the ERN in individuals who were low in Conscientiousness was greater for errors that were more motivationally salient (i.e., more valuable). Conversely, individuals who are high in impulsiveness

exhibit a smaller ERN than those who are low in this factor (Ruchsow, Spitzer, Grön, Grothe, & Kiefer, 2005, although see Luu, Collins, & Tucker, 2000).

Negative Affect/ Negative Emotionality (NE). The relationship between an enhanced ERN and high levels of NA/ NE has been well-established (Hajcak et al., 2004; Luu et al., 2000; Pailing & Segalowitz, 2004, although see Santesso et al., 2005). These results suggest a possible mechanism by which the ERN is associated with internalizing disorders. According to Clark and Watson's tripartite model (1991), depression and anxiety are both associated with high levels of NE. This suggests that individuals with a variety of internalizing disorders should demonstrate an enhanced ERN.

NE is a complex construct that includes anger, fear, and sadness (e.g., Rothbart, Ahadi, & Evans, 2000). Thus, it is possible that there are more specific associations between these subcomponents of NE and the ERN. For example, Gray's (1982) motivational systems theory posits a Behavioral Inhibition System (BIS), which is associated with NE, particularly the fear/ anxiety component. There is some evidence to suggest that individuals who are high in punishment sensitivity (high in BIS activity) exhibit a larger ERN than individuals who are higher in reward seeking (Boksem, Tops, Wester, Meijman, and Lorist, 2006; although see Cavanagh & Allen, 2007). However, a follow-up study by Boksem and colleagues (2008) found a more complex relationship, with individuals who were high in punishment sensitivity demonstrating an enhanced ERN only during a task in which errors were punished. The current study will examine these relationships in young children in an attempt to further clarify the associations between the ERN and NE.

Positive Affect/ Positive Emotionality (PE). The tripartite model predicts that depression, but not anxiety, is associated with lower levels of PE (Clark & Watson, 1991). However, little work has been done to examine associations of the ERN with PE. The two studies that have directly examined this question both found no association between ERN amplitude and PE in either adults or children (Luu et al., 2000; Santesso et al., 2005). However, Grey's (1982) model also proposes a Behavioral Activation System (BAS), which is associated with PE, and Boksem et al. (2008) found that individuals who were higher in reward seeking (higher in BAS activity) exhibited an enhanced ERN during a task in which errors were associated with an inability to acquire rewards.

The tripartite model proposes that depression is associated with the combination of low PE and high NE. Unfortunately, there have been no studies of the relationship between the ERN and the joint effects of PE and NE. The current study will also examine these associations.

Behavioral Inhibition (BI). BI is also a temperament construct that has been associated with the later development of anxiety (e.g., Biederman, Hirshfeld-Becker, Rosenbaum, Hérot, Friedman et al., 2001; Gladstone, Parker, Mitchell, Wilhelm, & Malhi, 2005; Gladstone & Parker, 2006; Hirshfeld-Becker, Biederman, Henin, Faraone, Davis et al., 2007) and depression (e.g., Jaffee, Moffitt, Caspi, Fombonne, & Martin, 2002; Muris, Merckelbach, Schmidt, Gadet, & Bogie, 2001). Despite this link with psychopathology, there has been very little work examining associations between BI and the ERN. The one study that did investigate this found that individuals who were classified as high BI during childhood exhibited a more negative ERN during adolescence than those who were classified as low BI during childhood (McDermott, Perez-Edgar, Henderson, Chronis-Tuscano, Pine, & Fox, 2009). The current study will explore this association when the ERN is measured in a younger sample.

The Correct Response Negativity

On correct trials, there is a smaller ERN-like negative deflection, known as the Correct Response Negativity (CRN; Coles, Scheffers, & Holroyd, 2001; Falkenstein et al., 2000; Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000), similar to the ERN in terms of its morphology and scalp topography (Vidal et al., 2000). Although the functional significance of the CRN remains unclear (Coles et al., 2001; Suchan, Jokisch, Skotara, & Daum, 2007; Vidal et al., 2003), there is some evidence that increased cognitive and affective demands, but not evaluation of errors, modulate the amplitude of the CRN (Simon-Thomas & Knight, 2005). Importantly, Hajcak et al. (2005) found no difference in the amplitude of the CRN in high and low motivational conditions. Few studies have examined the CRN in young children, but those that have suggest that the CRN is larger in children than in adults (Davies et al., 2004; although see Santesso et al., 2006, whose results varied depending on the way in which they computed the CRN). Interestingly, the amplitude of the CRN

does not appear to differ between young and older adolescents (Santesso & Segalowitz, 2008). A goal of the current study is to further characterize the CRN in young children.

The CRN and Psychopathology

Anxiety disorders. Only a handful of studies have examined associations between the CRN and anxiety disorders. A CRN of higher amplitude was demonstrated in individuals with high levels of obsessive-compulsive characteristics (Hajcak & Simons, 2002) and in individuals with high levels of worry (Hajcak et al., 2003), suggesting that anxiety is associated with enhanced response monitoring, regardless of error commission. These findings were not replicated in adults (Ruchsow et al., 2005) or children with obsessive-compulsive disorder (Hajcak et al., 2008). Clearly, more work is needed to clarify this relationship.

Depression. The association between depression and the CRN has received very little attention in the literature. In fact, only Holmes and Pizzagalli (2008) focused on this relationship and they found that the CRN was comparable in depressed and non-depressed individuals.

The CRN and Temperament

The association between CRN amplitude and temperament has also been little studied. Hajcak et al. (2004) found a larger CRN in individuals high in NE, which suggests enhanced response monitoring that is not specific to actual error commission, in this population. No work has been conducted on the relationship between the CRN and PE, nor have the joint effects of PE and NE been examined. The current study will attempt to further clarify the nature of the CRN by examining its associations with parental psychopathology and temperament in young children.

The Error Positivity

Response-locked ERP studies have isolated a third component associated with response monitoring: a large positivity known as the Error Positivity (Pe), that appears within 200 – 500 ms following an erroneous response (Falkenstein et al., 2000; Santesso et al., 2006). A review by Overbeek, Nieuwenhuis, and Ridderinkhof (2005) concluded that although the exact function of the Pe is unknown, it appears to be independent of the ERN. Specifically, some source localization studies have demonstrated that the Pe is localized in more posterior regions than the ERN (Burgio-Murphy, Klorman, Shaywitz,

Fletcher, Marchione, Holahan et al., 2007; Ullsperger & von Cramon, 2006) and there is evidence that it is differentially affected by a number of factors, such as awareness of error commission (Nieuwenhuis et al., 2001), and task demands (Mathewson et al. (2005). However, like the CRN, it is influenced by degree of response conflict and the affective salience of the stimuli (Simon-Thomas & Knight, 2005). Finally, there is evidence that the Pe does not change over development at more posterior sites (Davies et al., 2004; Wiersema et al., 2007). However, Santesso et al. (2006) found that adults demonstrated a larger Pe than children at anterior sites. Additional work is necessary to further characterize the Pe in young children.

The Pe and Psychopathology

Anxiety disorders. Unlike the ERN, the Pe has not been consistently associated with anxiety disorders. For example, Ruchsow et al. (2005) found no difference in the amplitude of the Pe in adults with OCD and healthy controls. Similar findings have been demonstrated in comparisons of anxious and non-anxious children (Ladouceur et al., 2006; Hajcak et al., 2008). However, Santesso et al. (2006) found that children high in obsessive-compulsive behaviors had a larger Pe compared to children low in these behaviors.

Depression. As was discussed in a previous section, fewer studies have examined response monitoring in depression and only a subset of those have focused attention on the Pe. Preliminary evidence suggests that there is no relationship between Pe amplitude and depression (Chiu & Deldin, 2007; Compton et al., 2008; Holmes & Pizzagalli, 2008). Interestingly, Compton and colleagues (2008) found a slightly different scalp distribution for the Pe in undergraduates who were high and low in self-reported depressive symptomatology. For the group that endorsed few depressive symptoms, the Pe was greatest in a slightly more anterior location than it was for the group that endorsed many depressive symptoms, although the Pe was more anterior for both groups than has been found in the majority of the literature examining this component. No studies have examined associations between the Pe and comorbid depression and anxiety, nor has work been conducted on the relationship between the Pe and parental psychopathology.

The Pe and Temperament

The little work that has been conducted on the Pe and temperament suggests that temperament does not influence the amplitude of the Pe in the way that it is associated with the ERN. For example, Hajcak et al. (2004) found that the Pe was smaller in individuals high in NA compared to those who were low-NA; however, this relationship was found after both correct and error trials. The only study to date that has explicitly examined associations between the Pe and PE found no relationship between the two in children (Santesso et al., 2005). However, in the studies that have examined the BIS and BAS, individuals who were higher in reward sensitivity demonstrated a larger Pe than individuals who were lower (Boksem et al. 2006; although see Cavanagh & Allen, 2008). Boksem and colleagues (2008) further clarified this relationship by demonstrating a positive association between Pe amplitude and BAS when individuals committed an error during a task in which there would earn rewards for correct responses, and a negative association between the Pe and BAS when individuals were completing a task in which errors were punished. Finally, adults who were high and low in agreeableness did not demonstrate differences in this component (Tops, Boksem, Wester, Lorist, & Meijman, 2006); nor did children high and low in socialization (Santesso et al., 2005).

The Present Study

The results from these studies suggest the need for a more thorough description of these ERP components and an examination of individual differences in ERPs related to response monitoring in younger children. The current work will use a simple Go/ No-Go paradigm to characterize the ERN (defined as both the ERN – CRN difference and the ERN alone), CRN, and Pe (defined as both the Pe – Correct Trial Positivity and the Pe alone), and to examine associations between parental psychopathology, child temperament, and these ERP components in a large community sample of children between 5- and 7-years old. The first aim of the current study is to describe the ERP components in a young population and examine associations with demographic variables.

The second aim is to examine the relationship between parental depression and/ or anxiety and offspring ERP components, including the ERN – CRN, ERN, CRN, Pe – Correct Trial Positivity, and Pe. It is hypothesized that having a parent with an anxiety disorder or with depression will be associated with a larger ERN – CRN in children. Further, it is hypothesized that there will be an interaction between parental depression

and anxiety that will be associated with a more negative ERN - CRN amplitude in their children. Similar hypotheses are proposed for these associations with the ERN as were described for the ERN – CRN difference. It is hypothesized that having a parent with an anxiety disorder will be associated with an enhanced CRN in children, but this relationship is not expected for children with a depressed parent. Further, it is hypothesized that the CRN will be enhanced in children of parents with both depression and anxiety, but not larger than will be found for children with anxious parents only.

It is hypothesized that there will be no relationship between parental psychopathology and either the Pe – Correct Trial Positivity or the Pe.

A third aim of the present study is to examine the association between child temperament, specifically NE and PE, and the ERP components. It is hypothesized that children who have high levels of NE will demonstrate a larger ERN – CRN than children who have low levels of NE. It is hypothesized that there will be no relationship between the ERN – CRN and PE; however, it is hypothesized that there will be an interaction between NE and PE that will be associated with a larger ERN – CRN. This aim also includes examining the relationship between child temperament and ERN and CRN, separately. Similar predictions are proposed for the associations with the ERN as were described for the ERN – CRN difference. It is hypothesized that children who have high levels of NE will demonstrate a larger CRN than children who have low levels of NE. It is hypothesized that there will be no relationship between the ERN and PE. No relationship between child temperament and the Pe – Correct Trial Positivity or between child temperament and the Pe is expected.

A fourth aim is to explore whether child temperament moderates or mediates the relationship between parental psychopathology and offspring ERN – CRN and ERN. It is hypothesized that the interaction between child NE and PE will be the mechanism by which parental depression and/ or anxiety are associated with a larger ERN – CRN difference and an enhanced ERN.

II. Methods

Participants

Participants included 413 children (54.48% male, 45.52% female) from a suburban community who were assessed twice over a three year period. The mean age of

the children during the first assessment was 3.56 years (*SD* = .26 years, range = 2.93 – 4.18 years) and was 6.14 years (*SD* = .42 years, range = 5.15 – 7.57 years) during the second assessment. The mean age of parents during the first assessment was 36.24 years (*SD* = 4.47) for mothers and 38.52 years (*SD* = 5.52) for fathers. Participants were recruited via a commercial mailing list and were initially contacted by the Stony Brook University Center for Survey Research. Eligible families had a child between three and four years of age, with no significant medical conditions or developmental disabilities, and at least one English-speaking biological parent. The vast majority of the children was Caucasian (87.41%), came from two-parent homes (95.40%), had at least one parent who was a college graduate (69.25%), and had mothers who worked outside the home part- or full-time (53.03%). Children's mean scores on the Peabody Picture Vocabulary Test (*M* = 103.19, *SD* = 13.16) (PPVT; Dunn & Dunn, 1997) and the Expressive One-Word Picture Vocabulary Test (*M* = 100.78, *SD* = 12.42) (EOWPVT; Brownell, 2000) were in the average range.

Age 3 Assessment Procedures

Child Laboratory Assessment. The laboratory visit lasted approximately two hours, during which children participated in a standardized set of twelve laboratory episodes adapted from the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith, Reilly, Lemery, Longley, & Prescott, 1995). The episodes in the Lab-TAB were drawn from previous studies of child development spanning a number of different research questions (Kochanska & Knaack, 2003; Pfeifer, Goldsmith, Davidson, & Rickman, 2002). The selected episodes were designed to elicit different temperament traits or emotional displays. Between each episode, the child was given play breaks to return to a neutral state before entering a new situation. Each task was videotaped through a one-way mirror and later coded. The episodes are described below in the order that they were presented to the children.

Risk Room (BI; activity level). This episode was originally used in researching BI (Kagan, 1997). The episode allowed children to explore a set of novel and ambiguous stimuli, including a Halloween mask, balance beam, and a black box.

Tower of Patience (inhibitory control; interest). The child and experimenter alternated taking turns in building a tower together. The experimenter increased the

amount of time before placing her block on the tower during each of her turns. The child was made to wait until the experimenter took her turn.

Arc of Toys (PE; interest; NE). The child played independently with toys. After a few minutes, the experimenter asked the child to clean up the toys.

Stranger Approach (BI). The child was left alone briefly in the assessment room while the experimenter left to look for toys. In the experimenter's absence, a male research accomplice entered the room and spoke to the child while slowly walking closer.

Make that Car Go (PE, interest). The child and experimenter raced remote controlled cars.

Transparent Box (persistence, interest, NE). The experimenter locked an attractive toy in a transparent box. The child was then left alone with a set of keys to attempt to open the box. After a few minutes, the experimenter returned to the child and told him/her that she had left the wrong set of keys. The child was then encouraged to use the new keys to open the box and play with the toy.

Exploring New Objects (BI; activity level). This episode allowed the child to explore a set of novel and ambiguous stimuli, including a mechanical spider, a mechanical bird, and sticky water-filled soft gel balls.

Pop-up Snakes (PE). The child and experimenter surprised the child's mother with a can of potato chips that actually contained coiled snakes.

Impossibly Perfect Green Circles (NE, persistence). The experimenter repeatedly asked the child to draw a circle on a large piece of paper. Each attempt was mildly criticized.

Popping Bubbles (PE, interest). The child and experimenter played with a bubble-shooting toy.

Snack Delay (inhibitory control). The child was instructed to wait for the experimenter to ring a bell before eating a snack. The experimenter systematically increased the delay before ringing the bell.

Box Empty (NE). The child was given an elaborately wrapped box to open, under the impression that a toy was inside. After the child discovered that the box was empty, the experimenter returned with several small toys for the child to keep.

Tape Coding Procedures. Coding procedures followed those in previous studies (Durbin, Klein, Hayden, Buckley, & Moerk, 2005). Different coding methods were employed for positive and negative affect, the behavior variables, and behavioral inhibition. These ratings are associated with independent home observations and are moderately stable over time (Durbin, Hayden, Klein, & Olino, 2007).

With the exception of behavioral inhibition, all variables were coded during all 12 episodes. The rating of NE considered qualitative and quantitative aspects of displays of fear, anger, and sadness. Each instance of facial, vocal, and bodily anger, sadness, and fear was rated separately on a four-point intensity scale and then summed across each episode. Ratings of facial, vocal, and bodily anger, sadness, and fear were then each averaged across the 12 episodes to yield composite scores for anger ($\alpha = .68$), sadness ($\alpha = .81$), and fear ($\alpha = .63$). Interrater reliability, assessed using the intraclass correlation coefficient (ICC), for anger, sadness, and fear were .73, .79, and .64, respectively ($N = 35$).

NE was calculated by summing the ratings for anger, sadness, and fear, yielding a composite score of NE ($\alpha = .82$). Interrater reliability for NE was ICC = .74 ($N = 35$).

The ratings of positive affect considered qualitative and quantitative aspects of displays of joy and enthusiasm. Each instance of facial, vocal, and bodily positive affect was rated on a four-point intensity scale, and summed across each episode. Then the PA scores for each episode were averaged across the 12 episodes to yield a composite score of positive affect ($\alpha = .87$). Interrater reliability for positive affect was ICC = .92 ($N = 35$).

For the behavior rating of interest, which is related to the construct of PE, only a single rating was made per episode. This single rating was based on all behaviors thought to be relevant to interest during that episode. Global interest ratings ($\alpha = .68$) were based on how engaged the child appeared in play. Interrater reliability for interest was ICC = .75 ($N = 35$).

PE was calculated by summing the ratings for PA and interest, yielding a composite score of PE ($\alpha = .82$). Interrater reliability for PE was ICC = .89 ($N = 35$).

Most previous studies of BI have employed a micro-coding approach, using a small number of episodes specifically designed to elicit BI (Kagan, 1997; Pfeifer et al.,

2002). Thus, we used a micro-coding system for BI that incorporates variables from the three situations designed specifically to elicit BI (Risk Room, Exploring New Objects, and Stranger Approach). The micro-level coding system, based on Goldsmith et al. (1995), consisted of coding highly specific behaviors and emotions at 20-30 second intervals for each episode. A summary variable was computed for each variable coded in each episode by computing average ratings over the entire episode. Aggregate variables were then computed as averages across all episodes that coded that variable.

Variables from Risk Room and Exploring New Objects included latency to touch objects, total number of objects touched, tentative play, references and proximity to parent, references to experimenter, time spent playing, and latency to verbalize. A startle variable from Exploring New Objects was also included. Variables from Stranger Approach included gaze aversion, latency to vocalize, approach to and avoidance of stranger, and verbal/nonverbal interaction with stranger. Finally, variables included from all three episodes consisted of fearful facial, vocal, bodily affect, and latency to first fear response. The micro BI scale was comprised of an average of z-scored codes for these variables ($\alpha = .80$). Interrater reliability ($N = 28$) was ICC = .88.

Parental Psychopathology. Biological mothers and fathers of the children were interviewed during the assessment at age 3 using the Structured Clinical Interview for DSM-IV non-patient version (SCID-NP; First, Spitzer, Gibbon, & Williams, 1996). The SCID is currently one of the most widely used diagnostic instruments and has established acceptable levels of reliability and validity (Williams, Gibbon, First, Spitzer, Davies, Borus et al., 1992). Interviews were conducted by telephone, which generally yields comparable results to face-to-face interviews (Rohde, Lewinsohn, & Seeley, 1997; Sobin, Weissman, Goldstein, Adams, Wickramaratne, Warner et al., 1993). Two Masters-level raters conducted the diagnostic interviews. Based on audiotapes of 30 assessments (20 with mothers and 10 with fathers), inter-rater reliability (assessed with kappa) for presence/absence of a lifetime depressive disorder was .93; and for presence/absence of a lifetime anxiety disorder diagnosis was .91. When one parent could not be directly interviewed, diagnostic information on that parent was obtained by interviewing the co-parent using the family history method. The total number of mothers who completed the SCID was 409. The total number of fathers who either completed the SCID or for whom

the co-parent completed the family history interview was 406, with 350 fathers who completed the SCID and 56 fathers for whom the family history interview was conducted.

Age 6 Assessment Procedures

Child Laboratory Psychophysiological Assessment. Participants returned to the laboratory for the psychophysiological assessment when they were approximately 6 years old.

Task. A Go/ No-Go paradigm adapted from that described in Kim et al. (2007) was administered using Presentation software (Neurobehavioral Systems, Inc.). The stimuli were green equilateral triangles, 1.5 cm on each side, in four different orientations. There were a total of 240 trials, which were divided into 4 blocks of 60 trials each. In each block, 60% of the triangles were vertically aligned and pointed up, 20% were vertically aligned and pointed down, 10% were tilted slightly to the left, and 10% were tilted slightly to the right. All stimuli were presented on a black background.

Each trial started with the presentation of one of the four triangles for 1,200 ms in the middle of the monitor. Following this, a small gray fixation cross was displayed in the middle of the monitor for between 300 to 800 ms before the next trial commenced with the presentation of a new triangle. At the end of each block, the number of points won by the participant was displayed in white numbers. Figure 1 depicts the sequence of events for a trial.

Procedure. After the EEG sensors were attached, the children were taken into the recording chamber, which was decorated to look like a pirate ship (adapted and altered from Fox, Rubin, Calkins, Marshall, Coplan, Porges et al., 1995). The walls and ceiling of the room were lined with black fabric and covered with stickers of stars and planets. The child was instructed to sit in a large chair facing a computer monitor in the chamber.

A series of practice blocks were administered to ensure that the participant understood the various aspects of the task. First, each of the stimuli was presented on a card to the child. Participants were instructed to press a button with their thumb only when the vertically-aligned upward-pointing triangle was displayed (Go stimulus) and not to respond when the other three types of triangles (No-Go stimuli) or the fixation cross were presented. Participants were then presented with 8 triangles, (2 go stimuli, 6

no-go stimuli), and were given as much time as necessary to decide whether or not to press the button. Each Go trial ended when the participant pressed the response button. For the No-Go trials, an experimenter advanced to the next trial only when it was clear that the participant understood not to press the button when those stimuli were displayed.

The next practice block contained 20 trials. In addition to the triangles and fixation cross, participants also received feedback after each trial consisting of a "thumbs-up" or "thumbs-down" stimulus (1.5 cm^2) presented in the middle of the monitor, indicating whether their performance was correct or incorrect on the preceding trial. In addition to helping the participants learn to differentially respond to the triangle stimuli, the feedback stimulus also emphasized the importance of speedy response: if participants did not respond to Go stimuli within 1,300 msec, the thumbs-down feedback was presented.

The final practice block was identical to the task, as described above; however, there was no feedback to indicate whether the participant's responses were correct or incorrect on a trial-by-trial basis and there were 30 trials. Following completion of this practice block, the children were told that the actual game was going to begin and that for each block, they would earn one point for correct responses on Go trials and for withholding responses on No-Go trials. They were told that if they earned enough points, they could win up to $5.00. Speed of response was emphasized to the children. Between each block, the experimenter told the participants how many points they earned and reminded the children of the task instructions. If necessary due to poor performance, the children were shown the cards depicting the triangle stimuli and were instructed to tell the experimenter how they would respond to each stimulus. Additionally, the importance of response speed was re-emphasized before each block commenced. Following completion of the task, all children were told that they won the maximum number of points and received $5.00.

Psychophysiological Recording. Data was acquired using the Active Two system (Biosemi, Amsterdam, Netherlands). A stretch Lycra cap was placed on the child's head and 32 Ag/AgCl-tipped electrodes arranged according to the American Electroencephalographic Society labeling system (1994). A small amount of electrolyte (Signa Gel; Bio-Medical Instruments Inc., Warren, Michigan) was applied to the child's

scalp at each electrode position. The offsets of all electrodes were between ±20 μV; when necessary, the scalp was slightly abraded using a plastic syringe tip to reduce impedance. Additionally, flat electrodes were placed at supra and infra orbital sites of the right eye to monitor vertical eye movements and on the outer canthi of the left and right eyes to monitor horizontal eye movements; an electrode was also placed on the tip of the nose. All data was sampled at 512 Hz. Per BioSemi's design, the ground electrode during acquisition was formed by the common mode sense active electrode and the driven right leg passive electrode.

Offline, all data processing was performed 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. From the continuous EEG, 1500 ms segments were extracted beginning 500 ms prior to correct and erroneous responses. ERP data were corrected for blinks and eye-movements using the method developed by Gratton, Coles, and Donchin (1983). Additional artifacts were rejected when any of the following criteria are met: a voltage step of more than 50 mV between data points, a voltage difference of 300 mV within a single trial, or a voltage difference of less than .5 mV within 100 ms intervals. Data were also visually inspected for any remaining artifacts. ERP averages were then created separately for each trial type (correct and error) and were baseline corrected by subtracting from each data point the average activity in a 500- to 300- ms window prior to the response. Trials were not included in ERP averages if the reaction time occurred outside of a 200 – 1,300 ms window.

Consistent with previous studies, the ERN and CRN were evaluated along the midline (Fz, Cz, and Pz) and were defined as the average voltage in the window from 0 ms to 100 ms after the response. The ERN – CRN was computed by subtracting the average voltage on correct trials from the average voltage on error trials. The Pe was also evaluated along the midline and was defined as the average voltage in the window 200 ms to 500 ms following the response. The Pe – Correct Trial Positivity was computed by subtracting the average voltage on correct trials from the average voltage on error trials. For each study aim, the results of the analyses examining the ERN – CRN amplitude will be followed by the analyses separately examining the ERN and the CRN. The results of

the analyses examining the Pe – Correct Trial Positivity will be followed by the analyses separately examining the Pe.

In analyses aimed at examining the entire sample, behavioral measures were analyzed using repeated measures analysis of variance (ANOVA) and all ERP components were statistically evaluated using repeated measures ANOVA with the Greenhouse-Geisser epsilon correction (Jennings & Wood, 1976) applied to *p* values to counteract heterogeneity of variance-covariance matrices associated with repeated measures. Simultaneous regression analyses were used to examine associations between the behavioral measures, ERP components, parental psychopathology, and child temperament and hierarchical regression analyses were used to examine interaction effects.

III. Results

Aim 1: Characterize ERN – CRN, ERN, CRN, Pe – Correct Trial Positivity, and Pe in Young Children

Because Olvet and Hajcak (2009) found that 6 or more error trials are needed to demonstrate a stable ERN, data from 85 out of 413 (20.58%) children in total were excluded from further analyses (69 due to committing 5 or fewer errors, 16 subjects were excluded from analyses due to having 5 or fewer artifact-free error trials). Due to technical errors, the behavioral data from 7 participants were lost; however, the ERP data for these subjects were included in the analyses. The ERP data for one participant were lost due to technical error; however the behavioral data were included in the aggregate analyses. The ERP data from one additional participant were excluded due to values that were different from the grand mean of the data points by multiple standard deviations; however, the behavioral data were included in the aggregate analyses. This left a total of 321 subjects included in the behavioral analyses and 326 subjects included in the ERP analyses[1].

[1] Independent samples *t*-test indicated that children whose data were included in the final analyses were not significantly different from those children whose data were excluded in terms of child age at first assessment ($t = .87$, $p = ns$), child age at second assessment ($t = -1.61$, $p = ns$), PPVT score ($t = -.97$, $p = ns$), EOWPVT score ($t = -.94$, p = ns), or maternal age at first assessment ($t = -.21$, $p = ns$). Children included in the final analyses had slightly younger fathers than children who were excluded ($t = -2.26$, $p < .05$). Chi square analyses confirmed that there were no significant differences between groups in the proportion of children with mothers who worked at least part-time outside of the home, who came from two-parent homes, and who had at least one parent with a college degree. There were also no statistically

Behavioral Measures – Entire Sample

Table 1 presents RT and accuracy data for all participants. Both the number of errors and percentage of correct trials are presented because they provide different information. The number of errors refers only to errors of commission, whereas the percentage of correct trials accounts for both correct responses to Go stimuli and correct withholding of responses to No-Go stimuli. Post-hoc paired sample *t*-tests confirmed that reaction times on error trials were significantly faster than reaction times on correct Go trials ($t(1,320) = -34.41$, $p <. 001$). Post-hoc paired sample *t*-tests indicated that reaction times on correct trials that followed errors were significantly slower than all correct Go trials ($t(1,320) = 6.26$, $p <. 001$).

Response-Locked ERPs – Entire Sample

Error-Related Negativity (ERN) and Correct-Response Negativity (CRN)

The average ERP values for correct and error trials at the Fz, Cz, and Pz sites for both low- and high-value trials are presented in Table 2. Consistent with the impression from these data, a 2 (Region: Fz, Cz, and Pz) x 2 (Trial Type: Correct and Error) ANOVA was conducted and confirmed that both error and correct trial averages were more negative at the central site, $F(2,650) = 54.54$, $p < .001$. Consistent with the presence of the ERN on error trials, errors were associated with a greater negativity than correct trials, $F(1,325) = 280.17$, $p < .001$. Moreover, the difference between error and correct trials varied as a function of Electrode Site, $F(2, 650) = 98.35$, $p < .001$. Post-hoc paired sample *t*-tests indicated that the ERN was more negative than the correct trial average at all three electrode sites ($t(1,325) = -10.36$, $p <. 001$ at Fz; $t(1,325) = -18.09$, $p <. 001$ at Cz; $t(1,325) = -15.46$, $p <. 001$ at Pz). The difference between the error and correct trials (ERN – CRN) was larger at both Cz and Pz compared to Fz, ($t(1,325) = -11.77$, $p < .001$; $t(1,325) = -5.60$, $p < .001$, respectively) and was significantly larger at Cz compared to Pz, $t(1,325 = -3.98$, $p < .001$), suggesting that the maximum ERN- CRN difference was at Cz.

Post-hoc paired sample *t*-tests indicated that the ERN was more negative at Fz compared to Cz and Pz ($t(1,325) = -2.03$, $p < .05$; $t(1,325) = -4.74$, $p < .001$, respectively)

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significant differences between the number of boys and girls or child race/ethnicity between groups that were included and excluded from the final analyses.

and was significantly more negative at Cz compared to Pz ($t(1,325) = -4.24$, $p < .001$), suggesting that the ERN was most negative at Fz.

Post-hoc paired sample *t*-tests indicated that the CRN was less positive at Fz compared to Cz and Pz ($t(1,325) = -22.38$, $p < .001$; $t(1,325) = -16.15$, $p < .001$, respectively), but was not significantly different at Cz and Pz ($t(1,325) = -.61$, $p = ns$), suggesting that the CRN was least positive at Fz.

Subsequent analyses were focused on the ERN – CRN difference at Cz, and the ERN and CRN at Fz only because these were the sites at which they were most negative.[2]

Error Positivity (Pe) and Correct Trial Positivity – Entire Sample

The average Pe values at the Fz, Cz, and Pz sites for both low- and high-value trials are presented in Table 2. Consistent with the impression from these data, a 2 (Region: Fz, Cz, and Pz) x 2 (Trial Type: Correct and Error) ANOVA was conducted and confirmed that both error and correct trial averages were more positive at central and posterior sites, $F(2,650) = 56.72$, $p < .001$. Consistent with the presence of the Pe on error trials, errors were associated with a greater positivity than correct trials, $F(1,325) = 269.32$, $p < .001$. Moreover, the difference between error and correct trials varied as a function of Electrode Site, $F(2, 650) = 223.32$, $p < .001$. Post-hoc paired sample *t*-tests indicated that the Pe was more positive than the Correct Trial Positivity at all three electrode sites ($t(1,325) = 12.41$, $p <. 001$ at Fz; $t(1,325) = 14.93$, $p <. 001$ at Cz; $t(1,325) = 18.17$, $p <. 001$ at Pz). The difference between the error and correct trials was larger at both Pz and Cz compared to Fz, ($t(1,325) = 9.04$, $p < .001$, $t(1,325) = 6.44$, $p < .001$, respectively) and was larger at Pz compared to Cz, $t(1,325) = 5.40$, $p < .001$, suggesting that the maximum Pe – Correct Trial Positivity difference was at Pz. Subsequent analyses were focused on Pe – Correct Trial Positivity and Pe at Pz only because this was the site at which they were maximal.[3]

Associations Between Outcome Measures and Demographic Variables

[2] The results of the analyses using the ERN – CRN difference at Fz and Pz, and the ERN and CRN at Cz and Pz can be found in the Appendix.

[3] The results of the analyses using the Pe – Correct Trial Positivity difference and the Pe at Fz and Pz can be found in the Appendix.

Simultaneous regression analyses were used to explore the relationships between behavioral and ERP measures and several demographic variables, including child age at ERP assessment, child gender, child PPVT scores, and parent education.

Behavioral Measures

Total Correct Responses on Go Trials

Table 3 depicts the results of the analyses examining total correct responses on Go Trials. Higher levels of maternal education were significantly associated with more correct responses on Go Trials. There was no association between the other demographic variables and total correct responses on Go trials.

Reaction Time on Correct Responses to Go Trials

Table 3 depicts the results of the analyses examining reaction time on correct Go trials. Sex of child was associated with this outcome measure; specifically, girls were characterized by slower RTs on Go trials than boys. Child age was also significantly associated with RT: older children responded faster than younger children. Additionally, paternal education was associated with RT, with children with more educated fathers responding faster on correct Go trials than children with less educated fathers.

Total Correct No-Go Trials

Analyses examining total correct No-Go trials yielded no associations with the demographic variables (Table 3).

Total Errors of Commission

Analyses examining total errors of commission suggested there were no associations with the demographic variables (Table 4).

Reaction Time of Errors of Commission

Table 4 depicts the results of the analyses examining reaction time on errors of commission. Sex of child and child age at ERN assessment were associated with reaction time on errors of commission. Specifically, girls responded more slowly on errors of commission than boys, and older children had faster reaction times than younger children. There was a trend toward children with more educated fathers responding faster on errors of commission than children with less educated fathers.

Total Errors of Omission

Table 4 depicts the results of the analyses predicting total errors of omission, which were defined as responses occurring outside of the 200 – 1300 ms window. Sex of child and child age at ERN assessment were associated with total errors of omission; specifically, girls had more errors of omission than boys and older children had fewer errors of omission than younger children. Higher child PPVT scores and higher levels of paternal education were also significantly associated with fewer errors of omission.

Total Correct Go Trials Following Errors of Commission

Analyses examining total correct Go trials following errors of commission suggested there were no associations with the demographic variables (Table 5).

Reaction Time of Correct Go Trials Following Errors of Commission

Table 5 depicts the results of analyses examining reaction time on correct Go trials following errors of commission. Sex of child and child age at ERN assessment were significantly associated with reaction time on trials following errors of commission, with girls and younger children responding more slowly than boys and older children, respectively, on these trials.

Total Accuracy

Table 5 depicts the results of analyses examining total accuracy. Child age at ERN assessment, child PPVT score, and paternal education were all significantly associated with total accuracy, with older children, children with higher PPVT scores, and children with more educated fathers being more accurate than younger children, children with lower PPVT scores, and children with less educated fathers, respectively.

Response-Locked ERP Components

ERN – CRN

Table 6 depicts the results of the analyses examining ERN – CRN amplitude at Cz. There was a significant effect of child age, suggesting that older children demonstrated a more negative ERN – CRN than younger children.

ERN

Table 6 depicts the results of analyses examining ERN amplitude at Fz. Although there was a trend toward girls demonstrating a more negative ERN than boys, none of the demographic variables were significantly associated with ERN amplitude.

CRN

Analyses examining CRN amplitude at Fz suggested there were no associations with the demographic variables (Table 6).

Pe - Correct Trial Positivity

Table 7 depicts the results of analyses examining Pe – Correct Trial Positivity amplitude at Pz. Child PPVT score was significantly associated with Pe – Correct Trial Positivity difference; children with higher PPVT scores had a more positive Pe – Correct Trial Positivity difference than children with lower PPVT scores. There was also a trend toward older children demonstrating a greater Pe – Correct Trial Positivity difference than younger children.

Pe

Table 7 depicts the results examining associations with the Pe at Pz. There were significant effects of child age at ERN assessment and child PPVT score, such that older children and children with higher PPVT scores demonstrated a more positive Pe than younger children and children with lower PPVT scores, respectively.

Aim 2: Examine the relationship between parental depression and/ or anxiety and offspring ERP Components.

Simultaneous regression analyses were used to explore the relationships between behavioral and ERP measures and parental depression and/ or anxiety. Separate analyses were conducted to examine the impact of maternal and paternal psychopathology. Due to evidence suggesting that specific phobias may have a different underlying genetic structure than the other anxiety disorders (Hettema, Prescott, Myers, Neale, & Kendler, 2005) and a finding by Hajcak et al., (2003) that there was no difference in the ERN between phobic and non-phobic individuals, the impact of parental specific phobia on the amplitude of the ERN in their children was separately analyzed. In order to test the specificity of the association between parental internalizing psychopathology and increased ERN in offspring, we also examined the effects of parental substance use disorders. Based on Fein and Chang (2008)'s findings, we expected parental substance use disorders to be associated with diminished offspring ERN. Thus, maternal and paternal psychopathology were each defined by four variables: lifetime diagnosis of depression; lifetime diagnosis of anxiety disorder, excluding specific phobia; lifetime diagnosis of specific phobia; and lifetime diagnosis of substance dependence. Each set of

four parental psychopathology variables were entered simultaneously in each model.[4] Demographic variables that were significantly associated with specific outcome measure were included as covariates in the models.

Behavioral Measures

Total Correct Responses on Go Trials

Analyses examining total correct responses on Go trials suggested there were no associations with either maternal or paternal psychopathology (Table 8).[5]

Reaction Time on Correct Responses to Go Trials

Table 8 depicts the results of the analyses examining reaction time on correct responses on Go trials. Although there were no significant associations with parental psychopathology, there was a trend toward children of mothers with a history of substance dependence responding faster on Go trials than children whose mothers did not have a history of substance dependence.

Total Correct No-Go Trials

Analyses examining total correct No-Go trials suggested there were no associations with either maternal or paternal psychopathology (Table 8).

Total Errors of Commission

Analyses examining total errors of commission suggested there were no associations with the parental psychopathology (Table 9).

Reaction Time on Errors of Commission

Analyses examining reaction time on errors of commission suggested there were no associations with the parental psychopathology (Table 9).

Total Errors of Omission

Analyses examining total errors of omission suggested there were no associations with the parental psychopathology (Table 9), although there was a trend toward children

[4] Separate analyses were also conducted to examine the impact of having zero, one, or two parents with a lifetime diagnosis of depression; zero, one, or two parents with a lifetime diagnosis of anxiety disorder (excluding specific phobia); zero, one, or two parents with a lifetime diagnosis of specific phobia; and zero, one, or two parents with a lifetime diagnosis of substance dependence. Most of the results were identical to those obtained when maternal psychopathology variables were included in the model; however, footnotes are included when the results differed. Additionally, all results for these analyses are in Appendix I.

[5] There was an effect of parental loading of an anxiety disorder that approached significance when analyses examining parental loading of psychopathology were conducted. Specifically, there was a trend toward more total correct responses on Go trials being associated with increased parental loading of an anxiety disorder.

of mothers with a lifetime history of depression responding less frequently within the 200 – 1300 ms window than children whose mothers did not have a history of depression.

Total Correct Go Trials Following Errors of Commission

Analyses examining total correct Go trials following errors of commission suggested there were no significant associations with parental psychopathology (Table 10), although there was a trend toward children with mothers or fathers who had a history of depression having fewer correct Go trials following errors of commission than children whose mothers or fathers did not.

Reaction Time of Correct Go Trials Following Errors of Commission

Table 10 depicts the results of analyses examining reaction time on correct Go trials following errors of commission. Maternal substance abuse was significantly associated with reaction time following errors of commission, with children whose mothers had a history of substance dependence responding faster than children whose mothers did not. Interestingly, there was an association with paternal substance dependence in the opposite direction that was significant, such that children whose fathers had a lifetime diagnosis of substance dependence responded more slowly on trials following errors than children whose fathers did not have a substance dependence history.

Total Accuracy

Analyses examining total accuracy suggested there were no associations with the parental psychopathology (Table 10).

Response-Locked ERP Components

ERN – CRN

Table 11 depicts the results of the analyses examining ERN – CRN amplitude at Cz. There was a significant effect of maternal anxiety, suggesting that children who had mothers with a lifetime history of an anxiety disorder demonstrated a less negative ERN – CRN difference than children whose mothers did not have an anxiety disorder. A similar association between the ERN – CRN difference and maternal specific phobia approached significance.[6] There were no significant associations with paternal psychopathology.

[6] This trend was significant when the model included parental loading of psychopathology variables.

ERN

Table 11 also depicts the results of analyses examining ERN amplitude at Fz. There was a significant association between ERN amplitude and maternal anxiety, with children whose mothers had a history of an anxiety disorder demonstrating a less negative ERN than children whose mothers did not have an anxiety disorder. There were no significant associations with paternal psychopathology.

CRN

Analyses examining CRN amplitude at Fz suggested a significant association with maternal depression; specifically, children who had mothers with a history of a depression demonstrated a less negative CRN than children whose mothers did not have depression (Table 11). There was also an association with maternal specific phobia that approached significance, suggesting that children who had mothers with a lifetime history of a specific phobia trended toward demonstrating a more negative CRN than children whose mothers did not have a specific phobia. There were no associations with paternal psychopathology.

Pe - Correct Trial Positivity

Analyses examining the Pe – Correct Trial Positivity difference at Pz suggested there were no significant associations with either maternal or paternal psychopathology (Table 12).

Pe

Analyses examining Pe amplitude at Pz suggested there were no significant associations with either maternal or paternal psychopathology (Table 12).

Aim 3: Examine the relationship between child temperament and ERP components.

Several simultaneous regression analyses were conducted to examine associations between child temperament variables and several behavioral measures. The two broader temperament constructs of positive and negative emotionality (PE and NE, respectively), were the predictor variables in one model. In order to examine whether the constructs from which NE is comprised differentially predict behavioral measures, the second regression model included sadness, fear, and anger, as well as PE. Another model

included PE and BI, instead of NE.[7] Demographic variables that were significantly associated with specific outcome measure were included as covariates in the models.

Behavioral Measures

Total Correct Responses on Go Trials

Analyses examining total correct responses on Go trials suggested there were no significant associations with child temperament (Table 13), although there was a trend toward children with higher levels of anger having more total correct responses on Go trials than children with lower levels of anger.

Reaction Time on Correct Responses to Go Trials

Analyses examining reaction time on correct responses to Go trials suggested there were no associations with child temperament (Table 13).

Total Correct No-Go Trials

Table 13 depicts the results of analyses examining total correct No-Go trials. There was a significant effect of child anger, with children who demonstrated higher levels of anger exhibiting fewer correct No-Go trials than children with lower levels of anger.

Total Errors of Commission

Analyses examining total errors of commission suggested there were no associations with child temperament (Table 14), although there was an effect of child BI that approached significance. Specifically, children who exhibited higher levels of BI demonstrated fewer total errors of commission than children who exhibited lower levels of BI.

Reaction Time on Errors of Commission

Analyses examining reaction time on errors of commission suggested there were no associations with child temperament (Table 14), although there was an effect of child sadness that approached significance. Specifically, children who exhibited higher levels of sadness demonstrated faster reaction times on errors of commission than children who exhibited lower levels of sadness.[8]

Total Errors of Omission

[7] All analyses were repeated using positive affect (PA) as a predictor variable in place of PE. As was described above, PE is comprised of both PA and child interest. Results were similar, except where noted.
[8] The effect of child sadness was significant when the model included PA instead of PE.

Table 14 depicts the results of analyses examining total errors of omission. There was a significant effect of NE, with children who exhibited higher levels of NE committing more errors of omission than children who exhibited lower levels of NE. There was a main effect of sadness as well, suggesting that children who exhibited more sadness responded less frequently within the 200 – 1300 ms window than children who exhibited less sadness.

Total Correct Go Trials Following Errors of Commission

Analyses examining total correct Go trials following errors of commission suggested there were no significant associations with child temperament (Table 15), although there was a trend toward children with higher levels of BI demonstrating fewer total correct Go responses following errors of commission than children with lower levels of BI.

Reaction Time of Correct Go Trials Following Errors of Commission

Analyses examining reaction time on correct Go trials following errors of commission suggested there were no associations with child temperament (Table 15).

Total Accuracy

Table 15 depicts the results of analyses examining total accuracy. There were significant effects of NE and sadness. Specifically, children who exhibited higher levels of NE and sadness were less accurate than children who exhibited lower levels of NE and sadness, respectively.

Response- Locked ERP Components

ERN – CRN

Table 16 depicts the results of the analyses examining ERN – CRN amplitude at Cz. Child NE was significantly associated with the ERN – CRN difference; specifically, higher levels of NE predicted smaller (i.e., less negative) ERN amplitude than lower levels of NE. Fear was also significantly associated with ERN – CRN amplitude, with higher levels of fear associated with a smaller (i.e., less negative) ERN – CRN difference.

ERN

Table 16 depicts the results of analyses examining ERN amplitude at Fz. There was a significant association between ERN amplitude and NE, with children who exhibited higher levels of NE demonstrating a less negative ERN than children who

exhibited lower levels of NE.[9] There was an association with fear that approached significance, with higher levels of fear associated with a less negative ERN.

CRN

Analyses examining CRN amplitude at Fz suggested there were no significant associations with child temperament (Table 16).

Pe - Correct Trial Positivity

Analyses examining the Pe – Correct Trial Positivity difference at Pz suggested there were no significant associations with child temperament (Table 17), although there was a trend toward children who exhibited higher levels of sadness demonstrating a smaller (i.e., less positive) Pe – Correct Trial Positivity difference.

Pe

Analyses examining Pe amplitude at Pz suggested there were no significant associations with child temperament (Table 17).

Associations between ERN - CRN Amplitude and Interaction between PE and NE

A series of hierarchical regression analyses were conducted to examine the effects of the interaction between PE and NE on ERN – CRN difference at Cz. Because age had been significantly associated with ERN – CRN amplitude, this was included in the models as an independent variable and in interaction terms with PE and NE. Additionally, the cross-product of PE and NE was included in the analyses. As depicted in Table 18, there were significant main effects of NE and child age at ERN assessment. There were no other significant effects, suggesting that the interaction between NE and PE is not associated with the ERN – CRN difference. [10]

Associations between ERN Amplitude and Interaction between PE and NE

A series of hierarchical regression analyses were conducted to examine the effects of the interaction between PE and NE on ERN amplitude at Fz (Table 19). PE and NE were included in the models as independent predictors, as well as their cross-product. There was a significant main effect of NE; however, there were no other significant

[9] When PA was used in the model instead of PE, the effect of NE approached significance.

[10] These analyses were also conducted using PA instead of PE. Results were identical. Analyses were also conducted excluding age from the model. NE continued to significantly predict ERN amplitude; there continued to be no main effect or interaction involving PE. Child BI was included in the model in place of child NE. There was a main effect of child age at assessment; however, there were no other main or interaction effects.

effects, suggesting that the interaction between NE and PE is not associated with ERN amplitude. [11]

Aim 4: Explore whether child temperament moderates or mediates the relationship between parental psychopathology and offspring

Child NE as a Moderator of Relationship Between Maternal Anxiety and Child ERN – CRN Difference

Hierarchical regression analyses were conducted to examine the effects of the interaction between child NE and maternal anxiety on the ERN – CRN difference at Cz (Table 20). As with the above analyses, because age had been significantly associated with ERN – CRN amplitude, this was included independently in the first step of the model and in cross-product terms with maternal anxiety and child NE, in addition to the cross-product of maternal anxiety and child NE, in the second step of the model. There was a main effect of maternal anxiety; however, there were no other main or interaction effects suggesting that child NE does not moderate the association between maternal anxiety and the ERN – CRN difference. These analyses were repeated with child age at assessment, maternal anxiety, and child NE included as independent predictors, but the model did not include a second step with cross-product terms. There were main effects of all three variables (maternal anxiety: b = 2.63, S.E. = 1.10, $t = 2.39$, $p < .05$; child NE: b = 4.58, S.E. = 1.91, $t = 2.40$, $p < .05$; child age at ERN assessment: b = -2.86, S.E. = 1.15, $t = -2.48$, $p < .05$).[12]

Child NE as a Moderator of Relationship Between Maternal Anxiety and Child ERN Amplitude at Fz

Hierarchical regression analyses were conducted to examine the effects of the interaction between child NE and maternal anxiety on the ERN amplitude at Fz (Table 21). Maternal anxiety and child NE were included in the models as independent predictors, as well as their cross-product, in the second step of the model. There was a main effect of maternal anxiety; however, there was no main effect of child NE and no

[11] These analyses were also conducted using PA instead of PE and the results were identical. Results were also similar when child BI was included in the model instead of child NE and PA was included instead of PE. However, when PE was included in the model with BI, the interaction between PE and BI approached significance ($b = -1.17$, S.E. = .69, and $t = -1.70$, $p < .10$).

[12] Analyses were repeated using parental loading of a lifetime anxiety disorder instead of maternal anxiety. Results were identical.

significant interaction between NE and maternal anxiety suggesting that child NE does not moderate the association between maternal anxiety and the ERN. These analyses were repeated with maternal anxiety and child NE included as independent predictors, but the model did not include a second step with cross-product terms. There were main effects of both variables (maternal anxiety: b = 3.42, S.E. = .99, t = 3.47, p = .001; child NE: b = 3.45, S.E. = 1.71, t = 2.02, $p < .05$).[13]

Child Fear as a Moderator of Relationship Between Maternal Anxiety and Child ERN – CRN Difference

Hierarchical regression analyses were conducted to examine the effects of the interaction between child fear and maternal anxiety on the ERN – CRN difference (Table 22). As with the analyses examining associations with child NE and maternal anxiety, because age had been significantly associated with ERN – CRN amplitude, this was included in the models as an independent variable in the first step of the model and in cross-product terms with maternal anxiety and child fear, in addition to the cross-product of maternal anxiety and child fear, in the second step of the model. There was a main effect of maternal anxiety; however, there were no other main or interaction effects suggesting that child fear does not moderate the association between maternal anxiety and the ERN – CRN difference. These analyses were repeated with child age at assessment, maternal anxiety, and child fear included as independent predictors, but the model did not include a second step with cross-product terms. There were main effects of all three variables (maternal anxiety: b = 2.71, S.E. = 1.10, t = 2.46, $p < .05$; child fear: b = 3.26, S.E. = 1.39, t = 2.34, $p < .05$; child age at ERN assessment: b = -3.11, S.E. = 1.15, t = -2.70, $p < .01$).[14]

Child NE, Child Fear, and Child BI as Potential Mediators of the Relationship Between Maternal Anxiety and Child ERN

[13] Analyses were repeated using parental loading of a lifetime anxiety disorder instead of maternal anxiety. Results were identical.

[14] Analyses were repeated using parental loading of a lifetime anxiety disorder instead of maternal anxiety. Results were identical.

There was no significant correlation between maternal anxiety and child NE, fear, or BI (r = .03, .00, and -.01, respectively), which precluded mediational analyses.[15]

Discussion

Aim 1: Characterize ERN – CRN, ERN, CRN, Pe – Correct Trial Positivity, and Pe in Young Children

The current study used a simple Go/No-Go paradigm to examine ERP components related to response monitoring in a sample of 328 children who were between 5- and 7- years old and demonstrated that an ERN can be elicited in a young population. The ERN – CRN was both temporally and spatially similar to the ERN – CRN that has been described in the adult literature (e.g., Cavanagh & Allen, 2008; Johannes et al., 2001; Ruchsow et al., 2006), peaking between 0 and 100 ms after the response and having a central maximum. Although the ERN – CRN difference was largest at Cz, the ERN alone was most negative at Fz. This is consistent with other findings in adults (e.g., Brázdil et al., 2005; Hajcak & Simons, 2002; Holmes & Pizzagalli, 2008) and children (e.g., Kim et al., 2005).

Prior to this study, there had been mixed evidence regarding whether or not an ERN could be reliably elicited in children (Davies et al., 2004; Santesso et al., 2006; Kim et al., 2007; Wiersema et al., 2007) and only one study had examined a sample so young (Torpey et al., 2009). The current results suggest that these mixed findings may have been the result of task complexity and that the ERN can be demonstrated in young children if the task is simple enough.

Despite the fairly narrow age range of the population, there was a developmental effect, with older children demonstrating a more negative ERN – CRN. These findings are consistent with other studies that have examined the development of the ERN over the lifespan (Davies et al., 2004; Kim et al., 2007 (although they did not find a difference between the ERN in 7-11 year-olds and adults, older children demonstrated a more negative ERN than younger children); Santesso et al., 2006; Santesso & Segalowitz, 2008; Wiersema et al., 2007). These results support previous findings that suggest that these developmental changes are due to the later maturation of the ACC, from where it

[15] Correlations between parental loading for an anxiety disorder and child NE, fear, and BI were also assessed and were not significant (rs = .03, .02, and -.01, respectively). Because of this, no mediational analyses were conducted.

has been hypothesized the ERN originates (e.g., Mathewson et al., 2005; Mathalon et al., 2005; Brázdil et al., 2005).

As has been demonstrated in previous studies (e.g., Vidal et al., 2000), the CRN was less negative than the ERN but was similar to the ERN both temporally and in terms of scalp topography. Similar to the findings by Santesso and Segalowitz (2008) that the amplitude of the CRN was not different between young and older adolescents, this study also found no association between CRN amplitude and child age.

The present study found that young children demonstrate a Pe that is spatially and temporally similar to the Pe described in the adult literature (e.g., Falkenstein et al., 2000; Ullsperger & von Cramon, 2006), peaking between 200 and 500 ms after error commission and maximal in the posterior. However, although prior research has suggested that the Pe does not change over development (Davies et al., 2004; Wiersema et al., 2007), the current study found that older children demonstrate a more positive Pe than younger children. There is some evidence that the Pe is attenuated in participants who commit more errors (e.g., Falkenstein et al., 2000) and, in the present sample, younger children were less accurate than older children, although this appears to be due to more errors of omission, potentially explaining this developmental effect.

Pe amplitude was also positively correlated with PPVT score, suggesting that Pe amplitude is affected by cognitive ability. This is consistent with other work that has found associations between the Pe and awareness of error commission (Nieuwenhuis et al., 2001), as it is likely that more cognitively advanced children better understood the task and when they were committing errors. It is also likely that children with higher PPVT scores understood and were more motivated by the abstract association between errors and not acquiring points that would later be redeemed for money. This would be evidence supporting other findings that the Pe is associated with the affective salience of an error (Simon-Thomas & Knight, 2005). The current associations with PPVT scores also support the hypothesis that Pe amplitude is inversely correlated with task difficulty (Mathewson et al., 2005). Specifically, it seems likely that children who were less cognitively advanced were more challenged by the task demands (and perhaps were less aware when they committed an error), which led to a smaller Pe.

The behavioral associations demonstrated by this young population were also similar to those found in adults and older children. Specifically, the children in this study responded more quickly on error than correct trials, suggesting increased impulsivity on these trials (e.g., Hajcak et al., 2005; Luu et al., 2003; Santesso et al., 2006; Wiersema et al., 2007). Additionally, the young participants demonstrated post-error slowing, which is thought to be a compensatory mechanism to increase performance following error commission (e.g., Davies et al., 2004; Falkenstein et al., 2000, Gehring & Fencsik, 2001; Hajcak et al., 2004; Santesso et al., 2006).

There were also a number of interesting associations between the behavioral measures and the demographic variables. Older children, children with higher PPVT scores, and children with more educated fathers were more accurate than younger children, children with lower PPVT scores, and children who had fathers with fewer years of education, respectively. Importantly, none of these variables were associated with total errors of commission, total correct responses on Go trials, or total correct No-Go trials. Only maternal education was positively associated with total correct Go trials, with children who had more educated mothers having more total correct responses on Go trials than children with less educated mothers. However, age, PPVT score, and paternal education were all associated with total errors of omission/ responses that were outside of the 200 -1300 ms window. Specifically, younger children, children with lower PPVT scores, and children with less educated fathers committed more errors of omission than older children, children with higher PPVT scores, and children with fathers who were more educated, respectively, suggesting that the differences in total accuracy may be due to the total errors of omission. Girls also committed more errors of omission than boys did. It remains unclear whether these errors are absolute response omissions or if they are due to responses that were made too quickly or too slowly. It is not possible to determine this because there are no reaction times available for errors of omission.

In terms of reaction times, boys responded faster than girls on correct responses, errors, and correct Go responses immediately following errors of commission. Older children also responded faster on correct, error, and Go trials following errors of commission trials. Paternal, but not maternal, education was inversely associated with reaction times as well. Taken together with the absence of differences in the accuracy of

these three types of responses, these findings may suggest that boys, older children, and children with more educated fathers were able to complete the task with greater ease than girls, younger children, and children with less educated fathers, respectively. Few studies have examined sex differences, although Kim and colleagues (2007) also found that boys tended to respond faster than girls. There is significant evidence that age is inversely correlated with reaction times and this has been found even in examinations of children (e.g., Davies et al., 2004; Wiersema et al., 2007). For example, Kim et al. (2005) demonstrated that 9 – 11 year-olds had faster reaction times than 7 – 8 year-olds. The present results are in contrast to those found by Santesso and Segalowitz (2008) in which older adolescents demonstrated more post-error slowing than younger adolescents and by Hogan et al. (2005), in which post-error slowing increased with age in adolescents and adults. However, the current sample was significantly younger than those participants, and spanned a much smaller age range, and it is possible that the youngest children were unable to respond any faster.

Aim 2: Examine the relationship between parental depression and/ or anxiety and offspring ERP Components.

Associations between the ERN – CRN and ERN and Maternal Psychopathology

This was the first study to examine associations between parental psychopathology and the ERN in children. Interestingly, although there were associations with maternal anxiety, they were in the opposite direction as was hypothesized. Because of studies that suggested that clinical (Gehring et al., 2000; Hajcak et al., 2008; Johannes et al., 2001; Ladouceur et al., 2006; Ruchsow et al., 2005) and subclinical (e.g., Hajcak & Simons, 2002; Santesso et al., 2006) levels of anxiety are associated with a more negative ERN, the current results finding a less negative ERN in children of anxious mothers were not predicted. However, it is important to note that many offspring of mothers with an anxiety disorder do not go on to develop anxiety disorders themselves (e.g., Li, Sundquist, & Sundquist, 2008), hence studies of at-risk samples may not always produce results that are consistent with studies of individuals with the disorder. Nonetheless, it is very surprising that the findings from these two populations would be in opposite directions.

A possible explanation of these findings is that the present results reflect the differential maturation of the rostral and dorsal areas of the ACC. Previous findings have suggested that differentiating between these two areas in an ERP study was dependent on the nature of the task used to elicit the ERN. Gründler et al. (2009) found that individuals who were high in obsessive-compulsive symptoms demonstrated an enhanced ERN when they committed errors during a flanker task, but exhibited a less negative ERN when they committed errors during a probabilistic learning task. These findings led Gründler and colleagues to hypothesize the existence of two different neural mechanisms that are responsible for the ERN, one of which is associated with learning to avoid maladaptive choices and may be localized to the rostral ACC, whereas the other allows the same behavior (the erroneous response) to be repeated despite their failure to produce a desired outcome and may be localized to the dorsal ACC. Specifically, they posited that in individuals who are high in obsessive-compulsive symptoms, there is hypoactivity in the system that is associated with avoidance and hyperactivity in the system that is associated with the perpetuation of a response that has been undesirable. Go/No-Go and Flanker paradigms may create conflict primarily in the hyperactive response perpetuating system in adults, whereas probabilistic learning paradigms may create conflict primarily in the hypoactive avoidance system, but both of these systems are dysfunctional in adults with obsessive-compulsive symptoms.

There is evidence to suggest that the mechanism of error processing changes over the course of development due, in part, to the maturation of the ACC. Velanova, Wheeler, and Luna (2008) examined the development of the ACC from middle childhood through adulthood and found that the rostral ACC showed significant deactivation during correct but not error trials for all ages, whereas only adults exhibited dorsal ACC activation for error versus correct trials. It may be that Go/No-Go and flanker paradigms actually do affect both the rostral and dorsal ACC in adolescents and in adults, but that the rostral ACC hypoactivity is not detected because of the hyperactivity in the dorsal ACC, which leads to the enhanced ERN. However, the dorsal ACC may not be developed well enough in children as young as those in the current sample and only the hypoactivity in the rostral ACC is detected, despite the nature of the paradigm. This would suggest that a maternal history of an anxiety disorder is associated with a

dysfunctional rostral ACC. Interestingly, although this theory would lead to the expectation that children with mothers who had a history of an anxiety disorder would demonstrate significantly more correct No-Go trials and commit fewer errors of commission, these neural differences were not associated with changes in behaviors, which is consistent with other studies, including Gründler et al. (2009).

Future studies are necessary to investigate whether these children demonstrate an enhanced ERN during Go/No-Go paradigms at an older age when the dorsal ACC has more fully developed. Further, comparisons of the ERN using Go/No-Go and probabilistic learning paradigms need to be conducted in these children to determine if maternal history of anxiety is specifically associated with rostral ACC dysfunction or affects the dorsal ACC over the course of development as well.

There was a similar relationship between maternal specific phobia and the ERN – CRN, although this appears to be due to children of mothers with a history of a specific phobia demonstrating a more negative CRN. This will be discussed below. There was no relationship with the ERN alone, providing further evidence that there is a difference between specific phobias and other anxiety disorders. Only one prior study was conducted in which the ERN was compared in individuals with and without a specific phobia (Hajcak et al., 2003) and the current results are similar to those findings with maternal specific phobia being unrelated to ERN amplitude.

There were no associations between maternal depression and the ERN – CRN or the ERN. The relationship between the ERN and depression has not been clear, with some evidence suggesting that depressed subjects exhibit a more negative ERN than non-depressed individuals (Holmes & Pizzagalli, 2008; Chiu & Deldin, 2007), that patients with remitted depression demonstrate a less negative ERN, but only on error trials that follow error trials (Ruchsow et al., 2004), and that there is no association between depression and the ERN (Compton et al., 2008).

There were also no associations between a history of maternal substance abuse or dependence and the ERN – CRN or the ERN. This was more surprising, given the Fein and Chang (2008) study which demonstrated a smaller ERN was associated with increased family loading of alcohol dependence. There were several differences between the Fein and Chang (2008) work and the present study; most importantly, Fein and Chang

measured a feedback-ERN in adult participants who were actively alcohol dependent. Notably, only about 1/5 of the mothers in the current sample met criteria for a history of substance abuse or dependence and the majority of those that did were abused alcohol for a finite period in late adolescence, suggesting a much less severe, and potentially less heritable, disorder than was examined by Fein and Chang (2008), which may impact the association with ERN amplitude.

Associations between the CRN and Maternal Psychopathology

There were also associations between the CRN and maternal psychopathology. Because some previous work suggested that a more negative CRN may be associated with high levels of obsessive-compulsive symptoms (Hajcak & Simons, 2002) and high levels of worry (Hajcak et al., 2003), the current finding that there was no relationship between maternal anxiety and the CRN was not predicted. As was discussed above, this could be because having an anxious mother does not always result in offspring anxiety. However, although this relationship was not significant, children who had mothers with a history of a specific phobia did demonstrate a more negative CRN. This suggests that a maternal history of a specific phobia may be associated with increased response monitoring, independent of error commission. In contrast, maternal depression was associated with a less negative CRN. This association, in combination with the behavioral findings that maternal depression was non-significantly associated with fewer correct Go responses following errors of commission and more errors of omission, suggest that a history of maternal depression may be associated with decreased response monitoring in young offspring, particularly following error commission. This finding was also not predicted because of previous results in which the CRN was comparable in depressed and non-depressed adults (Holmes & Pizzagalli, 2008).

It was hypothesized that there would be an interaction between parental depression and anxiety that would be associated with an enhanced ERN in their children; however, this was not supported by the data. There are several potential explanations for this, including that depression does not affect the ERN at all. Alternatively, it is possible that the differential maturation of the rostral and dorsal ACC may result in associations that are not detectable until later in development. Future research will have to clarify this.

Associations between ERN – CRN, ERN, CRN, and Paternal Psychopathology

It is notable that there were no associations with paternal psychopathology. One explanation for this could be that maternal psychopathology appears to have a greater impact on offspring than parental psychopathology. A meta-analysis by Connell and Goodman (2002) found that maternal psychopathology was more strongly related to both internalizing and externalizing problems in young children. Connell and Goodman (2002) proposed that this relationship could be due to the more prominent role mothers have in the care of younger children and greater influence on their early development. This is consistent with the current sample, in which almost all of the fathers worked outside of the home at least part-time; whereas only slightly over half of the mothers did, suggesting they were devoting more time to child-care. Connell and Goodman (2002) found that paternal psychopathology has a greater impact on offspring internalizing and externalizing problems during adolescence, when fathers have a larger influence on child-care and it will be important to continue to examine this as the sample ages.

Associations between the Pe and Parental Psychopathology

As was hypothesized, there were no associations between the Pe and either maternal or paternal psychopathology. The current results are consistent with other studies, which have found no association between the Pe and anxiety disorders in adults (Ruchsow et al., 2005) or children (e.g., Hajcak et al., 2008; Ladouceur et al., 2006; although see Santesso et al., 2006), or between the Pe and depression (Chiu and Deldin, 2007; Compton et al., 2008; Holmes & Pizzagalli, 2008).

Associations between Behavioral Measures and Parental Psychopathology

Because most previous research did not demonstrate associations between performance and subclinical (e.g., Hajcak & Simons, 2002; Hajcak et al., 2003) and clinical anxiety in adults (e.g., Johannes et al., 2001; Ruchsow et al., 2005) and children (e.g., Ladouceur et al., 2006), the current findings of no relationships between the behavioral measures and parental anxiety disorders were predicted.

The behavioral associations with maternal depression were discussed above in order to clarify the relationship with CRN; although, it is notable that children with mothers who had a history of depression did not exhibit increased post-error slowing.

Because increased post-error slowing had been demonstrated in depressed adults by Compton and colleagues (2008), the current findings were not predicted.

Although there were no associations between the ERP components and parental substance abuse or dependence, there were several associations with reaction time. Maternal substance abuse or dependence was associated with decreased post-error slowing, which was predicted because of results found by Stieben et al. (2007), in which externalizing children demonstrated less post-error slowing than both control children and children who had both internalizing and externalizing symptoms. Notably, children who had fathers with a history of substance abuse or dependence exhibited increased post-error slowing. As described above, these conflicting findings may be related to restricted range in the severity of the disorder in this population. Like the mothers, the majority of the fathers met criteria for alcohol abuse and did so during late adolescence for a finite time period. It is unclear whether the results of the current analyses would have been different if more of the parents, especially the fathers, had a more extensive substance dependence history.

One of the limitations of this study is that parental psychopathology was assessed 2 – 3 years before the children completed the ERN task. It seems possible that the results may have differed had the parents met criteria for a disorder at the time of assessment. Future work that examines the ERN and CRN in children who have parents with current and/or more extensive histories of psychopathology could further clarify the relationships that were identified here. Additionally, it will be important to monitor the development of psychopathology in the children themselves because only a subset of children of parents with a psychological disorder will develop a disorder themselves, and to continue to examine associations with the ERN, CRN, and behavioral measures.

Aim 3: Examine the relationship between child temperament and ERP components.

Associations between the ERN – CRN, ERN, and Temperament

As predicted, the ERN – CRN and the ERN were associated with NE; however, this relationship was also different than was hypothesized. Specifically, similarly to the results with maternal anxiety, higher levels of NE exhibited when children were 3 years-old were associated with a less negative ERN – CRN difference and a less negative ERN. This is in contrast to a number of other findings that demonstrated that higher levels of

NE were associated with an enhanced ERN (Hajcak et al., 2004; Luu et al., 2000; Pailing & Segalowitz, 2004). Although there have been no studies with designs like the Gründler et al. (2009) work that establish two different but overlapping neural mechanisms underlying the ERN and their associations with temperament, it is possible that the explanation for these results is similar to that posited for the relationship between maternal anxiety and a less negative ERN described above.

Although a number of studies have examined the relationship between the ERN and NE, the current work is the first to directly investigate various facets of this construct. There were no relationships between the ERN and child anger or sadness; however, higher levels of child fear were associated with a less negative ERN – CRN as well as a trend toward a less negative ERN. As was hypothesized above in the discussion about the similar association between the ERN- CRN and maternal anxiety, these findings may also be related to the differential activity of the two neural mechanisms of error processing proposed by Gründler et al. (2009). Specifically, it is possible that child fear is associated with hypoactivity in the rostral ACC, which may be involved in learning to avoid maladaptive responding and matures earlier than the dorsal ACC, which is thought to be responsible for allowing erroneous responses to be repeated despite their failure to produce a desired outcome. This would suggest that child fear is associated with a dysfunctional rostral ACC. This is a particularly interesting finding in light of evidence suggesting that the rostral-ventral region of the ACC is part of a network that includes the amygdala and is responsible for affective processing, whereas the dorsal region of the ACC is part of a network associated with cognitive functions, (for a review, see Bush, Luu, & Posner, 2000).

As has been demonstrated in other studies (Luu et al., 2000; Santesso et al., 2005), there was no relationship between PE and the ERN – CRN or the ERN. Santesso et al. (2005) suggested that this lack of association may be due to individuals who are high in PE not experiencing distress when they commit an error.

Interestingly, although one previous study had demonstrated an association between high BI measured during early childhood and an enhanced ERN measured during adolescence (McDermott et al., 2009), the current study found no association between BI and either the ERN – CRN or the ERN. This difference may be due to

maturational differences, as the participants in the McDermott et al. (2009) study examined much older children than the present investigation. It will be important to continue to examine the associations between BI measured in early childhood and ERN amplitude over the course of development. The current results also suggest that BI is a different construct than either NE or fear. Although fear was averaged across all twelve of the episodes, BI was calculated from only three episodes that were designed to elicit BI. The results suggest that more pervasive fear is associated with ERN amplitude, whereas situation-specific BI is not.

Associations between the CRN and Temperament

Little was known about the relationship between the CRN and temperament, with only one previous study finding a more negative CRN in individuals who were high in NE (Hajcak et al., 2004). The current investigation did not find any relationships between the CRN and temperament. This suggests that NE and fear are specifically associated with error commission.

It was hypothesized that there would be an interaction between NE and PE that would be associated with a larger ERN – CRN and a more negative ERN; however, this was not supported by the data. PE appears to have no relationship with the ERN, either alone or in combination with NE.

Associations between the Pe and Temperament

As was hypothesized based on previous findings (e.g., Santesso et al., 2005), there were also no significant associations between the Pe and temperament. Although the exact function of the Pe is unknown, the Pe has been found to be influenced by a number of factors, such as awareness of error commission (Nieuwenhuis et al., 2001), task demands (Mathewson et al., 2005); response conflict and affective salience of the stimuli (Simon-Thomas & Knight, 2005). It is possible that temperament constructs could interact with these factors to affect Pe amplitude; however, the current investigation used a paradigm with affectively neutral stimuli and for which task demands and response conflict were relatively low because the participants were so young. Future studies may benefit from using a more complex paradigm in order to examine associations between the Pe and temperament.

Associations between Behavioral Measures and Temperament

Behaviorally, children who exhibited higher levels of anger when they were 3 years-old may have been responding more often, as evidenced by both fewer correct No-Go trials and a trend toward more correct Go trials, suggesting they were more impulsive than children who exhibited lower levels of anger. Interestingly, anger was not associated with faster reaction times, only more frequent responding.

NE was also related to a number of behavioral measures, although these associations appear to be driven primarily by child sadness. Both appeared to be associated with slower reaction times. Total accuracy was lower for children who exhibited higher levels of NE and for children who demonstrated more sadness and this was likely due to more errors of omission. These results, in combination with a trend toward sadder children responding more slowly on errors of commission, suggest that NE, and specifically sadness, may be associated with slower response monitoring. Notably, child sadness and maternal history of depression were significantly correlated (r = .18, p = .001), which may explain why children of mothers with a history of depression also demonstrated non-significantly more errors of omission.

There was also a trend toward children who were more behaviorally inhibited having fewer correct responses following errors. Because there is no evidence that higher levels of BI were associated with fewer correct Go trials overall, this may suggest that they are particularly distracted by error commission and have difficulty re-engaging in the task immediately following an error.

One of the primary limitations to the current analyses is that the temperament assessment occurred approximately three years before the ERP assessment. Future work will examine associations between the ERN and child temperament when they are assessed more closely together. In addition, approximately one-quarter of the sample had to be excluded from the analyses because these participants committed too few errors to generate a reliable ERN (Olvet & Hajcak, 2009). Although the age range of the participants in this study appears to be relatively narrow, the exclusion of so many children due to near-perfect performance suggests the difficulty in designing a task that is appropriately challenging for a young group that has such cognitive variability.

A number of exploratory analyses revealed that child temperament neither moderated nor mediated the relationship between the ERN and maternal anxiety. This, in

combination with the findings that both maternal anxiety and child NE, and maternal anxiety and child fear are independently associated with ERN amplitude, suggests that maternal psychopathology and child temperament are associated with the ERN via separate pathways. Future studies should examine other potential factors that moderate or mediate these relationships.

In conclusion, this study characterized the ERP components associated with response monitoring in a large community sample of children between the ages of 5- and 7- years-old. Specifically, an ERN, CRN, and Pe were reliably elicited in these young children and were temporally and spatially similar to the ERN, CRN, and Pe that have been demonstrated in adults. Additionally, the current investigation also demonstrated that a maternal history of an anxiety disorder, child NE, and child fear were all associated with a less negative ERN – CRN and ERN. It was predicted that there would be relationships between these variables, but based on the literature examining adolescents and adults with anxiety and anxiety-relevant temperament traits, it was anticipated that the direction of the association would be opposite from what was observed. This is surprising; however, the fact that the findings for each of these two risk factors are similar suggests that there may be meaningful developmental changes in the relation between the ERN and risk for anxiety disorders. Specifically, this may be due to the differential maturation of the rostral and the dorsal portions of the ACC, with children as young as those in the current sample having only a rostral pathway that is developed enough to influence response monitoring. There was also an association between a maternal history of depression and a less negative CRN, which suggested that children at risk for depression may have decreased response monitoring. Finally, there were no associations between the ERP components and paternal psychopathology, the CRN and child temperament, or between the Pe and child temperament, providing further evidence that each of these components has a unique function and is differentially modulated by various factors.

Table 1

Mean Reaction Times and Accuracy Measures (Standard Deviations)

N = 321

Behavioral Measures	Accuracy/ Reaction Time
Number of errors of commission	16.18 (7.72)
Number of correct responses on Go trials	132.79 (13.71)
Accuracy (% correct)	88.29 (6.81)
Error Reaction Time (ms)	509.42 (86.85)
Correct Reaction Time (ms)	626.77 (72.48)
Number of correct trials following error trials	8.82 (4.46)
Reaction Time (ms) on correct trials following error trials	654.65 (117.85)
Number of errors of omission	10.16 (11.27)

Table 2

Mean (S.D.) of ERN and Pe Amplitude (μV) in Error Trials and Amplitude (μV) in Correct Trials at Midline Sites

N = 326

	Electrode Site		
ERP Component	Fz	Cz	Pz
ERN	-.77 (8.08)	-.05 (9.09)	1.51 (8.92)
Correct Trials Averaged	4.14 (4.56)	9.02 (5.76)	9.16 (6.24)
Pe (Error Trials)	7.47 (11.25)	13.45 (12.45)	14.22 (12.02)
Correct Trial Positivity	-.21 (3.64)	3.33 (4.29)	2.03 (4.38)

Table 3

Simultaneous Regression Analyses Examining Associations Between Total Correct Responses on Go Trials, Reaction Time on Correct Responses to Go Trials, and Total Correct No-Go Trials and Demographic Characteristics of the Sample

N = 304	Total Correct Responses on Go Trials			Reaction Time on Correct Responses to Go Trials			Total Correct No-Go Trials		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	1.35	.94	1.43	30.31	7.89	3.84***	-.66	1.41	-.47
Child Age at Assessment	-.61	1.08	-.56	-50.01	9.06	-5.52***	1.16	1.62	-.72
Child PPVT Score	.02	.04	.49	-.44	.32	-1.35	.03	.06	.52
Maternal Education	.98	.48	2.01*	1.98	4.06	.49	-.39	.72	-.55
Paternal Education	.52	.47	1.11	-8.57	3.96	-2.16*	-.08	.71	-.12

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 4

Simultaneous Regression Analyses Examining Associations Between Total Errors of Commission, Reaction Time on Errors of Commission, and Total Errors of Omission and Demographic Characteristics of the Sample

N = 304	Total Errors of Commission			Reaction Time on Errors of Commission			Total Errors of Omission		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	-.54	.91	-.59	40.58	9.44	4.30***	3.75	1.21	3.11**
Child Age at Assessment	-.26	1.04	-.25	-60.79	10.84	-5.61***	-6.81	1.38	-4.92***
Child PPVT Score	-.04	.04	-1.12	-.44	.39	-1.13	-.11	.05	-2.22*
Maternal Education	-.55	.47	-1.19	-.01	4.85	.00	.71	.62	1.15
Paternal Education	-.30	.45	-.66	-8.35	4.74	-1.76†	-1.28	.61	-2.11*

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 5

Simultaneous Regression Analyses Examining Associations Between Total Correct Trials Following Errors of Commission, Reaction Time on Correct Go Trials Following Errors of Commission, and Total Accuracy and Demographic Characteristics of the Sample

N = 304	Total Correct Trials Following Errors of Commission			Reaction Time on Correct Go Trials Following Errors of Commission			Total Accuracy		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	-.05	.53	-.09	40.81	13.29	3.07**	-2.89	1.79	-1.62
Child Age at Assessment	.26	.60	.42	-71.51	15.27	-4.68***	7.83	2.05	3.82***
Child PPVT Score	.01	.02	.28	-.26	.55	-.47	.17	.07	2.26*
Maternal Education	-.26	.27	-.97	-3.77	6.83	-.55	.03	.92	.03
Paternal Education	.13	.26	.48	-1.88	6.67	-.28	1.76	.90	1.96*

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 6

Simultaneous Regression Analyses Examining Associations Between ERN – CRN at Cz and ERN and CRN Amplitudes at Fz and Demographic Characteristics of the Sample

(*N* = 309)	ERN – CRN at Cz			ERN at Fz			CRN at Fz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	-.82	1.04	-.78	-1.64	.94	-1.74†	-.55	.53	-1.03
Child Age at Assessment	-2.74	1.20	-2.28*	-1.39	1.09	-1.27	.21	.61	.35
Child PPVT Score	.04	.04	1.02	.05	.04	1.33	.01	.02	.29
Maternal Education	.21	.54	.38	-.09	.49	-.19	-.16	.27	-.58
Paternal Education	.33	.53	.63	.19	.48	.40	.08	.27	.29

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 7

Simultaneous Regression Analyses Examining Associations Between Pe – Correct Trial Positivity Difference and Pe Amplitude from Demographic Characteristics of the Sample

(*N* = 309)	Pe – Correct Trial Positivity at Pz			Pe at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	-.84	1.40	-.60	-.02	1.38	-.02
Child Age at Assessment	3.05	1.62	1.88†	3.25	1.59	2.04*
Child PPVT Score	.14	.06	2.34*	.15	.06	2.57*
Maternal Education	-1.03	.72	-1.43	-.75	.71	-1.05
Paternal Education	.26	.71	.36	.37	.70	.53

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 8

Simultaneous Regression Analyses Examining Associations Between Total Correct Responses on Go Trials, Reaction Time on Correct Responses to Go Trials, and Total Correct No-Go Trials and Parental Psychopathology

	Total Correct Responses on Go Trials			Reaction Time on Correct Responses to Go Trials			Total Correct No-Go Trials		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
		N = 313			*N* = 307			*N* = 318	
Maternal Depression	-.22	1.00	-.22	1.76	8.62	.20	.97	1.51	.64
Maternal Anxiety Disorder (excluding specific phobia)	1.33	1.05	1.27	-2.56	8.97	-.29	-.44	1.56	-.28
Maternal Specific Phobia	1.27	1.36	.93	11.79	11.80	1.00	-1.41	2.03	-.69
Maternal Substance Dependence	.59	1.13	.52	-16.81	9.89	-1.70†	-1.13	1.68	-.67
		N = 311			*N* = 305			*N* = 316	
Paternal Depression	.89	1.30	.69	-15.55	11.18	-1.39	.87	1.94	.45
Paternal Anxiety Disorder (excluding specific phobia)	1.22	1.31	.94	3.38	11.24	.30	-2.90	1.93	-1.50
Paternal Specific Phobia	-.58	1.91	-.30	-6.54	16.59	-.39	3.04	2.86	1.06
Paternal Substance Dependence	-.15	.94	-.16	10.52	8.01	1.31	.73	1.39	.53

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 9

Simultaneous Regression Analyses Examining Associations Between Total Errors of Commission, Reaction Time on Errors of Commission, and Total Errors of Omission and Parental Psychopathology

	Total Errors of Commission			Reaction Time on Errors of Commission			Total Errors of Omission		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
		N = 318			*N* = 318			*N* = 305	
Maternal Depression	-.74	.96	-.77	11.65	10.16	1.15	2.54	1.32	1.93†
Maternal Anxiety Disorder (excluding specific phobia)	-.46	1.00	-.46	-1.35	10.52	-.13	-.77	1.38	-.56
Maternal Specific Phobia	.08	1.30	.06	15.03	13.69	1.10	.05	1.81	.03
Maternal Substance Dependence	.82	1.08	.77	-15.28	11.40	-1.34	1.38	1.51	.91
		N = 316			*N* = 316			*N* = 303	
Paternal Depression	-1.62	1.24	-1.31	-11.41	13.17	-.87	-.33	1.72	-.19
Paternal Anxiety Disorder (excluding specific phobia)	1.48	1.24	1.20	-5.09	13.22	-.39	-.20	1.73	-.12
Paternal Specific Phobia	-2.03	1.83	-1.11	-6.09	19.42	-.31	-1.23	2.55	-.48
Paternal Substance Dependence	-.97	.89	-1.09	8.81	9.45	.93	.29	1.23	.24

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 10

Simultaneous Regression Analyses Examining Associations Between Total Correct Trials Following Errors of Commission, Reaction Time on Correct Go Trials Following Errors of Commission, and Total Accuracy and Parental Psychopathology

	Total Correct Trials Following Errors of Commission			Reaction Time on Correct Go Trials Following Errors of Commission			Total Accuracy		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
		N = 318			*N* = 318			*N* = 305	
Maternal Depression	-.93	.56	-1.67†	11.86	14.17	.84	-2.05	1.95	-1.05
Maternal Anxiety Disorder (excluding specific phobia)	-.17	.58	-.30	-3.01	14.67	-.21	1.97	2.03	.97
Maternal Specific Phobia	-.24	.75	-.32	19.54	19.09	1.02	-.08	2.67	-.03
Maternal Substance Dependence	.49	.62	.79	-34.19	15.90	-2.15*	-2.70	2.23	-1.21
		N = 316			*N* = 316			*N* = 303	
Paternal Depression	-1.25	.72	-1.74†	-28.92	18.13	-1.60	1.34	2.54	.53
Paternal Anxiety Disorder (excluding specific phobia)	.61	.72	.85	4.51	18.20	.25	-1.04	2.54	-.41
Paternal Specific Phobia	-.91	1.06	-.86	4.48	26.73	.17	3.73	3.76	.99
Paternal Substance Dependence	-.18	.52	-.34	27.98	13.00	2.15*	.50	1.82	.27

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 11

Simultaneous Regression Analyses Examining Associations Between ERN – CRN at Cz and ERN and CRN Amplitudes at Fz and Parental Psychopathology

	ERN – CRN at Cz			ERN at Fz			CRN at Fz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
		N = 322			*N* = 322			*N* = 322	
Maternal Depression	-1.08	1.11	-.97	-.70	1.00	-.70	1.13	.57	1.99*
Maternal Anxiety Disorder (excluding specific phobia)	2.46	1.16	2.12*	3.57	1.04	3.43***	.90	.59	1.53
Maternal Specific Phobia	2.62	1.50	1.75†	.51	1.35	.38	-1.43	.76	-1.87†
Maternal Substance Dependence	.91	1.25	.72	.08	1.12	.07	.35	.64	.55
		N = 321			*N* = 321			*N* = 321	
Paternal Depression	-.63	1.45	-.43	-.88	1.31	-.67	.21	.74	.28
Paternal Anxiety Disorder (excluding specific phobia)	.87	1.44	.61	1.88	1.29	1.45	-.16	.73	-.22
Paternal Specific Phobia	1.54	2.11	.73	1.51	1.90	.80	-.01	1.08	-.01
Paternal Substance Dependence	-.49	1.04	-.48	-.41	.94	-.44	-.05	.53	-.10

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 12

Simultaneous Regression Analyses Examining Associations Between Pe – Correct Trial Positivity Difference and Pe Amplitudes at Pz and Parental Psychopathology

	Pe – Correct Trial Positivity at Pz			Pe at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
		N = 320			*N* = 320	
Maternal Depression	1.01	1.51	.67	1.20	1.49	.80
Maternal Anxiety Disorder (excluding specific phobia)	2.11	1.58	1.34	1.79	1.56	1.15
Maternal Specific Phobia	-1.36	2.05	-.66	-2.47	2.03	-1.22
Maternal Substance Dependence	2.78	1.70	1.64	2.36	1.68	1.41
		N = 319			*N* = 319	
Paternal Depression	3.20	1.96	1.63	2.22	1.94	1.14
Paternal Anxiety Disorder (excluding specific phobia)	-1.76	1.92	-.91	-1.05	1.90	-.55
Paternal Specific Phobia	2.33	2.82	.83	1.23	2.79	.44
Paternal Substance Dependence	.54	1.40	.39	.36	1.38	.26

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 13

Simultaneous Regression Analyses Examining Associations Between Total Correct Responses on Go Trials, Reaction Time on Correct Responses to Go Trials, and Total Correct No-Go Trials and Child Temperament

	Total Correct Responses on Go Trials			Reaction Time on Correct Responses to Go Trials			Total Correct No-Go Trials		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
		N = 316			N = 310			N = 321	
Child PE	-.02	.26	-.07	.06	2.26	.03	.23	.39	.59
Child NE	.93	1.72	.54	7.12	14.68	.49	-2.93	2.58	-1.13
Child PE	-.03	.26	-.11	-.03	2.27	-.02	.25	.39	.65
Child Sadness	-.03	1.75	-.02	2.61	15.00	.17	-1.44	2.60	-.55
Child Fear	-1.22	1.31	-.93	-2.51	11.08	-.23	2.11	1.93	1.09
Child Anger	2.47	1.47	1.68†	6.08	12.65	.48	-4.46	2.17	-2.05*
Child PE	.01	.27	.03	-.19	2.31	-.08	.34	.39	.86
Child BI	.86	1.10	.78	-2.67	9.51	-.28	1.38	1.66	.83

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 14

Simultaneous Regression Analyses Examining Associations Between Total Errors of Commission, Reaction Time on Errors of Commission, and Total Errors of Omission and Child Temperament

	Total Errors of Commission			Reaction Time on Errors of Commission			Total Errors of Omission		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
		N = 321			*N* = 321			*N* = 304	
Child PE	-.17	.25	-.68	-1.91	2.66	-.72	-.45	.35	-1.29
Child NE	1.32	1.67	.79	9.22	17.38	.53	6.07	2.22	2.73**
Child PE	-.17	.25	-.69	-1.66	2.66	-.62	-.42	.35	-1.19
Child Sadness	1.00	1.69	.59	33.67	17.59	1.92†	6.44	2.25	2.86**
Child Fear	-.91	1.25	-.73	-15.65	13.05	-1.20	.34	1.69	.20
Child Anger	1.69	1.41	1.20	-3.43	14.76	-.23	.55	1.91	.29
Child PE	-.28	.25	-1.12	-2.48	2.72	-.91	-.51	.36	-1.41
Child BI	-1.91	1.06	-1.80†	-7.88	11.26	-.70	.73	1.46	.50

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 15

Simultaneous Regression Analyses Examining Associations Between Total Correct Trials Following Errors of Commission, Reaction Time on Correct Go Trials Following Errors of Commission, and Total Accuracy and Child Temperament

	Total Correct Trials Following Errors of Commission			Reaction Time on Correct Go Trials Following Errors of Commission			Total Accuracy		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
		N = 321			*N* = 321			*N* = 308	
Child PE	-.06	.14	-.43	.89	3.69	.24	.33	.51	.64
Child NE	.13	.96	.14	-27.07	24.15	-1.12	-8.79	3.29	-2.67**
Child PE	-.07	.15	-.48	.96	3.71	.26	.29	.51	.57
Child Sadness	-.39	.98	-.39	4.15	24.56	.17	-8.67	3.33	-2.61**
Child Fear	-.30	.72	-.41	-26.25	18.23	-1.44	.24	2.46	.10
Child Anger	.93	.82	1.14	-8.87	20.61	-.43	-2.61	2.80	-.93
Child PE	-.12	.15	-.82	.56	3.78	.15	.50	.52	.96
Child BI	-1.15	.61	-1.87†	-13.01	15.67	-.83	1.10	2.14	.51

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 16

Simultaneous Regression Analyses Examining Associations Between ERN – CRN at Cz, and ERN and CRN Amplitudes at Fz and Child Temperament

	ERN – CRN Amplitude			ERN at Fz			CRN at Fz		
Variables Entered	b	Standard Error N = 326	*t*	b	Standard Error N = 326	*t*	b	Standard Error N = 326	*t*
Child PE	-.17	.29	-.58	.16	.26	.62	.07	.15	.48
Child NE	4.46	1.93	2.32*	3.72	1.73	2.14*	1.23	.98	1.25
Child PE	-.20	.29	-.69	.15	.26	.58	.08	.15	.51
Child Sadness	-1.07	1.95	-.55	.59	1.76	.34	1.33	1.00	1.34
Child Fear	3.05	1.45	2.11*	2.43	1.31	1.86†	-.06	.74	-.08
Child Anger	2.51	1.63	1.54	.92	1.46	.63	.16	.83	.20
Child PE	-.18	.30	-.61	.09	.26	.35	.03	.15	.19
Child BI	.91	1.24	.73	-.35	1.11	-.31	-.52	.63	-.82

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 17

Simultaneous Regression Analyses Examining Associations Between Pe – Correct Trial Positivity and Pe at Pz Amplitudes and Child Temperament

	Pe – Correct Trial Positivity at Pz			Pe at Pz		
Variables Entered	b	Standard Error N = 324	*t*	b	Standard Error N = 324	*t*
Child PE	-.06	.39	-.14	-.16	.39	-.42
Child NE	-1.17	2.60	-.45	-.07	2.57	-.03
Child PE	-.08	.39	-.21	-.18	.39	-.45
Child Sadness	-4.43	2.62	-1.69†	-3.24	2.59	-1.25
Child Fear	2.34	1.95	1.20	2.80	1.93	1.45
Child Anger	.47	2.18	.22	.00	2.17	.00
Child PE	.02	.40	.06	.09	.40	-.23
Child BI	1.38	1.66	.83	1.49	1.64	.91

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Table 18

Hierarchical Multiple Regression Analyses Examining Associations Between ERN - CRN at Cz and Child Age at Assessment, Child PE, and Child NE

(*N* = 325)	ERN – CRN		
Variables Entered	b	Standard Error	*t*
Child Age at Assessment	-2.55	1.17	-2.18*
Child PE	-.16	.29	-.55
Child NE	4.53	1.94	2.33*
Child Age x Child PE	.49	.67	.73
Child Age x Child NE	2.98	4.22	.71
PE x NE	.56	1.24	.45

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Table 19

Hierarchical Multiple Regression Analyses Examining Associations Between ERN Amplitude at Fz and Child PE and Child NE

(*N* = 325)	ERN Amplitude		
Variables Entered	b	Standard Error	*t*
Child PE	.16	.26	.61
Child NE	3.81	1.75	2.18*
PE x NE	.30	1.10	.27

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Table 20

Simultaneous Regression Analyses Examining Associations Between ERN – CRN Amplitude at Cz, Child NE, and Maternal Anxiety

(*N* = 321)	ERN – CRN at Cz		
Variables Entered	b	Standard Error	*t*
Child Age at Assessment	.23	3.56	.06
Maternal Anxiety Disorder (excluding specific phobia)	2.55	1.11	2.30*
Child NE	5.41	5.77	.94
Child Age x Maternal Anxiety	-2.47	2.72	-.91
Child Age x NE	3.08	4.17	.74
NE x Maternal Anxiety	-.54	4.09	-.13

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Table 21

Simultaneous Regression Analyses Examining Associations Between ERN Amplitude at Fz, Child NE, and Maternal Anxiety

(*N* = 321)	ERN at Fz		
Variables Entered	b	Standard Error	*t*
Maternal Anxiety Disorder (excluding specific phobia)	3.41	.99	3.45***
Child NE	5.81	5.14	1.13
NE x Maternal Anxiety	-1.78	3.64	-.49

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

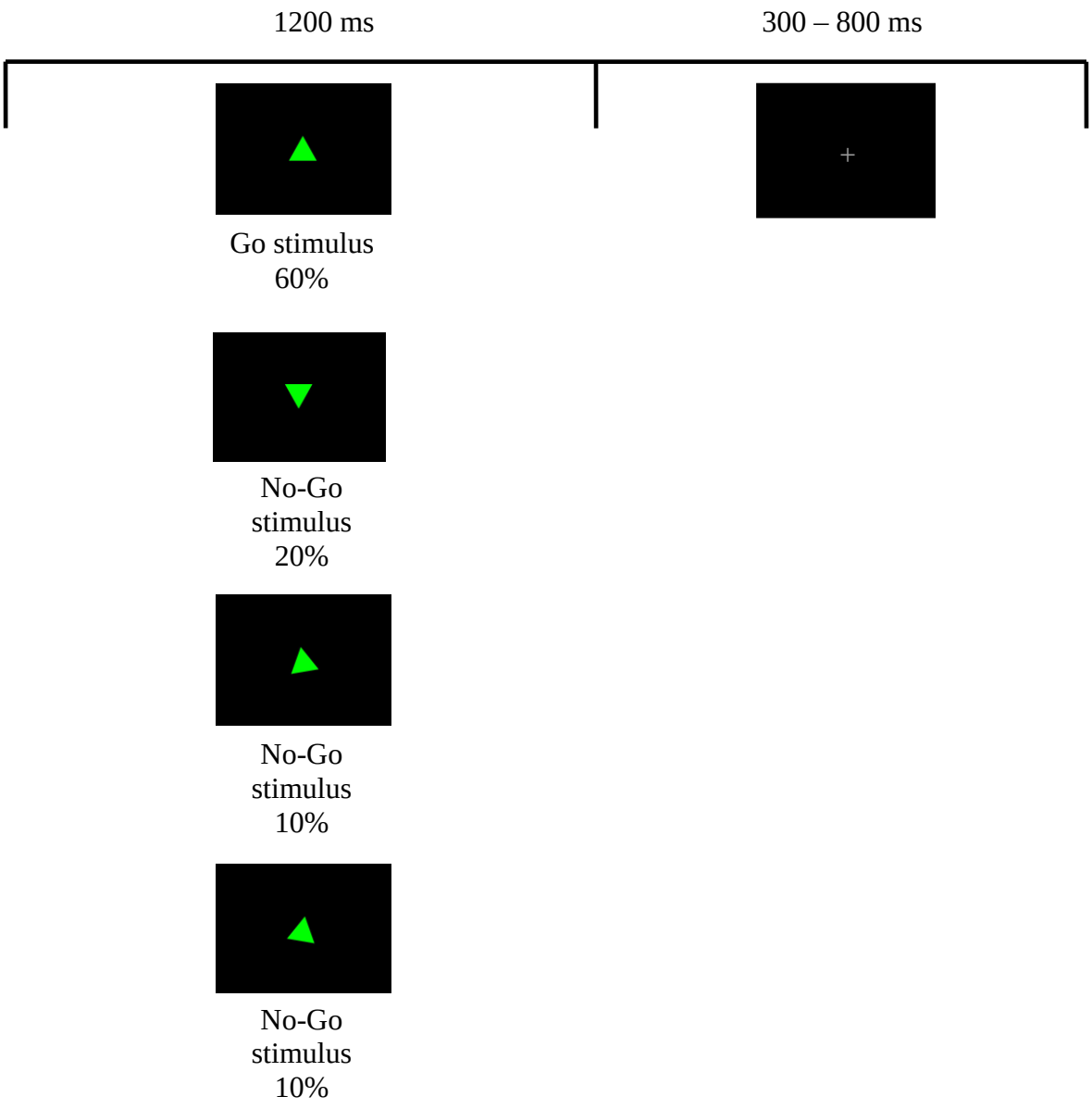
Table 22

Simultaneous Regression Analyses Examining Associations Between ERN – CRN at Cz, Child Fear, and Maternal Anxiety

(*N* = 321)	ERN – CRN at Cz		
Variables Entered	b	Standard Error	*t*
Child Age at Assessment	-.90	3.57	-.25
Maternal Anxiety Disorder (excluding specific phobia)	2.70	1.11	2.44*
Child Fear	2.64	4.22	.63
Child Age x Maternal Anxiety	.89	3.03	.29
Child Age x Fear	-1.80	2.72	-.66
Fear x Maternal Anxiety	.47	3.08	.15

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Figure 1. Stimulus presentation in Go/No-Go paradigm.



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Appendix A

Characterize ERN – CRN, ERN, CRN, Pe – Correct Trial Positivity, and Pe in Young Children

Associations Between ERP Measures and Demographic Variables

Simultaneous regression analyses were used to explore the relationships between ERP measures (ERN – CRN, ERN, and CRN at Fz and Pz, and Pe – Correct Trial Positivity and Pe at Fz and Cz) and several demographic variables, including child age at ERP assessment, child gender, child PPVT scores, and parent education.

Response-Locked ERP Components

ERN – CRN at Fz

Appendix B depicts the results of the analyses examining ERN – CRN amplitude at Fz. There were no significant associations with the demographic variables.

ERN – CRN at Pz

Appendix B depicts the results of the analyses examining ERN – CRN amplitude at Pz. There was a significant effect of child age, suggesting that older children demonstrated a more negative ERN than younger children.

ERN at Cz

Appendix C depicts the results of analyses examining ERN amplitude at Cz. Although there were trends toward younger children and children with higher PPVT scores demonstrating a less negative ERN, none of the demographic variables were significantly associated with ERN amplitude.

ERN at Pz

Analyses examining ERN amplitude at Pz suggested there were no associations with the demographic variables (Appendix C).

CRN at Cz

Analyses examining CRN amplitude at Cz suggested there were no associations with the demographic variables (Appendix D).

CRN at Pz

Appendix D depicts the results of analyses examining CRN amplitude at Pz. There was a significant effect of child age, with older children demonstrating a less negative CRN than younger children.

Pe - Correct Trial Positivity at Fz

Appendix E depicts the results of analyses examining Pe – Correct Trial Positivity at Fz. Child PPVT score was significantly associated with Pe – Correct Trial Positivity difference; children with higher PPVT scores had a more positive Pe – Correct Trial Positivity difference than children with lower PPVT scores.

Pe - Correct Trial Positivity at Cz

Appendix E depicts the results of analyses examining Pe – Correct Trial Positivity at Cz. Child PPVT score was significantly associated with Pe – Correct Trial Positivity difference; children with higher PPVT scores had a more positive Pe – Correct Trial Positivity difference than children with lower PPVT scores.

Pe at Fz

Appendix F depicts the results examining associations with the Pe at Fz. The results were similar to those from the analyses examining the Pe – Correct Trial Positivity at Fz.

Pe at Cz

Appendix F depicts the results examining associations with the Pe at Cz. The results were similar to those from the analyses examining the Pe – Correct Trial Positivity at Cz.

Examine the relationship between parental depression and/ or anxiety and offspring ERP components.

Simultaneous regression analyses were used to explore the relationships between behavioral and ERP measures and parental depression and/ or anxiety. Maternal and paternal psychopathology were each defined by four variables: lifetime diagnosis of depression; lifetime diagnosis of anxiety disorder, excluding specific phobia; lifetime diagnosis of specific phobia; and lifetime diagnosis of substance dependence. Each set of four parental psychopathology variables were entered simultaneously in each model. Demographic variables that were significantly associated with specific outcome measure were included as covariates in the models.

Response-Locked ERP Components

ERN – CRN at Fz

Associations with Maternal Psychopathology

Appendix G depicts the results of the analyses examining ERN – CRN amplitude at Fz. There was a significant effect of maternal anxiety, suggesting that children who had mothers with a lifetime history of an anxiety disorder demonstrated a less negative ERN – CRN difference than children whose mothers did not have an anxiety disorder. An association between ERN – CRN difference and maternal depression approached significance, with a trend toward children who had mothers with a lifetime history of depression demonstrating a more negative ERN – CRN at Fz.

Associations with Paternal Psychopathology

There were no significant associations with paternal psychopathology.

ERN – CRN at Pz

Analyses examining the ERN – CRN amplitude at Pz suggested there were no significant associations with maternal or paternal psychopathology (Appendix G).

ERN at Cz

Associations with Maternal Psychopathology

Appendix H also depicts the results of analyses examining ERN amplitude at Cz. There was a significant association between ERN amplitude and maternal anxiety, with children whose mothers had a history of an anxiety disorder demonstrating a less negative ERN than children whose mothers did not have an anxiety disorder.

Associations with Paternal Psychopathology

There were no significant associations with paternal psychopathology.

ERN at Pz

Analyses examining the ERN amplitude at Pz suggested there were no significant associations with maternal or paternal psychopathology (Appendix H).

CRN at Fz

Associations with Maternal Psychopathology

Analyses examining CRN amplitude at Fz suggested a significant association with maternal specific phobia (Appendix I); specifically, children who had mothers with a history of a specific phobia demonstrated a more negative CRN than children whose mothers did not have specific phobia.

Associations with Paternal Psychopathology

There were no associations with paternal psychopathology.

CRN at Pz

Associations with Maternal Psychopathology

Analyses examining CRN amplitude at Pz suggested a significant association with maternal specific phobia; specifically, children who had mothers with a history of a specific phobia demonstrated a more negative CRN than children whose mothers did not have a specific phobia (Appendix I).

Associations with Paternal Psychopathology

There were no associations with paternal psychopathology.

Pe - Correct Trial Positivity at Fz

Analyses examining the Pe – Correct Trial Positivity difference at Fz suggested there were no significant associations with either maternal or paternal psychopathology (Appendix J).

Pe - Correct Trial Positivity at Cz

Analyses examining the Pe – Correct Trial Positivity difference at Cz suggested there were no significant associations with either maternal or paternal psychopathology (Appendix J).

Pe at Fz

Analyses examining the Pe at Fz suggested there were no significant associations with either maternal or paternal psychopathology (Appendix K).

Pe at Cz

Analyses examining the Pe at Cz suggested there were no significant associations with either maternal or paternal psychopathology (Appendix K).

Examine the relationship between parental loading of psychopathology and offspring ERP components.

Simultaneous regression analyses were used to explore the relationships between behavioral and ERP measures and the impact of having zero, one, or two parents with a lifetime diagnosis of depression; zero, one, or two parents with a lifetime diagnosis of anxiety disorder (excluding specific phobia); zero, one, or two parents with a lifetime diagnosis of specific phobia; and zero, one, or two parents with a lifetime diagnosis of substance dependence. Maternal and paternal psychopathology were each defined by four variables: lifetime diagnosis of depression; lifetime diagnosis of anxiety disorder, excluding specific phobia; lifetime diagnosis of specific phobia; and lifetime diagnosis of substance dependence. Each set of four parental psychopathology variables were entered simultaneously in each model. Demographic variables that were significantly associated with specific outcome measure were included as covariates in the models.

Behavioral Measures

Total Correct Responses on Go Trials

Appendix L depicts the results of analyses examining total correct responses on Go trials. There were no significant associations with parental loading of psychopathology.

Reaction Time on Correct Responses to Go Trials

Appendix L depicts the results of the analyses examining reaction time on correct responses on Go trials. There were no significant associations with parental loading of psychopathology.

Total Correct No-Go Trials

Analyses examining total correct No-Go trials suggested there were no associations with parental loading of psychopathology (Appendix L).

Total Errors of Commission

Analyses examining total errors of commission suggested there were no associations with parental loading of psychopathology (Appendix M).

Reaction Time on Errors of Commission

Analyses examining reaction time on errors of commission suggested there were no associations with parental loading of psychopathology (Appendix M).

Total Errors of Omission

Analyses examining total errors of omission suggested there were no associations with the parental loading of psychopathology (Appendix M).

Total Correct Go Trials Following Errors of Commission

Analyses examining total correct Go trials following errors of commission suggested that an increased parental loading for depression was associated with fewer offspring total correct responses on Go trials following errors of commission (Appendix N).

Reaction Time on Correct Go Trials Following Errors of Commission

Analyses examining reaction time on correct Go trials following errors of commission suggested there were no associations with the parental loading of psychopathology (Appendix N).

Total Accuracy

Analyses examining total accuracy suggested there were no associations with the parental loading of psychopathology (Appendix N).

Response-Locked ERP Components

ERN – CRN at Fz

Appendix O depicts the results of the analyses examining ERN – CRN amplitude at Fz. There was a significant effect of parental loading of an anxiety disorder, suggesting that increased parental loading of anxiety was associated with children demonstrating a less negative ERN – CRN. There was a trend toward increased loading for a parental specific phobia being associated with a less negative ERN in offspring, as well as a trend toward increased loading for parental depression being associated with a more negative ERN.

ERN – CRN at Cz

Appendix O depicts the results of the analyses examining ERN – CRN amplitude at Cz. There was a significant effect of parental loading of an anxiety disorder, suggesting that increased parental loading of anxiety was associated with children

demonstrating a less negative ERN – CRN. A similar association was present between the ERN – CRN difference and parental loading of a specific phobia.

ERN – CRN at Pz

Appendix O depicts the results of the analyses examining ERN – CRN amplitude at Pz. Although there were no significant associations with parental loading of psychopathology, there was a trend toward increased loading for a parental specific phobia being associated with a less negative ERN in offspring.

ERN at Fz

Appendix P depicts the results of analyses examining ERN amplitude at Fz. There was a significant association between ERN amplitude and parental loading of anxiety, with an increased loading of parental anxiety associated with children demonstrating a less negative ERN.

ERN at Cz

Appendix P also depicts the results of analyses examining ERN amplitude at Cz. Results were similar to those found in the analyses examining the ERN at Fz with a significant association between a less negative ERN amplitude and increased parental loading of anxiety.

ERN at Pz

Analyses examining associations with ERN amplitude at Pz suggested there were no significant associations with parental loading of psychopathology (Appendix P).

CRN at Fz

No significant associations were found in analyses examining CRN amplitude at Fz (Appendix Q).

CRN at Cz

Appendix Q depicts the results of analyses examining the CRN at Cz. There was a trend toward an association with parental loading for a specific phobia; specifically, an increased parental loading of a specific phobia was associated with children demonstrating a more negative CRN.

CRN at Pz

Appendix Q depicts the results of analyses examining the CRN at Pz. Although there were no significant associations, there was a trend toward a similar association between CRN amplitude and parental loading of a specific phobia as was found at Cz.

Pe - Correct Trial Positivity at Fz

Analyses examining the Pe – Correct Trial Positivity difference at Fz suggested there were no significant associations with parental loading of psychopathology (Appendix R).

Pe - Correct Trial Positivity at Cz

Analyses examining the Pe – Correct Trial Positivity difference at Cz suggested there were no significant associations with parental loading of psychopathology (Appendix R).

Pe - Correct Trial Positivity at Pz

Analyses examining the Pe – Correct Trial Positivity difference at Pz suggested there were no significant associations with parental loading of psychopathology (Appendix R).

Pe at Fz

Analyses examining Pe amplitude at Fz suggested there were no significant associations with parental loading of psychopathology (Appendix S).

Pe at Cz

Analyses examining Pe amplitude at Cz suggested there were no significant associations with parental loading of psychopathology (Appendix S).

Pe at Pz

Analyses examining Pe amplitude at Pz suggested there were no significant associations with parental loading of psychopathology (Appendix S).

Aim 3: Examine the relationship between child temperament and ERP components.

Several simultaneous regression analyses were conducted to examine associations between child temperament variables and several behavioral measures. The two broader temperament constructs of positive and negative emotionality (PE and NE, respectively), were the predictor variables in one model. In order to examine whether the constructs from which NE is comprised differentially predict behavioral measures, the second regression model included sadness, fear, and anger, as well as PE. Another model included PE and BI, instead of NE.[16] Demographic variables that were significantly associated with specific outcome measure were included as covariates in the models.

Response- Locked ERP Components

ERN – CRN at Fz

Appendix T depicts the results of the analyses examining ERN – CRN at Fz. Although there was a trend toward increased levels of child fear being associated with a less negative ERN – CRN, there were no significant associations with child temperament.

ERN – CRN at Pz

Appendix T depicts the results of the analyses examining ERN – CRN at Pz. There was a significant effect of NE; specifically, higher levels of child NE predicted less negative ERN - CRN than lower levels of NE. Fear was also significantly associated with ERN – CRN, with higher levels of fear associated with a smaller (i.e., less negative) ERN – CRN difference.

ERN at Cz

Appendix U also depicts the results of analyses examining ERN amplitude at Cz. There was a significant association between ERN amplitude and NE, with children who exhibited higher levels of NE demonstrating a smaller (i.e., less negative) ERN than children who exhibited lower levels of NE.

ERN at Pz

Appendix U depicts the results of analyses examining ERN amplitude at Pz. There was a significant association between ERN amplitude and NE, with children who exhibited higher levels of NE demonstrating a less negative ERN than children who exhibited lower levels of NE. There was a significant association with fear, with children who exhibited higher levels of fear demonstrating a less negative ERN than children who exhibited lower levels of fear.

CRN at Fz

[16] All analyses were repeated using positive affect (PA) as a predictor variable in place of PE. As was described above, PE is comprised of both PA and child interest. Results were similar, except where noted.

Analyses examining CRN amplitude at Cz suggested there were no significant associations with child temperament (Appendix V).

CRN at Pz

Analyses examining CRN amplitude at Pz suggested there were no significant associations with child temperament (Appendix V).

Pe - Correct Trial Positivity at Fz

Analyses examining the Pe – Correct Trial Positivity difference at Fz suggested there were no significant associations with child temperament (Appendix W).

Pe - Correct Trial Positivity at Cz

Analyses examining the Pe – Correct Trial Positivity difference at Cz suggested there were no significant associations with child temperament (Appendix W), although there was a trend toward children who exhibited higher levels of sadness demonstrating a smaller (i.e., less positive) Pe – Correct Trial Positivity difference. [17]

Pe at Fz

Analyses examining Pe amplitude at Fz suggested there were no significant associations with child temperament (Appendix X).

Pe at Cz

Analyses examining Pe amplitude at Cz suggested there were no significant associations with child temperament (Appendix X).

Associations between ERN – CRN Difference at Fz and Pz and Interaction between PE and NE

Hierarchical regression analyses were conducted to examine the effects of the interaction between PE and NE on ERN – CRN difference at Fz and at Pz. Because age had been significantly associated with the ERN – CRN difference at Pz, this was included in the model examining ERN – CRN at Pz as an independent variable and in interaction terms with PE and NE, but not in the model examining ERN – CRN at Fz. Both analyses included the cross-product of PE and NE. As depicted in Appendix Y, there were no significant main or interaction effects in the model examining ERN – CRN at Fz. [18] Appendix Z contains the results of the analyses examining the ERN – CRN at Pz. There were significant main effects of child age at assessment and NE; however, there were no other significant main or interaction effects, suggesting that the interaction between NE and PE is not associated with the ERN – CRN difference at Pz.[19]

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[17] When these analyses were repeated using PA instead of PE, there was no longer a trend toward a significant effect of child fear.

[18] These analyses were also conducted using PA instead of PE. Results were identical. Child BI was included in the model in place of child NE. There were no main or interaction effects in models examining BI and PE or BI and PA associations .

[19] These analyses were also conducted using PA instead of PE. Results were identical. Analyses were also conducted excluding age from the model. There was a main effect of NE; however, there continued to be no main effect or interaction involving PE or PA. Child BI was included in the model in place of child NE. There was a main effect of child age at assessment and an interaction between BI and PE that approached significance (b = -1.43, S.E. = .75, and t = -1.91, $p < .10$). These results were similar when PA was included in the model in place of PE. Analyses were also conducted excluding age from the models. Results were similar when PA was included in the model; however when the model include PE, the interaction between BI and PE was significant (b = -1.41, S.E. = .69, and t = -2.04, $p < .05$). In order to further characterize this interaction, the median split for PE and BI were independently calculated and the sample was divided into four groups: children who demonstrated low PE and low BI, children who demonstrated high PE and low BI, children who demonstrated low PE and high BI, and children who

Associations between ERN Amplitude at Cz and Pz and Interaction between PE and NE

A series of hierarchical regression analyses were conducted to examine the effects of the interaction between PE and NE on ERN amplitude at Cz (Appendix AA). PE and NE were independently included in the models, as well as their cross-product. As depicted in Appendix AA, there was a significant main effect of NE. There were no other significant effects, suggesting that the interaction between NE and PE is not associated with ERN amplitude. [20]

Aim 4: Explore whether child temperament moderates or mediates the relationship between parental psychopathology and offspring

Child NE as a Moderator of Relationship Between Maternal Anxiety and Child ERN Amplitude at Cz

Because maternal anxiety and child NE were both independently associated with child ERN amplitude at Cz, hierarchical regression analyses were conducted to examine the interactive effects of child NE and maternal anxiety (Appendix BB). Maternal anxiety and child NE were included in the models as independent variables in the first step, as was their cross-product, in the second step of the model. There was a main effect of maternal anxiety; however, there was no main effect of child NE and no significant interaction between NE and maternal anxiety suggesting that child NE does not moderate the association between maternal anxiety and the ERN at Cz.

Child Fear as a Moderator of Relationship Between Maternal Anxiety and Child ERN Amplitude at Cz

Hierarchical regression analyses were conducted to examine the effects of the interaction between child fear and maternal anxiety on the ERN at Cz (Appendix CC). Maternal anxiety and child fear were included in the models as independent variables, as well as their cross-product, in the second step of the model. There was a main effect of maternal anxiety; however, there was no main effect of child fear, nor was there a significant interaction between fear and maternal anxiety, suggesting that child fear does not moderate the association between maternal anxiety and the ERN.[21]

Child NE as a Moderator of Relationship Between Parental Loading for Anxiety and Child ERN – CRN at Cz

Because parental loading for an anxiety disorder and child NE were both independently associated with child ERN - CRN amplitude at Cz, hierarchical regression analyses were conducted to examine the interactive effects of child NE and parental loading for anxiety (Appendix DD). Child age had been significantly associated with the ERN – CRN amplitude and was included in the model as an independent variable and in interaction terms with child NE and parental loading of an anxiety disorder. Parental

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demonstrated high PE and high BI. A one-way ANOVA was conducted; however, the results were not significant, suggesting there were no differences between the groups. Independent-samples *t*-tests were conducted in order to directly compare these groups. There was a significant difference only between the low PE/low BI group and the low PE/high BI group, with the low PE/ high BI group demonstrating a significantly less negative ERN – CRN at Pz than the low PE/ low BI group.

[20] These analyses were also conducted using PA instead of PE and the results were identical. Results were also similar when child BI was included in the model instead of child NE. Analyses were also conducted to examine the CRN at Cz; however, there were no significant main or interaction effects.

[21] Analyses were repeated using parental loading of a lifetime anxiety disorder instead of maternal anxiety. Results were identical.

loading for anxiety and child NE were included in the models as independent predictors in the first step, as was their cross-product, in the second step of the model. There was an effect of parental loading for anxiety that approached significance; however, there was no main effect of child NE and no significant interaction between NE and parental loading for anxiety suggesting that child NE does not moderate the association between parental loading of an anxiety disorder and the ERN – CRN at Cz.[22]

Child Fear as a Moderator of Relationship Between Parental Loading for Anxiety and Child ERN – CRN at Cz

Because parental loading for an anxiety disorder and child fear were both independently associated with child ERN - CRN amplitude at Cz, hierarchical regression analyses were conducted to examine the interactive effects of child fear and parental loading for anxiety (Appendix FF). Child age was included in the model, independently and in interaction terms with child fear and parental loading of an anxiety disorder. Parental loading for anxiety and child fear were included in the models as independent variables in the first step, as was their cross-product, in the second step of the model. There was a main effect of parental loading for anxiety; however, there was no main effect of child fear and no significant interaction between fear and parental loading for anxiety suggesting that child fear does not moderate the association between parental loading of an anxiety disorder and the ERN – CRN difference at Cz.[23]

Child NE as a Moderator of Relationship Between Parental Loading for Anxiety and Child ERN at Cz and Fz

Because parental loading for an anxiety disorder and child NE were both independently associated with child ERN amplitude at both Cz and Fz, hierarchical regression analyses were conducted to examine the interactive effects of child NE and parental loading for anxiety (Appendix HH). Parental loading for anxiety and child NE were included in the models as independent variables in the first step, as was their cross-product, in the second step of the model. There was a main effect of parental loading for anxiety at both sites; however, there was no main effect of child NE (although there was a trend toward an effect at Cz) and no significant interaction between NE and parental loading for anxiety suggesting that child NE does not moderate the association between parental loading of an anxiety disorder and the ERN at either Cz or Fz.

Child NE, Child Fear, and Child BI as Potential Mediators of the Relationship Between Maternal Anxiety and Child ERN

There was no significant correlation between maternal anxiety and child NE, fear, or BI (r = .03, .00, and -.01, respectively), which precluded mediational analyses.[24]

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[22] These analyses were repeated with child age excluded from the model. The results were similar, except that there was a significant main effect of parental loading for anxiety. These analyses were also repeated examining parental loading of a specific phobia instead of parental loading for an anxiety disorder and the results were similar (Appendix EE), although there was a significant main effect of parental loading of a specific phobia.

[23] These analyses were repeated with child age excluded from the model. The results were similar. These analyses were also repeated examining parental loading of a specific phobia instead of parental loading for an anxiety disorder and the results were similar (Appendix GG), although in the model excluding child age, the effect of parental loading of a specific phobia only approached significance.

[24] Correlations between parental loading for an anxiety disorder and child NE, fear, and BI were also assessed and were not significant (rs = .03, .02, and -.01, respectively). Because of this, no mediational analyses were conducted.

Appendix B

Simultaneous Regression Analyses Examining Associations Between the ERN – CRN and Demographics of the Sample

(*N* = 309)	ERN – CRN at Fz			ERN – CRN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	-1.09	.99	-1.11	.05	1.04	.05
Child Age at Assessment	-1.60	1.14	-1.40	-2.48	1.20	-2.06*
Child PPVT Score	.05	.04	1.12	.03	.04	.76
Maternal Education	.07	.51	.13	.00	.54	.00
Paternal Education	.11	.50	.22	.54	.53	1.03

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix C

Simultaneous Regression Analyses Examining Associations Between ERN Amplitude at Cz and Pz and Demographics of the Sample

(*N* = 309)	ERN at Cz			ERN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	-1.65	1.06	-1.55	-.07	1.05	-.07
Child Age at Assessment	-2.34	1.23	-1.91†	-.58	1.21	-.47
Child PPVT Score	.07	.04	1.66†	.05	.04	1.20
Maternal Education	-.03	.55	-.05	-.22	.54	-.41
Paternal Education	.06	.54	.12	.45	.53	.86

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix D

Simultaneous Regression Analyses Examining Associations Between CRN Amplitude at Cz and Pz and Demographics of the Sample

(*N* = 309)	CRN at Cz			CRN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	-.83	.67	-1.23	-.12	.72	-.17
Child Age at Assessment	.40	.78	.52	1.90	.83	2.30*
Child PPVT Score	.03	.03	1.04	.02	.03	.65
Maternal Education	-.23	.35	-.67	-.22	.37	-.60
Paternal Education	-.26	.34	-.78	-.09	.36	-.25

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix E

Simultaneous Regression Analyses Examining Associations Between the Pe – Correct Trial Positivity and Demographics of the Sample

(N = 309)	Pe – Correct Trial Positivity Amplitude at Fz			Pe – Correct Trial Positivity Amplitude at Cz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	-1.86	1.28	-1.45	-1.91	1.42	-1.35
Child Age at Assessment	1.98	1.48	1.34	2.07	1.63	1.27
Child PPVT Score	.18	.05	3.47***	.21	.06	3.52***
Maternal Education	-.54	.66	-.82	-.33	.73	-.46
Paternal Education	.40	.65	.61	-.17	.71	-.24

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix F

Simultaneous Regression Analyses Examining Associations Between Pe Amplitude and Demographics of the Sample

(*N* = 309)	Pe at Fz			Pe at Cz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
Sex of Child	-1.43	1.29	-1.11	-.95	1.45	-.66
Child Age at Assessment	1.70	1.49	1.14	1.11	1.67	.66
Child PPVT Score	.17	.05	3.26***	.21	.06	3.55***
Maternal Education	-.59	.67	-.89	-.25	.74	-.34
Paternal Education	.52	.65	.80	.06	.73	.09

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix G

Simultaneous Regression Analyses Examining Associations Between the ERN – CRN at Fz and Pz and Maternal and Paternal Psychopathology

	ERN – CRN at Fz			ERN – CRN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
		N = 322			*N* = 322	
Maternal Depression	-1.83	1.06	-1.73†	.92	1.11	.83
Maternal Anxiety Disorder (excluding specific phobia)	2.70	1.10	2.45*	.26	1.16	.22
Maternal Specific Phobia	1.93	1.43	1.35	2.19	1.50	1.46
Maternal Substance Dependence	-.28	1.19	-.23	.94	1.25	.75
		N = 321			*N* = 321	
Paternal Depression	-1.01	1.38	-.73	1.43	1.43	1.00
Paternal Anxiety Disorder (excluding specific phobia)	1.98	1.37	1.45	.39	1.41	.28
Paternal Specific Phobia	1.59	2.01	.79	2.34	2.08	1.13
Paternal Substance Dependence	-.34	.99	-.35	.71	1.02	.70

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix H

Simultaneous Regression Analyses Examining Associations Between the ERN Amplitude at Cz and Pz and Parental Psychopathology

	ERN at Cz			ERN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
		N = 322			*N* = 322	
Maternal Depression	-.92	1.13	-.81	.66	1.13	.59
Maternal Anxiety Disorder (excluding specific phobia)	3.24	1.18	2.76**	1.11	1.17	.95
Maternal Specific Phobia	.42	1.53	.28	.07	1.52	.05
Maternal Substance Dependence	1.20	1.27	.94	.57	1.26	.45
		N = 321			*N* = 321	
Paternal Depression	-.77	1.47	-.52	78	1.45	.54
Paternal Anxiety Disorder (excluding specific phobia)	.71	1.45	.49	.26	1.43	.18
Paternal Specific Phobia	1.05	2.13	.49	1.73	2.10	.83
Paternal Substance Dependence	-.59	1.05	-.56	.64	1.03	.62

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix I

Simultaneous Regression Analyses Examining Associations Between CRN Amplitude at Cz and Pz and Parental Psychopathology

	CRN at Cz			CRN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
		N = 322			*N* = 322	
Maternal Depression	.16	.72	.22	-.26	.77	-.34
Maternal Anxiety Disorder (excluding specific phobia)	.83	.74	1.11	.85	.80	1.07
Maternal Specific Phobia	-2.21	.97	-2.29*	-2.12	1.04	-2.04*
Maternal Substance Dependence	.27	.81	.34	-.37	.87	-.42
		N = 321			*N* = 321	
Paternal Depression	.00	.94	.01	-.63	1.00	-.63
Paternal Anxiety Disorder (excluding specific phobia)	-.26	.93	-.29	-.16	.99	-.16
Paternal Specific Phobia	-.35	1.36	-.26	-.58	1.45	-.40
Paternal Substance Dependence	-.07	.67	-.10	-.07	.72	-.09

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix J

Simultaneous Regression Analyses Examining Associations Between Pe – Correct Positivity Trial at Fz and Cz and Parental Psychopathology

	Pe – Correct Trial Positivity at Fz			Pe – Correct Trial Positivity at Cz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
		N = 320			*N* = 320	
Maternal Depression	.18	1.39	.13	.58	1.52	.38
Maternal Anxiety Disorder (excluding specific phobia)	.85	1.45	.58	1.77	1.59	1.11
Maternal Specific Phobia	1.28	1.89	.68	-1.17	2.07	-.57
Maternal Substance Dependence	1.07	1.56	.69	.96	1.71	.56
		N = 319			*N* = 319	
Paternal Depression	.32	1.80	.18	2.62	1.97	1.33
Paternal Anxiety Disorder (excluding specific phobia)	-.99	1.77	-.56	-1.90	1.93	-.98
Paternal Specific Phobia	.60	2.60	.61	.29	2.84	.10
Paternal Substance Dependence	-.17	1.29	-.13	-.69	1.40	-.49

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix K

Simultaneous Regression Analyses Examining Associations Between Pe Amplitude at Fz and Cz and Parental Psychopathology

	Pe at Fz			Pe at Cz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
		N = 320			*N* = 320	
Maternal Depression	.67	1.40	.48	.98	1.55	.63
Maternal Anxiety Disorder (excluding specific phobia)	1.62	1.6	1.11	2.16	1.62	1.34
Maternal Specific Phobia	.52	1.90	.27	-2.21	2.11	-1.05
Maternal Substance Dependence	1.04	1.57	.66	.81	1.74	.47
		N = 319			*N* = 319	
Paternal Depression	.51	1.82	.28	2.18	2.01	1.09
Paternal Anxiety Disorder (excluding specific phobia)	-.62	1.79	-.35	-1.53	1.97	-.78
Paternal Specific Phobia	.84	2.62	.32	-.11	2.89	-.04
Paternal Substance Dependence	.00	1.30	.00	-.48	1.43	-.34

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix L

Simultaneous Regression Analyses Examining Associations Between Total Correct Responses on Go Trials, Reaction Time on Correct Responses to Go Trials, and Total Correct No-Go Trials and Parental Loading of Psychopathology

	Total Correct Responses on Go Trials			Reaction Time on Correct Responses to Go Trials			Total Correct No-Go Trials		
Variables Entered	b	Standard Error $N = 309$	*t*	b	Standard Error $N = 303$	*t*	b	Standard Error $N = 314$	*t*
Zero, One, or Both Parents with Depression	.27	.74	.37	-3.11	6.39	-.49	.68	1.10	.62
Zero, One, or Both Parents with Anxiety Disorder (excluding specific phobia)	1.24	.80	1.54	-2.41	6.92	-.35	-1.69	1.18	-1.43
Zero, One, or Both Parents with Specific Phobia	.68	1.10	.62	4.57	9.57	.48	-.15	1.64	-.09
Zero, One, or Both Parents with Substance Dependence	.06	.69	.09	-.43	6.17	-.07	.19	1.03	.18

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix M

Simultaneous Regression Analyses Examining Associations Between Total Errors of Commission, Reaction Time on Errors of Commission, and Total Errors of Omission and Parental Loading of Psychopathology

	Total Errors of Commission			Reaction Time on Errors of Commission			Total Errors of Omission		
Variables Entered	b	Standard Error $N = 314$	*t*	b	Standard Error $N = 314$	*t*	b	Standard Error $N = 301$	*t*
Zero, One, or Both Parents with Depression	-.91	.70	-1.30	3.21	7.51	.43	1.26	.98	1.29
Zero, One, or Both Parents with Anxiety Disorder (excluding specific phobia)	.65	.75	.88	-2.55	8.08	-.32	-.77	1.06	-.73
Zero, One, or Both Parents with Specific Phobia	-.38	1.04	-.37	7.69	11.17	.69	-.17	1.47	-.12
Zero, One, or Both Parents with Substance Dependence	-.32	.66	-.49	-1.65	7.08	-.23	.52	.95	.55

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix N

Simultaneous Regression Analyses Examining Associations Between Total Correct Trials Following Errors of Commission, Reaction Time on Correct Go Trials Following Errors of Commission, and Total Accuracy and Parental Loading of Psychopathology

	Total Correct Go Trials Following Errors of Commission			Reaction Time on Correct Go Trials Following Errors of Commission			Total Accuracy		
Variables Entered	b	Standard Error $N = 314$	*t*	b	Standard Error $N = 314$	*t*	b	Standard Error $N = 301$	*t*
Zero, One, or Both Parents with Depression	-.89	.41	-2.20*	-2.41	10.46	-.23	-.70	1.44	-.49
Zero, One, or Both Parents with Anxiety Disorder (excluding specific phobia)	.30	.43	.68	-2.42	11.26	-.22	.54	1.5	.35
Zero, One, or Both Parents with Specific Phobia	-.37	.60	-.62	11.92	15.56	.77	.79	2.15	.37
Zero, One, or Both Parents with Substance Dependence	.07	.38	.18	2.71	9.86	.27	-.38	1.39	-.27

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix O

Simultaneous Regression Analyses Examining Associations Between ERN – CRN at Fz, Cz, and Pz and Parental Loading of Psychopathology

	ERN – CRN at Fz			ERN – CRN at Cz			ERN – CRN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
N = 318									
Zero, One, or Both Parents with Depression	-1.34	.78	-1.72†	-.91	.82	-1.11	.92	.81	1.13
Zero, One, or Both Parents with Anxiety Disorder (excluding specific phobia)	2.06	.83	2.48*	1.72	.87	1.98*	.09	.86	.10
Zero, One, or Both Parents with Specific Phobia	1.91	1.15	1.65†	2.57	1.21	2.13*	2.33	1.20	1.94†
Zero, One, or Both Parents with Substance Dependence	-.16	.73	-.21	.09	.77	.12	.64	.76	.83

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix P

Simultaneous Regression Analyses Examining Associations Between ERN Amplitude at Fz, Cz, and Pz and Parental Loading of Psychopathology

	ERN at Fz			ERN at Cz			ERN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
N = 318									
Zero, One, or Both Parents with Depression	-.73	.74	-.99	-.90	.83	-1.08	.56	.83	.68
Zero, One, or Both Parents with Anxiety Disorder (excluding specific phobia)	2.66	.78	3.40***	2.24	.89	2.53*	.58	.88	.66
Zero, One, or Both Parents with Specific Phobia	1.08	1.09	.99	1.01	1.23	.82	.72	1.22	.59
Zero, One, or Both Parents with Substance Dependence	-.09	.69	-.13	.20	.78	.25	.59	.78	.76

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix Q

Simultaneous Regression Analyses Examining Associations Between CRN Amplitude at Fz, Cz, and Pz from Parental Loading of Psychopathology

	CRN at Fz			CRN at Cz			CRN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
N = 318									
Zero, One, or Both Parents with Depression	.64	.42	1.54	.06	.53	.11	-.35	.57	-.61
Zero, One, or Both Parents with Anxiety Disorder (excluding specific phobia)	.56	.44	1.27	.45	.56	.80	.48	.60	.80
Zero, One, or Both Parents with Specific Phobia	-.80	.62	-1.29	-1.52	.78	-1.94†	-1.60	.84	-1.91†
Zero, One, or Both Parents with Substance Dependence	.07	.39	.17	.11	.50	.22	-.04	.53	-.08

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix R

Simultaneous Regression Analyses Examining Associations Between Pe – Correct Trial Positivity Amplitude at Fz, Cz, and Pz and Parental Loading of Psychopathology

	Pe – Correct Trial Positivity at Fz			Pe – Correct Trial Positivity at Cz			Pe – Correct Trial Positivity at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
N = 316									
Zero, One, or Both Parents with Depression	.04	1.03	.04	1.00	1.13	.88	1.27	1.12	1.14
Zero, One, or Both Parents with Anxiety Disorder (excluding specific phobia)	.41	1.09	.38	.53	1.19	.44	.85	1.19	.71
Zero, One, or Both Parents with Specific Phobia	1.50	1.52	.99	-.45	1.67	-.27	.13	1.65	.08
Zero, One, or Both Parents with Substance Dependence	.28	.97	.29	-.06	1.06	-.05	1.30	1.05	1.23

$^{\dagger}p \leq .10$, $^{*}p \leq .05$, $^{**}p \leq .01$, $^{***}p \leq .001$

Appendix S

Simultaneous Regression Analyses Examining Associations Between Pe Amplitude at Fz, Cz, and Pz and Parental Loading of Psychopathology

	Pe at Fz			Pe at Cz			Pe at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*	b	Standard Error	*t*
N = 316									
Zero, One, or Both Parents with Depression	.36	1.03	.35	1.09	1.15	.95	1.14	1.10	1.04
Zero, One, or Both Parents with Anxiety Disorder (excluding specific phobia)	.96	1.10	.87	.85	1.22	.70	.95	1.17	.82
Zero, One, or Both Parents with Specific Phobia	.79	1.53	.52	-1.28	1.70	-.75	-1.05	1.63	-.64
Zero, One, or Both Parents with Substance Dependence	.36	.97	.37	.03	1.08	.03	1.07	1.04	1.03

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix T

Simultaneous Regression Analyses Examining Associations Between ERN – CRN Amplitude at Fz and Pz and Child Temperament

	ERN – CRN at Fz			ERN – CRN at Pz		
Variables Entered	b	Standard Error N = 326	*t*	b	Standard Error N = 326	*t*
Child PE	.09	.28	.33	-.04	.29	-.15
Child NE	2.48	1.84	1.35	4.45	1.90	2.35*
Child PE	.08	.28	.28	-.05	.29	-.16
Child Sadness	-.74	1.86	-.40	.54	1.92	.28
Child Fear	2.49	1.39	1.80†	3.64	1.43	2.55*
Child Anger	.76	1.55	.49	.49	1.60	.30
Child PE	.07	.28	.23	-.03	.29	-.11
Child BI	.17	1.18	.14	1.38	1.22	1.13

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix U

Simultaneous Regression Analyses Examining Associations Between ERN Amplitude at Cz and Pz and Child Temperament

	ERN at Cz			ERN at Pz		
Variables Entered	b	Standard Error N = 326	*t*	b	Standard Error N = 326	*t*
Child PE	-.18	.29	-.63	-.09	.29	-.32
Child NE	4.66	1.95	2.40*	4.35	1.91	2.27*
Child PE	-.21	.29	-.71	-.09	.29	-.28
Child Sadness	.29	1.97	.15	2.19	1.93	1.14
Child Fear	2.29	1.47	1.56	3.25	1.43	2.26*
Child Anger	2.27	1.64	1.38	-.69	1.60	-.43
Child PE	-.26	.30	-.87	-.10	.29	-.34
Child BI	-.23	1.25	-.18	1.08	1.23	.88

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix V

Simultaneous Regression Analyses Examining Associations Between CRN Amplitude at Cz and Pz and Child Temperament

	CRN at Cz			CRN at Pz		
Variables Entered	b	Standard Error N = 326	*t*	b	Standard Error N = 326	*t*
Child PE	.07	.19	.36	-.04	.20	-.17
Child NE	.10	1.24	.08	-.12	1.33	-.09
Child PE	.08	.19	.43	-.01	.20	-.06
Child Sadness	1.49	1.26	1.18	1.68	1.35	1.25
Child Fear	-.60	.94	-.64	-.35	1.00	-.35
Child Anger	-.64	1.05	-.61	-1.28	1.13	-1.13
Child PE	.02	.19	.12	-.05	.20	-.22
Child BI	-.90	.79	-1.14	-.25	.85	-.30

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix W

Simultaneous Regression Analyses Examining Associations Between Pe – Correct Trial Positivity at Fz and Cz and Child Temperament

	Pe – Correct Trial Positivity at Fz			Pe – Correct Trial Positivity at Cz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
		N = 324			*N* = 324	
Child PE	-.20	.36	-.56	-.31	.39	-.78
Child NE	-.12	2.38	-.05	-1.13	2.61	-.44
Child PE	-.22	.36	-.62	-.34	.39	-.86
Child Sadness	-3.46	2.40	-1.44	-4.39	2.63	-1.67†
Child Fear	2.47	1.79	1.38	1.33	1.96	.68
Child Anger	.81	2.00	.41	1.69	2.19	.77
Child PE	-.18	.36	-.49	-.25	.40	-.63
Child BI	.52	1.52	.34	.85	1.66	.51

† $p \leq .10$, * $p \leq .05$, ** $p \leq .01$, *** $p \leq .001$

Appendix X

Simultaneous Regression Analyses Examining Associations Between Pe Amplitude at Fz and Cz and Child Temperament

	Pe at Fz			Pe at Cz		
Variables Entered	b	Standard Error N = 324	*t*	b	Standard Error N = 324	*t*
Child PE	-.17	.05	-.48	-.37	.40	-.93
Child NE	.52	2.40	.22	-.35	2.65	-.13
Child PE	-.19	.36	-.52	-.39	.40	-.97
Child Sadness	-2.53	2.42	-1.05	-2.97	2.68	-1.11
Child Fear	2.47	1.80	1.37	1.52	1.99	.76
Child Anger	.58	2.02	.29	.92	2.23	.41
Child PE	-.16	.37	-.43	-.32	.41	-.79
Child BI	.57	1.53	.37	1.02	1.69	.60

$^{\dagger}p \leq .10$, $^{*}p \leq .05$, $^{**}p \leq .01$, $^{***}p \leq .001$

Appendix Y

Hierarchical Multiple Regression Analyses Examining Associations Between ERN – CRN at Fz and Child PE and NE

(*N* = 325)	ERN – CRN at Fz		
Variables Entered	B	Standard Error	*t*
Child PE	.08	.27	.30
Child NE	2.57	1.85	1.39
PE x NE	-.33	1.16	-.28

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Appendix Z

Hierarchical Multiple Regression Analyses Examining Associations Between ERN – CRN at Pz and Child Age at ERN Assessment, Child PE, and Child NE

(*N* = 325)	ERN – CRN at Pz		
Variables Entered	B	Standard Error	*t*
Child Age at Assessment	-2.60	1.15	-2.25*
Child PE	-.03	.29	-.10
Child NE	4.44	1.92	2.31*
Child Age x Child PE	.31	.67	.46
Child Age x Child NE	-.96	4.18	-.23
PE x NE	-.20	1.22	-.16

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Appendix AA

Hierarchical Multiple Regression Analyses Examining Associations between ERN Amplitude at Cz and at Pz and Child PE and NE

(*N* = 325)	ERN at Cz			ERN at Pz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
Child PE	-.18	.29	-.63	-.09	.29	-.32
Child NE	4.95	1.95	2.53*	4.48	1.93	2.33*
Child PE x Child NE	1.64	1.23	1.33	.67	1.21	.55

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Appendix BB

Simultaneous Regression Analyses Examining Child NE as a Moderator of the Relationship Between the ERN at Cz and Maternal Anxiety

(*N* = 321)	ERN at Cz		
Variables Entered	b	Standard Error	*t*
Maternal Anxiety Disorder (excluding specific phobia)	3.19	1.12	2.85**
Child NE	6.97	5.81	1.20
NE x Maternal Anxiety	-1.72	4.12	-.42

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Appendix CC

Simultaneous Regression Analyses Predicting ERN Amplitude at Cz from Child Fear and Maternal Anxiety

(*N* = 321)	ERN at Cz		
Variables Entered	b	Standard Error	*t*
Maternal Anxiety Disorder (excluding specific phobia)	3.28	1.12	2.92**
Child Fear	.53	4.27	.12
Fear x Maternal Anxiety	1.58	3.12	.51

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Appendix DD

Simultaneous Regression Analyses Examining Child NE as a Moderator of the Relationship Between the ERN – CRN Difference at Cz and Parental Loading of an Anxiety Disorder (excluding specific phobia)

(*N* = 317)	ERN – CRN at Cz		
Variables Entered	b	Standard Error	*t*
Child Age at Assessment	-2.02	3.19	-.63
Zero, One, or Two Parents with an Anxiety Disorder	1.60	.84	1.90†
Child NE	7.60	5.21	1.46
Child Age x Zero, One, or Two Parents with an Anxiety Disorder	-.54	2.12	-.26
Child Age x NE	3.31	4.17	.80
NE x Zero, One, or Two Parents with an Anxiety Disorder	-1.85	3.28	-.56

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Appendix EE

Simultaneous Regression Analyses Examining Child NE as a Moderator of the Relationship Between the ERN – CRN at Cz and Parental Loading of a Specific Phobia

(*N* = 317)	ERN – CRN at Cz		
Variables Entered	b	Standard Error	*t*
Child Age at Assessment	-.82	3.18	-.26
Zero, One, or Two Parents with a Specific Phobia	2.56	1.18	2.16*
Child NE	4.50	5.99	.75
Child Age x Zero, One, or Two Parents with a Specific Phobia	-1.6	2.43	-.70
Child Age x NE	3.24	4.18	.78
NE x Zero, One, or Two Parents with a Specific Phobia	.28	4.75	.06

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Appendix FF

Simultaneous Regression Analyses Examining Child NE as a Moderator of the Relationship Between the ERN – CRN at Cz, and Parental Loading of an Anxiety Disorder (excluding specific phobia)

(*N* = 317)	ERN – CRN at Cz		
Variables Entered	b	Standard Error	*t*
Child Age at Assessment	-2.70	3.19	-.85
Zero, One, or Two Parents with an Anxiety Disorder	1.67	.85	1.98*
Child Fear	4.66	3.74	1.25
Child Age x Zero, One, or Two Parents with an Anxiety Disorder	-.27	2.11	-.13
Child Age x Fear	.50	3.06	.16
Fear x Zero, One, or Two Parents with an Anxiety Disorder	-.74	2.43	-.31

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Appendix GG

Simultaneous Regression Analyses Examining Child Fear as a Moderator of the Relationship Between the ERN – CRN at Cz and Parental Loading of a Specific Phobia

(*N* = 317)	ERN – CRN at Cz		
Variables Entered	b	Standard Error	*t*
Child Age at Assessment	-1.65	3.20	-.51
Zero, One, or Two Parents with a Specific Phobia	2.41	1.20	2.01*
Child Fear	.13	4.30	.03
Child Age x Zero, One, or Two Parents with a Specific Phobia	-1.21	2.46	-.49
Child Age x Fear	.73	3.07	.24
Fear x Zero, One, or Two Parents with a Specific Phobia	2.73	3.37	.81

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Appendix HH

Simultaneous Regression Analyses Examining Child NE as a Moderator of the Relationship Between the ERN at Cz and at Fz and Parental Loading of an Anxiety Disorder (excluding specific phobia)

(*N* = 317)	ERN at Cz			ERN at Fz		
Variables Entered	b	Standard Error	*t*	b	Standard Error	*t*
Zero, One, or Two Parents with an Anxiety Disorder	2.05	.85	2.43*	2.49	.75	3.32***
Child NE	8.42	5.23	1.61†	6.20	4.63	1.33
NE x Zero, One, or Two Parents with an Anxiety Disorder	-2.29	3.29	-.70	-1.73	2.92	-.59

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$