

ABSTRACT

Title of Dissertation: **SOCIAL INFLUENCES OF ERROR
MONITORING**

 Tyson V. Barker, Doctorate of Philosophy, 2016

Dissertation directed by: **Professor Nathan A. Fox, Department of Human
Development and Quantitative Methodology**

Adolescence is characterized by dramatic hormonal, physical, and psychological changes, and is a period of risk for affective and anxiety disorders. Pubertal development during adolescence plays a major role in the emergence of these disorders, particularly among girls. Thus, it is critical to identify early biomarkers of risk. One potential biomarker, the error-related negativity (ERN), is an event-related potential following an erroneous response. Individuals with an anxiety disorder demonstrate a greater ERN than healthy comparisons, an association which is stronger in adolescence, suggesting that pubertal development may play a role in the ERN as a predictor of anxiety. One form of anxiety often observed in adolescence, particularly among girls, is social anxiety, which is defined as anxiety elicited by social-evaluative contexts. In adults, enhancements of the ERN in social-evaluative contexts is positively related to social anxiety symptoms, suggesting that the ERN in social contexts may serve as a biomarker for social anxiety.

This dissertation examined the ERN in and its relation with puberty and social anxiety among 76 adolescent girls. Adolescent girls completed a flanker task in two different conditions. In the social condition, adolescents were informed that two other adolescents would be observing their performance. In the nonsocial condition, adolescents completed a flanker task alone. Results revealed that self-report of puberty predicted developmental changes in the ERN. Furthermore, the ERN was enhanced in social contexts as compared to nonsocial contexts, and the greatest enhancements were observed among early pubertal adolescents. In contrast to predictions, puberty did not moderate the association between social anxiety and enhancements of the ERN in social contexts. However, reductions of the ERN in social contexts was related to more depressive symptoms. This study is the first to demonstrate the role of puberty in influencing the ERN. Additionally, these findings are further evidence that the ERN is sensitive to social factors, particularly in adolescence. Lastly, reductions of the ERN in social contexts may be a possible biomarker for depression.

SOCIAL INFLUENCES OF ERROR MONITORING

by

Tyson V. Barker

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Advisory Committee:
Professor Nathan A. Fox, Chair
Professor Donald J. Bolger
Professor Andrea Chronis-Tuscano
Dr. Daniel S. Pine
Professor Tracy T. Riggins

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Chapter 1. Introduction

Learning from the consequences of actions is critical for adaptive behavior (Thorndike, 1911; Thorndike, 1927). One of the hallmarks of learning is the ability to recognize mistakes and adjust behavior to prevent future errors (Segalowitz & Dywan, 2009). The ability to identify errors, known as error monitoring (or more generally known as performance monitoring), is a complex skill that undergoes a protracted rate of development beginning in early childhood and continuing through adolescence (see Tamnes, Walhovd, Torstveit, Sells, & Fjell, 2013 for review). Brain regions that support error monitoring, namely the prefrontal cortex (PFC) and anterior cingulate cortex (ACC; Carter et al., 1998; Luria, 1966; Van Veen & Carter, 2002), exhibit a similar delayed developmental pattern (Gogtay et al., 2004; Tamnes et al., 2010; Westlye et al., 2010). Individual differences in error monitoring are in part biological based (Anokhin, Golosheykin, & Heath, 2008). However, the error monitoring system can also be influenced by a number of environmental factors, particularly those which influence motivation (Luu, Tucker, Derryberry, Reed, & Poulsen, 2003a; Proudfit, Inzlicht, & Mennin, 2013), via reward (Cavanagh, Frank, & Allen, 2011; Frank, Woroch, & Curran, 2005; Holroyd & Coles, 2002), and/or punishment (Riesel, Weinberg, Endrass, Kathmann, & Hajcak, 2011; Riesel, Weinberg, Moran, & Hajcak, 2013). Abnormalities in the error monitoring system have been observed in numerous psychological disorders, such as anxiety (Hajcak, 2012; Ursu, Stenger, Shear, Jones, & Carter, 2003), depression (Holmes & Pizzagalli, 2008), and attention deficit hyperactivity disorder (ADHD; van Meel, Heslenfeld & Oosterlaan, 2007). Thus, the study of the development of the error monitoring system

may lead to a better understanding of biological and environmental factors that heighten risk for psychopathology.

Adolescence, which is typically defined by the onset of puberty, is a transition period in development characterized by dramatic hormonal, physical, and psychological changes. Adolescence is also characterized by increased risk-taking behavior and susceptibility to peer pressure (Casey et al., 2010). Changes in adolescent behavior are theorized to be driven by the reorganization of neural circuits critical for social motivation and reward processing (Crone & Dahl, 2012; Nelson, Leibenluft, McClure, & Pine, 2005; Steinberg et al., 2008). For example, the PFC and ACC, two regions critical for both cognitive control and motivation (Bush, Luu, & Posner, 2000), continue to undergo dramatic maturation into adolescence (Gogtay et al., 2004; Tamnes et al., 2010). The mesencephalic dopaminergic system, originating in the midbrain with many projections to the ACC and PFC, plays an important role in motivation and reward-seeking behavior (Schultz, 2007). The mesencephalic dopaminergic system undergoes dramatic changes during pubertal development (Sisk & Foster, 2004; Sisk & Zehr, 2005), suggesting that increases in sex hormones during puberty may initiate the reorganization of the adolescent brain (Forbes & Dahl, 2010). Such reorganization may result in the heightened social, affective and reward processing observed in adolescence (Dahl & Crone, 2012).

Due to such dramatic biological and social changes, adolescence has been characterized as a window of risk for the development of psychopathology (Dahl, 2004). Pubertal development plays a major role in the emergence of psychopathology in adolescence, particularly among girls (Angold et al., 1999; Reardon et al., 2009).

Adolescent girls enter puberty earlier than boys (Sun et al., 2002), and are twice as likely to develop an depressive or anxiety disorder (Kessler et al., 2005; Lewinsohn et al., 1998), suggesting that this heightened risk in girls is in part driven by the effects of puberty on the function and structure of neural circuits supporting affective processing (Crone & Dahl, 2012; Nelson et al., 2005). Indeed, a number of neuroanatomical differences between sexes emerge in adolescence (Lenroot et al., 2007), which are influenced by differences in pubertal hormone concentrations (Herting et al., 2014). As such, it is important to utilize neural markers of affective processing to explore the role of puberty in the emergence of anxiety and depressive disorders, particularly among adolescent girls.

One potential biomarker for anxiety disorders, the error-related negativity (ERN), is a neural correlate of the error monitoring system. The ERN is a negative deflection observed in the event-related potential (ERP) following an erroneous motor response (see figure 1; Gehring et al., 1993). The ERN is theorized to be generated in the ACC (Dehaene, Posner, & Tucker, 1994; Holroyd, Dien, & Coles, 1998), by a dip in mesencephalic dopaminergic activity, which disinhibits firing of neurons in the ACC (Frank et al., 2005; Holroyd & Coles, 2002). The ERN emerges in early childhood (Grammer, Carrasco, Gehring, & Morrison, 2014) and exhibits a delayed developmental pattern (Davies, Segalowitz, & Gavin, 2004). The most notable changes in the ERN are observed from early to late adolescence (Ladouceur, Dahl, & Carter, 2004, 2007; Santesso & Segalowitz, 2008), suggesting that puberty may explain such developmental changes. Sex differences in the ERN emerge after adolescence (Davies et al., 2004; Larson, South, & Clayson, 2011), further suggesting

pubertal influences. However, no research has examined whether pubertal development explains changes in the magnitude of the ERN in adolescence. A related component, the positive error (Pe), is a positive-deflection in the ERP waveform that occurs approximately 200-400 ms after an error (Overbeek, Nieuwenhuis, & Richard, 2005; see figure 1). In contrast to the ERN, the Pe exhibits very little change in adolescence (Davies et al., 2004). It is also unknown whether puberty influences the Pe.

Many studies have examined the importance of the ERN as a measure of motivation and reward processing (Frank et al., 2005; Proudfit et al., 2013). The ERN is enhanced when accuracy is emphasized over speed (Gehring et al., 1993), when errors are punished (Hajcak & Foti, 2008), and when errors incur a monetary cost (Hajcak, Moser, Yeung, & Simons, 2005). In motivationally salient social contexts, such as when performance is critically evaluated, or when errors are observed by a peer, the magnitude of the ERN is enhanced (Barker, Troller-Renfree, Pine, & Fox, 2015; Hajcak et al., 2005; Kim, Iwaki, Uno, & Fujita, 2005). However, it is unknown

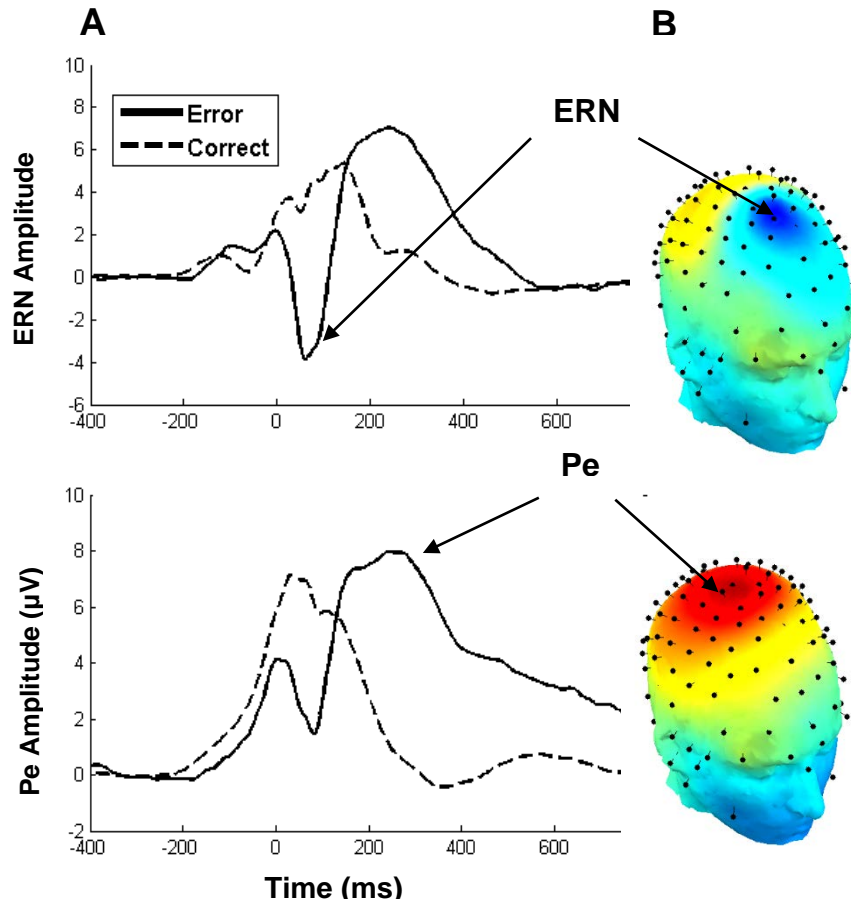


Figure 1. A) Response-locked ERP waveforms on correct (dashed line) and error (solid line) responses for adults ($N = 44$). The top row is the average of fronto-central electrodes where the ERN is maximal. The bottom row is an average of centro-parietal electrodes where the Pe is maximal. B) Top image is the scalp topography of the ERN at 80 ms after an erroneous response. The bottom image is the scalp topography of the Pe after 250 ms after an erroneous response.

whether the ERN is also influenced by social factors in adolescence, a period in development characterized by increased importance of social evaluation (La Greca & Lopez, 1998), and increased social motivation and reward processing (Crone & Dahl, 2012). Enhanced social motivation in adolescence is theorized to be caused in part by changes in sex hormone concentrations during puberty (Forbes & Dahl, 2010). However, it is unknown if puberty affects the degree to which the ERN is enhanced in social contexts in adolescence.

A large body of literature has demonstrated that the ERN is elevated among individuals with an anxiety disorder (Carrasco, Hong, et al., 2013; Endrass, Riesel, Kathmann, & Buhmann, 2014; Gehring, Himle, & Nisenson, 2000; Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006; Weinberg, Olvet, & Hajcak, 2010). The ERN has also been found to relate to dimensional aspects of anxiety, particularly anxious apprehension (i.e., worry; Moser, Moran, & Jendrusina, 2012; Moser, Moran, Schroder, Donnellan, & Yeung, 2013; Zambrano-Vazquez & Allen, 2014). The relation between dimensional aspects of anxiety and the ERN are strongest among females (Moran, Taylor, & Moser, 2012; Moser, Moran, Kneip, Schroder, & Larson, 2016). Additionally, the association between dimensional aspects of anxiety are stronger in adolescence than in childhood (Meyer et al., 2012). Such differences in the strength of the association between the ERN and dimensional aspects in anxiety suggest that puberty may moderate the association between the ERN and anxiety, particularly among adolescent girls. However, it has yet to be examined whether pubertal development moderates the relation between the ERN and dimensional aspects of anxiety among adolescent girls.

It has been theorized that the larger ERN observed in anxious individuals is due to an increased defensive motivation to threat (Proudfit et al., 2013; Weinberg, Meyer, Hale-Rude, et al., 2016). Errors are a distressing event, particularly for anxious individuals (Proudfit et al., 2013). Contextual factors can also interact with individual differences in anxiety to influence the degree to which errors are perceived as distressing. For example, individual differences in anxiety are related to the degree to which the ERN is enhanced when errors are punished (Riesel, Weinberg, et al.,

2011; Riesel et al., 2013). Social observation can increase perceived distress from errors (Geen, 1991). Social anxiety, which is defined by social-evaluative anxiety, predicts the degree to which the ERN is enhanced in social contexts (Barker et al., 2015). Thus, enhancements of the ERN in social contexts may reflect defensive responses to perceived threats in social contexts. However, it is unknown if social anxiety is related to an enhanced ERN in social contexts in adolescence, a period in development characterized by enhanced social motivation and social-evaluative anxiety (La Greca & Lopez, 1998).

Given the above stated gaps in the literature, the overarching goal of the proposed study was to explore how puberty influences the magnitude of the ERN (and the Pe) under different social contexts, and whether puberty moderates the relation between neural correlates of error monitoring and social anxiety. To accomplish this goal, 76 adolescent girls between 8-17 years of age participated in the present study. Only adolescent girls were recruited since girls are more sensitive to peer evaluation (La Greca & Lopez, 1998), exhibit different brain and pubertal maturation patterns (Lenroot et al., 2007; Sun et al., 2002), and are at a greater risk for the development of depression and social anxiety than boys (Kessler et al., 2005; Lewinsohn et al., 1998). Furthermore, the ERN is larger in females after adolescence (Davies et al., 2004; Grammer et al., 2015; Larson et al., 2011), and the ERN and anxiety relation is strongest among females (Moran et al., 2012; Moser et al., 2016).

In the present study, adolescent pubertal status was collected via parent-report and self-report (Petersen et al., 1988). In addition, parents and adolescents reported on adolescent anxiety and depressive symptoms (Muris et al., 1998). To measure the

ERN and Pe, adolescents completed a flanker task while electroencephalogram (EEG) was collected under two different conditions. In the social condition, adolescents were told that two adolescents located in another lab would observe and evaluate their performance. In the nonsocial condition, adolescents completed the flanker task alone.

The first aim of the proposed study was to examine whether pubertal status predicted changes in the ERN and Pe across adolescence. The second aim of the proposal was to examine the effect of puberty in modulating the ERN and Pe under different social contexts. The third aim of the proposal was to examine the relation between the degree to which the ERN and the Pe were enhanced in social contexts and symptoms of social anxiety. In addition, the proposal examined whether pubertal status moderated the relation between enhancements of the error monitoring system in social contexts and social anxiety symptoms.

Exploring how puberty influences the error monitoring system will aide in the understanding of how pubertal hormones influences the mesencephalic dopamine system and the function of brain structures such as the ACC. Furthermore, the proposed study will gain insight into the usefulness of the ERN and Pe as a biomarker for affective disorders in adolescence.

Chapter 2. Background

The aim of the current chapter is to present theoretical and empirical support for the hypothesis that changes in the ERN in adolescence are likely due to puberty, reflecting reorganization of brain circuits critical for social motivation and reward

processing. This reorganization increases risk for anxiety and depressive disorders, and this risk can be measured by biomarkers such as the ERN (and perhaps the Pe). As such, the proposal presents empirical support that the ERN is a measure of social motivation, and changes in the ERN during social contexts may be viable biomarker for social anxiety in adolescent girls. First, the chapter will review adolescent development with a specific focus on the role of puberty in reorganizing the mesencephalic dopamine system and structures such as the ACC. Second, the chapter will review behavior and neural indices of the error monitoring system, with a specific focus on the ERN and the Pe. Third, the chapter will review empirical findings for the neural basis of the ERN and Pe. Fourth, theories of the functional significance of the ERN and the Pe will be reviewed. Fifth, empirical studies on the development of the error monitoring system, both behavioral (e.g., post-error slowing) and psychophysiological indices of the system (e.g., ERN, Pe) are reviewed, with a specific focus on changes observed during adolescence. Fifth, empirical findings of the association between the ERN/Pe and anxiety and depression are reviewed. Last, the chapter will review the ERN as a biomarker for affective disorders in adolescent girls.

2.1 Adolescent Development: The Role of Puberty

Adolescence is a critical transition period in development characterized by rapid biological, cognitive, and emotional changes. The beginning of adolescence is traditionally defined by the onset of puberty. However, the end of adolescence is less well-defined, and is typically based upon cultural expectations in which the emerging

adult can take on adult roles and responsibilities in society (Arnett, 1999, 2000). In many domains of development, adolescence is viewed as a period of prosperity, due to increases in physical health, as well as cognitive and emotional growth. Yet, despite these improvements, adolescence has been referred to as a period of “storm and stress”, due to increasing conflict with parents, sharp swings in mood, risky behavior, and poor decision making (Arnett, 1999; Hall, 1916). These changes in behavior are also accompanied by increases in mortality (Eaton et al., 2012; Resnick et al, 1997) as well as the emergence of psychopathology, such as depressive and anxiety disorders (Kessler et al., 2005; Lewinsohn et al., 1998). Thus, Dahl (2004) has referred to this disparity as the health paradox of adolescence. Researchers have increasingly focused on why adolescents demonstrate such dramatic changes in behavior (Arnett, 1999; Crone & Dahl, 2012; Dahl, 2004; Steinberg, 2005, 2008). One promising line of research has focused on the role of puberty in reorganizing adolescent brain and behavior (Dahl, 2004; Sisk & Foster, 2004; Sisk & Zehr, 2005; Steinberg, 2008).

One of the most noticeable changes in adolescent behavior is the increasing desire for peer affiliation (Larson & Richards, 1991; Larson, Richards, Moneta, Holmbeck, & Duckett, 1996; Rubin, Bukowski, & Parker, 1998). Typically beginning in middle school, adolescents transition from spending time with family to spending time with peers (Steinberg & Silverberg, 1986), and become increasingly sensitive to peers’ perceptions, opinions, and approval (Bradford, 1990). Along with increased propensity to seek peer relationships, adolescents are also known to engage in risky behavior (Steinberg, 2007). Adolescents are more likely to engage in drug and

alcohol use, unprotected sex, and driving under the influence of alcohol than adults (Eaton et al., 2008). Interestingly, adolescents are comparable to adults in reporting the degree of perceived risk and their vulnerability to these risks (Reyna & Farley, 2006), as well as in their judgments about the consequences of risky behavior (Beyth-Marom, Austin, Fischhoff, Palmgren, & Jacobs-Quadrel, 1993). Thus, it has been suggested that adolescent risk behavior is not the result of cognitive deficits, but rather due to the relative immaturity of neural regions responsible for reward, affective, and social processing (Steinberg, 2007).

Puberty is thought to play an important role in the reorganization of neural networks responsible for social and affective processing (Dahl, 2004; Ernst, Romeo, & Andersen, 2009). Pubertal development is initiated by increases in hormones secreted by the adrenal and gonadal glands (see Buck Louis, et al., 2008 for review). On average, physical changes associated with puberty begin in girls around 10 years of age, with girls typically reaching sexual maturation by 16 years of age (Sun et al., 2005). For boys, physical changes begin approximately 1.5 years later (Sun et al., 2005). However, there is large variability in pubertal timing for both sexes (Sun et al., 2005). Although the beginning of puberty is typically viewed to begin with physical changes, increases in pubertal hormone secretions occur earlier (Dorn, Dahl, Woodward, & Biro, 2006). Both adrenal and gonadal hormones play major roles in organizing adolescent brain function and structure (Dahl, 2004; Romeo & McEwen, 2006) and may influence affective processing and motivational behaviors (Ernst et al., 2009). However, the exact nature of how these systems organize behavior is still relatively unknown (Steinberg, 2008).

Adrenarche, known as the awakening of the adrenal gland, is the first change associated with puberty. Adrenarche typically begins between 6-9 years of age (Grumbach, 2002). During adrenarche, androgen steroids, such as dehydroepiandrosterone (DHEA), are released at increasing levels from the adrenal gland (Nakamura, Gang, Suzuki, Sasano, & Rainey, 2009). However, other adrenal hormones, such as basal corticoids, are relatively stable in late childhood and are not released at increasing levels until much later in adolescence (Apter, Pakarinen, Hammond, & Vihko, 1979; Elmlinger, Kühnel, & Ranke, 2005; Netherton, Goodyer, Tamplin, & Herbert, 2004). Adrenal hormones such as DHEA cause changes in pubic hair and body odor, which are the first physical changes associated with puberty (Petersen et al., 1988). Gonadarche, which is the second phase of puberty, is associated with the maturation of the gonads. Gonadarche typically begins around 9-10 years of age in girls, and typically a year later in boys (Grumbach, 2002). The activation of the gonads is triggered by the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). In girls, gonadal development leads to the release of estradiol and progesterone by the ovaries, which causes the development of primary and secondary sexual characteristics and the development of the menstrual cycle (Marshall & Tanner, 1969, 1970). In boys, the gonads release androgens from the testes, such as testosterone, which result in the development of primary and secondary sexual characteristics (Marshall & Tanner, 1969, 1970). Menarche, which is the onset of the menstrual cycle in girls, occurs relatively late in pubertal development, on average at 12.5 years (Anderson et al., 2005). Since menarche

occurs relatively late in pubertal development, it has been suggested that menarche status is not a strong measure of pubertal development (Dorn et al., 2006).

The release of pubertal hormones is theorized to play a causal role in the observed changes in social motivation and reward processing observed in adolescence (Forbes & Dahl, 2010). Many gender differences in peer relationships emerge in adolescence (Paquette & Underwood, 1999) suggesting that the organizational effects of puberty may differentially influence behavior between sexes. For example, adolescent girls, relative to males, are more sensitive to social signals in adolescence (McClure, 2000), and report greater concern for social acceptance during adolescence (La Greca & Lopez, 1998). Pubertal development also has direct effects on behavior. For example, pubertal status predicts sensation seeking behavior even after controlling for age (Martin et al., 2002; Spear, 2000). The startle response, a measure of threat sensitivity, increases in magnitude in response to threat across puberty (Schmitz, Grillon, Avenevoli, Cui, & Merikangas, 2014), and late pubertal adolescents exhibit greater startle potentiation to facial expressions than early pubertal adolescents (Quevedo, Benning, Gunnar, & Dahl, 2009). Changes in hormone concentrations have also been linked to changes in reward seeking and social motivation. Testosterone concentrations in adolescents are negatively correlated with functional activation of the striatum, a region critical for reward processing (Forbes et al., 2010). Furthermore, increases in testosterone concentration during puberty predict increased activation of the amygdala to threat cues (Spielberg et al., 2014). Circulating hormone concentrations also affect structural brain development. Estradiol and testosterone concentrations predict white matter growth

across adolescence (Herting et al., 2014), demonstrating that pubertal hormones play a direct role in changes in brain development during adolescence.

2.2 Structural Brain Changes in Adolescence

Early studies examining post-mortem brain tissue have found that the frontal lobe regions such as the PFC continue to develop into adolescence. Although peak synaptic density is reached early in life in sensory regions of the brain, the synaptic density of the PFC does not reach its peak until early adolescence (Huttenlocher, De Courten, Garey, & Van der Loos, 1982; Peter, 1979). Similarly, it has been found that the myelination of sensory regions of the brain are completed by the second year of life whereas the myelination of the PFC continues into adulthood (Yakovlev & Lecours, 1967). Most of the dramatic changes in synaptic pruning occur around the beginning of puberty (Woo, Pucak, Kye, Matus, & Lewis, 1997), suggesting that pubertal hormones may influence synaptic pruning. These findings suggest that the PFC continue to mature and organize through synaptic pruning and myelination during adolescence. However, the role of puberty in this organization is complex and not well-understood (Steinberg, 2007).

With the advent of magnetic resonance imaging (MRI), researchers have increasingly explored structural changes in brain development during adolescence. Cross-sectional studies of brain development in infancy have found that the PFC exhibits slow increases in grey matter growth followed by rapid increases beginning in the second year of life (Gilmore et al., 2012; Knickmeyer et al., 2008). Throughout childhood and adolescence, there appears to be a broadly defined brain development

pattern from posterior to anterior, with the most protracted regions in grey matter development occurring in the PFC (Gogtay et al., 2004). A cross-sectional imaging study comparing grey matter in children, adolescents, and adults found grey matter volume acceleration in the PFC into adolescence followed by a slow grey matter reduction (Sowell, Thompson, Tessner, & Toga, 2001). There is also sex differences in timing of peak grey matter. Grey matter volume in the frontal lobe peaks at 9.5 years of age for girls and 10.5 years of age for females (Giedd et al., 1999). Similar changes in grey matter over the course of development have been observed in the ACC. To estimate structural changes in cortical regions such as the ACC across development, Tamnes et al. (2013b) conducted a cross sectional study of structural brain maturation in participants from 8-30 years of age, and found that both the ACC and the PFC showed a slower decrease in cortical density changes throughout development compared to other cortical regions. In addition, regions of the lateral PFC, which have extensive connections with the ACC, demonstrated a faster rate of volume reduction in adolescence.

The emergence of sex differences in grey matter maturation in adolescence are theorized to be due to pubertal hormones (Blakemore, Burnett, & Dahl, 2010). Peper et al. (2009a) examined the relations between puberty and grey matter volume and found that the onset of secondary sexual characteristics was related to decreases in frontal grey matter volume. In a subsequent study, pubertal hormones were measured and it was found that higher estradiol levels in girls predicted grey matter decreases in the PFC (Peper, et al., 2009b). Tanner stage, in addition to estradiol, have been shown to predict grey matter decreases in girls (Herting et al., 2014).

Taken together, there appears to be a rapid increase in grey matter volume in the PFC and ACC, reaching its peak in adolescence, followed by a gradual decline. Such findings suggest that pubertal development may moderate the association between PFC and ACC changes in adolescence (Blakemore & Choudhury, 2006).

Contrary to the patterns of grey matter observed through development, white matter exhibits a general increase in volume throughout childhood and adolescence (Blakemore & Choudhury, 2006). The PFC exhibits a linear increase in white matter volume growth from childhood through adulthood (Tamnes et al., 2010). Linear white matter growth in the PFC has been also observed in a longitudinal sample through childhood and adolescence (Giedd et al., 1999). Similar findings have been found using diffuse tensor imaging (DTI), an imaging methodology used to explore the density of white matter tracts. Studies of DTI across the lifespan have found maturation of PFC white matter tracts into the third decade of life (Lebel & Beaulieu, 2011; Westlye et al., 2010). In a cross-sectional study using DTI in children and adolescents, it was found that white matter tract development in the PFC was positively related to age (Barnea-Goraly et al., 2005). Structural increase in white matter have also been observed in the ACC. Lebel and colleagues (2011, 2008) examined white matter tract changes from 5 to 33 years of age and found that the cingulate bundle tract matured later than other white tract bundles. Taken together, there is increasing evidence of a similar delayed maturation pattern of white matter for both the PFC and ACC.

Sex differences in white matter development have also been observed. Girls exhibit more gradual white matter development as compared to boys, particularly in

adolescence (Bellis et al., 2001; Lenroot et al., 2007), suggesting pubertal hormones may play a role in the emergence of these differences. Herting et al. (2014) found that testosterone, estradiol, and physical pubertal development positively predicted total white matter volume. It has also been found that the degree of gene expression coding for androgen receptors was positively related to white matter growth in the frontal lobe (Perrin et al., 2008; Perrin et al., 2009), further confirming the positive association between testosterone and white matter. Taken together, studies of white matter development suggest a general linear increase of white matter through adulthood, which in part may be modulated by pubertal hormones in adolescence (Blakemore et al., 2010).

2.3 Development of the Mesencephalic Dopamine System

Developmental maturation of the mesencephalic dopamine system may account for some of the observed changes in adolescent social behavior. The mesencephalic dopamine system has extensive connection with the ACC and PFC (Spear, 2000), two regions which continue to mature into late adolescence (Giedd et al., 1999; Tamnes et al., 2010; Westlye et al., 2010). Mesencephalic dopamine neurons are thought to play a key role in motivation and reward processing (Fibiger & Phillips, 2011), working memory (Sawaguchi & Goldman-Rakic, 1991), contingency learning (Romo & Schultz, 1990), error monitoring (Frank, 2005; Holroyd & Coles, 2002), and the execution of motor actions (Kalivas, Churchill, & Klitenick, 1993). Mesencephalic dopamine neurons are located in regions of the midbrain, including the ventral tegmental area and substantia nigra, and project to the striatum, basal

ganglia, which then project to cortical regions such as the ACC and PFC (e.g., mesocortical pathway; Andersson, Jensen, Parmar, Guillemot, & Björklund, 2006). There is a high density of dopamine receptors in the ACC and PFC (Berger et al., 1991; Gaspar et al., 1989; Williams & Goldman-Rakic, 1993). Dopamine receptors are traditionally divided into two types: D1 and D2 receptors (Kebabian & Calne, 1979). D1 and D2 receptors are differentially activated and inhibited by phasic changes in dopamine (Frank, Loughry, & O'Reilly, 2001; Schultz, 2007). Specifically, a phasic increase in midbrain dopamine causes an excitatory response in D1 receptors but an inhibitory response in D2 receptors. In contrast, a phasic decrease in midbrain dopamine causes an excitatory response in D2 receptors and an inhibitory response of D1 receptors (Frank, Loughry, & O'Reilly, 2001; Schultz, 2007). Different responses of D1 and D2 receptors to phasic changes in dopamine are thought to play a role in learning to execute motor actions and learning to withhold motor actions (Frank, 2005).

A number of changes in the mesencephalic dopamine system occur in adolescence (Spear, 2000). Dopamine receptors in the midbrain increase in density in the first few weeks of life and peak during adolescence before decreasing (Andersen, Thompson, Rutstein, Hostetter, & Teicher, 2000; Gelbard, Teicher, Faedda, & Baldessarini, 1989; Teicher, Andersen, & Hostetter, 1995). There is also an increase in dopaminergic innervation of surviving neurons in the midbrain during adolescence (Rosenberg & Lewis, 1995). Dopamine synthesis patterns appear to be different depending on the cortical region. For example, dopamine synthesis in the PFC increases until adolescence then decreases, whereas dopamine synthesis in the ACC

decreases until adolescence then increases (Andersen, Dumont, & Teicher, 1997; Teicher et al., 1993). An increasing rate of pruning of dopamine receptors in the striatum as also been observed from adolescence to adulthood (Seeman et al., 1987). There is also a clear peak observed in both D1 and D2 receptors in the ACC during early adolescence followed by a loss in both receptors (Tarazi, Tomasini, & Baldessarini, 1999, 1998). However, there is evidence to suggest that only D1 receptors demonstrate developmental changes (Montague, Lawler, Mailman, & Gilmore, 1999). Findings of a direct relationship between pubertal hormones and dopamine levels in the midbrain are unclear (Andersen, Thompson, Krenznel, & Teicher, 2002), suggesting a more complex picture of the relations between pubertal hormones and dopamine during adolescence (Dahl, 2004; Steinberg et al., 2006). Taken together, there is growing evidence to suggest that the mesencephalic dopamine system develops into adolescence (Spear, 2000). Such changes may explain the reorganization of neural networks critical for social and affective processing (Ernst et al., 2009; Nelson et al., 2005).

2.4 The Neural Basis of Error Monitoring

A number of event-related potentials (ERP's) have been identified that reflect the engagement of the error monitoring system. The error-related negativity (ERN), also known as the error negativity (Ne), is a negative-going deflection in the ERP waveform that occurs approximately 50-80 ms after an erroneous response (see figure 1; Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993). In both children and adults, the ERN has been

hypothesized to be generated in the ACC (Dehaene, Posner, & Tucker, 1994; Holroyd, Dien, & Coles, 1998; Van Veen & Carter, 2002). The ERN is broadly theorized to reflect performance monitoring of errors (Gehring, Orr, & Carp, 2012). However many theories exist in explaining the functional significance of the ERN (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Holroyd & Coles, 2002; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Yeung, Botvinick, & Cohen, 2004).

A number of related ERP components have also been linked to error monitoring. The positive error (Pe) is a positive going deflection in the ERP waveform that occurs approximately 300 ms after an incorrect response (Falkenstein et al., 1991). The Pe is considered functionally distinct from the ERN and is suggested to relate to error awareness (Falkenstein et al., 2000; Overbeek et al., 2005). The Pe appears to be two separate components; an early Pe which follows the ERN that has a similar fronto-central topography as the ERN, and a late Pe, which has a slower onset and has a more parietal topography (Arbel & Donchin, 2009, 2011; Overbeek et al., 2005). The late Pe has become of more interest in that it resembles the stimulus-locked P3b (Ridderinkhof, Ramautar, & Wijnen, 2009).

Initially, there was little interest in the ERP waveform on correct trials (Falkenstein et al., 1991; Gehring et al., 1993), but researchers often noticed a small negativity on correct trials (Coles, Scheffers, & Holroyd, 2001; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). Due to the similar latency and topography as the ERN, this component has been coined the correct-response negativity, or the CRN (Ford, 1999). There has been debate of the exact functional significance of the CRN, but the

most common theory of the CRN is that it reflects a general response monitoring mechanism (Falkenstein et al., 2000).

EEG source analysis studies have identified that the ERN is likely generated in the dorsal ACC (dACC; Dehaene et al., 1994; Holroyd et al., 1998; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Van Veen & Carter, 2002). However, it has been noted that there is variability in the exact loci of the ERN (see Agam et al., 2011 for review). The ERN has been found to be localized to the dACC regardless of input modality (visual, auditory) or motor output modality (hand errors, foot errors, saccade errors), suggesting the ERN is input and output independent (Holroyd et al., 1998). In addition, the type of task used to elicit errors does not substantially influence the location of the generator of the ERN (Santesso & Segalowitz, 2008). Source analysis studies using magnetoencephalography (MEG) have also found the ERN to be generated in the dACC (Keil, Weisz, Paul-Jordanov, & Wienbruch, 2010; Miltner et al., 2003). In addition, the ERN has also been found to be generated in the dACC in children and adolescents. Ladouceur et al. (2006) localized the ERN to the dACC in both anxious and non-anxious children. A similar dipole in the dACC has been found in adolescents (Ladouceur et al., 2007). Taken together, there is strong evidence to suggest that the dACC is the main contributor of the generation of the ERN.

It is important to note that source localization of EEG is hampered by the inverse problem, such that it is impossible to localize an unknown electric source from the brain using electrodes on the scalp (Pascual-Marqui, 1999). Thus, it is impossible to know the true neural generator of the ERN using EEG. Studies using intracerebral recordings offer one solution to explore the actual electrical currents in

the brain. In patients who have had electrodes implanted on the surface of the cortex, it has been found that ERN-like and Pe-like deflections are present in the ACC (Brázdil et al., 2002). However, ERN-like deflections have also been observed in dorsolateral PFC and the orbitofrontal cortex (Brázdil, Roman, Daniel, & Rektor, 2005). Local field potential and single unit recordings in macaque monkeys have also found ERN-like activity in the ACC (Godlove et al., 2011; Ito, Stuphorn, Brown, & Schall, 2003), further supporting the theory that the ERN is generated by the ACC.

Source localization studies of the CRN have found that both the CRN and ERN are likely generated from overlapping regions of the ACC (Gentsch, Ullsperger, & Ullsperger, 2009; Roger, Bénar, Vidal, Hasbroucq, & Burle, 2010; Wessel, Danielmeier, Morton, & Ullsperger, 2012). However, there is still debate whether the CRN actually reflects neural activity on correct responses, or if the CRN is an artifact of error trials averaged into the ERN waveform (Coles et al., 2001). Source localization studies of the Pe have been relatively rare. Intercerebral recordings have found Pe-like signals from regions of the ACC (Brázdil et al., 2002, 2005). Herrmann and colleagues (2004) found that the generator of the Pe was slightly more rostral within the ACC than the source of the ERN. Rostral ACC contributions to the Pe have been replicated (van Boxtel, van der Molen, & Jennings, 2005). However, the early Pe and late Pe may be generated by different cortical areas (Overbeek et al., 2005). Van Veen & Carter (2002) observed that the early Pe had a similar generator as the ERN. However, the late Pe had a generator in the rostral ACC and another generator in the left superior parietal cortex. Given that the late Pe resembles the P3b, source localization studies of the P3b may shed light on the cortical involvement for

the Pe (Ullsperger, Fischer, Nigbur, & Endrass, 2014). Source analysis of the P3b have typically found a distributed cortical network, including, the parietal, cingulate, and temporal cortices (Volpe et al., 2007). It has been theorized that the main contributor of the P3b is generated in the temporal parietal junction (Bledowski et al., 2004; Knight, Scabini, Woods, & Clayworth, 1989). Intercerebral recording also suggest the involvement of the hippocampus and the superior temporal sulcus (Halgren, Marinkovic, & Chauvel, 1998).

Functional magnetic resonance imaging (fMRI) has also been utilized to explore the neural correlates of the error monitoring system. A number of studies have found that the dACC is reliably activated during error (Braver, Barch, Gray, Molfese, & Snyder, 2001; Carter et al., 1998; Critchley, Tang, Glaser, Butterworth, & Dolan, 2005; Fiehler, Ullsperger, & Von Cramon, 2004; Garavan, Ross, Kaufman, & Stein, 2003; Garavan, Ross, Murphy, Roche, & Stein, 2002; Ullsperger & von Cramon, 2001). Carter et al., (1998) first demonstrated that the dACC was more activated on error responses as compared to correct responses. However, the authors also observed enhanced dACC activity on high-conflict correct trials, suggesting that the dACC may not be specific to errors. Using a Go/NoGo task, Kiehl and colleagues (2000) found increased activation in the rostral ACC as well as the dorsolateral PFC. To further delineate differential contributions of the ACC, Menon and colleagues (2001) examined ACC activation during correct NoGo trials and error NoGo trials and found that the dACC was activated in both error and correct NoGo trials, whereas the rostral ACC was more activated only on NoGo error trials. In contrast, Ullsperger and von Cramon (2001) found that only the dACC was engaged during error

responses. Both error correction as well as error detection have been found to activate the dACC (Fiehler et al., 2004). Although the dACC is more active during errors than correct responses, there appears to be a wide range in the degree of activation, which may be due to individual differences, such as sustained attention, processing efficiency, and age (Hester, Fassbender, & Garavan, 2004).

A number of studies have utilized combined EEG/fMRI recordings to directly examine the relation between the ERN and blood-oxygen-level dependent (BOLD) activity in the ACC. Mathalon, et al. (2003) found that the ERN was significantly related to rostral ACC activity (Kiehl et al., 2000; Menon et al., 2001). However, the authors only compared average ERN and the average BOLD activation, making it unclear whether the magnitude of the ERN was directly related to ACC activity on a trial-by-trial basis. To address this limitation, Debener et al., (2005) collected combined EEG/fMRI during a flanker task and found that the magnitude of the ERN predicted concurrent activation in the dACC. Similarly, Huster et al. (2011) found that the magnitude of the ERN was correlated with the concurrent activation of the dACC but also found that the ERN was related to activation in the pre-supplementary motor area and the basal ganglia. It has been suggested that components of the ERN relate to the ACC differently; one component is related to rostral ACC and the other related to dACC (Edwards, Calhoun, & Kiehl, 2012). Furthermore, when allowing for multiple dipole solutions, a number of regions outside the dACC, both cortical and subcortical, may contribute to error processing and the generation of the ERN on the scalp (Doñamayor, Heilbronner, & Münte, 2012). Taken together, functional imaging studies have established that the dACC is reliably engaged during error processing.

However, other regions, specifically the rostral ACC, PFC and pre-supplementary motor area may also play a role in error processing.

It is important to note that there is wide variability in findings of the exact cortical location in the ERN. EEG source analysis studies have typically found the loci of the ERN at a more posterior location than error activity reported in imaging studies (Agam et al., 2011). Source analysis studies typically report a dipole for the ERN in the dACC or the posterior cingulate cortex (Mathewson, Dywan, & Segalowitz, 2005; Santesso & Segalowitz, 2008; Vlamings, Jonkman, Hoeksma, Van Engeland, & Kemner, 2008). In contrast, a number of fMRI studies have found error-related brain activity localized to rostral regions of the ACC (Kiehl et al., 2000; Menon et al., 2001). Furthermore, it cannot be assumed that error brain activity during an fMRI scan is directly related to the neural generator responsible the ERN. To address this issue, Agam et al., (2011) estimated the location of the ERN using EEG source analysis and compared the source estimation to concurrent fMRI activity during errors. Interestingly, the source of the ERN was found to be in the posterior cingulate cortex (Brodmann's area 23/31), whereas BOLD activation during errors was found in the dACC (Brodmann's area 24/32). These findings suggest that although the ERN is correlated with concurrent fMRI activity in the dACC during errors (Debener et al., 2005), the ERN may actually be generated in the posterior cingulate cortex (Agam et al., 2011). However, further studies are needed to explore this possibility.

2.4 Post-Error Adjustments during Error Monitoring

A number of adjustments in behavior have been observed following an error. Rabbitt (1966, 1968) observed that incorrect responses were often immediately corrected by a correct response, suggesting that participants were actively trying to fix their mistakes. Rabbitt (1966, 1968) additionally found that response times following errors were significantly slower than response times following correct responses. This phenomena, known as post-error slowing (PES) has been extensively studied in over the past 50 years and has been observed across a variety of performance monitoring tasks (see Danielmeier & Ullsperger, 2011 for review). Response slowing following an error is often observed to occur with increased accuracy following an error, termed post-improvements in accuracy (PIA; Laming, 1979). Lastly, it has been observed that the interference effect of incongruent stimuli, which typically is associated with response slowing, is reduced following an error (Ridderinkhof et al., 2002).

Theories for the observation for such post-error behaviors (PES, PIA, PERI) have been speculated. Original theories by Rabbit (1966, 1968) and Laming (1979) suggested that slowing after errors was a behavioral mechanism in which to improve subsequent accuracy. Such theories are coherent with the observed speed-accuracy trade-off, such that slower of response times are associated with better accuracy (D. E. Meyer, Kornblum, Abrams, & Wright, 1990; Wickelgren, 1977). More recently, it has been theorized that such improvements are related to the engagement of cognitive control strategies following errors (Botvinick, Braver, Barch, Carter, & Cohen, 2001). Similarly, response conflict as seen in the PERI effect, also signals increased

cognitive control (Ridderinkhof, 2002). Neuroimaging evidence for this theory come from studies that have found that the degree of activation of the ACC during errors and high-conflict situations is related to the degree of PES, PEA, and PERI (Garavan et al., 2002; Kerns et al., 2004; Klein et al., 2007; Wessel & Ullsperger, 2011; West & Travers, 2008). There is also some evidence that the magnitude of the ERN is related to post-error adjustments, such that a larger ERN is related to larger PES and PIA (Cavanagh, Cohen, & Allen, 2009; Debener et al., 2005; Gehring et al., 1993; West & Travers, 2008). However, a number of studies have not found any association between the ERN and post-error adjustments (Endrass, Reuter, & Kathmann, 2007; Gehring & Fencsik, 2001; Gehring & Knight, 2000; Hajcak, McDonald, & Simons, 2003b; Hajcak & Simons, 2002; Riesel, Endrass, Kaufmann, & Kathmann, 2011; Scheffers, Humphrey, Stanny, Kramer, & Coles, 1999; Scheffers et al., 1999). Such mixed findings suggest that the relation between the ERN and post-error adjustments may be indirect (Weinberg, Riesel, & Hajcak, 2012).

Another account of PES suggest that slowing after errors is a general orienting response rather than an adaptive behavioral adjustment (Castellar, Kühn, Fias, & Notebaert, 2010; Notebaert et al., 2009; Van der Borgh, Braem, & Notebaert, 2014). Errors are salient events because errors occur less frequency than correct responses. It has been found that PES is larger when less errors are committed, suggesting that the frequency of errors may modulate the degree of PES (Danielmeier & Ullsperger, 2011). To test the salience theory of PES, Notebaert and colleagues (2009) created a task where errors were frequent and correct responses rare. The authors found response slowing following the rare correct responses, but not following the frequent

error responses. In a follow-up study, the authors conducted the same experiment while recording EEG and found that the ERN was unrelated to PES and PIA. However, the authors found that the P3 component, which reflects orienting to motivationally salient stimuli, was correlated with PES (núñez Castellar, Kühn, Fias, & Notebaert, 2010), further suggesting that PES is an orienting response. In line with these findings, it has been theorized that PES is not a beneficial behavior adjustment to errors, but is rather due to a failure in disengaging from the orienting response to the error (Carp & Compton, 2009; Compton, Arnstein, Freedman, Dainer-Best, & Liss, 2011).

2.5 Functional Significance of the ERN, CRN and Pe

Since the discovery of the ERN approximately 20 years ago, a number of theories have arisen to account for the functional significance of the ERN. The discovery of the ERN by Gehring and colleagues (1993) and Falkenstein and colleagues (1991) gave rise to the first theory of the ERN, known as the error detection/error mismatch theory (Falkenstein et al., 2000). The theory argues that the ERN is specifically involved in error detection, where the ACC compares the output of the motor response to the intended response. The ERN represents the degree of mismatch between these responses (Coles et al., 2001). In support of this theory, it has been demonstrated that the ERN is greater in magnitude (i.e., more negative) when the actual response is more dissimilar to the intended correct response (Bernstein et al., 1995). This error signal is then used to improve future performance and make strategic adjustments in behavior (Gehring et al., 1993). The error

detection/error mismatch theory has fallen out of favor and has been updated using reinforcement learning principles (Holroyd & Coles, 2002).

One of the most influential theories of the function of the ERN comes from the conflict monitoring theory. Originally articulated by Botvinick et al. (2001), the theory is not specific to the ERN, but to the broader role of the ACC in monitoring conflict. The theory suggest that in situations of high conflict, there is increased ACC activity, signaling the need for more behavioral control. Errors are instances of particularly high conflict, where the error motor response interferes with the correct motor response. Thus, the ERN is not specific to errors, but is more broadly associated with high conflict. Similarly, the stimulus-locked N2 component, observed on high conflict trials, is also a signal of competing motor conflict. The differences between the components is that the N2 reflects conflict before a correct response is executed, whereas the ERN reflects conflict immediate after the error response was executed. Simulation studies of the ERN and N2 have confirmed the theory, where the N2 and ERN represent maximal conflict before and after motor responses respectively (Yeung, Botvinick, et al., 2004).

Behavioral evidence for the conflict monitoring theory comes from the observation that error motor response are often immediately self-corrected (Rabbitt, 1966, 1968), suggesting that the competing correct motor response is simultaneously activated with the error motor response (Botvinick et al., 2001). In addition, the observation of behavioral adjustments following errors (PES, PIA, PERI), suggest that processing during errors and high conflict situations leads to improvements in subsequent performance. Neuroimaging studies confirm the main predictions of

involvement of the ACC in both high conflict situations and during motor errors (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Braver et al., 2001; Carter et al., 1998, 2000; Ridderinkhof et al., 2004; Ullsperger & von Cramon, 2001). Furthermore, the degree of ACC activity during high conflict situations predicts subsequent behavioral adjustments (Danielmeier, Eichele, Forstmann, Tittgemeyer, & Ullsperger, 2011; Debener et al., 2005; Garavan et al., 2002; Kerns et al., 2004). The ERN has also been found to predict subsequent behavioral adjustments, such as PES and PIA (Debener et al., 2005; Gehring et al., 1993; Wessel & Ullsperger, 2011; West & Travers, 2008). Time-frequency analysis of the ERN and N2 suggest that theta rhythms underlie both components (Cavanagh, Zambrano-Vazquez, & Allen, 2012), both of which have been localized to the ACC (Bekker, Kenemans, & Verbaten, 2005; Jonkman, Sniedt, & Kemner, 2007; Van Veen & Carter, 2002).

One of the most prominent theories of the ERN, is the reinforcement learning theory of the ERN (RL-ERN; Holroyd & Coles, 2002). The RL-ERN is a computational model that posits that during motor errors, the mesencephalic dopamine system sends a negative reinforcement learning signal to the frontal and cingulate cortices, which disinhibits neurons in the ACC. The goal of this system is to exert control over motor behavior through reinforcement learning via the release of midbrain dopamine to ensure correct motor actions. Thus, the generation of the ERN in the ACC serves as a learning signal to regions such as the basal ganglia, an interconnected set of structures responsible for motor movement and reward (Walsh & Anderson, 2012). The RL-ERN theory is heavily influenced by studies of the FRN (originally called the feedback-ERN), which is a negative ERP deflection with a

similar topography as the ERN that is elicited when observed outcomes are worse than expected (Gehring & Willoughby, 2002; Miltner, Braun, & Coles, 1997). The FRN is thought to reflect a prediction error measuring the degree to which an outcome was better or worse than expected. Thus, when outcomes are better than expected (i.e., positive prediction error), there is an increase in midbrain dopamine, which in turn inhibits neurons in the ACC, resulting in a reduced (or no observable) FRN. When outcomes are worse than expected (i.e., negative prediction error), there is a reduction in midbrain dopamine, which in turn disinhibits neuron firing in the ACC, resulting in a larger (i.e., more negative) FRN. The FRN has a similar topography and source localization as the ERN (see Hauser et al., 2014 for review), and it has been hypothesized that the ERN and FRN are the same component, reflecting reinforcement learning signals (Holroyd & Coles, 2002) (Holroyd & Coles, 2002; Miltner et al., 1997). The ERN is a special instance of the FRN, where the mapping of correct responses is based on previous reinforcements of the current response (Holroyd, Yeung, Coles, & Cohen, 2005). Thus, when a mismatch occurs between the current response and the desired responses, there is disinhibition of ACC neurons resulting in a negative deflection observed on the scalp. Thus, the ERN reflects a prediction error, measuring the degree to which the occurrence of a negative outcome (i.e., error response) was different from what was predicted (Holroyd & Coles, 2002; Holroyd et al., 2005).

Empirical evidence for the RL-ERN theory is that during probabilistic learning tasks where subjects have not yet learned the mapping of correct and incorrect responses, the ERN is diminished on errors but the FRN to feedback about

performance is enhanced (Holroyd & Coles, 2002). However, when subjects learn the mapping of correct and incorrect responses, the ERN is larger in magnitude and the FRN is reduced (Holroyd & Coles, 2002). These findings suggest that the error monitoring system learns about performance first through externally monitored through feedback (i.e., FRN), and once learned, the performance is internally monitored (Holroyd et al., 2005; Walsh & Anderson, 2012). Evidence for the role of dopamine in the RL-ERN theory comes from pharmacological studies, which have found that the administration of a dopamine agonist causes an increase in the magnitude of the ERN (de Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004), and an administration of a dopamine antagonist leads to a decrease in the magnitude of the ERN (de Bruijn, Sabbe, Hulstijn, Ruigt, & Verkes, 2006). Furthermore, individuals with Parkinson's or Huntington's disease, which are both characterized by reduced dopamine in the basal ganglia, exhibit a reduced ERN (Beste, Saft, Andrich, Gold, & Falkenstein, 2006; Falkenstein et al., 2001; Stemmer, Segalowitz, Dywan, Panisset, & Melmed, 2007).

More recent models have advanced the RL-ERN theory to explain the ERN (and FRN) as an prediction error signal which causes subsequent changes in decision making for both motor avoidance to negative outcomes and motor initiation to positive outcomes (Cavanagh & Frank, 2014; Frank et al., 2005). This theory is based on work demonstrating that deficits in learning in Parkinson's disease, a disorder characterized by decreased basal levels of midbrain dopamine, are caused by reduced firing of dopamine neurons during positive reinforcement (see Frank, 2005 for review). The release of dopamine during rewards plays a key role in in the

modulation of the basal ganglia, an interconnected set of midbrain structures responsible for reward and movement. A phasic burst of dopamine in the basal ganglia causes an increase in learning to initiate correct motor movements (i.e., “Go” signal) while inhibiting incorrect motor movements (i.e., “NoGo” signal; Hikosaka, 1989). In contrast, dips in phasic dopamine causes increased NoGo learning to actively suppress non-reinforced future responses. Thus, increased phasic dopamine is responsible for learning from positive outcomes whereas decreased phasic dopamine is responsible for learning to avoid negative outcomes. Empirical support for the model is that individuals with Parkinson’s disease have difficulty learning from positive outcomes due to lack of phasic dopamine to produce the Go signal, but conversely are better at learning to avoid negative outcomes (Frank, Seeberger, & O’Reilly, 2004). Furthermore, medication that increases dopamine levels reverses this learning bias and prevents NoGo learning (Frank et al., 2004).

Frank and colleagues (2005) explored whether the ERN reflects NoGo learning during a probabilistic learning task and found that the magnitude of the ERN was correlated with the degree to which individuals learned more from negative than from positive outcomes from their choices. Furthermore, individuals who learned more from negative feedback had an enhanced FRN during feedback. These findings suggest that the ERN serves as a learning mechanism to change subsequent behavior to avoid negative events. Indeed, the magnitude of the FRN after negative feedback predicts future behavioral choices (Cohen & Ranganath, 2007; Holroyd & Krigolson, 2007). Individual differences in biases of negative learning predict enhancements of the ERN in unrelated tasks (Frank, D’Lauro, & Curran, 2007). Recently, it has been

suggested that such negative learning biases may account for the enhanced ERN observed in anxiety disorders (Endrass, Kloft, Kaufmann, & Kathmann, 2011).

Other theories have attempted to explain the function of the ERN by exploring the similarities in topography and oscillatory activity between the ERN and FRN (Cavanagh et al., 2009; Cavanagh & Shackman, 2014.; Luu & Tucker, 2001; Luu et al., 2003a). Luu and Tucker (2001) first noted that the ERN appeared to reflect oscillatory activity in the theta frequency (frontal-midline theta) which is only visible in the ERP waveform after removing overlapping slow-wave components (i.e., stimulus-locked P3) through high-pass filtering. EEG source analyses studies suggest that theta oscillations observed during errors are generated in the ACC (Luu et al., 2003a). This finding was extended to the FRN, where it was found that the FRN was also the result of oscillatory activity in the theta range, and localized to the ACC (Luu, Tucker, Derryberry, Reed, & Poulsen, 2003b). These findings suggest that both the ERN and FRN may reflect similar action monitoring processes since both the ERN and FRN are generated by theta oscillations in the ACC (Luu, Tucker, & Makeig, 2004). More recently, the N2 has been found to also reflect phase-locked theta activity (Cavanagh et al., 2012). However, it should be noted that some researchers have argued that the observed theta oscillations observed in the ERN and FRN may be due to filtering artifacts, and that it is impossible to determine with certainty whether these components are the result of phase-locked oscillations rather than phasic burst of activity (Yeung, Bogacz, Holroyd, & Cohen, 2004).

Frontal midline theta has been suggested to play a key role in learning and working memory (Gevins, Smith, McEvoy, & Yu, 1997; Schacter, 1977), and is

generally localized to the ACC (Gevins et al., 1997). Studies of the ACC in primates has found that the ACC reliably generates theta activity following contexts that require enhanced executive control (Tsujimoto, Shimazu, & Isomura, 2006). Thus, frontal midline theta, which gives rise to the ERN and FRN, and even perhaps the N2 (Cavanagh et al., 2012) may reflect the monitoring of actions and the outcomes of these actions, through the engagement of cognitive control and learning (Botvinick et al., 2001; Luu et al., 2003b, 2004). A unifying theory of frontal midline theta by Cavanagh & Frank (2014) state that the phase-locked theta frequency reflects a neural signal for increased cognitive control when there is uncertainty about performance and outcomes.

Motivational theories of the ERN suggest that the ERN reflects a motivational defensive response to committing an error (Luu, Collins, & Tucker, 2000; Proudfit et al., 2013; Weinberg, Meyer, Hale-Rude, et al., 2016; Weinberg, Riesel, et al., 2012). Committing an error is a distressing event, as mistakes can lead to disastrous outcomes, such as death or injury (Hewitt et al., 2003; Weinberg, Meyer, Hale -Rude, et al., 2016). Thus, errors are an endogenous threat, reflecting uncertainty about the consequence of erroneous actions (Weinberg, Meyer, Hale -Rude, et al., 2016). A number of physiological changes have been observed during error commission, such as potentiation of the startle reflex (Hajcak & Foti, 2008), heart rate deceleration (Hajcak et al., 2003b), elevated skin conductance (Hajcak et al., 2003b), pupil dilation (Critchley et al., 2005), and contraction of the corrugator (i.e., frowning) muscle (Lindström, Mattsson-Mårn, Golkar, & Olsson, 2013).

A recent revision of the motivation theory of the ERN emphasizes that the ERN is an evaluative signal of the degree to which errors are important in an individual's environment (Weinberg et al., 2016). Thus, in contexts which are more threatening, an enhanced ERN serves as an evaluative signal of the motivational salience of the threatening context. A number of experimental studies provide evidence that the ERN in part reflects defensive motivation toward errors in specific contexts (Hajcak et al., 2005; Kim et al., 2005; Themanson, Ball, Khatcherian, & Rosen, 2014; Van Meel & Van Heijningen, 2010). The ERN is enhanced when errors are punished (Riesel, Weinberg, et al., 2011), or when there is a monetary cost to committing an error (Hajcak et al., 2005). In motivationally salient social contexts, such as when the performance is critically evaluated, or during interpersonal competition, the magnitude of the ERN is enhanced (Barker et al., 2015; G. Hajcak et al., 2005; Van Meel & Van Heijningen, 2010). The ERN can also be reduced in magnitude, such as when subjects are socially excluded (Themanson et al., 2014). Thus, based on the motivational impact of the environment, the ERN signals the need for more behavioral control.

Much less is known about the functional significance of the Pe (Overbeek et al., 2005). Early studies of the Pe suggested that variations in the Pe reflect the degree of error awareness. For example, Nieuwenhuis and colleagues (2001) had participants complete an anti-saccade task and rate whether they made an error after each trial. Anti-saccade task often result in many automatic and unperceived saccade errors. The authors found that the Pe, but not the ERN, was reduced when errors were not perceived. The finding of a reduced/absent Pe during unperceived errors has been

replicated numerous times (Endrass, Franke, & Kathmann, 2005; Endrass et al., 2007; O'Connell et al., 2007). It has also been noted that the Pe resembles a stimulus-locked P3, and that the Pe may actually be a specific instance of the P3b component (Overbeek et al., 2005). The P3b, also known as the classic P300, is elicited when subjects are asked to respond to the presence of a target stimulus (Polich, 2007). The P3b has been thought to reflect context updating of motivational salient stimuli (Polich, 2007). Evidence that the Pe and the P3b are the same component comes from findings that parametric changes in the magnitude of the stimulus-locked P3 are correlated with the Pe (Ridderinkhof et al., 2009). Furthermore, both the Pe and P3 follow similar developmental patterns (Polich, 1997; Polich, Ladish, & Burns, 1990; Ridderinkhof & van der Molen, 1995; van Dinteren, Arns, Jongsma, & Kessels, 2014). Thus, the Pe might be a specific instance of the P3b (Overbeek et al., 2005).

Pharmacological studies have demonstrated that the Pe is not influenced by dopamine agonist and antagonist drugs (Bruijn et al., 2004; de Bruijn et al., 2006; Ridderinkhof et al., 2002). Furthermore, patients with Parkinson's disease, which is characterized by reduced dopamine in the basal ganglia, exhibit no difference in the Pe as compared to healthy controls (Falkenstein, Willemsen, Hohsbein, & Hielscher, 2005). These pharmacological findings suggest that unlike the ERN, the Pe is not related to the mesencephalic dopamine system (Overbeek et al., 2005). Rather, it has been suggested that the Pe, similar to the P3b, may reflect phasic activity in the locus coeruleus-norepinephrine system (Nieuwenhuis, Aston-Jones, & Cohen, 2005; Overbeek et al., 2005). Specifically, during motivationally salient events, there is a phasic increase in norepinephrine in the brain stem, which causes enervation of the

neocortical regions, such as the temporal-parietal junction (Nieuwenhuis et al., 2005; Polich, 2007; Soltani & Knight, 2000). Integrative theories of the Pe suggest a similar account, where the Pe is a reflection of a burst of norepinephrine, which activates the anterior insula, causing an orienting response during errors (Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010).

A number of theories have attempted to explain the existence of the CRN. Early theories suggested that the CRN reflected general performance monitoring and/or response comparison processes (Falkenstein et al., 2000; Vidal et al., 2000). The CRN has a similar topography as the ERN, and is also generated by the ACC (Gentsch et al., 2009; Roger et al., 2010; Wessel & Ullsperger, 2011). However, others have argued that the CRN is actually not a unique component. For example, it has argued that the CRN is the result of uncertainty of correct responses (i.e., some ERN trials are averaged into the CRN on trials where subjects pressed the correct response but actually believe they made an error the ERN; Coles et al., 2001; Scheffers & Coles, 2000). Another theory is that the CRN is the reflection of simultaneous motor activation of both correct and error responses on correct trials (Coles et al., 2001; Scheffers & Coles, 2000). However, it has been found that there is still a CRN when controlling for uncertainty of simultaneous motor responses (Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003; Vidal et al., 2000), suggesting that the CRN is a unique component. Taken together, the CRN likely reflects general performance monitoring.

2.6 The Development of the Error Monitoring System

Early studies of the development of the ERN and the error monitoring system have found that the ERN was not present before adolescence (Davies et al., 2004; Ladouceur et al., 2006, 2004). In the first large cross-sectional study on the development of error monitoring, Davies and colleagues (2004) measured the magnitude of the ERN in children and adolescents from 7 to 18 years of age. The authors observed that the ERN exhibited small changes between 7-12 years of age followed by a quadratic growth up to 18 years of age. In addition, the authors found that females demonstrated an earlier maturation of the ERN than boys, with the sex differences emerging in early adolescence, suggesting that puberty may impact the maturation of the ERN.

Additional studies have confirmed that the error monitoring system undergoes rapid maturation in adolescence (Ladouceur et al., 2004; Santesso & Segalowitz, 2008; Santesso, Segalowitz, & Schmidt, 2006). Santesso and colleagues (2006) compared the ERN between 10-year-old children and adults. Consistent with Davies and colleagues (2004), the authors found that children demonstrated a significantly smaller ERN than the adults. To more closely explore developmental changes in adolescence, Ladouceur and colleagues (2004) explored the development of the error monitoring system in early (aged 8-14 years) and late (aged 14-17 years) adolescents using an arrow version of the flanker task. The authors found that the older adolescents demonstrated a significantly larger ERN than younger adolescents. In a follow-up study comparing the ERN in adolescents and adults (Ladouceur et al., 2007), it was found that younger adolescents (mean age = 12.36 years) exhibited a

significantly smaller ERN than both older adolescents (mean age = 16.53) and adults. Santesso and Segalowitz (2008) more closely examined the development of error monitoring in later adolescence by measuring the ERN during a flanker task and a Go/NoGo task in younger (aged 15-16 years) and older (aged 18-20 years) adolescents. The authors found that the magnitude of the ERN for both tasks was significantly larger in the older adolescents than the younger adolescents. In addition, the authors found no differences in accuracy or response times between groups, suggesting that age-related differences in the ERN are not due to general differences in performance.

More recently, researchers have begun to examine the neural correlates of error monitoring during earlier periods in development. Richardson and colleagues (2011) examined the ERN in 7- and 9-year-old children using a flanker task. The authors found that both age groups had a more negative ERN as compared to the CRN, demonstrating that children as young as 7 years of age reliably demonstrate physiological correlates of error monitoring. However, the ERN was unrelated to age, suggesting that the ERN is relatively stable in middle childhood. To test the development of error monitoring in middle childhood, van Meel and colleagues (2012) compared the ERN in younger children (aged 6-9 years), older children (aged 10-12 years), and young adults using a flanker task. The authors found no differences in the magnitude of the ERN between older and younger children, but both groups had a significantly smaller ERN than adults. Kim and colleagues (2005) developed a simple Go/NoGo task that was successful in eliciting an ERN on incorrect No-Go responses in younger children. In a follow-up study, the authors found that younger

children (7-8 years of age) demonstrated a significantly smaller ERN than the older children (9-11 years of age). Torpey and colleagues (2009) measured the error monitoring system in 5- 7 year-old children using the Go/NoGo task developed by Kim et al., (2007), and found that the ERN was significantly more negative than the CRN, suggesting that the error physiological indices of the error monitoring system are detectable by 5 years of age. In a follow-up study of a large community sample of 5-7 year-olds, the authors replicated that the ERN was present by middle childhood, and found that age was only weakly associated with the magnitude of the ERN in middle childhood (Torpey, Hajcak, Kim, Kujawa, & Klein, 2011). Taken together, these studies suggest that the ERN is relatively stable in middle childhood.

The findings reviewed above suggest that the physiological indices of the error monitoring system are detectable by 5 years of age. To explore the developmental patterns of error monitoring at earlier ages, Grammer et al. (2014) recruited a large community sample of children from 3-7 years of age and had children complete a Go/NoGo task. The authors found that the ERN was significantly larger than the CRN, suggesting that the neural correlates of error monitoring may be observed as young as 3 years of age. However, correlational analyses revealed that there was no relation between age and the magnitude of the ERN in the sample, further suggesting that the ERN is stable in childhood. In a recent longitudinal study, DuPuis et al., (2015) examined the development of the ERN in early childhood (5-9 years of age) in a large community sample and found that the ERN was associated with age. In addition, the authors utilized time-frequency analysis to decompose signal strength and temporal consistency of the ERN and

found that only temporal consistency of the signal (i.e., the consistency of timing of the ERN on each trial) predicted changes of the ERN across childhood. These findings suggest that changes in the magnitude of the ERN are not due to increased neural activity, but to greater temporal consistency.

The development of the Pe is less understood. However, it appears that the Pe demonstrates a different developmental pattern than the ERN (Santesso et al., 2006). Davies et al., (2004) found that the Pe was present by 7 years of age but did not show any developmental changes from 7-18 years of age. Furthermore, the Pe was present even in the absence of the ERN, suggesting that the components are relatively independent and undergo different developmental patterns. Similarly, Wiersema et al. (2007) found that unlike the ERN, the Pe did not demonstrate any developmental changes from middle childhood through adolescence. Other studies examining error monitoring at different periods of adolescence have found no differences in the magnitude of the Pe (Ladouceur et al., 2004; Santesso & Segalowitz, 2008). Thus, it appears that the Pe undergoes relatively little developmental changes in adolescence. For example, 10-year old children demonstrate a similar Pe as compared to adults (Santesso et al., 2006), suggesting that the Pe reaches an adult-like magnitude prior to adolescence.

Less is known about the development of the Pe at earlier ages. Grammer et al. (2014) examined the development of the Pe in 3-7 year-olds. Interestingly, the Pe, which is relatively invariant in adolescence, demonstrated growth during this period of development. In addition, changes in the Pe occurred concomitantly with changes in behavioral performance. These findings suggest that the Pe appears to mature in

early childhood. The stimulus-locked P3, which may be a similar component as the Pe (Overbeek et al., 2005), exhibits a similar developmental pattern of the Pe, with only small changes observed in adolescence (Polich, 1997; Polich et al., 1990). However, some developmental changes of the P3 in adolescence have been observed (van Dinteren et al., 2014).

Taken together, developmental studies of the ERN and the Pe have generally found that the two components demonstrate vastly different patterns of development. Both components are present early in childhood (Grammer et al., 2014.; Torpey et al., 2011). The Pe appears to follow an early maturational pattern with little developmental changes observed past childhood (Davies et al., 2004; Grammer et al., 2014). In contrast, the ERN is relatively stable in childhood (Richardson, Anderson, Reid, & Fox, 2011; van Meel et al., 2012), followed by rapid maturation in adolescence (Davies et al., 2004; Ladouceur et al., 2007). However, given some mixed findings in late childhood/early adolescence (Kim, Iwaki, Imashioya, Uno, & Fujita, 2007) it is likely other factors besides age, such as pubertal status, may contribute to changes in the ERN in adolescence. In contrast, the Pe is unrelated to chronological age in adolescence (Davies et al., 2004; Ladouceur et al., 2007). However, no research has examined whether puberty explains developmental changes in the ERN or Pe.

A number of studies have examined developmental changes in post-error adjustments (see Smulders, Soetens, & van der Molen, 2016 for review). PES appears to be present by early childhood (Jones, Rothbart, & Posner, 2003). However, the developmental direction of changes in PES have been debated, with some studies

finding increased PES across development, and other studies findings decreased PES across development (Smulders et al., 2016). Fairweather (1978) first observed that PES was present by early childhood, but also found that the amount of PES decreased throughout childhood and adolescence. A number of studies, particularly those which employ simple response tasks, have also found decreases in PES across development (Brewer & Smith, 1989; Schachar et al., 2004). Observed reductions in PES throughout development are theorized to be due to increases in processing efficiency (Fairweather, 1978). However, a number of studies, particularly those which employ a conflict task, have found increases in PES through development (Hogan, Vargha-Khadem, Kirkham, & Baldeweg, 2005; Santesso et al., 2006), or no developmental changes (Davies et al., 2004; Ladouceur et al., 2007; Wiersema et al., 2007). Task difficulty may be one reason for mixed findings. Using two different response tasks varying in difficulty, Hogan and colleagues (2005) found that older adolescents exhibited more post-error slowing than younger adolescents only on a difficult task. Taken together, these findings suggest that PES is present by early childhood, but the developmental pattern of PES may be dependent on task difficulty.

A number of related behavioral constructs have been used to examine the development of performance monitoring. Inhibitory control is the ability to inhibit a dominant motor response (Hasher, Zacks, & May, 1999) and is typically measured as the ability accurately withhold motor responses on stop trials or NoGo trials on a speeded-response task. A consistent finding in the developmental literature is that there are large improvements in inhibitory control in childhood and early adolescence (Bedard et al., 2002; Williams, Ponsse, Schachar, Logan, & Tannock, 1999).

Davidson and colleagues (2006) found that even 4-5 year-olds were able to successfully inhibit dominant motor responses. Luna and colleagues (2004) examined cognitive control using an oculomotor tasks and observed steep improvements in inhibitory control from 8 years of age into adolescence followed by more graduate improvements thereafter. Hooper and colleagues (2004) similarly found that older adolescents (14-17 year-olds) demonstrated better response inhibition (less number of false alarms on a Go/NoGo task) than 11-13 year olds and 9-10 year olds. A related way to measure inhibitory control is through response interference, which is observed when peripheral stimuli contradicts information presented by the target stimuli. This is typically observed on the flanker task, when the flanking arrows are incongruent to the target arrow (Eriksen & Eriksen, 1974). Similar to developmental changes in inhibitory control, there is a large decrease in response interference (as measured by the difference between congruent and incongruent response times) in childhood, which reaches adult levels by adolescence (Ridderinkhof & van der Molen, 1995; Rueda, Posner, Rothbart, & Davis-Stober, 2004). Taken together, the developmental of inhibitory control is similar to observed development of post-error behavior, suggesting that the two systems may rely on similar neural structures (Danielmeier & Ullsperger, 2011)

It is likely that many of the observed developmental changes of the ERN in adolescence are due to neurochemical changes associated with pubertal development (Davies et al., 2004). Prior to puberty, dopamine and serotonin levels fluctuate before demonstrating a dramatic increase at puberty (Goldman-Rakic & Brown, 1982). Furthermore, there is a large developmental increases in the dopaminergic innervation

of neurons in the PFC, which reaches its maximal level during puberty (Lambe, Krimer, & Goldman-Rakic, 2000). Changes in the dopaminergic system during puberty is consistent with the biochemical model of the ERN (Holroyd & Coles, 2002), which argues that the ERN is generated by dips in midbrain dopamine. Thus, the development of the ERN in adolescence may be partly dependent on neurochemical changes that take place during puberty.

There is indirect evidence to suggest that puberty may influence developmental changes in the ERN in adolescence. In childhood, no differences in the ERN between sexes have been observed (Grammer et al., 2015; Torpey et al., 2011, 2009). However, adult females have a significantly larger ERN than adult males (M. J. Larson et al., 2011). Sex differences in the ERN appears to emerge in adolescence (Davies et al., 2001). Furthermore, the timing of developmental changes in the ERN occurs earlier in females than males (Davies et al., 2001), consistent with differences in pubertal timing between sexes. The relation between the ERN and dimensional aspects of anxiety is influenced by sex, such that the relation is strongest among females (Moran et al., 2012; Moser et al., 2016). For the Pe, there is little evidence that puberty may influence the Pe. However, one study has examined the influence of puberty on the P3, and found that pubertal status was related to the amplitude of the P3 for girls, but not for boys (Brumback, Arbel, Donchin, & Goldman, 2012). Taken together, there is strong evidence to suggest that puberty influences developmental changes in the ERN, and perhaps for the Pe.

2.7 The Development of the Error Monitoring System using fMRI

A number of studies have utilized fMRI to investigate the development of the error monitoring system. Rubia et al. (2007) investigated the functional activation of the ACC and PFC during error trials in children and adults and found that adults demonstrated increased BOLD activation of the ACC. In addition, the authors conducted a regression analysis controlling for behavioral performance and found that age continued to predict activation in the ACC following errors. Fitzgerald and colleagues (2010) investigated developmental changes in inhibitory control and error monitoring in children and adults and that found both children and adults engaged regions of the PFC and ACC during errors and during high conflict correct trials. Furthermore, interference- and error-related activation increased with age independent of performance. Velanova and colleagues (2007) investigated error activity during an anti-saccade task in children and young adults and found decreased activation in the rostral ACC and increased activation in the dorsal ACC during errors as compared to correct trials. The findings by Velanova and colleagues (2007) suggest that the functional development of the ACC may not demonstrate the same developmental pattern in dorsal and rostral regions. Neural regions involved in inhibitory control have also been examined using fMRI and have shown similar developmental changes in activation of the ACC and PFC across development (Braet et al., 2009; Fitzgerald et al., 2010; Rubia, Smith, Taylor, & Brammer, 2007; Velanova, Wheeler, & Luna, 2008). Taken together, functional imaging studies of error monitoring have found developmental increases in activation of the ACC and PFC through childhood and adolescence.

2.8 The Relation between the ERN and Anxiety

A growing body of literature has focused on understanding the relation between the ERN and psychopathology. Gehring and colleagues (2000) found that individuals with obsessive compulsive disorder (OCD) exhibited an enhanced ERN as compared to healthy controls. However, behavioral performance (i.e., response times, accuracy) were equivalent across groups, suggesting that an enhanced ERN in OCD patients is not directly related to performance differences. An enhanced ERN in individuals with OCD has been replicated numerous times (Endrass et al., 2010, 2014). An enhanced ERN has also been observed in anxiety disorders such as generalized anxiety disorder (GAD; Weinberg, Klein, & Hajcak, 2012; Weinberg et al., 2010), and social anxiety disorder (SAD; Endrass et al., 2014). These findings suggest that an enhanced ERN is a transdiagnostic marker of anxiety disorders (Weinberg, Riesel, et al., 2012).

An elevated ERN has been observed among trait anxious individuals (Hajcak, McDonald, & Simons, 2003a). Furthermore, the magnitude of the ERN is correlated with individual differences in anxiety among healthy individuals (Moser et al., 2012), suggesting that the relation between anxiety and the ERN is not strictly related to clinical populations. Imaging studies have also found increased BOLD activation in the ACC among anxious individuals. Ursu and colleagues (2003) found increased functional activation of the ACC in OCD patients and that ACC activation correlated with OCD symptom severity. Enhanced activation of the ACC in individuals with OCD has been replicated numerous times (Fitzgerald et al., 2010; Maltby, Tolin,

Worhunsky, O'Keefe, & Kiehl, 2005). However, less research has tested whether individuals with other types of anxiety disorders (e.g., GAD, SAD) demonstrate enhanced ACC activation during the processing of errors.

Numerous studies have also reported abnormal error monitoring in children with anxiety disorders. Ladouceur and colleagues (2006) first observed an enhanced ERN in 8-14 year old children diagnosed with an anxiety disorder. Similar to adult studies, the authors found no differences in PES or other behavioral measures of error monitoring between groups. To explore differences in error monitoring within different types of anxiety disorders in children, Carrasco and colleagues (2013) compared children with OCD, and non-OCD anxiety disorders (generalized anxiety disorder and separation anxiety disorder) to healthy controls on a flanker task. The authors similarly found no differences in behavioral indices of the error monitoring system, but found that both the OCD group and the non-OCD anxiety disorders group demonstrated a greater ERN as compared to healthy controls. An enhanced ERN among anxious children may be observed earlier in life. Meyer et al. (2013) found an enhanced ERN in 6-year-old children with a clinical diagnosis of anxiety. In a follow-up study, the authors found that a greater ERN at age 6 predicted the development of an anxiety disorder 2 years later (Meyer, Hajcak, Torpey-Newman, Kujawa, & Klein, 2015). However, in both studies, among the whole sample, the ERN was unrelated to individual differences in anxiety. Taken together, these findings suggest that, like adults, children with an anxiety disorder demonstrate a greater ERN. However, among a normative sample, the relation between individual differences in anxiety and the ERN may emerge later in life. In a study examining this issue, it was found that

age moderated the relation between the ERN and anxiety such that there was relation between ERN and anxiety was only among older adolescents (A. Meyer et al., 2012).

It has been suggested that the enhanced ERN observed among many anxiety disorders is driven by symptoms of general distress/anxious apprehension (Moser et al., 2012, 2013; Simons, 2010; Weinberg et al., 2010), which is a core symptom of most anxiety disorders (L. A. Clark & Watson, 1991; Watson, 2005). The association between the ERN and anxious apprehension/general distress is stronger than the association between the ERN and mixed anxiety symptoms (Moser et al., 2013). Thus, anxiety disorders may be characterized by an enhanced ERN inasmuch as the disorders are characterized by general distress/anxious apprehension (Moser et al., 2013). Specifying the association between the ERN and different anxiety symptoms was explored by Zambrano-Vazquez and Allen (2014), who compared the ERN in subjects characterized as high in general anxiety and moderately low in obsessive compulsive symptoms to individuals characterized as moderately low in general anxiety and high in obsessive compulsive symptoms. The authors found that only the high general anxiety group demonstrated an enhanced ERN, suggesting an enhanced ERN in individuals with OCD may be due to general anxiety symptoms.

Alterations of the ERN in depressive disorders have also been observed. Some studies have found an enhanced ERN in depressive disorders (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008, 2010). However, a number of studies have found no differences, or a reduced ERN in depression (Ladouceur et al., 2012; Olvet, Klein, & Hajcak, 2010; Ruchow et al., 2004; Schrijvers et al., 2009; Weinberg, Kotov, & Proudfit, 2015). Such mixed findings may be due to the high comorbidity observed

between anxiety and depressive disorders (Kessler, 2005), such that anxiety symptoms may be the driving factor of the enhanced ERN among some individuals with depression (Olvet & Hajcak, 2008; Weinberg, Riesel, et al., 2012). Indeed, individual differences in depressive symptoms are unrelated to the ERN when not controlling for anxiety (Olvet et al., 2010). Furthermore, it has been found that individuals with comorbid anxiety and depressive disorders do not exhibit an enhanced ERN, whereas anxious individuals without comorbid depression exhibit an enhanced ERN (Weinberg, Klein, et al., 2012), suggesting that depressive symptoms mask the effect of the ERN on anxiety. It was recently found that anxiety and depressive symptoms have opposing effect on the ERN, such that the anxiety symptoms are associated with a larger ERN, whereas depressive symptoms are associated with a reduced ERN (Weinberg et al., 2015). In line with accumulating evidence in adults, it has been found that children and adolescents with major depressive disorder exhibit a reduced ERN (Ladouceur et al., 2012). Furthermore, it was recently observed that, controlling for anxiety symptoms, individual differences in depressive symptoms are related to a reduced ERN (Weinberg, Meyer, Hale - Rude, et al., 2016). Taken together, there is growing evidence that depressive symptoms are associated with a reduced ERN in both adults and children.

2.9 The ERN as a Biomarker for Social Anxiety in Adolescence

Social anxiety is defined by fear and anxiety of social performance and/or social interaction, particularly in situations in which social evaluation and scrutiny may take place (Rapee & Heimberg, 1997; Rapee & Spence, 2004). Unlike other

forms of anxiety that are associated with general anxious apprehension across both social and nonsocial contexts (e.g., general distress; Clark & Watson, 1991), social anxiety is specific to anxious apprehension and arousal to perceived socially threatening contexts (Geen, 1991; Rapee & Heimberg, 1997; Schlenker & Leary, 1982). During anxiety-evoking social contexts (e.g., speech performance), individuals characterized as highly socially anxious exhibit greater anxious behavior and self-report of feelings of anxiety, enhanced heart rates, and increased salivary cortisol than low socially anxious individuals (Beidel, Turner, & Dancu, 1985; Furlan, DeMartinis, Schweizer, Rickels, & Lucki, 2001; Levin et al., 1993; Mauss, Wilhelm, & Gross, 2004). Socially anxious individuals also demonstrate altered neural patterns during socially threatening contexts, such as increased right-hemispheric lateralization (Davidson, Marshall, Tomarken, & Henriques, 2000), and altered functional activation of regions critical for processing of conflict (e.g., ACC; Amir et al., 2005; Lorberbaum et al., 2004). Taken together, these findings suggest that social anxiety is defined by enhanced anxious apprehension and arousal to social evaluative contexts, resulting in heightened neural, physiological and behavioral responses to perceived social threats.

Social anxiety disorder (SAD) typically emerges in early adolescence (Kessler, 2005; Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996; Ruscio et al., 2008), suggesting functional and structural changes in brain development during puberty may explain the rise in social anxiety in adolescence (Dahl, 2004; Nelson et al., 2005; Rudolph, 2014). Many studies find that advancing pubertal status predicts increasing internalizing symptoms, particularly among girls (Angold et al., 1999; Ge

et al., 2001; Reardon et al., 2009). Changes in risk for psychopathology during pubertal development may be driven by changes in hormone secretions. For example, increases in gonadal hormone levels are related to increased risk for depression in girls (Adrian Angold et al., 1999). Thus, puberty may signal a change in risk for internalizing disorders such as social anxiety (Forbes & Dahl, 2010). Adolescent girls demonstrate higher rates of social anxiety symptoms than boys (La Greca & Lopez, 1998), and pubertal developmental is related to more social anxiety symptoms only among adolescent girls (Deardorff et al., 2007). Thus, neurodevelopment during puberty may account for a shifting risk for SAD in girls. However, there is little support for a neural mechanism to account for the rise in social anxiety symptoms among girls in adolescence.

It has been suggested that changes in the motivational salience of social contexts in adolescence occurs in tandem with neurochemical and neuroanatomical changes associated with puberty, which results in the reorganization of networks critical to social and affective processing (Dahl & Crone, 2012). There is indirect evidence that puberty may play a role in enhanced ACC processing of social information in adolescence. Gunther and colleagues (2010) measured neural activation in response to social acceptance in middle childhood (8-10), early adolescence (12-14) and late adolescence (16-17 years). The authors found that all age groups demonstrated increased activation of the ACC when they received positive feedback as compared to negative feedback. Using the Cyberball game, Masten et al. (2009) found that, as compared to adults, early adolescents (ages 10-12 years) exhibited enhanced ACC activation during peer rejection. In a subsequent

study, increased activation of the ACC during social exclusion task was predictive of depressive symptoms 1 year later (Masten et al., 2011). Taken together, these studies suggest that the ACC is sensitive to social influences, and there is some evidence to suggest that adolescents may exhibit greater enhancements during social contexts. However, little research has explored how the ERN is influenced by social motivational factors in adolescence.

Although the relation between the ERN and anxiety is well established, little research has examined if socially anxious individuals are similarly characterized by an enhanced ERN. Endrass and colleagues (2014) found that both adults with SAD and adults with OCD exhibited an enhanced ERN as compared to healthy controls. However, the ERN was unrelated to symptoms of social anxiety, suggesting that other anxiety symptoms, such as anxious apprehension, may explain the elevated ERN observed in the SAD group. To explore the relation between individual differences in social anxiety and the ERN, Barker and colleagues (2015) examined the ERN in young adults characterized as low or high in social anxiety symptoms. Participants completed a flanker task across two different social motivational contexts. In one context, participants completed a flanker task and committed errors while alone in a room (i.e., alone condition). In the other condition, participants played the same flanker task and committed errors while being observed and evaluated by a peer (i.e., peer condition). The authors found that the ERN was enhanced in the peer condition as compared to the alone condition *only* among the high socially anxious individuals (see figure 2). Furthermore, the degree to which the ERN was enhanced in the peer condition from the alone condition, which was referred to as the *Social Effect ERN*,

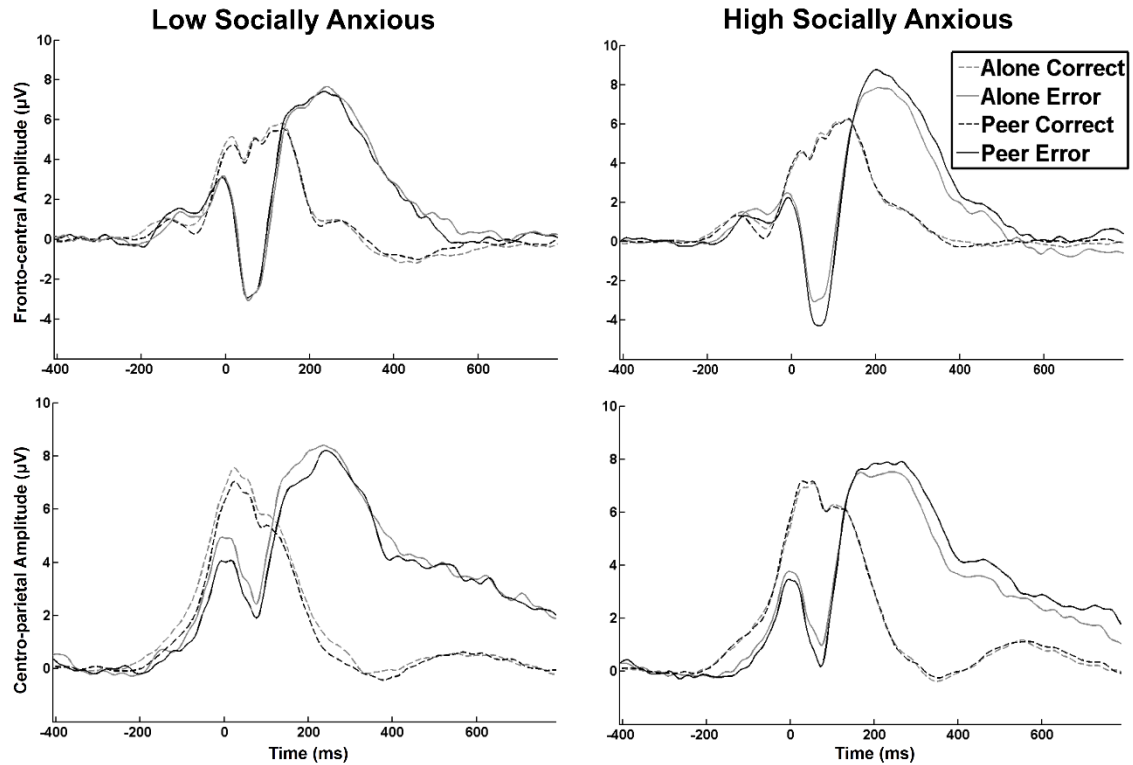


Figure 2. Response-locked event-related potential (ERP) waveforms on correct and error responses for high socially anxious and low socially anxious young adults ($N = 44$) who completed a flanker task and committed errors during peer evaluation (i.e., peer condition) and when errors were committed alone (i.e., alone condition). The top row is the fronto-central electrode grouping, where the error-related negativity (ERN) was maximal. The bottom row is the centro-parietal electrode grouping, where the positive error (Pe) was maximal. The ERN was enhanced in peer condition only among the high socially anxious individuals (Barker, Troller-Renfree, Pine, & Fox, 2015).

was negatively related to social anxiety symptoms such that a larger (i.e., more negative) ERN in the peer condition relative to the alone condition was related to more social anxiety symptoms. These findings suggest that social anxiety is characterized by enhanced neural activity to errors in social contexts. However, it is unknown whether the social effect ERN is related to social anxiety symptoms during adolescence, a period in which most anxiety disorders are initially diagnosed (Canino et al., 2004).

Chapter 3. The Current Study

3.1 Statement of the Problem

Adolescence is a transition period characterized by rapid growth and high risk for affective and anxiety disorders (Dahl, 2004; Pine et al., 1998). Pubertal development during adolescence plays a major role in the emergence of these disorders, particularly among girls (Angold et al., 1999; Reardon et al., 2009). Adolescent girls enter puberty earlier than boys (Sun et al., 2002), and are twice as likely to develop a depressive or anxiety disorder (Kessler et al., 2005; Lewinsohn et al., 1998), suggesting that this heightened risk in girls is in part driven by the effects of puberty on the function and structure of neural circuits supporting affective processing (Crone & Dahl, 2012; Nelson et al., 2005). Furthermore, pubertal status is associated with the emergence of social anxiety symptoms among adolescent girls (Deardorff et al., 2007). As such, it is important to utilize neural markers of affective processing to explore the role of puberty in the emergence of anxiety disorders in adolescent girls.

One potential biomarker for affective and anxiety disorders, the error-related negativity (ERN), is a negative deflection in the event-related potential following an erroneous response (Gehring et al., 1993). A related component, known as the positive error (Pe), is a positive deflection occurring after the ERN (Falkenstein et al., 1991). The ERN is theorized to be generated by dip in dopaminergic innervation of mesencephalidbrain dopamine (Frank et al., 2005; Holroyd & Coles, 2002), whereas the Pe is thought to reflect phasic increases in norepinephrine and acetylcholine (Ullsperger, Fischer, Nigbur, & Endrass, 2014). There is evidence to suggest such

neurotransmitters are influenced by pubertal hormones in adolescence (Blakemore et al., 2010; Forbes & Dahl, 2010; Spear, 2000). Thus, puberty may explain developmental differences in variations in the magnitude of the ERN and Pe. The ERN undergoes dramatic maturation in adolescence, whereas the Pe appears to be relatively stable (Davies et al., 2004; Ladouceur et al., 2004, 2007; Santesso & Segalowitz, 2008). However, no research has examined whether pubertal development can explain changes in the magnitude of the ERN and Pe in adolescence. This question was addressed in aim 1 of the proposal.

Theories on the ERN-anxiety association propose that the ERN represents an affective response to committing an error, which is in part influenced by motivational factors (Proudfit et al., 2013). The ERN is elevated when errors are committed during social evaluation and competition as compared to less motivationally salient contexts (Barker et al., 2015; G. Hajcak et al., 2005; Schillinger, Smedt, & Grabner, 2015; Van Meel & Van Heijningen, 2010). There is also evidence to suggest that the Pe is also enhanced by motivationally salient contexts (Hajcak et al., 2005). However, it is unknown whether social-motivational factors influence the ERN in adolescence. Furthermore, pubertal hormones are thought to influence changes in social motivation in adolescence (Blakemore et al., 2010; Ernst et al., 2009; Forbes & Dahl, 2010). It is also unknown if pubertal development modulates the degree to which the ERN and Pe are influenced by socially motivating contexts. These questions were addressed in aim 2 of the proposal.

Individuals with an anxiety disorder exhibit a greater ERN than healthy controls (Gehring et al., 2000; Ladouceur et al., 2006; Weinberg et al., 2010), and

variations in the ERN reflect dimensional aspects of anxiety (Hajcak et al., 2003a; Moser et al., 2012). In contrast, there is growing evidence that depressive symptoms are associated with a reduced ERN (Ladouceur et al., 2012; Weinberg et al., 2016, 2015). However, little research has examined how the ERN relates to symptoms of social anxiety. Social anxiety disorder (SAD), one of the most commonly diagnosed mental disorders in adolescence (Kessler et al., 2005; Ruscio et al., 2008), is characterized by excessive fear and anxiety of social-evaluative situations (Rapee & Spence, 2004). SAD is highly comorbid with major depressive disorder (Kessler, Stang, Wittchen, Stein, & Walters, 1999; Wittchen, Stein, & Kessler, 1999), and social anxiety in adolescence is associated with increased risk of the development of depressive disorders (Essau, Conradt, & Petermann, 1999; Stein et al., 2001). Recent evidence suggest that socially anxious adults exhibit an enhanced ERN in social-evaluative contexts (see figure 2; Barker et al., 2015), suggesting that the ERN in social contexts may be a biomarker of social anxiety. The relation between dimensional aspects of anxiety and the ERN emerges in adolescence (A. Meyer et al., 2012; Weinberg, Meyer, Hale, & Rodehorst, 2016). It is unknown if the ERN in social contexts is related to dimensional aspects of social anxiety during adolescence, a period of elevated fear of peer evaluation (La Greca & Lopez, 1998). Furthermore, it is unknown whether pubertal development modulates the association between the ERN and social anxiety in adolescence. These questions were also addressed in aim 3 of the proposal.

3.2 Overview of the Present Study

The present study is the first known study to investigate how pubertal development influences the function of neural structures that support social motivation and affective processing in adolescence (i.e., ERN, CRN, Pe), and provides initial evidence of possible neurobiological mechanisms for the development of social anxiety and depression in adolescence. Seventy-six adolescent girls between 9-17 years of age participated in the present study and completed the Pubertal Development Scales (PDS; Petersen et al., 1988), a self-report measure of current pubertal status. Adolescents then completed a flanker task (Eriksen & Eriksen, 1974) under two different social-motivational contexts; alone (nonsocial condition) and during social evaluation by peers (social condition). In addition, parents and adolescents completed questionnaires on adolescent development, general anxiety and social anxiety, and depression (Faulstich, Ruggiero, Enyart, & Gresham, 1986; La Greca & Lopez, 1998; Muris et al., 1998).

3.3 Research Questions and Hypotheses

Aim 1. The Influence of Puberty on Error Monitoring

To examine whether pubertal development is associated with developmental changes in the ERN, CRN, Pe, and behavioral indices of error monitoring in adolescence. The proposed study examined whether developmental changes in error monitoring are related to pubertal development, either through overlapping associations with chronological age, or in addition to the shared association with chronological age. Specifically, the study examined whether the ERN (in addition to

the CRN and Pe) are related to pubertal development. In addition, the study explored whether behavioral indices of error-monitoring (PES, PIA) are positively related to pubertal development. It was hypothesized that the ERN and CRN will be related to pubertal development above and beyond shared variance with age. However, it was expected that the Pe would be unrelated to pubertal development. In addition, it was expected that improvements in PES would be related to pubertal development.

Aim 2. Social Influences of Error Monitoring

To examine whether the ERN, CRN, Pe and behavioral indices of error monitoring are enhanced in social contexts in adolescence and whether puberty modulates the degree of enhancement. The present study examined whether the ERN, CRN, and Pe, as well as the behavioral indices of error monitoring are enhanced in social contexts as compared to the nonsocial contexts. It was hypothesized that among all adolescents, the ERN, CRN and Pe would be enhanced in social contexts as compared to the nonsocial contexts. In addition, it was hypothesized that adolescents would exhibit better behavioral performance (improved accuracy, faster response times) post-error adjustments (larger PES and PIA) in social contexts as compared to nonsocial contexts.

Aim 3. Associations with Anxiety/Moderating Role of Puberty

To examine whether puberty moderates the relation between enhancements of the ERN (and other indices of error monitoring) in social contexts and social anxiety symptoms. The present study examined whether pubertal status moderated the relation between the degree to which the ERN was enhanced in social contexts as compared to nonsocial contexts (i.e., social effect ERN) and social anxiety symptoms (see figure

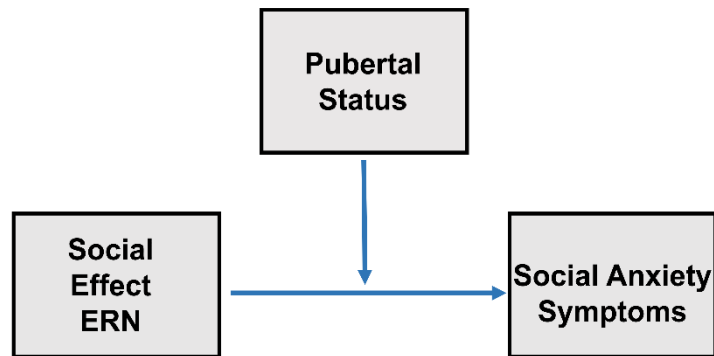


Figure 3. Hypothesized moderation model: Pubertal Status moderates the relation between *Social Effect ERN* (social ERN - nonsocial ERN) and social anxiety symptoms such that there is an association between the social effect ERN and social anxiety only among mid/late pubertal adolescents. A similar moderation effect was hypothesized for the *Social Effect Pe* (social Pe - nonsocial Pe).

3). It was hypothesized that pubertal status would moderate the relation between the social effect ERN and social anxiety such that there would be a significant relation between the social effect ERN and social anxiety only among late pubertal adolescents. No relation between social effect ERN and social anxiety was expected to be observed among early pubertal adolescents. Similarly, it was hypothesized that pubertal status would moderate the relation between the social effect Pe (social Pe - nonsocial Pe) and social anxiety symptoms. No moderation with behavioral measures was expected.

Chapter 4. Methods

4.1 Participants

Participants were 76 adolescents girls ($M_{\text{age}} = 11.87$ years; $SD = 2.2$ years; range 8.7 - 17.1 years; see figure 4 for histogram). Subsequent to the approval of the University of Maryland Institutional Review Board, adolescent girls and their families were recruited through an interdepartmental database of families who agreed

to be contacted for participation in psychological studies at the University of Maryland, College Park. Participants with known developmental disorders (e.g., autism, Down syndrome), known birth defects, or severe visual impairment that could not be corrected with glasses were excluded from participation. Ethnicity and race were self-reported as: 56% Caucasian, 17% African American, 12% multi-racial, 5% Hispanic, 4% Asian, and 6% unreported.

For aim 1, the nonsocial condition served as the baseline measure to examine the development of the ERN and related error monitoring indices. Participants were excluded if accuracy was below 60% in the nonsocial condition (1 participant). In addition, one participant did not complete the nonsocial condition due to experimenter error. Thus, the sample for behavioral analyses was 74 participants. For EEG analyses, three additional participants were excluded due to inability to collect EEG (due to hair braids). All remaining participants who completed EEG collection had at least 6 artifact-free error trials for ERN analysis (Olvet & Hajcak, 2009). Thus, the final sample for EEG analysis was 71 participants. For aim 2 and aim 3 (social

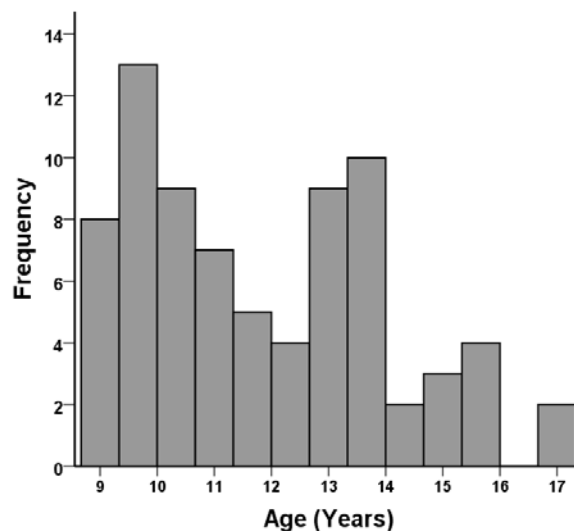


Figure 4. Histogram distribution of participant age (in years).

influences of error monitoring), participants were excluded if accuracy was below 60% in either the social or the nonsocial condition (1 participant). In addition, three subjects did not complete the social condition due to experimenter error. Thus, behavioral analyses for aim 2 and aim 3 were based on 72 participants. For EEG analysis, six additional subjects were excluded due to fewer than 6 artifact-free error trials in each the social condition and nonsocial condition. Thus, the final sample for EEG analysis for aim 2 and aim 3 was 66 participants.

4.2 Procedure

At the beginning of the experiment, adolescents and parents were explained the procedures of the study and informed consent was obtained from the parent and assent was obtained from the adolescent. Following consent/assent, parents and adolescents completed the study questionnaires (see below for description of questionnaires). Next, adolescents were fitted with the EEG net (see below) and performed the flanker task in one of two conditions, which were counterbalanced across participants. During the nonsocial condition, adolescents were informed that they would be receiving computer generated feedback about their performance, and were asked to adjust their performance based on the feedback. The feedback received was based on the participant's accuracy on the previous block (see flanker task for more information). In the social condition, participants were informed that two adolescents located in another lab would be observing them through a webcam while they played the flanker task, and that the adolescents located in the other lab would be giving feedback about their performance (see figure 5). However, in actuality,

participants were not observed by other adolescents, and all feedback was computer generated. The criteria for feedback received in the social condition was identical to criteria of nonsocial condition. For participants who completed the social condition first, during the nonsocial condition it was emphasized that no one was watching their performance. Following the procedure, adolescents were administered a debriefing questionnaire which includes questions to check deception of the social manipulation, and self-report of effort and anxiety in each condition. Of adolescents who completed both the social and nonsocial condition ($n = 68$), 94% of adolescents reported that they were deceived by the social manipulation. Adolescents who were not deceived by the manipulation were older ($M = 14.07$, $SD = 1.2$) than those that were deceived ($M = 11.6$, $SD = 2.1$), $t(70) = 2.29$, $p = .025$. However, there were no differences in pubertal development, behavioral measures, or ERP's between those who were and were not deceived, p 's $> .20$. Thus, all participants regardless of deception were included in all analyses.

4.3 Measures

Questionnaires

Pubertal Development Scale (PDS; Petersen et al., 1988): Adolescent girls completed the PDS, a 6-item, standardized, well-validated measure of puberty. Parents also completed the parent-report version of the PDS. Questions that reflect the main axes of puberty for females, growth (item 1), adrenal (items 2, 3), and gonadal (item 5), were separately averaged for parent-report and adolescent-report (Dorn et al., 2006; Quevedo et al., 2009). Next, parent and child PDS reports were

averaged together to create a composite score. The correlation between parent-report and adolescent-report of PDS was very high, $r(74) = .88, p < .001$. For some analyses, adolescents were categorized into pubertal groups (prepubertal, early puberty, midpubertal, late pubertal) based on the categorization criteria suggested by Peterson et al. (1988) using the parent-report version of the PDS. The PDS is a strong measure of pubertal development; The PDS is highly concordant with clinician-administered exams and picture-based interviews of pubertal development, and is predictive of basal sex hormone concentrations (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Shirtcliff, Dahl, & Pollak, 2009).

Screen for Child Anxiety Related Emotional Disorders (SCARED-R; Muris et al., 1998): Adolescents completed the SCARED-R, a 66-item questionnaire that assesses child anxiety symptoms across 8 domains of anxiety. Parents also completed a parent-report version of the SCARED-R. Analyses focused on the Total Anxiety Score (sum of all 8 domains), The SCARED-R has been successfully utilized to explore the relation between anxiety and the ERN in children (Lahat et al., 2014) and adolescents (Meyer et al., 2012). The SCARED-R demonstrates excellent psychometric properties and is able to reliability distinguish anxiety disorders from other psychological disorders in adolescence (Muris et al., 1998).

Social Anxiety Scale for Adolescents (SAS-A; La Greca & Lopez, 1998): Adolescents completed the SAS-A, a 22-item self-report questionnaire designed to examine social anxiety symptoms in adolescence, with a particular focus on social-evaluative anxiety. Parents also completed a parent-report version of the SAS-A. Adolescents rated on a 5-point scale according to how true each description is to their

own experiences, ranging from 1 (*not at all*) to 5 (*all the time*). The SAS-A demonstrates excellent psychometric properties, good internal reliability, and good test-retest reliability (La Greca & Lopez, 1998; Storch, Masia-Warner, Dent, Roberti, & Fisher, 2004).

Penn State Worry Questionnaire for Children (Chorpita, Tracey, Brown, Collica, & Barlow, 1997; T. J. Meyer, Miller, Metzger, & Borkovec, 1990). Adolescents completed the PSWQ-C, a 14-item self-report questionnaire that assesses symptoms of extreme worry and rumination associated with generalized anxiety disorder (GAD). Adolescents rated on a scale ranging from 1 (*never true*) to 4 (*always true*) how much they agreed with statements about the frequency and intrusiveness of their worries. PSWQ-C has been utilized for children and adolescent studies of anxiety (Chorpita et al., 1997). In addition the PSWQ-C successfully discriminates GAD from other anxiety disorders in children (Chorpita et al., 1997) and is highly correlated with other questionnaire measures of GAD symptoms (Muris, Meesters, & Gobel, 2001).

Center for Epidemiologic Studies Depression Scale (CES-DC; Faulstich, Ruggiero, Enyart, & Gresham, 1986; Radloff, 1977). Adolescents completed the CES-DC, a 20 item self-report questionnaire that assesses current depressive symptoms. Adolescents rated on a scale ranging from 1 (*not at all*) to 4 (*a lot*) how much they agreed with a series of statements about how they felt in the past week. The CES-DC demonstrates high internal consistency and good test-retest reliability in children and adolescents (Faulstich et al., 1986; Radloff, 1977). The CES-DC has

been shown to discriminate clinically depressed adolescents from non-depressed adolescents (Roberts, Lewinsohn, & Seeley, 1991), and predict the development of major depressive disorders (Garrison, Jackson, Marsteller, McKeown, & Addy, 1990).

Self-report of mood and effort: Adolescents completed mood scales (1) before the first flanker task completed, (2) between the first and second flanker condition, and (3) after the second flanker condition. At the very end of the experiment, adolescents reported how hard they tried in each condition on a likert scale from 1 (*did not try at all*) to 10 (*tried really hard*).

Experimental Design

Flanker Task: An adapted arrow version of the flanker task (Eriksen & Eriksen, 1974) was administered using e-prime software (Psychology Software Tools, Inc., Sharpsburg, PA). On each trial, participants viewed five horizontal arrowheads. On half of the trials, arrowheads were congruent (<<<<<<, >>>>>>) and on the other half of the trials the arrowheads were incongruent (<<<><<, >><>>>). The order of presentation of the arrowheads were presented randomly. All stimuli were presented for 200 ms with an intertrial interval (ITI) that varies randomly 800-1200 ms following the response. Prior to beginning the task, participants were explained to press a button depending on the direction of the middle arrow and then completed a practice block of 16 trials. Following, adolescents completed the actual flanker task, which consisted of 10 blocks of 32 trials (320 trials total). After each block, participants received a short break and feedback about their performance (Weinberg et al., 2010). If performance was 75% or below, participants received a message to be

more accurate. If performance was above 90%, participants received a message to respond faster. If performance between 75% and 90%, participants received a message that they were doing a good job. In the nonsocial condition, during feedback breaks, white text was presented on the screen that displayed “Be more accurate”, “Good Job”, or “Respond Faster” for the respective feedback types (i.e., computer generated feedback). In the social condition, adolescents received images (i.e., emoji/emoticon) accompanied with text that was unique to each feedback type (see figure 5). Although the source of feedback was manipulated between the nonsocial flanker condition and the social flanker condition, the accuracy criteria in which they received the feedback were identical between conditions.

4.4 EEG Recording and Data Reduction

Continuous EEG was recorded using a 128-channel Geodesic Sensor Net and sampled at 250 Hz using EGI software (Electrical Geodesic, Inc, Eugene, OR). Before data collection, all electrode impedances were reduced to below 50 k Ω . All electrodes were referenced online to Cz and re-referenced to the average off-line. All EEG/ERP processing was completed using ERP PCA Toolkit (Dien, 2010). Data were filtered off-line using a digital band-pass FIR filter from .3-30 Hz. Responses-locked trials were separately segmented for error and correct trials 600 ms before the response to 600 ms after the response. Channels were marked bad if the amplitude for

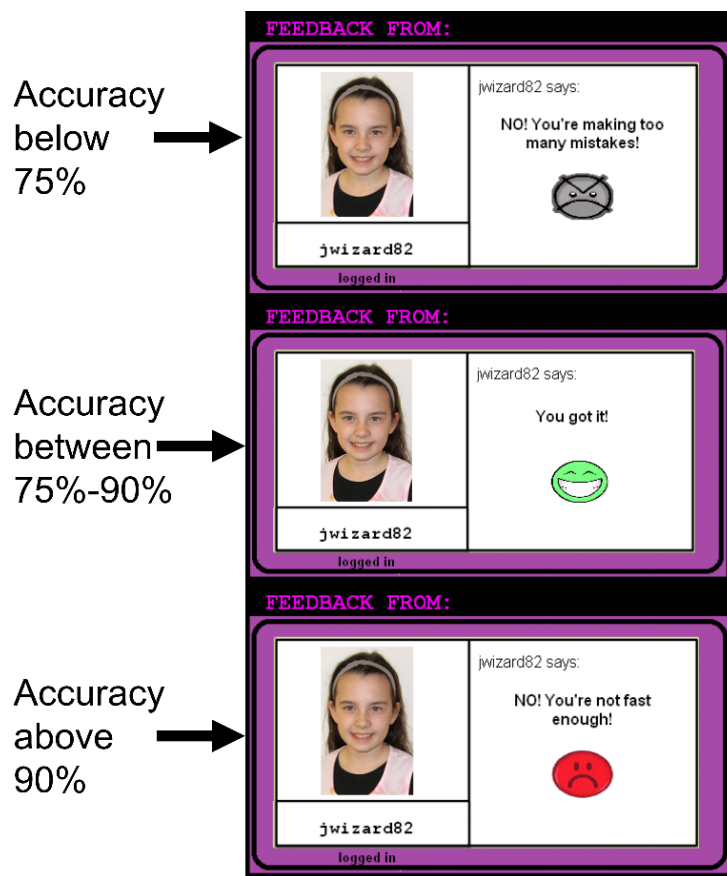


Figure 5. Examples of the social feedback received during breaks between trial blocks for participants in the social flanker condition. Like the nonsocial condition, feedback that participants received in the social condition was dependent on participant accuracy on the previous block.

a trial exceeded 145 μV or if the difference between a channel and neighboring channels was greater than 45 μV for an individual segment. Channels were marked globally bad if the correlation between neighboring channels was less than .30 or if the channel was bad on greater than 20% of trials. Individual trials were marked bad if more than 15% of channels were determined to be bad (social condition 8.8% of trials; nonsocial condition: 2.1% of trials). Bad channels on remaining good trials were replaced using spherical spline interpolation (Perrin et al., 1989, 1990). Participants needed at least 6 artifact-free error trials for each respective condition to be included in analyses (Larson, Baldwin, Good, & Fair, 2010; Meyer, Bress, & Proudfit, 2014; Olvet & Hajcak, 2009). There were no differences in the number of artifact-free error trials between conditions (nonsocial: $M = 36.23$, $SD = 17.1$, social: $M = 35.39$, $SD = 16.5$), $t(65) = 0.49$, $p = .63$, or the number of artifact-free correct trials between conditions (nonsocial condition: $M = 241.77$, $SD = 50.2$, social condition: $M = 246.24$, $SD = 46.4$), $t(65) = 1.00$, $p = .32$. Pubertal status and age were unrelated to the number of artifact-free error trials in either condition, p 's $> .20$. However, pubertal status was positively correlated with the number of artifact-free correct trials (nonsocial condition: $r(64) = .58$, $p < .001$; social condition: $r(64) = .50$, $p < .001$), such that more advanced pubertal status was associated with more artifact-free correct trials. In addition, in the nonsocial condition, the number of artifact-free error trials was negatively correlated with accuracy, $r(67) = -.59$, $p < .001$, such that less artifact-free error trials was related to better accuracy. This correlation was also observed in the nonsocial condition, $p < .001$.

To avoid possible confounds of neural measures with number of trials averaged into each subject's ERP waveform, a mean amplitude measure was utilized. Mean amplitude has been demonstrated to be an unbiased estimate of neural activity associated with ERP's, and is recommended when trial numbers vary between conditions (Clayson, Baldwin, & Larson, 2013; Keil et al., 2014, Luck 2014). Furthermore, the ERN is stable across increasing trials, such that the ERN on early error trials are of a similar magnitude as the ERN on later error trials (A. Meyer, Bress, & Proudfit, 2014b; A. Meyer, Riesel, & Proudfit, 2013; Olvet & Hajcak, 2009; Weinberg & Hajcak, 2011), suggesting that all error trials can be averaged together for each subject. Taken together, the utilization of mean amplitude measures obviates the need for alternative methods to control for trial number confounds (e.g., trial titration, controlling for the number of trials in analyses).

To measure the ERN and CRN, a quasi peak-to-peak measure was created by first baseline correcting all waveforms 140 ms-40 ms before the response. This baseline was chosen to reduce the influence of overlapping P3 variation (see Pailing, Segalowitz, Dywan, & Davies, 2002), which may underestimate developmental effects of the ERN and CRN (Davies et al., 2004). However, employing an early baseline (-600 to 400 ms before the response; Davies et al., 2004) yielded similar but less robust results. The ERN and CRN were then evaluated as the mean activity 0-100 ms following the error and correct responses respectively at an average from 3 fronto-central electrodes along the midline (electrode numbers on 128 ch. geodesic net: 5, 6 (FCz), 12; see figure 6). The Pe was evaluated as the average activity 150-350 ms following the response from the average of 8 centro-parietal electrodes (Cz, 31, 54,

55 [PCz], 62 [Pz], 79, 80; see figure 6). For the ERN, to examine brain activity specific to errors, a difference wave was created by subtracting brain activity on correct trials from brain activity on error trials (i.e., ERN - CRN), which is referred to as the Δ ERN. Similarly, a change score was calculated for the Pe by subtracting the Pe on correct trials from the Pe on error trials (Δ Pe). In addition, in order to examine the change in neural activity across conditions, neural activity from the nonsocial condition was subtracted from neural activity in the social condition for ERP measures of interest (e.g., Social ERN – Nonsocial ERN, Social Δ ERN – Nonsocial Δ ERN; Social Pe – Nonsocial Pe).

Trials with response times faster than 200 and slower than 1200 were removed from the analyses. Errors of omission (i.e., nonresponses) were not included in any calculations. Accuracy was calculated as the number of correct trials divided by the number total trials with a response. Response times were separately averaged for correct trials and error trials for each condition. Post-error improvements in accuracy

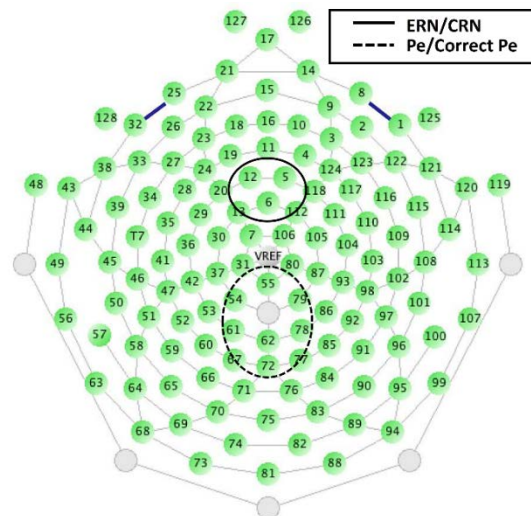


Figure 6. Electrode map of the HGSN 129-channel EEG net. The solid black circle represents the electrode grouping that was used to measure the ERN and CRN. The dotted black circle represents the electrode grouping that was used to measure the Pe and Correct Pe.

(PIA) was calculated as the differences in accuracy between post-correct trials and post-error trials. Post-error slowing (PES) was calculated as the difference in response times between post-error trials and post-correct trials.

4.5 Data Analysis Plan

Prior to exploring the main study aims, multiple Pearson's correlations were conducted to explore the intercorrelations between age, pubertal status, and study questionnaires.

Aim 1. The Influence of Puberty on Error Monitoring

The nonsocial flanker was utilized as the baseline measure to explore developmental changes in error monitoring. For all models, pubertal status was mean centered and included as a covariate. Significant main and interaction effects were explored using paired sample *t*-test for within-subjects effects and independent samples *t*-test for between-subjects effects, and was corrected for multiple comparisons when necessary. The significance level was set at .05 for all analyses.

For all analysis where pubertal status was a significant predictor (main or interaction effect), identical analyses were conducted replacing mean-centered age as the covariate in the model. If both pubertal status and age reached significance in a given statistical model, then both variables were entered into regression analyses to explore if age and puberty uniquely predicted outcome variables above and beyond the overlapping variance with the other predictor. If both age and pubertal status were entered into a model and neither predictor reached significance but the overall model reach significance, then such findings were interpreted that both age and puberty have

overlapping effects on the dependent variables (Steinberg, 1987; Steinberg & Monahan, 2007). If an individual predictor was significant even when controlling for the other predictor, then such findings were interpreted that the significant predictor explained the dependent variables above the overlapping variance of puberty and age (Steinberg, 1987; Steinberg & Monahan, 2007).

Behavioral measures (i.e., accuracy, response time, PES, PIA) and physiological measures (i.e., ERN, CRN, Pe) were separately evaluated. First, to explore developmental changes of the flanker effect, mixed-model ANCOVA's were conducted with congruency (incongruent vs. congruent) and response (error vs. correct) as a within-subjects factors for response times and PES. Similarly, for accuracy and PIA, mixed-model ANCOVA's were conducted with congruency (incongruent vs. congruent) as a within-subjects variable. Interactions with pubertal status were followed-up using separate Pearson's correlations. Next, regression analyses were conducted with age and pubertal status as predictors to explore the degree to which developmental variables predicted changes in behavioral measures.

Since large differences in accuracy and response times were observed between incongruent and congruent trials (see analyses below), a series of repeated measures t-tests were conducted to explore whether flanker congruency influenced neural signatures of error monitoring for the ERN, CRN, and Pe. To explore the developmental differences in error monitoring, mixed-model ANCOVA's were conducted with response (error vs. correct) as a within-subjects factor for the ERN/CRN and the Pe/Correct Pe. Following, regression analyses were conducted with age and pubertal status as predictors to explore the degree to which

developmental variables predicted changes in neural measures. Next, pubertal status was categorized into groups (prepubertal, early pubertal, midpubertal, late pubertal) based on suggestions by Petersen et al. (1997). Multiple ANOVA's were then conducted using this categorization for all ERP components, and follow-up *t*-tests were performed when needed.

Lastly, multiple Pearson's correlations were conducted between behavioral and neural measures to examine if the ERN, CRN, and Pe were related to processing efficiency and post-error adjustments.

Aim 2. Social Influences of Error Monitoring

Mean-centered pubertal status was included as a covariate in all ANCOVA models. First, mixed-model ANCOVA's were conducted to explore differences in self-reported effort and anxiety between conditions. To explore social influences on the error monitoring system, behavioral measures (i.e., accuracy, response time, PES, PIA) and physiological measures (i.e., ERN, CRN, Pe) were then separately evaluated. A mixed-model ANCOVA was conducted with condition (social, nonsocial) and response (correct, incorrect) as the within-subjects factor for response time and PES. Similarly, for accuracy and PIA, mixed-method ANCOVA's were conducted with response (correct, error) as a within-subjects variable. For ERP analyses, multiple 2 (condition) x 2 (response) mixed-model ANCOVA's were conducted. Next, multiple mixed-model ANOVA's were conducted using the pubertal group categorization for ERN/CRN and Pe/Correct Pe. Follow-up *t*-tests were performed when necessary.

Aim 3. Associations with Anxiety/Moderating Role of Puberty

In order to create one variable to use in moderation analyses that indicates how behavioral performance changed across conditions, behavioral measures in the nonsocial condition were subtracted from behavioral measures in the social condition (e.g., social accuracy – nonsocial accuracy). This change score is referred to as a *social effect* (e.g., social effect accuracy), and was created for all behavioral measures. Similarly, this change score was computed for changes in neural measures across conditions. For example, for the social effect ERN, neural activity from the nonsocial condition was subtracted from neural activity in the social condition (i.e., social ERN – nonsocial ERN). Next multiple moderation analyses were conducted to explore if pubertal status moderated the association between the social effect variables and self-report measures of general anxiety, social anxiety, and depression. Moderation analyses were conducted following the guidelines set forth by Baron and Kenny (1986). Age was included as a covariate in all moderation regression models. All predictors were mean-centered prior to analysis. The interaction variable for analyses was computed as the mean-centered product of the moderating variable (i.e., PDS) and the social effect variable. If the interaction variable was significant in the regression model, then follow-up analyses were conducted at high (1 *SD* above) and low (1 *SD* below) levels of the moderator (Baron and Kenny, 1986).

Chapter 5. Results

5.1 Intercorrelations between Study Variables

Table 1 presents the correlations between age, puberty, and self-report and parent-report questionnaires of anxiety and depression. As expected, chronological age was highly correlated with PDS scores, such that older adolescents had a more advanced pubertal status, $r(74) = .81, p < .001$, (see figure 7). In addition, chronological age was positively correlated with PSWQ-C general anxiety such that older adolescents reported more general anxiety symptoms, $r(74) = .32, p < .05$.

Correlations were moderate between child-report and parent-report version for SCARED total anxiety, $r(74) = .38, p < .001$, SCARED social anxiety, $r(74) = .31, p < .01$, SCARED general anxiety, $r(74) = .41, p < .001$, and SAS-A social anxiety, $r(74) = .41, p < .001$. Since parent-report and child-report were only moderately related, versions were separately evaluated in relation to the ERN and other neural measures. For both child-report and parent-report, anxiety and depressive symptoms were moderately correlated with one another, p 's $< .01$. For example, adolescents who reported more SCARED total anxiety symptoms also reported more CES-DC depressive symptoms, $r(74) = .74, p < .001$, and SAS-A social anxiety symptoms, $r(76) = .78, p < .001$.¹

Variable	1.	2.	3.	4.	5.	6.	7.	Mean (SD)
1. Age (years)	-							11.87 (2.2)
2. PDS Pubertal Status	.81**	-						2.2 (.8)
3. SCARED Anxiety (sr)	.18	.13	-					25.86 (14.1)
4. SAS-A Social Anxiety (sr)	.15	.05	.76**	-				44.79 (15.1)
5. CES-DC Depression (sr)	.19	.13	.73**	.62**	-			13.24 (10.0)
6. PSWQ General Anxiety (sr)	.32*	.16	.75**	.72**	.60**	-		10.50 (7.3)
7. SCARED Anxiety (pr)	.12	.11	.38*	.36*	.26*	.34*	-	12.77 (9.3)
8. SAS-A Social Anxiety (pr)	.12	.11	.30*	.40**	.31*	.39*	.74*	42.98 (12.8)

Table 1. Intercorrelations between age, pubertal status, and study questionnaires ($N = 76$). sr = self-report. pr = parent-report. Note: * $p < .05$; ** $p < .01$.

5.2 Aim 1. The Influence of Puberty on Error Monitoring

Development of Behavioral Performance

Overall accuracy was 86.31% ($SD = 5.9\%$; range: 68% to 96%). Analysis for accuracy revealed that overall, participants were significantly more accurate on congruent trials ($M = 93.2\%$, $SD = 5.6\%$) as compared to incongruent trials ($M = 79.24\%$, $SD = 8.8\%$), $F(1, 72) = 196.74$, $p < .001$, $\eta^2 = .73$. In addition, there was a marginal main effect of pubertal status, such that later pubertal development was associated with faster response times, $F(1, 72) = 3.89$, $p = .052$, $\eta^2 = .05$. However, these main effect were qualified by a congruency x pubertal status interaction, $F(1,72) = 5.66$, $p = .020$, $\eta^2 = .07$. Separate correlations for congruent and incongruent responses with puberty revealed that there was a significant positive

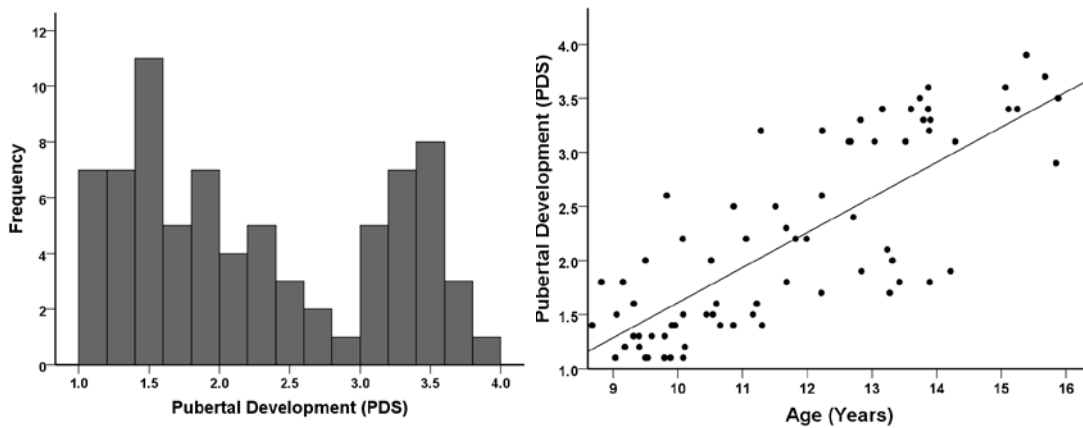


Figure 7. Left Panel: Distribution of PDS. Right Panel: Scatterplot between age and PDS. A linear function best fit this relation between age and PDS, $r(74) = .81$, $p < .001$.

relation between pubertal status and accuracy on congruent trials, $r(72) = .45$, $p < .001$, such that more advanced pubertal status was associated with better accuracy on

congruent trials. However, for incongruent trials, this relation was not significant, $r(72) = .02, p = .91$.

Analyses substituting age instead of pubertal status as a covariate yielded identical results. Thus, to determine the unique influence of age and puberty on accuracy, a multiple regression analysis was conducted with age and pubertal status as predictors. As expected, for incongruent accuracy, the model was not significant, $p = .94$. However, for congruent trials, the overall model was significant, $F(2, 71) = 12.70, p < .001$, with pubertal status and age explaining 26% of the variation in congruent trial accuracy. Age positively predicted congruent trial accuracy independent of puberty, $\beta = .01, t(71) = 2.43, p = .02$. However, puberty did not predict accuracy when controlling for age, $\beta = .008, t(71) = .69, p = .50$. Thus, chronological age predicted changes in congruent accuracy beyond the shared variance with puberty.

Mean response times for individual subjects on the flanker task ranged from 288 ms to 731 ms. Analysis of response times revealed a main effect of response type, $F(2, 67) = 121.04, p < .001, \eta^2 = .64$, such that error response times ($M = 414.39, SE = 11.6$) were significantly faster than correct response times ($M = 509.84, SE = 10.4$). In addition, there was a main effect of congruency, $F(2, 67) = 46.95, p < .001, \eta^2 = .41$, such that congruent trials were significantly faster ($M = 438.51, SE = 11.1$) than incongruent trials ($M = 485.71, SE = 10.3$). However, these main effects were qualified by a congruency x response interaction, $F(2, 67) = 37.05, p < .001, \eta^2 = .36$. Analysis of this interaction revealed that the flanker interference effect on response times (i.e., incongruent response time minus congruent response time) was

greater on correct trials ($M = 82.70$, $SE = 5.6$) as compared to error trials ($M = 11.70$, $SE = 11.4$), $F(1, 67) = 37.05$, $p < .001$, $\eta^2 = .36$. In addition, this was qualified by a response type x pubertal status interaction, $F(1, 67) = 8.78$, $p = .004$, $\eta^2 = .12$, such that there was a significantly larger correlation between pubertal status and response times on correct trials, $r(72) = -.58$, $p < .001$, as compared to error trials, $r(72) = -.40$, $p < .001$. These correlations suggest that more advanced pubertal status is associated with faster response times, particularly on correct trials.

Analyses using age instead of pubertal status as a covariate yielded identical results. Thus, a multiple regression analysis was conducted with age and puberty as predictors. Analyses focused on response type (error and correct responses) since puberty was found to specifically interact with response type. For error trials, the overall model was significant, $F(2, 71) = 8.30$, $p = .001$, $R^2 = .19$. However, neither age nor pubertal status independently predicted error response times, p 's $> .10$. For correct trials, the overall regression model was significant, $F(2, 71) = 23.10$, $p < .001$, with pubertal status and age together explaining 39% of the variation in response times. When controlling for puberty, age was significantly negatively related to response times such that older adolescents exhibited faster responses, $\beta = -.40$, $t(71) = 2.56$, $p = .013$. In addition, pubertal status was marginally negatively related to response times, such that more advanced pubertal status predicted faster response times, $\beta = -.26$, $t(71) = 1.70$, $p = .093$. Thus, both age and pubertal status independently predict developmental changes in response times.

Analysis of post-error improvements in accuracy (PIA) revealed no main or interaction effects, p 's $> .20$. Analysis of post-error slowing (PES) revealed a

marginally significant interaction between trial type (post-response error, post-response correct) and pubertal status, $F(2, 72) = 3.23, p = .072, \eta^2 = .04$, suggesting the degree of post-error slowing (PES) changes over development. Substituting age instead of pubertal status confirmed this developmental trend, $F(2, 72) = 5.31, p = .024, \eta^2 = .07$. Correlation analyses confirmed a positive association between the degree of PES (post-error response times minus post-correct response times) and puberty such that more advanced pubertal status was associated with a greater degree of PES, $r(73) = .21, p = .072$ (see figure 8). To determine the unique influence of age and puberty on response times, a multiple regression analysis was conducted with age and puberty as predictors. Analysis revealed that the overall model was marginally significant, $F(2, 71) = 2.62, p = .08, R^2 = .07$. However, neither predictor was significant when controlling for the other, p 's $> .10$, suggesting that puberty and age have an overlapping effect in predicting developmental improvements in PES.

Development of the ERN/CRN

For correct trials, no differences were observed between the incongruent CRN and the congruent CRN, $t(70) = .42$, $p = .67$. However, for the Pe component on correct trials (i.e., Correct Pe), incongruent trials were significantly more negative than congruent trials, $t(70) = 3.63$, $p < .001$. Among subjects who had committed enough congruent and incongruent errors to be included in the analyses ($n = 40$), the ERN was significantly larger (i.e., more negative) when errors occurred on congruent trials as compared to incongruent trials, $t(39) = 2.24$, $p = .031$. For the Pe, there was no effect of congruency, $t(39) = .79$, $p = .79$. Since sample size was dramatically smaller when excluding participants who did not enough errors on both congruent trials, congruent and incongruent trials were collapsed in order to preserve sample size. In addition, the correlation between the ERN including both trial types averaged together and the ERN on incongruent trials was extremely high, $r(67) = .95$, $p <$

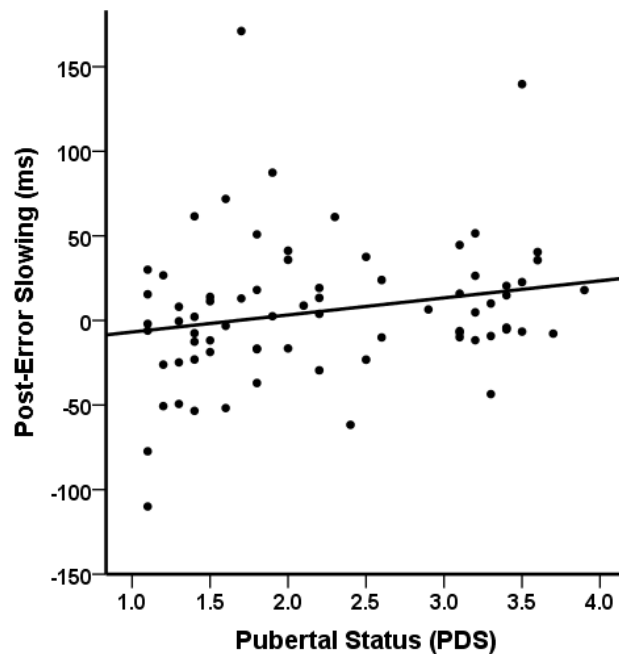


Figure 8. Scatterplot of post-error slowing (PES; response times after error trials minus response times after correct trials) and pubertal status, $p = .072$. A positive PES value indicates slower response times following errors than after correct responses.

.001). All results were identical when only using incongruent trials unless otherwise noted.

For the ERN/CRN, analyses revealed a main effect of response such that the magnitude of the ERN was larger (i.e., more negative) than the CRN, $F(1, 69) = 52.62, p < .001, \eta^2 = .43$. In addition, there was a main effect of puberty, where more advanced pubertal status was related to a larger (i.e., more negative) ERN and CRN, $F(1, 69) = 19.92, p < .001, \eta^2 = .22$. Figure 9 displays the waveforms for the CRN and

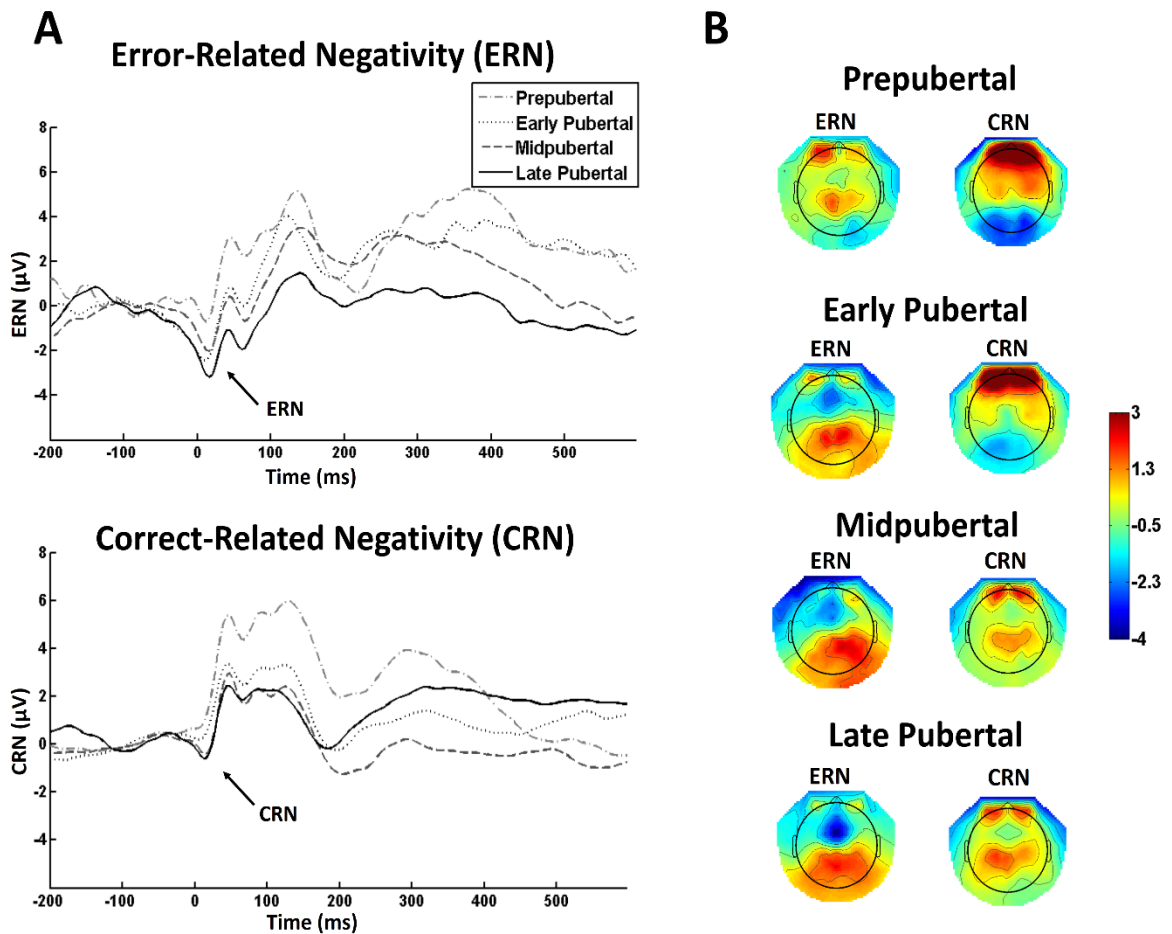


Figure 9. Left Panel (A): ERP waveforms for the ERN (top) and the CRN (bottom) for different pubertal status groups. For both the ERN and CRN, more advanced pubertal status was associated with a larger (i.e., more negative) ERP component. Right Panel (B): Scalp topographies for the ERN (left side) and CRN (right side) for each pubertal groups at 20 ms post-response.

ERN respectively for pubertal groups. Substituting categorical groupings of puberty, analyses demonstrated a significant main effect of pubertal group, such that both the ERN and the CRN were significantly different between pubertal groups $F(3, 67) = 6.95, p < .001, \eta^2 = .24$. Post-hoc Tukey tests revealed that prepubertal adolescents exhibited a smaller ERN and CRN than midpubertal adolescents, $p = .03$, and late pubertal adolescents, $p < .001$. No other differences were observed between pubertal groups.

Similar results were obtained substituting age for pubertal status as a covariate. Next, regression analyses were separately conducted for ERN and CRN with age and pubertal status as predictors. For the CRN, the overall model was significant, $F(2, 68) = 7.12, p = .002, R^2 = .17$. However, neither predictor was uniquely significant, p 's $> .10$. For the ERN, the overall model was significant, $F(2, 68) = 11.95, p < .001$, with 26% of the variation in the ERN explained by

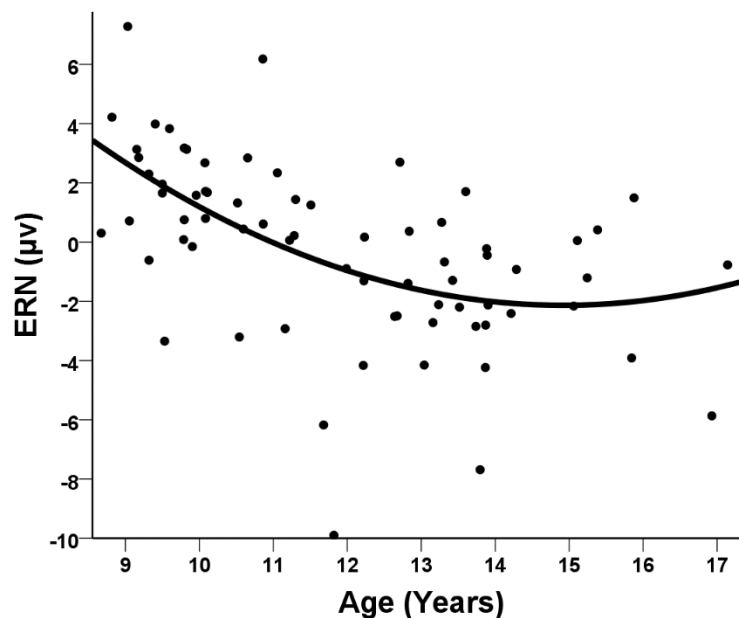


Figure 10. Scatterplot of the ERN and chronological age. Age continued to predict changes in the ERN above the overlapping association with pubertal status, $p < .05$. A quadratic function best fit the relation between age and the ERN, $p < .05$.

chronological age and pubertal status. Although pubertal status did not uniquely predict the ERN beyond the overlapping association with age, $p > .20$, chronological age predicted changes in the ERN beyond the overlapping variance associated with puberty, $\beta = -.65$, $t(68) = 2.74$, $p = .008$. Figure 10 displays the relation between age and the ERN. A quadratic function best explains this relation, $F(3, 67) = 9.93$, $p < .001$, $R^2 = .31$, above and beyond that of a linear function, $F(1, 67) = 4.62$, $p = .035$, $\Delta R^2 = .05$.

Development of the Pe

Analyses revealed that the magnitude of the Pe on error trials was larger than the Pe on correct trials, $F(1, 69) = 173.07$, $p < .001$, $\eta^2 = .72$. No other main or interaction effects reached significance. Similar results were obtained substituting age for pubertal status. Thus, neither chronological age nor pubertal status influenced the Pe in adolescence. Figure 11 displays the waveforms for the Pe and Correct Pe for the entire sample.

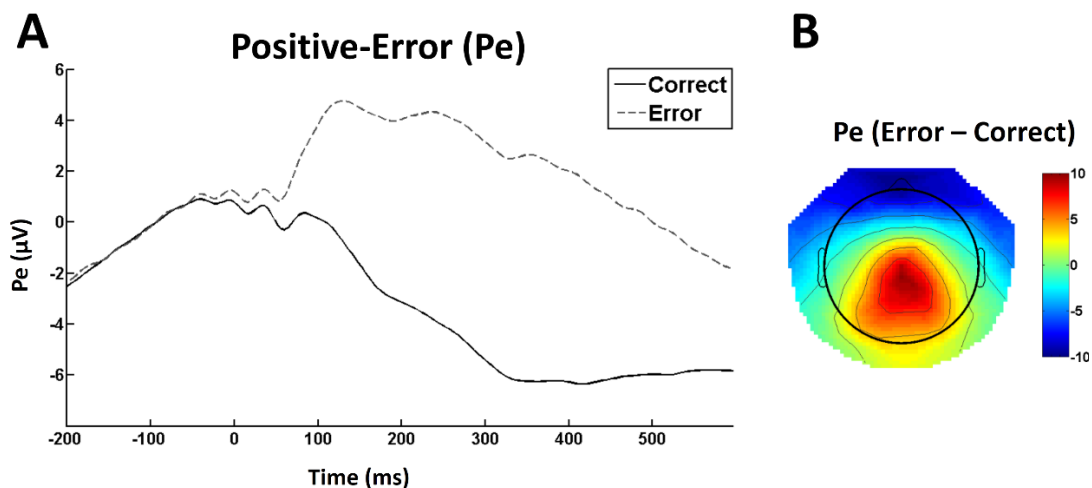


Figure 11. Left Panel (A): ERP waveforms for the Pe on error trials and the correct Pe. Right Panel (B): Scalp topography for the difference wave (Error-Correct) at 200 ms post-response. Age and pubertal status were not related to the Pe or Correct Pe.

Intercorrelations between Behavior and Neural Measures

Table 2 presents a correlation matrix between behavioral and neural measures. The ERN was positively correlated with response times on correct trials, $r(69) = .53$, $p < .001$, and error trials, $r(69) = .43$, $p < .001$, such that a larger ERN was related to faster response times. These effects remained significant when controlling for age and pubertal status, p 's $< .01$, suggesting that the relation between the ERN and response times is independent of developmental changes in response times. In addition, the ERN was marginally related to PES, $r(69) = -.23$, $p = .051$, such that a greater ERN was associated with greater response slowing after errors. The ERN was unrelated to overall accuracy and PIA, p 's $> .18$. For the CRN, there was a similar correlation with PES, $r(69) = -.24$, $p = .047$, such that a more negative CRN was related to greater response slowing after errors. The CRN was unrelated to PIA, overall accuracy, and response times, p 's $> .30$. However, the CRN was related to accuracy on congruent trials, $r(69) = -.23$, $p = .050$, such that a more negative CRN was related to better congruent accuracy. For the Pe, there was a significant positive correlation between the Pe and accuracy on congruent trials, $r(69) = .29$, $p = .015$, such that a larger Pe was related to higher accuracy on congruent trials. In addition, the Pe was marginally related to PIA, $r(69) = .22$, $p = .062$, such that a larger Pe was related greater accuracy on trials following errors. Conversely, the correct Pe was negatively related to congruent accuracy, $r(69) = -.29$, $p = .015$, suggesting a greater difference between Pe on correct and error trials is related to improved accuracy.

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. ERN	-								
2. CRN	.45**	-							
3. Pe	-.05	.19	-						
4. Correct Pe	.19	.04	.01	-					
5. PES	-.23 ⁺	-.24*	-.11	-.04	-				
6. PIA	-.16	-.03	.22	-.20	-.19	-			
7. Congruent Acc	-.20	-.23	.29*	-.29*	-.05	.32*	-		
8. Incongruent Acc	.01	.01	-.04	-.12	.14	-.06	.30*	-	
9. Error RT	.43**	.18	-.20	.10	.27	-.50**	-.33*	.33*	-
10. Correct RT	.53**	.15	-.12	.01	.04	-.29*	-.32*	.26*	.82**

Table 2. Intercorrelations between behavioral and neural measures during the nonsocial condition ($N = 71$). ERN: Error-related negativity; CRN: Correct-related negativity; Pe: Positive error; PES: Post-error slowing; PIA: Post-error improvements in accuracy; Acc.: Accuracy; RT: Response Time. Note: ⁺ $p < .10$; * $p < .05$; ** $p < .01$.

5.3 Aim 2. Social Influences of Error Monitoring

Social Influences on Behavior

Adolescents reported more effort during the social condition ($M = 8.27$, $SD = 1.7$) as compared to the nonsocial condition, ($M = 7.4$, $SD = 2.0$), $F(1, 69) = 23.81$, $p < .001$, $\eta^2 = .20$. This main effect was qualified by a marginally significant pubertal status x effort interaction, such that *less advanced* pubertal status was associated with larger self-report of effort in the social condition as compared to the nonsocial condition, $F(1, 69) = 3.13$, $p = .081$, $\eta^2 = .04$. Self-report of mood scales of anxiety during each condition revealed no significant main or interaction effect, p 's $> .20$.

Table 3 displays response times, accuracy, PIA, and PES for the social and nonsocial condition. For accuracy, analyses focuses on congruent trials since these trials had the largest development changes. Analyses of congruent trial accuracy revealed a main effect of condition, such that adolescents were more accurate in the

social condition as compared to the nonsocial condition, $F(1, 70) = 5.51, p = .022, \eta^2 = .07$. In addition, there was a main effect of pubertal status, such that more advanced pubertal status was associated with better congruent trial accuracy, $F(1, 70) = 5.57, p = .022, \eta^2 = .07$. No interaction effects reached significance. For PES and PIA, no main or interaction effects reached significance, p 's $> .20$.

Analyses of response times revealed a main effect of response type, such that errors were significantly faster than correct responses in both conditions, $F(1, 70) = 341.23, p < .001, \eta^2 = .83$. In addition, there was a main effect of condition, such that response times for both error and correct responses were faster in the social condition as compared to the nonsocial condition, $F(1, 70) = 9.50, p = .003, \eta^2 = .12$. Additionally, there was a main effect of pubertal status, such that more advanced pubertal status was associated with faster response times, $F(1, 70) = 29.80, p < .001, \eta^2 = .30$. These main effects were qualified by a pubertal status x response interaction, such that the greatest differences in response time between correct and error responses was observed among earlier pubertal adolescents, $F(1, 70) = 16.40, p < .001, \eta^2 = .19$. No other interaction effects reached significance.

Social Influences on ERN/CRN

Analysis of the ERN/CRN revealed that error responses (i.e., ERN) were significantly larger (i.e., more negative) than correct responses (i.e., CRN) in both the social and nonsocial condition, $F(1, 64) = 61.72, p < .001, \eta^2 = .49$. In addition, more advanced pubertal status was associated with a larger ERN and CRN, $F(1, 64) = 14.42, p < .001, \eta^2 = .18$. Furthermore, there was a main effect of condition, such that both the CRN and ERN were larger (i.e., more negative) in the social condition as compared to the nonsocial condition, $F(1, 64) = 5.94, p = .018, \eta^2 = .09$. This was qualified by a marginal pubertal status x condition interaction effect, $F(1, 64) = 3.00, p = .088, \eta^2 = .05$, suggesting the degree that the ERN/CRN changed across conditions was influenced by puberty.

To further explore the effect of puberty on the ERN/CRN, pubertal status was dichotomized into two groups (pre/early puberty, mid/late puberty) using the pubertal categorizations by Peterson et al. (1987). Figure 12 displays the waveforms in the

Behavior Measures	Nonsocial	Social
Error response time (ms)	401.61 (86.4)*	389.48 (85.5)*
Correct response time (ms)	495.07 (104.4)*	472.98 (86.7)*
Accuracy on congruent trials (%)	93.53 (5.4)*	94.27 (5.3)*
Post-error slowing-PES (ms)	5.13 (39.2)	6.86 (33.3)
Post-error accuracy-PIA (%)	-1.2 (10.8)	1.3 (7.8)
ERP's (μV)		
ERN	-0.43 (3.0)*	-1.24 (3.2)*
CRN	2.09 (2.1)*	1.59 (2.5)*
ΔERN	-2.52 (2.8)	-2.83 (3.4)
Pe	4.05 (3.9)*	5.09 (4.4)*
Correct Pe	-4.32 (3.6)	-4.71 (3.6)
ΔPe	8.38 (5.3)*	9.80 (4.7)*

Table 3. Means for behavioral performance and event-related potential (ERP) measures for the social and nonsocial condition ($N = 66$). * Indicates significant main effect of condition, $p < .05$.

social and nonsocial condition for pre/early and mid/late pubertal groups. Analyses using a categorical split of puberty revealed a similar pubertal status x condition interaction, $F(1, 64) = 4.90$ $p = .031$, $\eta^2 = .07$. In addition, this was qualified by a significant 3-way response x condition x pubertal status interaction, $F(1, 64) = 4.85$, $p = .031$, $\eta^2 = .07$. Exploration of this effect revealed that among pre/early pubertal participants, the ERN in the social condition was significantly larger ($M = -0.98$ $SD = 3.7$) than in the nonsocial condition ($M = 0.76$, $SD = 2.9$), $t(32) = 2.71$, $p = .011$. However, for mid/late pubertal adolescents, there were no differences between the ERN in the social condition ($M = -1.47$, $SD = 2.8$) and the nonsocial condition ($M = -1.57$, $SD = 2.8$), $t(32) = .27$, $p = .78$. For both groups, there were no significant differences between the CRN in the social and nonsocial conditions, p 's $> .10$.

Social Influences on Pe

Analysis of the Pe revealed a main effect of response, such that the Pe on error trials was significantly larger than the Pe on correct trials, $F(1, 64) = 245.45, p < .001, \eta^2 = .79$. This was qualified by a significant condition x response interaction, $F(1, 64) = 11.52, p = .001, \eta^2 = .15$. Exploration of this interaction revealed that the Pe was enhanced in the social condition as compared to the nonsocial condition, $t(65) = 2.59, p = .012$. However, there were no differences for the Correct Pe between conditions, $t(65) = 1.13, p = .19$. No interactive effects with puberty reached significance using either continuous or categorical measures of pubertal status, p 's $> .09$.

Relation between Behavior and Neural Changes

To compare whether behavioral and neural measures across conditions were related, a change score was created by subtracting each nonsocial measure from the equivalent social measure (i.e., *social effect*). These change scores were then correlated with one another. Pearson's correlations revealed that the degree to which adolescents reported more effort/motivation in the social condition from the nonsocial condition (social effect effort) was negatively correlated with the degree to which the ERN was enhanced in the social condition from the nonsocial condition (social effect ERN), $r(63) = -.25, p = .046$. This correlation suggest that higher reports of effort and motivation in the social condition was associated with concomitant enhancements of the ERN in the social condition. Interestingly, report of effort was unrelated to the social effect Pe and all behavioral changes observed on the flanker task (p 's $> .20$). Furthermore, the social effect ERN was positively correlated with the degree to which response times changed across conditions on correct trials (i.e., social effect RT),

$r(65) = -.28, p = .025$, and on error trials, $r(64) = .33, p = .007$, such that a larger ERN in the social condition was associated with faster responses in the social condition. The social effect Pe was marginally related to the social effect congruent accuracy, $r(64) = .22, p = .08$, such that a larger Pe in the social condition was associated with better accuracy on congruent trials.

5.4 Aim 3. Associations with Anxiety/Moderating Role of Puberty

Partial correlations (controlling for age and pubertal status) revealed that behavioral indices of error monitoring (i.e., accuracy, response times, PES, PIA) in the social condition and the nonsocial condition were not associated with any measures of general anxiety, social anxiety, or depression, p 's > .40. However, the degree to which response times changed across conditions (i.e., social effect RT) was correlated with child-report SCARED total anxiety (correct trials: $r(68) = .26, p = .032$; error trials: $r(68) = .27, p = .025$), such that faster response times in the social condition as compared to the nonsocial condition were related to higher self-report of anxiety symptoms. Similarly, adolescent-report of SAS-A social anxiety was correlated with the social effect RT for correct trials, $r(68) = .20, p = .029$, and error trials, $r(62) = .23, p = .054$.² In addition, social effect PES (Social PES – nonsocial PES) was positively correlated with SAS-A social anxiety, such that greater response slowing after errors was related to more social anxiety symptoms, $r(68) = .33, p = .006$.

² Similarly, a large positive correlation was observed between SCARED social anxiety subscale and social effect RT on correct trials, $r(68) = .43, p = .017$, and error trials, $r(68) = .35, p = .003$.

To better explain the relation between social anxiety and behavioral changes across conditions, social anxiety groups (i.e., high social anxiety and low social anxiety) were created by creating a median split based on scores on the SAS-A. Analysis of response times using this categorization demonstrated a marginally significant social anxiety x condition interaction, $F(1, 68) = 3.63, p = .061, \eta^2 = .05$. This interaction reflects that among low socially anxious adolescents, there were significantly faster response times in the social condition as compared to the nonsocial condition for both error and correct responses, $F(1, 33) = 11.93, p = .002, \eta^2 = .27$. However, among high socially anxious adolescents, there were no differences in response times between conditions, $F(1, 33) = .77, p = .39, \eta^2 = .02$ (see figure 13).

Analysis of PES with social anxiety group as a factor revealed a significant social anxiety x condition interaction, $F(1, 68) = 6.50, p = .013, \eta^2 = .09$ (see figure 14). This interaction reflects that among high socially anxious adolescents, there was a significant increase in PES in the social condition as compared to the nonsocial

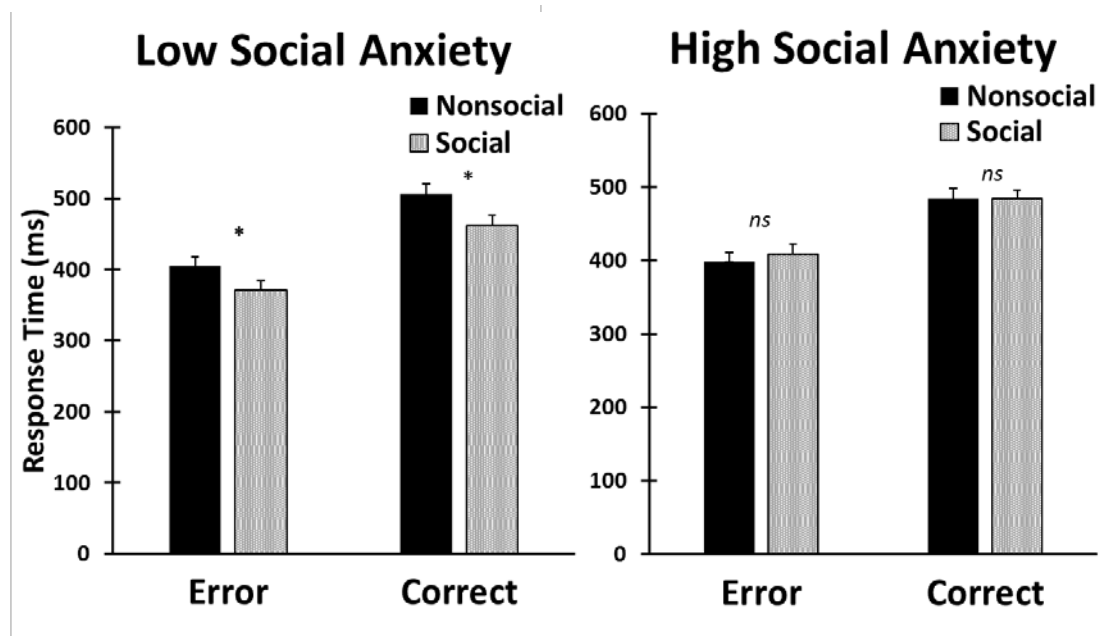


Figure 13. Responses times for error and correct trials for the low social anxiety group (left) and the high social anxiety group (right). For the low social anxiety group, there was a significant decrease in response times for both error and correct trials in the social condition as compared to the nonsocial condition ($p < .01$). However, for the high social anxiety group, there was no changes in response times between conditions ($p > .20$).

condition $F(1, 33) = 4.082, p = .052, \eta^2 = .11$. One-sample t -test revealed that for the high socially anxious group, PES was significantly greater than zero in the social condition, $t(35) = 3.94, p < .001$, but was not in the nonsocial condition, $t(35) = .68, p = .45$. However, for low socially anxious adolescents, there were no differences in PES between conditions, $F(1, 33) = 2.01, p = .17, \eta^2 = .06$, and PES slowing was not significantly larger than zero in either condition, p 's $> .20$.

For the ERN and CRN, there were no significant correlations with general anxiety or social anxiety symptoms, p 's $> .20$. However, self-report of depressive symptoms, as measured by the CES-C, was positively associated with the Δ ERN (ERN – CRN) in the social condition, such that a *smaller* Δ ERN was related to more depressive symptoms, $r(60) = .27, p = .031$. The Δ ERN and depression relation was

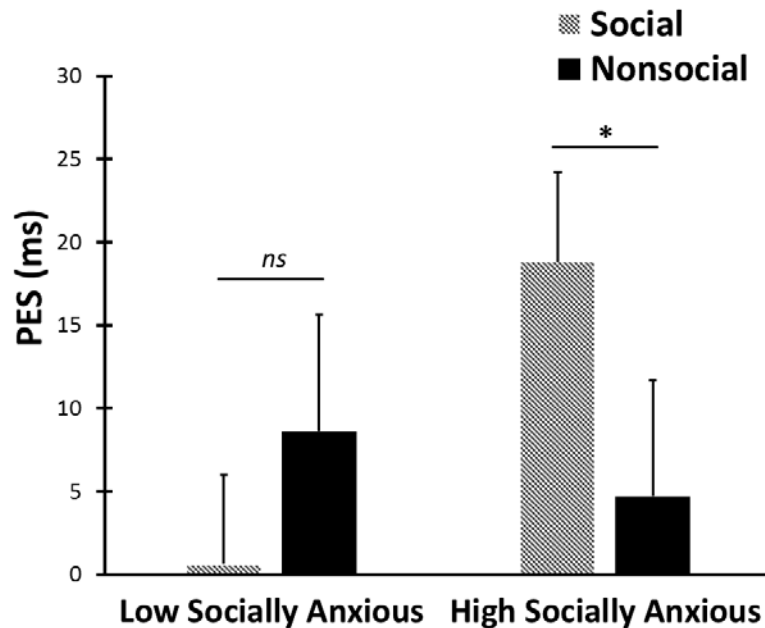


Figure 14. Post-error slowing (PES) for the low social anxiety group (left) and the high social anxiety group (right). For the high social anxiety group, there was a significant increase in PES in the social condition as compared to the nonsocial condition. In contrast, for the low socially anxious group, there were no differences between conditions * $p < .05$.

nonsignificant in the nonsocial condition, $p = .81$. Furthermore, the degree to which the Δ ERN was *smaller* in the social condition as compared to the nonsocial condition (social effect Δ ERN) was positively related to depressive symptoms, $r(60) = .30$, $p = .015$. This relation remained significant when controlling for SCARED total anxiety symptoms, $r(61) = .28$, $p = .027$, suggesting the relation was specific to depressive symptoms. As shown in figure 15, adolescents who exhibited a reduced Δ ERN in the social condition as compared to the nonsocial condition reported more depressive symptoms.

Moderation analyses for the social effect ERN and the social effect Δ ERN revealed that neither variable interacted with pubertal status in predicting general anxiety or social anxiety, p 's $> .20$. Substituting age as the moderator into analyses revealed similar nonsignificant results, p 's $> .20$. Thus, neither pubertal status nor age moderated the association between the degree of enhancement of the ERN in social

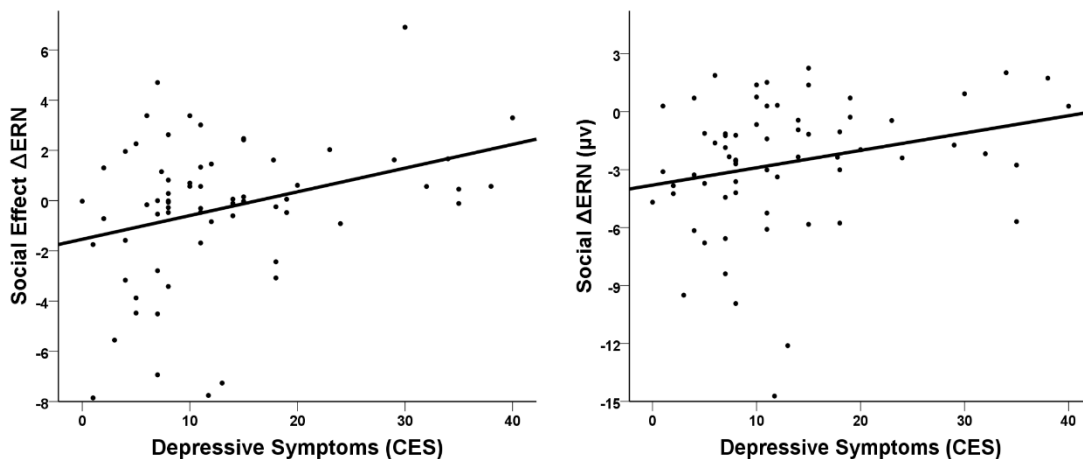


Figure 15. Left Panel: Scatterplot between Social Effect Δ ERN (Social Δ ERN – Nonsocial Δ ERN) and CES-DC depressive symptoms. Right Panel: Correlation between Social Δ ERN) and CES depressive symptoms. The correlation between the Social Effect Δ ERN and depression remained significant when controlling for SCARED total anxiety symptoms.

contexts and anxiety symptoms. For depressive symptoms, the social effect ERN was positively related to CES depressive symptoms, $t(61) = 2.42, p = .018$, such that a smaller ERN in the social condition as compared to the nonsocial condition was related with higher self-report of depression (see figure 15). However, the pubertal status x social effect ERN interaction variable was not significant, $p > .20$. Thus the strength of this effect was not influenced by pubertal status.

Moderation analyses for the social effect Pe (social Pe – nonsocial Pe) revealed that the social effect Pe x pubertal status interaction product significantly improved the regression model in predicting child-report of SCARED total anxiety symptoms, $F(1, 61) = 5.66, p = .02$. Analysis of this interaction revealed that among late pubertal adolescents (i.e., one standard deviation above mean; PDS score = 3.08), there was a positive, marginally significant association between anxiety and the social effect Pe, $t(61) = 1.83, p = .06$. However, among early pubertal adolescents (i.e., one standard deviation below mean; PDS = 1.34), the relation was negative but did not reach significance, $t(61) = 1.63, p = .11$. To explore whether this effect is driven by social anxiety, child-report on the SAS-A was substituted as the outcome variable in the regression model. Similarly, the social effect Pe x pubertal status interaction product was significant in the model, $F(1, 61) = 6.67, p = .012$. Among late pubertal adolescents there was a significant positive association between SAS-A social anxiety and the Social Effect Pe, $t(61) = 2.24, p = .03$. However, among early pubertal adolescents, the relation was not significant, $t(61) = 1.50, p = .14$) Figure 16 displays

the moderating effect of pubertal status on the social effect Pe for the SAS-A Social Anxiety.^{3, 4}

Chapter 6. Discussion

The overarching goal of the present study was to explore pubertal influences on the development of the error monitoring system, with a specific focus on the error-related negativity (ERN). It was found that the ERN, as well as the correct-related negativity (CRN), exhibited large changes across adolescent development, and such changes were associated with the overlapping variance of age and puberty. Furthermore, the positive error (Pe) was unrelated to both puberty and chronological age. In addition post-error slowing (PES) also was influenced by the overlapping variance of pubertal development and chronological age.

³ Regression analyses were also conducted using parent report of SCARED total anxiety and parent report of SAS-A social anxiety. Results revealed that the pubertal status x social effect Pe interaction variable did not reach significance in predicting anxiety for either model, p 's > .40, likely due to the relatively low correlations between parent-report and child-report of anxiety (see table 1 for correlations between child and parent report of anxiety).

⁴ To explore whether the social effect Pe is specific in predicting social anxiety, as opposed to more general anxiety symptoms, a similar multiple regression analysis was conducted including PSWQ-C as a covariate. PSWQ was positively related to SAS-A social anxiety, $t(60) = 6.75, p > .001$. In addition, the social effect Pe x pubertal status interaction variable was not significant, $t(60) = 1.48, p = .14$, likely due to the high correlation between PSWQ and SAS-A.

A second goal of the present study was to examine if social contexts enhanced the error monitoring system, and whether puberty modulates the degree to which error monitoring was enhanced in such contexts. As hypothesized, the ERN, CRN, and Pe were enhanced when adolescents committed errors in social contexts as compared to nonsocial contexts. However, contrary to the expected hypothesis, *earlier* pubertal status was associated with the greatest enhancements of the ERN in social contexts. In addition, adolescents exhibited faster response times and improved accuracy in social contexts as compared to nonsocial contexts. However, pubertal status did not modulate any of the behavioral measures across contexts.

A third goal of the present study was to examine whether the degree that neural indices of the error monitoring system were enhanced in social contexts was associated with social anxiety symptoms, and whether puberty moderated this

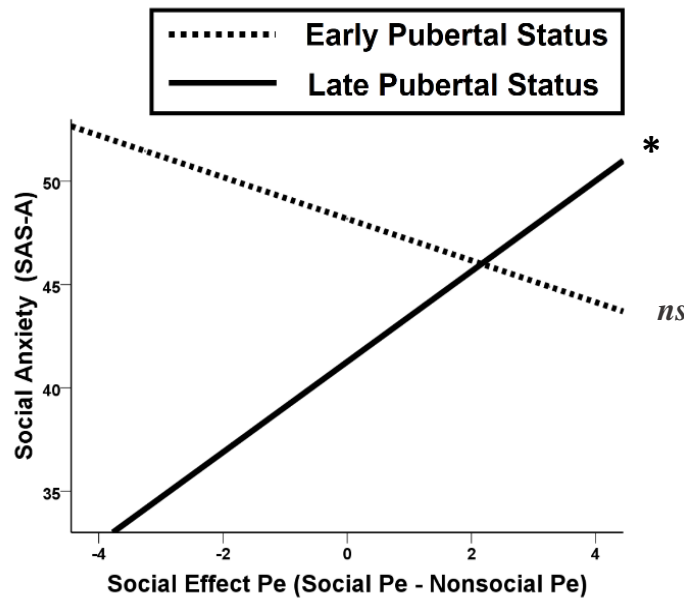


Figure 16. Results of the moderation analyses with the SAS-A social anxiety as the outcome variable. Among adolescents at later pubertal development, a larger social effect Pe was positively related to social anxiety symptoms. However, there was no relation between the social Effect Pe and social anxiety among adolescents at earlier pubertal development. Both moderations were significant even when controlling for chronological age. * $p < .05$.

association. Contrary to the expected hypothesis, enhancements of the ERN in social contexts was not related to social anxiety, nor did pubertal status moderate the association between the ERN and social anxiety. However, as hypothesized, pubertal status moderated the association between the Pe and social anxiety symptoms, such that there was a positive association between enhancements of the Pe in social contexts and social anxiety symptoms only among adolescents at a later pubertal status. In addition, it was found that a reduced ERN in social contexts as compared to nonsocial contexts was related to depressive symptoms. Lastly, social anxiety was related to increases in slowing after errors (i.e., PES) and altered response times in social context.

6.1 Influence of Puberty and Age on Error Monitoring

The present study found that both age and pubertal status predicted developmental changes in the ERN. Combined, the overlapping effects of age and puberty accounted for approximately 26% of the variation in the ERN amplitude across adolescence. These findings of large developmental changes in the ERN across adolescence are in line with previous research (Davies et al., 2004; Santesso & Segalowitz, 2008; Santesso et al., 2006). The ERN appears to be present as early as 3-5 years of age (Grammer et al., 2015). However, there appears to be little developmental changes of the ERN in middle childhood (Kim et al., 2007; Richardson et al., 2011; van Meel et al., 2012), further supporting the hypotheses that regions supporting the ERN continue to development well into adolescence. The anterior cingulate cortex (ACC), the likely generator of the ERN (Holroyd et al.,

1998), exhibits a protracted rate of development into adolescence and early adulthood (Tamnes et al., 2010; Westlye et al., 2010). Functional changes in ACC have also been observed in adolescence (Casey et al., 1995). Furthermore, the ERN has been shown to coincide with the degree of ACC activity (Debener et al., 2005; Mathalon et al., 2003), further suggesting that the ERN may measure the development of ACC function and structure. Thus, large developmental changes in the ERN in adolescence further support the notion that the ACC continues in development through adolescence.

As expected, the CRN demonstrated similar developmental changes across adolescence. However, this developmental effect was much smaller than the ERN. The overlapping effects of puberty and age accounted for approximately 13% of the variation in CRN amplitude. Theories of the CRN suggest that the CRN may represent general response monitoring (Falkenstein et al., 2000). The CRN also has a similar topography as the ERN and is generated by the ACC (Vidal et al., 2000). The CRN has also been found to exhibit developmental changes through adolescence (Davies et al., 2004). However, results are mixed (Santesso & Segalowitz, 2008). Similar to other reports, the development of the ERN appeared to be due to the emergence of a small negativity that occurred at the same time as the ERN (Ford, 1999). In addition, some of the developmental changes in the CRN visually appeared to be due to a reduction of an overlapping positivity, which is likely the stimulus-locked P3 (see figure 11). One possibility is that changes in response times across development changed the timing of the overlapping P3 in the response-locked waveform. However, in the current sample, the CRN was unrelated to response times

on correct trials when controlling for age, $r(68) = -.12$, $p = .34$, suggesting that differences in response times are not responsible for the emergence of the CRN. This finding is identical to the observation by Davies et al. (2001). Thus, changes in the CRN in adolescence appear to be independent of changes in response times, and appear to be due in part to the emergence of an ERN-like negativity on correct responses.

It has been argued that the small negativity of the CRN is not a unique ERP component, but rather the result of subject difficulty with stimulus-response mapping. Specifically, the negativity of the CRN is theorized to be the result of participants pressing the correct response button but actually believing they made an error, which results in error trials averaged into the CRN waveform (Coles et al., 2001; Scheffers & Coles, 2000). Likewise, ERN trials may also contain correct responses, thus reducing the ERN (Pailing & Segalowitz, 2004). Studies have shown that a smaller differentiation between the CRN and ERN due to stimulus-response mapping occurs when participants are unsure of the correct response, such as during high attentional demand (Pailing & Segalowitz, 2004), or when subjects are fatigued or distracted (Scheffers et al., 1999), both of which are common occurrences developmental samples. It has been observed that the difference between the ERN and CRN (i.e., ΔERN) is smaller during a difficult task for adolescents but not for adults (Hogan et al., 2005). This theory would suggest that the youngest participants in the present sample would have the most issues with stimulus-response mapping. Thus the ERN may be underestimated and the CRN may be overestimated among the youngest participants, resulting in a reduced ΔERN . In the present sample it was found that the

Δ ERN was positively correlated with age, although weakly. However, given the size of the age effect for both the CRN and ERN, the relation between the Δ ERN and age was relatively small, suggesting that this effect could be relatively minor. In addition, visual inspection of individual subject waveforms revealed no negativity for the CRN among the youngest participants. Thus, although stimulus-response mapping issues were likely to occur among the youngest participants, this effect on the ERN and CRN appears minimal.

As expected, the Pe was unrelated to age and pubertal status. This finding is in line with previous research that has found that the Pe is unrelated to chronological age in adolescence (Davies et al., 2004; Santesso et al., 2006). It has been theorized that the Pe is similar in topography and function as the stimulus-locked P3b (Overbeek et al., 2005; Ridderinkhof et al., 2009). Indeed, the stimulus-locked P3b is correlated with the response-locked Pe within individuals (Davies, Segalowitz, Dywan, & Pailing, 2001). Interestingly, studies of the development of the P3 have typically found that the P3 increases in amplitude until mid-adolescence, followed by a slow decline (van Dinteren et al., 2014). One study has examined the influences of puberty on the P3 and found that pubertal status was negatively related to P3 amplitude, such that more advanced pubertal status was related to a smaller P3 (Brumback et al., 2012). In the current study, it was found that the Pe was invariant across adolescence. However, given that the present sample encompassed the age in which the developmental direction of the P3 likely changes, the sample size may not have been large enough to detect this effect. However, based on the present findings, it does suggest that the Pe exhibits a different developmental pattern than the P3. Future

research should further explore the developmental patterns of the P3 and Pe within the same study.

The different developmental trajectories of the ERN and Pe are further evidence that the components are functionally distinct (Falkenstein et al., 2001). Theories of the Pe suggest that the Pe represents awareness to errors (Overbeek et al., 2005), in contrast to the ERN which is theorized to relate to more advanced developmental skills such as reward learning (Frank et al., 2005; Nieuwenhuis et al., 2002) and conflict monitoring (Botvinick et al., 2001; Van Veen & Carter, 2002). Interestingly, the Pe does undergo developmental changes in early childhood (3-7 years) whereas the ERN does not change (Grammer et al., 2015). Children began to recognize their own errors and attempt to correct mistakes around 3 years of age (Jones et al., 2003), which coincides with the emergence of the Pe (Grammer et al., 2015). These findings further suggest that the Pe emerges early in life and represents the development of basic error awareness. In contrast, the present findings further support the theory that the ERN undergoes dramatic changes in early adolescence.

Improvements in response time and accuracy were observed through adolescence. Puberty and age accounted for approximately 39% of the variation in response times and 26% of the variation in accuracy. Interestingly, both age and pubertal status independently predicted reductions in response time. Observed reductions in response times across adolescence are consistent with previous literature that has utilized the flanker task (Davies et al., 2004). Exponential decreases in response times are typically observed such that the largest changes occur in childhood with a slower decrease into adolescence (Hale, 1990; Kail, 1991; Luna, Garver,

Urban, Lazar, & Sweeney, 2004). Reductions in response times have been theorized to represent global improvements in processing efficiency (Hale, 1990), and/or development of fluid intelligence (Fry & Hale, 1996). It was also observed that puberty explained decreases in response times independent of age. Sex differences in motor development has been observed in adolescence (Thomas & French, 1985). Changes in reaction times have been observed around the onset of puberty, although pubertal status was not directly assessed (McGivern, Andersen, Byrd, Mutter, & Reilly, 2002). In a study examining response times to emotional and neutral stimuli, it was found that mid/late pubertal adolescents exhibited faster response times than pre/early pubertal adolescents (Silk et al., 2009). Thus, the current findings are further evidence that puberty may contribute to observed reductions in response times in adolescence.

Improvements in post-error adjustments were also observed. Pubertal status and age were related to improvements in PES such that older/more advanced pubertal status was related more slowing after errors. The present findings are consistent with other studies using conflict-related tasks that have found an increase in PES throughout development (Jones et al., 2003; Santesso & Segalowitz, 2008; Schachar et al., 2004). However, developmental findings of PES in children and adolescents have been inconsistent, with many studies finding no change or a decrease in PES across development (Smulders et al., 2016). In the present study, PES was related to ERN, such that a larger ERN was related to more slowing after errors. Such findings are consistent with the conflict monitoring theory, which suggests that the ERN represents a signal to adjust behavior after an error, such as through PES

(Danielmeier & Ullsperger, 2011). Thus, according to this theory, both PES and the ERN are expected to increase together throughout development, which is consistent with the observed findings. In addition, no relation between the Pe and PES was observed. Findings of a relation between PES and the Pe are less consistent (Danielmeier & Ullsperger, 2011). In contrast to PES, among the whole sample, post-error improvements in accuracy (PIA) was not observed and was unrelated to age. A number of studies have similarly observed no differences in accuracy after an error and after a correct response (Endrass et al., 2005; Gehring & Fencsik, 2001; Gehring & Knight, 2000; Hajcak et al., 2003b; G. Hajcak & Simons, 2002; Scheffers et al., 1999). Taken together, developmental changes in PES were observed, and these changes were associated with developmental changes in the ERN, likely due to puberty.

Pubertal influences on the mesencephalic dopaminergic system may partly explain observed developmental changes in ERN in adolescence (Davies et al., 2001; Spear, 2000). The ERN has been hypothesized to be generated by a dip in dopamine in the basal ganglia (Frank et al., 2005; Holroyd & Coles, 2002). Mesencephalic dopamine neurons play an important role in reward processing and motivation (Fibiger & Phillips, 2011), and the initiation of goal-directed behavior (Frank, 2005; Kalivas et al., 1993). Dopamine receptors in the midbrain peak in early adolescence (Andersen et al., 2000; Gelbard et al., 1989; Teicher et al., 1995). Peak in dopamine receptors is accompanied by an increase in dopaminergic innervation of surviving neurons in these limbic regions during adolescence (Rosenberg & Lewis, 1995). An increasing rate of pruning of dopamine receptors in the basal ganglia from

adolescence to adulthood has also been observed (Seeman et al., 1987). Sex hormones, such as testosterone and estradiol initiate reorganization of brain structure in adolescence (A. S. Clark & Goldman-Rakic, 1989; McEwen, 2001). Thus, pubertal hormones may indirectly modulate the ERN via changes in dopaminergic enervation of neurons in the ACC.

It is important to note that age explained developmental changes in the ERN independent of puberty. This finding can partly be explained by the observed quadratic relation between the ERN and age (see figure 10). Large developmental decreases in the ERN were observed among the youngest participants, where little variation in pubertal status was observed. Thus, age was able to account for developmental changes prior to the emergence of physical characteristics of puberty. These findings suggest that developmental changes not associated with puberty partly explain the development of the ERN. However, it is important to note though that increases in pubertal hormones begin much earlier than the emergence of physical characteristics associated with puberty. In girls, the activation of the adrenal axis (i.e., adrenarche) begins between 6-9 years of age (Grumbach, 2002). However, adrenal hormones do not reach levels high enough to cause physical changes until a few years later (Dorn et al., 2006). Thus, self-report of physical characteristics associated puberty may not adequately capture these developmental changes in the ERN that occur before 9-10 years of age. Future studies should collect hormone concentrations to better understand the direct effect that hormones have on the ERN and ACC function.

It was observed that adolescents exhibited a larger ERN on congruent trials than on incongruent trials. Although intuitively, one would expect a larger ERN on incongruent trials since those trials consist of contradictory stimuli information and are the trials that typically elicit errors. However, the current findings of a larger ERN on congruent trials is in line with the conflict monitoring theory (Botvinick et al., 2001; Yeung, Botvinick, et al., 2004). According to the conflict monitoring theory, congruent trials are characterized by higher motor input for the correct response than incongruent correct trials since congruent trials are unambiguous and have no stimuli to indicate alternative responses. However, on congruent error trials, there must be extensive motor activity for the incorrect motor response to override the typical correct response motor activity. Thus, there is high conflict due to high activation of the correct and error motor response on congruent error trials. Empirical and simulation studies also support this theory (Scheffers & Coles, 2000; Yeung, Botvinick, et al., 2004),

6.2 Social Influences of Error Monitoring

The present study found that the ERN was enhanced in social contexts as compared to nonsocial contexts among all adolescents. In addition, it was observed that the degree to which adolescents reported more effort in the social condition was correlated with enhancements of the ERN in social contexts, suggesting that enhancements in the ERN in social contexts are in part due to increased social motivation. These findings are further evidence that the ERN is sensitive to motivational factors. The ERN is enhanced when errors are punished or correct

responses are rewarded (Hajcak et al., 2005; Riesel, Weinberg, et al., 2011). In addition, a number of studies have found that the ERN is particularly sensitive to social motivation, such as when errors are observed (Barker et al., 2015; Hajcak et al., 2005), or during social competition (Van Meel & Van Heijningen, 2010). In children, the ERN is also enhanced when errors are observed by a peer (Kim et al., 2005). In contrast, monetary incentives do not enhance the ERN among children (Torpey et al., 2009), suggesting that the ERN is particularly sensitive to social factors in childhood and adolescence. Indeed, adolescence is characterized by increased social motivation toward peers (Blakemore et al., 2010; Crone & Dahl, 2012; Dahl, 2004; Ernst et al., 2009; Nelson et al., 2005; Steinberg et al., 2006). Thus, the current findings suggest that the ERN is particularly sensitive to social contexts in adolescence.

It was also observed that the Pe was enhanced by social contexts. Findings of enhancements of the Pe in motivational contexts has rarely been examined. It has been observed that the Pe is enhanced when errors are observed as compared to errors committed alone (Hajcak et al., 2005). Barker and colleagues (2015) found that the Pe was influenced by social context among socially anxious individuals, although the follow-up *t*-tests did not reach significance. However, others have found the Pe is not influenced by social context (van Meel et al., 2012). In children, the Pe is not modulated by monetary incentives (Torpey et al., 2009). The present study is the first to examine social modulations of the Pe in a developmental sample, and found that the Pe was enhanced in social contexts. However, unlike the ERN, enhancements of the Pe in social contexts was not related to self-report of effort. These findings suggest that the mechanism in which the Pe and the ERN are enhanced in social

contexts is different. Theories of the Pe suggest that the Pe represents error awareness (Overbeek et al., 2005). Thus, it is possible that adolescents were more aware when they committed errors during social observation.

Enhancements of the CRN in social contexts was also observed. Some research has found that the CRN is enhanced by motivational factors (Judah et al., 2016; Riesel, Weinberg, et al., 2011; Schillinger et al., 2015; Van Meel & Van Heijningen, 2010). However, others have found the CRN is not influenced by motivation (Barker et al., 2015; Hajcak et al., 2005). The CRN is theorized to reflect general performance monitoring (Vidal et al., 2003, 2000). Thus, an enhanced CRN may reflect that the performance monitoring system is more engaged during social motivational contexts. Another possibility is that a subset of error trials are averaged into the ERN waveform due to stimulus-response mapping issues (Coles et al., 2001), such that these enhanced ERN trials are driving the CRN finding. Future research should further explore motivational influences of the CRN.

The present study found that pubertal status influenced the degree to which the ERN was enhanced in social contexts. However, contrary to the hypothesis, early pubertal adolescents (and also younger participants) demonstrated the greatest enhancements of the ERN in the social condition. Among later pubertal adolescents (and older adolescents), the ERN was less influenced by social contexts. One possibility for this unexpected result is that older adolescents were less likely to be deceived by the social manipulation. However, there were no differences in the ERN in social contexts between adolescents who were and were not deceived. Another possibility is that among the youngest adolescents, external motivation from the

social condition was a stronger motivator than internal motivation needed in the nonsocial condition. In the nonsocial condition, internal motivation to complete the task was necessary. In contrast, in the social condition, motivation was likely driven by trying to receive positive feedback from peers. Indeed, the early pubertal adolescents reported more effort in the social condition than late pubertal adolescents. However, this possibility still leaves the question: why are younger adolescents more externally motivated by social factors and less internally driven than older adolescents? The findings that early pubertal status/younger adolescents demonstrated that largest enhancement of the ERN point to the role of pubertal hormones in social motivation. Pubertal hormones have large effects on brain function and structure and are associated with changes in social motivation (Forbes & Dahl, 2010). However, increases in pubertal hormones occur much earlier than physical changes associated with these hormones, (Dorn et al., 2006; Shirtcliff et al., 2009), and changes in hormone concentrations have fast and substantial effects on brain development (McEwen, 2001). Interestingly, the present study, the largest changes in ERN amplitude occurred among the youngest adolescents.

The present study found that a reduced ERN in social contexts as compared to nonsocial contexts was related to adolescent self-report of depressive symptoms. This effect remained even when controlling for anxiety. Although as a whole, adolescents exhibited an enhanced ERN in social contexts, adolescents reporting higher depressive symptoms appeared to have less enhancements of the ERN in these contexts. Although no specific hypotheses were made regarding the ERN and depression, this findings is in line with recent literature that has examined the

association of the ERN and depression, which has found that depressed adolescents exhibit a *reduced* ERN (Ladouceur et al., 2012; Weinberg, Meyer, Hale -Rude, et al., 2016). However, there have been mixed findings in adults (Holmes & Pizzagalli, 2008, 2010; Olvet et al., 2010), which may be due to opposing effects of anxiety on the ERN (Weinberg, Klein, et al., 2012). In the present study, depressive symptoms were strongly related to the ERN in social contexts but unrelated in nonsocial contexts. This finding suggest that social factors do not influence the ERN the same way in depressed individuals. One characteristic of depression is a reduced pleasure from previously rewarding stimuli and a lack of social motivation (Brown, Silvia, Myin-Germeys, & Kwapil, 2007). The ERN is highly sensitive to social motivational factors (Barker et al., 2015), and has been suggested to index a reward signal (Holroyd & Coles, 2002). Thus, the ERN in social contexts may index the degree that adolescents were motivated and rewarded by social stimuli.

Large changes in behavioral performance were observed when adolescents completed the flanker in social contexts. Adolescents reported trying harder in the social condition as compared to the nonsocial condition. Such increased effort was reflected in the fact that adolescents had faster response times and improved accuracy in the social condition as compared to the nonsocial condition. The current findings of improved accuracy in front of peers is consistent with the social facilitation drive theory, which postulates that the presence of others improves performance due to increased motivation (Zajonc, 1965). The mere presence of others increases physiological arousal (Cacioppo, Rourke, Marshall-Goodell, Tassinary, & Baron, 1990), which might partly be due to increased uncertainty about the social

environment (Zajonc & Paulus, 1980). Making mistakes in front of others is distressing (Hewitt et al., 2003; Leary & Kowalski, 1997; Schlenker & Leary, 1982), particularly for adolescents (La Greca & Lopez, 1998). Thus, the current observation of behavioral changes during social observation is consistent with previous theories of social facilitation drive.

It was observed that social anxiety was associated with increased post-error slowing (PES) in social contexts. It is theorized that PES reflects behavioral control following errors in order to prevent future mistakes (Danielmeier & Ullsperger, 2011). Thus, increased PES in socially anxious individuals may be due to a greater effort to avoid committing mistakes. Indeed, social anxiety is characterized by fear of performance mistakes during social observation (Rapee & Heimberg, 1997; Rapee & Spence, 2004). Interestingly, greater PES did not improve performance. Both high and low socially anxious adolescents were more accurate in social contexts. However, low socially anxious adolescents did not exhibit increased PES in social context, suggesting that PES was not associated with increased accuracy. An alternative theory, the orienting account, suggest that PES is not a beneficial mechanism to improve performance, but is rather the result of increasing attention to rare events (Notebaert et al., 2009). Thus, PES could be detrimental or beneficial to performance depending on context (Castellar et al., 2010). In the current study, PES among socially anxious adolescents may reflect greater orienting to error events.

Social anxiety was also associated with changes in reaction times between social and nonsocial contexts. The present study found that low socially anxious adolescents exhibited reductions in response times in social contexts whereas high

socially anxious adolescents exhibited no differences between contexts. Such discrepancies between high and low socially anxious adolescents could be explained by how individual differences in evaluation apprehension differentially modulate behavioral performance during social observation. The influence of anxiety on social facilitation of behavior was first postulated by (Cottrell, 1972), who suggested that changes in drive and motivation would only occur in the presence of others if individuals were concerned about social evaluation (i.e. evaluation apprehension). Concern about social evaluation is one of the defining features of social anxiety (Watson & Friend, 1969). Thus, it is likely that in social contexts, socially anxious adolescents are more careful of preserving performance accuracy (via slower response times), whereas less socially anxious adolescents exhibited social facilitation of performance with improved response times. Interestingly, among high socially anxious adolescents, preserved response times in social contexts did not improve accuracy.

6.3 Moderating role of puberty

The third main aim of the present study was to explore whether pubertal status moderated the relation between error monitoring and social anxiety. Contrary to the hypothesis of aim 3, pubertal status, nor age, moderated the association between an enhanced ERN in social contexts and social anxiety. Second, across the entire sample, enhancements of the ERN in social contexts was unrelated to social anxiety symptoms. Taken together, these findings suggest that the ERN, regardless of social context, was unrelated to individual differences in general anxiety or social anxiety.

Previous research has found that the ERN is elevated among individuals with an anxiety disorder (Carrasco, Hong, et al., 2013; Endrass et al., 2014; Gehring et al., 2000; Ladouceur et al., 2006; Weinberg et al., 2010). The ERN is also related to individual differences in dimensional anxiety symptoms in adults (Hajcak et al., 2003a; Moser et al., 2012). A number of studies have found the ERN is also heightened in children and adolescents with an anxiety disorder (Ladouceur et al., 2006; A. Meyer, Hajcak, et al., 2013). However, in children, no relation between the ERN and dimensional aspects of anxiety have been observed. (Meyer, Hajcak, et al., 2013; A. Meyer et al., 2015; Torpey et al., 2013), suggesting that the ERN is not related to normative levels of anxiety in childhood. It has been found that age moderates the association between the ERN and dimensional aspects of anxiety such that this relation emerges in adolescence (Meyer et al., 2012). Contrary to the findings of Meyer et al. (2012), the present findings did not find age or pubertal status moderated the association between the ERN and anxiety.

Pubertal status was found to moderate the association between enhancements of the Pe in the social condition from the nonsocial condition (social effect Pe) and social anxiety symptoms. Specifically, there was an association between the social effect Pe and social anxiety only among later pubertal adolescents. This effect was still present when controlling for chronological age, further supporting the idea that this association is specially moderated by puberty. Studies of the association between the Pe and anxiety have been mixed (Overbeek et al., 2005). A number of studies have found that the Pe was not related to anxiety in adults (Endrass et al., 2010; Ruchow et al., 2005) or children (Hajcak, Franklin, Foa, & Simons, 2008; Ladouceur

et al., 2006). However, Wienberg and colleagues (2012) found a larger Pe among adults with GAD. One likely issue in these mixed findings is differences in the measurement of the Pe, since the Pe overlaps with a more anterior positivity related to the ERN (Overbeek et al., 2005). However, it has been found that the Pe is influenced by social contexts (Hajcak et al., 2005). In addition, there is some evidence that socially anxious adults have an enhanced Pe in social contexts (Barker et al., 2015). Theories of the Pe suggest that the Pe represents the motivational salience of an error (Overbeek et al., 2005). Thus, enhancements of the Pe among socially anxious individuals may reflect enhanced salience of errors in social contexts. The present results also found that pubertal status remained a significant moderator even after controlling for age. Advancing pubertal status predicts the emergence of social anxiety symptoms (Deardorff et al., 2007), and internalizing symptoms (Angold, Costello, & Worthman, 1998; Adrian Angold et al., 1999). These findings suggest that enhancements of the Pe in social contexts among socially anxious individuals emerges in adolescence as a result of pubertal development.

6.4 The ERN and Pe as Biomarkers for Anxiety and Depression

Findings from the current study suggest that the ERN and Pe in social contexts could serve as biological endophenotype for affective and anxiety disorders. One possible mechanism to aid in the search of genetic liability and disease, known as an endophenotype, is an intermediary state between genetic risk and a disease state (Gottesman & Gould, 2003). The ERN is one possible endophenotype that could be utilized to understand the development of anxiety disorders in typical and atypical

developmental populations (Hajcak, 2012). Gottesman & Gould (2003) defined a number of requirements for a biological marker to be considered an endophenotype. First, the endophenotype must be associated with the disease. The first requirement has been consistently demonstrated for the ERN, where individuals with anxiety disorders demonstrate an elevated ERN (see Moser et al., 2013 for review). Second, an endophenotype should be state independent. There is some evidence in the literature that the ERN is state independent. Moser et al., (2005) measured the ERN in spider phobics during symptom provocation and found the ERN was unaltered in magnitude as compared to baseline levels. In addition, inducing negative affect also has no effect in changing the magnitude of the ERN (Larson, Perlstein, Stigge-Kaufman, Kelly, & Dotson, 2006). Furthermore, successful treatment of OCD symptoms has no effect in altering ERN magnitude (Hajcak, Franklin, Foa, & Simons, 2008). Thus, current findings suggest that the ERN is invariable to alterations in state anxiety and affect. However, it is interesting to note that the ERN is sensitive to motivation (Hajcak et al., 2005).

Addition requirements of an endophenotype as postulated by Gottesman & Gould (2003) include that the endophenotype must be in part heritable, and must be present in both individuals with the disorder as well as non-affected family members of the affected individual. To examine the heritability of common response monitoring components such as the ERN, Anokhin, and colleagues (2008) measured the ERN in monozygotic and dizygotic twins. The authors found that 47% of the variability in the ERN was due to genetic factors (the Pe and CRN also demonstrated similarly high estimates). To examine altered error monitoring in non-affected

relatives of individuals with an anxiety disorder, Riesel and colleagues (2011) compared the ERN in adults with OCD, unaffected first-degree relatives, and healthy controls. The authors found that the ERN was elevated in both individuals with OCD and their first-degree relatives, suggesting that an enhanced ERN is present regardless of presence of clinical symptoms. An enhanced ERN has also been observed in siblings of children with OCD, further implicating an enhanced ERN early in life as a biological endophenotype of anxiety (Carrasco, Harbin, et al., 2013). Overall, these findings suggest that variability in the magnitude of the ERN is largely heritable.

6.5 Limitations and Future Directions

There are a number of limitations in the present study that should be addressed. First, it should be noted that due to the high correlation between age and puberty, it was difficult to determine if age or puberty was the driving factor for many of the developmental effects observed. The issue of disentangling the effects of age and puberty is common in adolescent research (Steinberg, 1987; Steinberg & Monahan, 2007). Examination of puberty can typically be discussed as two constructs (Dorn et al., 2006). Pubertal status is defined as an adolescent's current level of progression through puberty based on physical changes associated with puberty. In contrast, pubertal timing is defined as the degree of pubertal development as compared to same-aged peers. Although subtle, this distinction has been noted in the study of the emergence of anxiety and depressive disorders in adolescence (Angold et al., 1998; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Reardon et al., 2009). One suggestion for examining pubertal status, as utilized in the present analysis, is to

examine both variables in a statistical model and to conclude that age and puberty have overlapping effects if neither predictor reaches significance but the overall model reaches significance (Steinberg, 1987). If puberty remains significant in the model when controlling for age, then the conclusion is that puberty is a predictor beyond the overlapping association with age. However, it is important to note that when controlling for age, the measurement of puberty becomes a measure of pubertal timing (Dorn et al., 2006). This analysis method is particularly effective when utilizing large samples (Martin et al., 2002; Steinberg & Monahan, 2007). Another method is to recruit participants who are within a narrow age window (e.g., 12 year-olds), thereby eliminating the correlation between age and puberty. One limitation of this design is that it is often difficult to recruit a large enough sample to provide adequate variability of puberty status, particularly at the extremes of the distribution. Another limitation, which is theoretical in nature, is that the question is one of pubertal timing, as opposed to pubertal status, since all adolescents are at the same age.

Another limitation is that no information on psychiatric status was collected. Thus, it is unknown if the ERN and the Pe were specifically related to anxiety and depressive disorders rather than dimensional aspects of anxiety and depression. Such a distinction is important to make since anxiety disorders are related to an enhanced ERN in childhood and adolescence but is not related to individual differences in anxiety in childhood (A. Meyer, Hajcak, et al., 2013; A. Meyer et al., 2015). The present study was an unselected sample of adolescent girls, which represents a typical distribution of dimensional aspects of anxiety. Thus, in order to better understand the

relation between social anxiety and the ERN, future studies should prescreen participants with high and low levels of social anxiety (Barker et al., 2015), or recruit participants with an existing anxiety disorder (Ladouceur et al., 2006).

It should be noted that a number of participants reported that they were not deceived by the social deception. Although identical findings were observed when adolescents who were not deceived were excluded from analysis, there may be a subset of adolescents who may have not believed the social manipulation but did not report it during debriefing. Thus, the present results may underestimate the degree to which the ERN was enhanced by social factors since the social manipulation did not include actual adolescents present. Future studies could have actual adolescents observe and evaluate the performance of another adolescent (Barker et al., 2015; Kim et al., 2005). In addition, it is important to note that social observation was inextricably linked with social feedback during the social condition. The manipulation was somewhat different than other studies that have explored social effects on the ERN, which have primarily used a passive observer who does not give feedback (Barker et al., 2015; Hajcak et al., 2005; Kim et al., 2005). The current manipulation was chosen because it allowed for the exploration of social influences on the ERN without having an actual adolescent present. Providing feedback between blocks also allowed a natural reminder that the adolescents were being observed and evaluated. Thus, the current social manipulation provided a standardized way to assess social influences of the ERN. However, it should be noted that social feedback was inextricably linked to the social context, making it difficult to separate the effects of passive observation and performance feedback on adolescent performance.

It is likely that the feedback given during breaks in the current study may have had different effects on the ERN between the social and the nonsocial condition. The ERN has been shown to be sensitive to feedback (Gehring et al., 1993) such that the ERN is larger when accuracy is emphasized and smaller when speed is emphasized. Thus, it is likely that in the current study, the ERN was larger on blocks following feedback emphasizing accuracy and smaller on blocks following feedback emphasizing speed. However, comparison of the ERN based on feedback type was not possible since not enough errors were committed in each feedback type. Another issue in the current study is that adolescents may have responded differently to feedback types between conditions. Indeed, it was observed that adolescents were more accurate and responded faster in the social condition than the nonsocial condition. This finding is contrary to speed-accuracy tradeoff typically observed (D. E. Meyer et al., 1990; Wickelgren, 1977), such that increased accuracy is typically coupled with slower response times. In addition, social observation of performance is typically associated with increased accuracy and slower response times (Robert Boleslaw Zajonc & others, 1965). One likely explanation for these findings is that when adolescents exhibited improved accuracy on a block of trials in the social condition, they would then receive feedback to emphasize speed (i.e., if accuracy was above 90%, adolescents were told to respond faster). Thus, it is likely that adolescents were attempting to exhibit a high level of accuracy due to the general social context, while at the same time trying to respond faster based on the social feedback delivered. Interestingly, the relation between speed and accuracy was different for high anxious adolescents such that high socially anxious adolescents did not exhibit faster response

times in the social condition, perhaps in order to better preserve accuracy. Future studies should separate social feedback and social context in order to better understand how the ERN is influenced by these separate constructs.

Another limitation in the present study is that only adolescent females participated, making it unknown whether the present findings generalize to adolescent males. A number of differences have been observed between the ERN and anxiety between sexes. Females exhibit a smaller ERN relative to males (Larson et al., 2011), particularly after puberty (Davies et al., 2004). The developmental trajectory of the ERN appears to be different between sexes, such that adolescent girls exhibit changes in the ERN earlier than boys (Davies et al., 2004). There is also evidence that the Pe is smaller in females (Larson et al., 2011). In addition, the association between the ERN and dimensional aspects of anxiety appears to be specific to females (Moran et al., 2012; Moser et al., 2016). Females are also more at risk for anxiety and depressive disorders (Kessler, 2005), and pubertal development is related to social anxiety only among adolescent girls (Deardorff et al., 2007). Taken together, these findings suggest that the ERN, and possibly the Pe, are distinctly different in females, and enhancements of the ERN and Pe may be a risk factor for internalizing disorders only for females. Future research should examine the ERN and Pe and the relation with anxiety in adolescent males.

6.6 Conclusion

Adolescence is a period in development characterized by dramatic changes, and is a period of risk for psychopathology (Blakemore et al., 2010; Crone & Dahl,

2012). Puberty plays a major role in the emergence of anxiety disorders, particularly among girls (Angold et al., 1998; Adrian Angold et al., 1999; Deardorff et al., 2007). One of the most commonly diagnosed anxiety disorders, social anxiety disorder (SAD), is a debilitating disorder which typically emerges in early adolescence and involves excessive fear in social-evaluative situations (American Psychiatric Association, 2013). Adolescent girls demonstrate higher rates of social anxiety symptoms than boys (Kessler, 2005; Wittchen et al., 1999), particularly after puberty (Deardorff et al., 2007), suggesting that neurodevelopmental changes associated with puberty may account for a shifting risk for SAD in girls. Thus, it is critical to identify early biomarkers of risk for SAD in adolescent girls.

One potential biomarker for anxiety disorders, the ERN, is a negative deflection in the event-related potential following an erroneous response (Gehring et al., 1993). Individuals with an anxiety disorder demonstrate a greater ERN than healthy comparisons (Carrasco, Harbin, et al., 2013; Gehring et al., 2000; Ladouceur et al., 2006; Weinberg et al., 2010), an association which arises in early adolescence (A. Meyer et al., 2012). Less is known about the Pe, a later positive component related to error awareness (Falkenstein et al., 1991). Little research has examined the relation between the ERN/Pe and social anxiety symptoms. Social anxiety symptoms are significantly related to the degree to which the ERN and the Pe are elevated in social-evaluative contexts (Barker et al., 2015). However, little is known how pubertal development modulates the ERN and Pe in socially motivating contexts and whether the degree of modulation is associated with social anxiety symptoms.

In the present study it was observed that pubertal development was related to the ERN, such that the later pubertal adolescents exhibited a larger ERN than earlier pubertal adolescents. In addition, chronological age predicted changes in the ERN in adolescence above the associated variance with puberty. In contrast, the Pe exhibited no changes across adolescence and was unrelated to age and puberty. In addition, it was observed that pubertal development influenced the degree that the ERN was enhanced in social contexts from nonsocial contrasts. The largest enhancement of the ERN in social contexts was observed among early pubertal adolescents. In contrast, the Pe was enhanced in social contexts regardless of pubertal status.

The present study also explored the relation between the ERN, Pe and internalizing symptoms. Enhancements of the ERN in social contexts was unrelated to social anxiety symptoms. However, a reduced ERN in social contexts was related to depressive symptoms, such that a smaller ERN in social contexts was related to more depressive symptoms. In addition, it was also explored whether pubertal status moderated the associations between the ERN/Pe and social anxiety. Puberty did not moderate the association between enhancements of the ERN in social contexts and social anxiety. However, pubertal status moderated the relation between enhancements of the Pe in social contexts and social anxiety symptoms, such that a larger Pe in social contexts was related to more anxiety symptoms only among later pubertal adolescents. These findings further suggest that the ERN and Pe may be utilized as biomarkers for affective disorders such as anxiety and depression.

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