Title
Social influences of error monitoring in adolescent girls

Permalink
https://escholarship.org/uc/item/4vm4p95r

Journal
Psychophysiology, 55(9)

ISSN
0048-5772

Authors
Barker, Tyson V
Troller-Renfree, Sonya V
Bowman, Lindsay C
et al.

Publication Date
2018-09-01

DOI
10.1111/psyp.13089

Peer reviewed

Social Influences of Error Monitoring in Adolescent Girls

Tyson V. Barker[1], **Sonya V. Troller-Renfree**[2], **Lindsay C. Bowman**[3], **Daniel S. Pine**[4], and **Nathan A. Fox**[2]

[1]Prevention Science Institute, University of Oregon, Eugene, Oregon 97403

[2]Department of Human Development and Quantitative Methodology, University of Maryland, College Park, MD 20742

[3]Department of Psychology, University of California, Davis, CA, 95616

[4]Section on Development and Affective Neuroscience, National Institute of Mental Health, Bethesda, MD 20895

Abstract

Adolescence is a developmental period characterized by increased social motivation and a heightened concern of peer evaluation. However, little research has examined social influences on neural functioning in adolescence. One psychophysiological measure of motivation, the error-related negativity (ERN), is an event-related potential following an error. In adults, the ERN is enhanced by contextual factors that influence motivation, such as social observation and evaluation. The current study examined relations among age and neural responses in social contexts in adolescence. Seventy-Six adolescent girls (9-17 years old) completed a flanker task under two different conditions. In the social condition, adolescent girls were informed that two other adolescents would be observing and providing feedback about their performance. In the nonsocial condition, adolescent girls completed a flanker task alone and were told feedback was computer generated. Results revealed that younger adolescents exhibited a larger ERN in social contexts than nonsocial contexts. In contrast, there were no differences in the ERN between contexts among older adolescents. In addition, enhancements of the ERN in social contexts among younger adolescents diminished the relation between the ERN and age. These findings suggest that the ERN is sensitive to social contexts in early adolescence and developmental changes in the ERN may be partially explained by contextual factors that influence motivation.

1. INTRODUCTION

Adolescence signals many changes including increasing social motivation (Crone & Dahl, 2012; Dahl, 2004; Shulman et al., 2016), and a heightened concern of social evaluation (Muris, Merckelbach, Meesters, & Brand, 2002; Parker, Rubin, Erath, Wojslawowicz, & Buskirk, 2015; Rubin, Bukowski, & Parker, 2007), particularly for adolescent girls (La Greca & Lopez, 1998). Such changes in social motivation may reflect increased neural

sensitivity to social contexts (Crone & Dahl, 2012; Nelson, Leibenluft, McClure, & Pine, 2005; Steinberg et al., 2008). However, more research is needed to directly examine neurodevelopmental changes in the sensitivity to social contexts across adolescence. The current study examines developmental changes in neural responses to errors during social and nonsocial contexts in adolescent girls.

1.1 Social motivation and error monitoring

One promising psychophysiological measure of the motivation, the error-related negativity (ERN), is an event-related potential (ERP) waveform following an erroneous motor response (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN is likely generated by a neural network including the anterior cingulate cortex (ACC; Dehaene, Posner, & Tucker, 1994; Holroyd, Dien, & Coles, 1998; Van Veen & Carter, 2002), as well as the posterior cingulate cortex (PCC), insula, and orbitofrontal cortex (Agam et al., 2011; Buzzell, Richards, et al., 2017; Liu, Hanna, Carrasco, Gehring, & Fitzgerald, 2013; Taylor, Stern, & Gehring, 2007; Westlye et al., 2010). Major theories view the ERN as an evaluative signal reflecting the motivational significance of an error (Pailing & Segalowitz, 2004b; Proudfit, Inzlicht, & Mennin, 2013; Weinberg et al., 2016; Weinberg, Riesel, & Hajcak, 2012). Alternative accounts suggest that the ERN serves as reinforcement signal for learning (Holroyd & Coles, 2002), detection of response conflict (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Carter & Veen, 2007; Yeung, Botvinick, & Cohen, 2004), or a general response detection mechanism (Coles, Scheffers, & Fournier, 1995; Gehring et al., 1993). Importantly, functional theories of the ERN support the notion that neural regions responsible for the generation of the ERN, such as the ACC, guide motivation and goal-directed behavior (Holroyd & Yeung, 2012; Segalowitz & Dywan, 2009).

It has been consistently observed that the magnitude of the ERN is influenced by the motivational significance of error commission (Boksem, Meijman, & Lorist, 2006; Boksem, Tops, Wester, Meijman, & Lorist, 2006; Pailing & Segalowitz, 2004b). In adults, the ERN is enhanced by nonsocial factors that increase error significance, such as when correct responses are rewarded (Hajcak, Moser, Yeung, & Simons, 2005; Stürmer, Nigbur, Schacht, & Sommer, 2011), when errors are punished (Potts, 2011; Riesel, Weinberg, Endrass, Kathmann, & Hajcak, 2012), or when accuracy is emphasized over speed (Gehring et al., 1993). Theoretical accounts for motivational influences on the ERN suggest that such enhancements of the ERN reflect increased motivation to the error in relation to current contextual demands (Weinberg et al., 2016). Indeed, other psychophysiological measures of motivation, such as startle potentiation, pupil dilation, and skin conductance, are also increased following error commission (Critchley, Tang, Glaser, Butterworth, & Dolan, 2005; Hajcak & Foti, 2008; Hajcak, McDonald, & Simons, 2003b, 2004; Riesel et al., 2012). Critically, these psychophysiological measures are also sensitive to contextual factors (Bradley, Zlatar, & Lang, 2018; Chiew & Braver, 2013), further emphasizing the role of motivation in influencing ERN magnitude.

The ERN is also influenced by social factors. The presence of social partners increases the perceived value of behavior (Geen, 1991, 1995), which increases arousal and evaluation

apprehension (Geen & Bushman, 1989; Zajonc & Paulus, 1980; Zajonc, 1965). Biopsychosocial models of social motivation suggest that social observation and evaluation increase physiological arousal, reflecting the mobilization of motivational systems towards the perceived significance of social contexts (Blascovich, Mendes, Hunter, & Salomon, 1999; Blascovich & Tomaka, 1996). In line with biopsychosocial theories of social motivation, the ERN is enhanced by social observation and evaluation (Barker, Troller-Renfree, Pine, & Fox, 2015; Hajcak et al., 2005; Masaki, Maruo, Meyer, & Hajcak, 2017; Schillinger, Smedt, & Grabner, 2015; Van Meel & Van Heijningen, 2010). In children and adolescents, social factors enhance the ERN (Buzzell, Troller-Renfree, et al., 2017; Kim, Iwaki, Uno, & Fujita, 2005), whereas nonsocial factors (i.e., monetary rewards) appear to not influence the ERN (Maruo, Sommer, & Masaki, 2017; Torpey, Hajcak, & Klein, 2009). However, children with attention deficit hyperactivity disorder (ADHD), a disorder in part characterized by altered motivational systems (Haenlein & Caul, 1987; Sergeant, 2000; Tripp & Wickens, 2008), display an increased ERN only when performance is extrinsically rewarded (Groom et al., 2013), suggesting the importance of within-individual valuation of context in influencing ERN magnitude in development.

Although there is strong evidence that the ERN is sensitive to social factors in adults, little research has examined whether the ERN is also influenced by social contexts in adolescence, a developmental period characterized by heightened social motivation and social evaluation (Crone & Dahl, 2012). We recently found that the ERN was enhanced during social contexts as compared to nonsocial contexts in middle adolescence (Buzzell, Troller-Renfree, et al., 2017). However, the age range of the sample was relatively restricted, limiting the ability to examine whether the degree of ERN enhancement in social contexts changes across adolescence. Theoretical accounts of adolescent neurobiology suggest that the sensitivity of neural regions associated with social motivation increase in early adolescence (Casey, Jones, & Hare, 2008; Shulman et al., 2016; Steinberg et al., 2008). Furthermore, mesencephalic dopamine (DA) neurons, which are proposed to play a key role in the generation of the ERN (Frank, Woroch, & Curran, 2005; Holroyd & Coles, 2002), reach peak density in early adolescence (Andersen, Thompson, Rutstein, Hostetter, & Teicher, 2000), and are theorized to contribute to changes in adolescent social motivation (Ernst, Romeo, & Andersen, 2009). Lastly, large increases in ERN magnitude have been observed in early adolescence, particularly among females (Davies, Segalowitz, & Gavin, 2004). Given that the magnitude of the ERN is theorized to reflect the interaction between within-individual and contextual factors (Riesel et al., 2012; Weinberg et al., 2016), it is likely that younger adolescents may exhibit a greater enhancement of the ERN in social contexts as compared to older adolescents. However, this has yet to be examined.

Two additional event-related potentials are related to the error monitoring system. The correct-related negativity (CRN) is a small negativity observed on correct trials, which has a similar topography as the ERN (Davies et al., 2004; Gentsch, Ullsperger, & Ullsperger, 2009; Roger, Bénar, Vidal, Hasbroucq, & Burle, 2010; Wessel & Ullsperger, 2011). Functional theories of the CRN suggest that the CRN may reflect a general response monitoring mechanism (Falkenstein et al., 2001; Vidal, Burle, Bonnet, Grapperon, & Hasbroucq, 2003; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000), or artifactual contamination of error trials in the CRN waveform due to stimulus-response mapping

uncertainty (Coles, Scheffers, & Holroyd, 2001; Pailing & Segalowitz, 2004a; Scheffers & Coles, 2000). The influence of motivation on the CRN are mixed (Barker et al., 2015; Hajcak et al., 2005; Kim et al., 2005), although there is evidence that the CRN is influenced by individual differences in personality factors (Hajcak et al., 2004; Hajcak & Simons, 2002; Moser, Moran, Schroder, Donnellan, & Yeung, 2013).

The last component of interest, the error positivity (Pe) is a positive deflection occurring approximately 200-300 ms after an error (Falkenstein et al., 1991). The Pe is thought to reflect error awareness (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001), and the motivational significance of an error (Hajcak et al., 2004; Overbeek, Nieuwenhuis, & Richard, 2005). There is evidence that the Pe is modulated by motivational factors (Schroder, Moran, Moser, & Altmann, 2012). In contrast to the ERN, the Pe has been found to directly relate to other physiological measures of motivation and arousal following error commission (Elkins-Brown, Saunders, & Inzlicht, 2016; Hajcak et al., 2003b).

1.2 Development of the error monitoring system

Studies of the development of the error monitoring system suggest that the ERN is relatively stable in childhood (Davies et al., 2004; Grammer, Carrasco, Gehring, & Morrison, 2014; Richardson, Anderson, Reid, & Fox, 2011), with notable increases in magnitude occurring in adolescence (Davies et al., 2004; Ladouceur, Dahl, & Carter, 2004; Santesso & Segalowitz, 2008; Santesso, Segalowitz, & Schmidt, 2006). In contrast, the CRN and Pe exhibit a different developmental pattern than the ERN (Davies et al., 2004; Grammer et al., 2014; Santesso et al., 2006; Wiersema, van der Meere, & Roeyers, 2007). Findings for the CRN typically find no developmental differences (Hogan, Vargha-Khadem, Kirkham, & Baldeweg, 2005; Santesso & Segalowitz, 2008; Santesso et al., 2006; although see Davies et al., 2004). Similarly, the Pe is invariant throughout adolescence (Davies et al., 2004; Ladouceur, Dahl, & Carter, 2007; Santesso et al., 2006).

It has been suggested that developmental increases in the ERN during adolescence reflect the structural development of neural regions supporting the generation of the ERN (Tamnes et al., 2010; Tamnes, Walhovd, Torstveit, Sells, & Fjell, 2013; Westlye et al., 2010). However, neural regions that support the generation of the ERN, such as the ACC and PCC (Buzzell, Richards, et al., 2017), are highly influenced by contextual factors, particularly in adolescence (Casey et al., 2008; Crone & Dahl, 2012; Ernst, Pine, & Hardin, 2006; Shulman et al., 2016). As such, little research has examined whether contextual factors influence developmental patterns of the ERN. Most studies finding developmental increases in ERN magnitude in adolescence have elicited the ERN in relatively low motivational contexts (Davies et al., 2004; Ladouceur et al., 2007; Santesso & Segalowitz, 2008; Santesso et al., 2006). Given that contextual factors that influence motivation can increase ERN magnitude (Barker et al., 2015; Boksem, Meijman, et al., 2006; Hajcak et al., 2005), it is possible that some of the observed increases in ERN magnitude in adolescence are due to contextual factors that influence motivation. However, this has yet to be examined.

1.3 The present study

This study examines developmental differences in neural indices of the error monitoring system in social and nonsocial contexts during adolescence. To examine social influences on the ERN, we developed a novel experimental paradigm where adolescent girls were led to believe that either two other adolescents (i.e., social condition) or a computer (i.e., nonsocial condition) would be observing and providing feedback on their performance (Buzzell, Troller-Renfree, et al., 2017). Seventy-six adolescent girls (9-17 years of age) completed a flanker task (Eriksen & Eriksen, 1974) in two contexts, alone (nonsocial condition) and during peer evaluation (social condition). Adolescent girls also provided information on their degree of effort in each condition as an index of motivation in social and nonsocial contexts.

We hypothesized that a social context, relative to nonsocial context, would increase the ERN in adolescent girls based on previous findings in adult and developmental populations (Barker et al., 2015; Buzzell, Troller-Renfree, et al., 2017; Hajcak et al., 2005; Kim et al., 2005), In addition, we hypothesized that age would moderate the degree to which the ERN was enhanced in social contexts, based on theoretical models of social motivation in adolescence (Casey et al., 2008; Ernst et al., 2009; Shulman et al., 2016). Specifically, we hypothesized that younger adolescent girls would exhibit a greater ERN enhancement from social to nonsocial contexts as compared to older adolescent girls. For the Pe, we similarly hypothesized that the Pe would be enhanced in social contexts as compared to nonsocial contexts. However, we did not expect age to moderate the degree to which the Pe was enhanced in social contexts based on findings that the Pe is not influenced by age (Davies et al., 2004; Ladouceur et al., 2007; Santesso et al., 2006).

We also explored how social contexts influence developmental patterns of the ERN. We hypothesized that in the nonsocial condition, increasing age would be associated with a larger ERN in adolescence, based on previous reports (Davies et al., 2004; Santesso & Segalowitz, 2008; Santesso et al., 2006). In contrast, we hypothesized that social contexts would significantly alter the typically observed association between the ERN and age. Specifically, we hypothesized that greater enhancements of the ERN in social contexts among younger adolescents would significantly diminish the typically observed relation between ERN and age.

2. METHOD

2.1 Participants

Participants were 76 adolescent girls (M_{age} = 11.87 years; SD = 2.18 years; range = 8.67 - 17.14 years). This study was part of a larger project examining social and cognitive development during adolescence. 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.

Participants were excluded if they reported that they were not deceived by the social manipulation ($n = 4$). One participant did not complete the nonsocial condition and three participants did not complete the social condition due to fatigue. Of the remaining participants, none performed below the 60% *a priori* accuracy threshold in either condition. Thus, behavioral analyses included 68 participants. For EEG analysis, six additional subjects were excluded if either or both the social and nonsocial condition contained fewer than 6 artifact-free error trials (Olvet & Hajcak, 2009). Thus, the final sample for electroencephalogram (EEG) analyses was 62 participants.

2.2 Experimental design

2.2.1 Nonsocial 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 was presented randomly. All stimuli were presented for 200 ms with an intertrial interval (ITI) that varied randomly 800-1200 ms following the response. Prior to beginning the task, participants were instructed to press a button depending on the direction of the middle arrow and then completed a practice block of 16 trials. Next, adolescents completed the actual task, which consisted of 10 blocks of 32 trials (320 trials total). After each block, adolescents were told that they would receive a short break and computer-based feedback about their performance, and were instructed to change their performance based on the feedback (see Figure 1; Weinberg, Olvet, & Hajcak, 2010). If performance accuracy was below 75 %, adolescents received a simple statement in white text indicating to increase accuracy (i.e., "Be more accurate"). If performance accuracy was above 90 %, adolescents received a statement indicating to increase response speed (i.e., "Respond faster"). If performance accuracy was between 75 % and 90 %, adolescents received a statement indicating to keep their performance at a similar level (i.e., "Good job").

2.2.2 Social flanker task—

In the social condition, adolescents completed an identical version of the flanker task, where feedback was presented at the same interval using the same accuracy parameters as the nonsocial flanker task, but adolescents were made to believe that the feedback between blocks was given by two peers who were evaluating their performance. The feedback text appeared to be typed through a chat room and consisted of phrases indicative of the language adolescents use in text/online conversations with peers. Social feedback was accompanied by images (i.e., emoji/emoticons) that were unique to each feedback type (see Figure 1). If performance accuracy was below 75 %, adolescents received emoji/emoticons and a statement indicating to be more accurate (e.g., "You made too many mistakes!"). If performance accuracy was above 90 %, adolescents received emoji/emoticons and a statement indicating to respond faster (e.g., "Go Faster!"). If performance accuracy was between 75 % and 90 %, adolescents received emoji/emoticons and a statement indicating to keep their performance at a similar level (e.g., "You did great!").

2.2.3 Self-report of motivation—After completing both conditions, adolescents reported their perceived effort for each condition on a Likert scale from 1 (*did not try at all*) to 10 (*tried really hard*).

2.3 Procedure

At the beginning of the experiment, adolescents and parents were informed of 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. Next, adolescents were fitted with the EEG net 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. 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. For participants who completed the social condition first, during the nonsocial condition, it was emphasized that no one was watching their performance, and the webcam was removed. Following the procedure, adolescents were administered a debriefing questionnaire which included questions to check deception of the social manipulation, and self-report of motivation in each condition. Of the adolescents who completed both the social and nonsocial condition, 96 % reported that they were deceived by the social manipulation. Similar results were obtained when including adolescents who reported they were not deceived by the social manipulation in analyses.

2.4 EEG collection 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 kΩ. 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. Reponses-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 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 9.29 % of trials; nonsocial condition: 2.16 % of trials). Bad channels on remaining good trials were replaced using spherical spline interpolation (Perrin, Pernier, Bertnard, Giard, & Echallier, 1987; Perrin, Pernier, Bertrand, & Echallier, 1989). 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: $M = 36.23$, $SD = 16.77$, social: $M = 34.94$, $SD = 16.34$), $t(61) = 0.75$, $p = .46$), or the number of artifact-free correct trials between conditions (nonsocial condition: $M =$

240.60, SD = 50.91, social condition: M = 245.73, SD = 47.43, $t(61)$ = 1.08, p = .28). In addition, in the nonsocial condition, the number of artifact-free error trials was negatively correlated with accuracy, $r(60)$ = −.81, p < .001, such that better accuracy was related to fewer artifact-free error trials. The correlation between artifact-free error trials and accuracy was also observed in the social condition, $r(60)$ = −.83, p < .001.

The correlation between age and the number of error trials averaged into the ERN was not significant for either the social condition $r(60)$ = .11, p = .39, or the nonsocial condition, $r(60)$ = .06, p = .67. In addition, the correlation between age and accuracy was nonsignificant for the social condition, $r(60)$ = .12, p = .34, or the nonsocial condition, $r(60)$ = .17, p = .18. Lastly, there were no differences in the number of artifact-free error trials between conditions, $F(1, 60)$ = 0.40, p = .53, and no interaction with age, $F(1, 60)$ = 0.26, p = .61.

All waveforms were first baseline corrected 600 ms to 400 ms before the response (Davies et al., 2004; Pailing, Segalowitz, Dywan, & Davies, 2002). The ERN and the CRN were then separately evaluated using a 20 ms moving window as the most negative average window from 50 ms before the response to 100 ms following the response at 3 fronto-central electrodes along the midline (electrode numbers on 128 ch. geodesic net: 5, 6 [FCz], 12). The Pe was evaluated as the average activity 200-400 ms following the response from the average of 7 centro-parietal electrodes (Cz, 31, 54, 55 [PCz], 62 [Pz], 79, 80). 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 minus CRN), which is referred to as the ERN. Similarly, a change score was calculated for the Pe by subtracting the Pe on correct trials from the Pe on error trials (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 slowing was calculated as the difference in response times between post-error trials and post-correct trials. Behavioral variability was calculated as the within-individual standard deviation of response time separately for each response and condition.

2.5 Data analysis

A univariate ANCOVA with age as a covariate was conducted to explore differences in self-reported effort between conditions and the influences of age on effort. Next, to explore social influences on the error monitoring system, mixed-model ACNOVA's with condition (social, nonsocial) and response (correct, incorrect) as the repeated measures with chronological age as a covariate were conducted. Behavioral measures (i.e., response time, behavioral variability, post-error slowing, and accuracy) and physiological measures (i.e., ERN/CRN, and Pe/Correct Pe) were separately evaluated. In all ANCOVA analyses, age was mean centered to evaluate main effects at the average age of the sample (Thomas et al., 2009). Note that interactive effects with response type for neural measures represent a change score (e.g., ERN, Pe).

Significant interactions with age were explored in two ways. First, to explore how age influenced the degree to which the ERN and CRN were influenced by social contexts, adolescents were categorized into groups based high and low terciles of age (i.e., younger adolescents, older adolescents). Then follow-up ANCOVA's were performed when interaction effects were significant. Second, to explore the influence of context on developmental patterns of neural components (i.e., ERN and CRN) across adolescence, Pearson's correlations were separately conducted for each condition. Differences in correlations between conditions were analyzed using Fisher's r-to-z test (Steiger, 1980).

Lastly, to explore whether behavioral variability explained changes in the ERN across conditions (DuPuis et al., 2015), mediation analyses were conducted using PROCESS (Hayes, 2009, 2012). Mediation analyses were conducted separately for each condition where behavioral variability on error responses was the mediator, age was the predictor, and the ERN was the outcome. Direct and indirect effects of each variable were tested using published guidelines (Hayes, 2009, 2012).

3. RESULTS

3.1 Influence of social context and age on behavior

Table 1 displays the behavioral measures for the social and nonsocial condition. Adolescents reported engaging in more effort during the social condition as compared to the nonsocial condition, $F(1, 65) = 17.20$, $p < .001$, $\eta^2 = .21$. This main effect was qualified by a trend for an age × effort interaction, $F(1, 65) = 3.54$, $p = .064$, $\eta^2 = .05$, suggesting that younger age was associated with a larger increase in reported effort in the social condition as compared to the nonsocial condition. Adolescents exhibited faster response times on error trials than correct trials across both conditions, $F(1, 66) = 326.78$, $p < .001$, $\eta^2 = .83$. In addition, response times in the social condition were faster than the nonsocial condition, $F(1, 66) = 10.11$, $p = .002$, $\eta^2 = .13$. Analyses of the effects of age revealed that response times decreased with chronological age across both conditions and responses, $F(1, 66) = 45.21$, $p < .001$, $\eta^2 = .41$. No interactive effects with age and condition reached significance, $p's > .20$. Lastly, no differences in accuracy or post-error slowing were observed between conditions, $p's > .20$.

Examination of behavioral variability revealed a main effect of response, such that incorrect responses had significantly less variability as compared to correct responses, $F(1, 66) = 4.99$, $p = .029$, $\eta^2 = .070$. In addition, there was a main effect of condition, such that response variability was significantly smaller in the social condition as compared to the nonsocial condition $F(1, 66) = 6.50$, $p = .013$, $\eta^2 = .09$. Analyses of the effects of age on behavioral variability revealed that increasing age was associated with decreases in response variability across both conditions and responses $F(1, 66) = 60.42$, $p < .001$, $\eta^2 = .48$. However, age did not interact with condition in predicting behavioral variability, $p > .20$.

3.2 Influence of social context and age on ERN

Figure 2 displays the ERP waveforms for the social and nonsocial condition. As expected, 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, $F(1, 60) = 41.16$, $p < .001$, $\eta^2 = .41$. In addition, both the CRN and ERN marginally increased with age, $F(1, 60) = 3.85$, $p = .054$, $\eta^2 = .01$.[1] This effect was qualified by a significant response × condition × age interaction, $F(1, 60) = 6.94$, $p = .011$, $\eta^2 = .10$, suggesting that adolescent age influenced the ERN and CRN differently across conditions.[2] No other main effect of condition was observed, nor any other interactive effects with condition reached significance, p's > .20.

Figure 3 presents the ERN across conditions for younger and older adolescents. Analyses using this categorical grouping continued to reveal a significant response condition × age interaction, $F(1, 40) = 9.04$, $p = .005$, $\eta^2 = .18$. Follow-up tests for each response revealed that for the CRN, there were no main or interactive effects with age group, p's > .20. However, for the ERN, there was a significant age × condition interaction, $F(1, 40) = 9.10$, $p = .004$, $\eta^2 = .19$. Follow-up analyses for each age group revealed that younger adolescents exhibited a larger (i.e., more negative) ERN in the social condition as compared to the nonsocial condition $F(1, 20) = 7.08$, $p = .015$, $\eta^2 = .26$. However, among older adolescents, the ERN was not different between conditions, $F(1, 20) = 2.09$, $p = .16$, $\eta^2 = .10$.

3.3 Influence of social context and age 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, $F(1, 60) = 233.49$, $p < .001$, $\eta^2 = .80$. In addition, there was a main effect of condition, such that the Pe on both correct and error trials in the social condition were significantly larger than the nonsocial condition, $F(1, 60) = 7.65$, $p = .008$, $\eta^2 = .11$. This effect was qualified by a significant condition × response interaction, $F(1, 60) = 4.81$, $p = .032$, $\eta^2 = .07$, indicating a larger Pe in the social condition as compared to the nonsocial condition. Follow-up tests for each response revealed that the Pe was significantly larger in the social condition than the nonsocial condition, $F(1, 60) = 7.19$, $p = .009$, $\eta^2 = .11$. However, the correct Pe was only marginally larger in the social condition than the nonsocial condition, $F(1, 60) = 3.89$, $p = .053$, $\eta^2 = .06$. Finally, there were no main or interactive effects with age, p's > .30.

3.4 Developmental patterns of ERN

Figure 4 depicts a scatterplot between the ERN and age in the social and nonsocial condition. As expected, the ERN in the nonsocial condition was negatively related to age, $r(60) = -.35$, $p = .005$, such that increasing age was associated with a larger (i.e., more negative) ERN. In contrast, the ERN in the social condition was unrelated to age, $r(60) = -.09$, $p = .88$. The difference between the social ERN and nonsocial ERN correlations with age was significant, $z(60) = 2.72$, $p = .006$, suggesting different developmental patterns of the

---

[1] Self-report of pubertal status was also collected via the Pubertal Development Questionnaire (PDS; Petersen, Crockett, Richards, & Boxer, 1988). The nonsocial ERN was negativity associated with the PDS such that increasing pubertal status was associated with a larger (i.e., more negative) ERN in the nonsocial condition, $r(60) = -.31$, $p = .014$, but not the social condition, $p = .88$. However, due to the high correlation between PDS and age, $r(60) = .80$, $p < .001$, PDS did not uniquely predict increases in the ERN, p's > .20.
[2] To examine if the influences of age and social context on the ERN and CRN were specifically related to increased neural activity as opposed to general decreases in behavioral variability, we also computed residualized scores for the ERN and CRN for each condition by removing the associated variance of behavioral variability. Critically, we continued to observe a significant age × condition × response interaction $F(1, 60) = 5.35$, $p = .016$, $\eta^2 = .09$, as well as significant follow-up tests, p's < .05.

ERN between conditions (see Figure 4). For the CRN, age was unrelated to the ERN in the social and nonsocial condition (p's > .10).

Mediation analyses were conducted to determine whether changes in behavioral variability explained developmental changes in the ERN observed in each condition. In the nonsocial condition, age and behavioral variability jointly predicted the ERN, R^2 = .12, $F(2, 59)$ = 4.13, p = .021. In addition, age continued to predict the ERN when controlling for behavioral variability, $t(59)$ = 2.01, p = .049. However, behavioral variability did not significantly predict the ERN when controlling for age, $t(59)$ = 0.21, p = .84. Furthermore, behavioral variability did not mediate the relation between the ERN and age, β = −0.05, 95 % CI [−.52, .44]. In the social condition, neither age nor behavioral variability predicted the ERN, p's > .20. In addition, behavioral variability did not significantly mediate the relation between the ERN and age, β = −0.20, 95 % CI [−.56, .10].

3.5 Relation between effort, ERN and Pe

The correlation between the change in magnitude of the ERN and Pe across conditions was not significant, $t(59)$ = .09, p = .47. In addition, change in effort across conditions was unrelated to change in ERN magnitude across conditions, $t(59)$ = .14, p = .29. However, for the Pe, change in effort across condition was significantly correlated with change in Pe magnitude across conditions, $t(59)$ = .29, p = .023, such that increased report of effort in the social condition as compared to the nonsocial condition was related to a larger Pe in the social condition as compared to the nonsocial condition. Next, to examine the impact of effort in explaining developmental changes in the ERN across conditions, we conducted mediation analyses with effort as the mediator, age as the predictor, and the ERN as the outcome measure for both the social and nonsocial conditions. Effort did not significantly mediate the relation between age and the ERN in either condition, p's > .10.

4. DISCUSSION

The primary goal of the present study was to examine developmental changes in the effect of social and nonsocial contexts on the error monitoring system in adolescent girls. As hypothesized, age influenced the degree to which the error-related negativity (ERN) was enhanced in social contexts. Specifically, we observed that younger adolescent girls exhibited a larger ERN in social contexts as compared to nonsocial contexts. In contrast, older adolescent girls exhibited no differences in the ERN between social and nonsocial contexts. For the error positivity (Pe), we found that the Pe was enhanced in social contexts across all adolescents, but the degree of enhancement was not influenced by age. The second goal of the study was to explore how contextual factors that increase motivation influence developmental patterns of the ERN. As hypothesized, we observed that increasing age was associated with a larger ERN in nonsocial contexts. In contrast, the ERN was unrelated to age in social contexts, suggesting contextual factors diminished the typically observed ERN and age association. Taken together, these findings suggest that sensitivity of the ERN to social contexts changes across adolescence, and motivational factors, such as social contexts, may explain a portion of the variance associated with developmental changes in the ERN across childhood and adolescence.

The present study found that adolescent girls reported higher motivation in social contexts as compared to nonsocial contexts, with the largest increases in motivation in social contexts observed among younger adolescents. Similarly, we observed a larger ERN in social contexts as compared to nonsocial contexts among younger adolescents. These findings are in agreement with previous research that has found enhancements of the ERN in motivationally significant contexts (Boksem, Tops, et al., 2006; Buzzell, Troller-Renfree, et al., 2017; Hajcak et al., 2005; Kim et al., 2005; Potts, 2011; Van Meel & Van Heijningen, 2010). In addition, our findings confirm previous work that the ERN is also sensitive to social factors (Barker et al., 2015; Buzzell, Troller-Renfree, et al., 2017; Kim et al., 2005), likely reflecting increased motivation (Blascovich et al., 1999; Blascovich & Tomaka, 1996).

We also observed developmental differences in the degree to which the ERN was enhanced by social contexts. Specifically, we found that only younger adolescent girls exhibited a larger ERN in social contexts as compared nonsocial contexts. In contrast, we observed no differences in the ERN between contexts among older adolescent girls. These findings suggest that the influence of social contexts on the ERN changes across adolescence. In addition, these observed changes support theories of adolescent neurobiology, which suggests that increased social motivation observed in adolescence is in part due to increased neural sensitivity to social cues (Casey et al., 2008; Crone & Dahl, 2012; Nelson et al., 2005; Shulman et al., 2016; Steinberg et al., 2008).

We found that the Pe was larger in social contexts as compared to nonsocial contexts. In addition, we found that age did not influence the degree to which the Pe was enhanced in social contexts. However, it is interesting to note that although the ERN and Pe were enhanced by social contexts, age influenced the magnitude of the effect only for the ERN. Given that age does not influence developmental patterns of the Pe (Davies et al., 2004; Santesso & Segalowitz, 2008; Santesso et al., 2006), it is possible that age similarly does not influence the degree that motivational and contextual factors impact the Pe.

As hypothesized, increasing age was associated with a larger ERN in nonsocial contexts, replicating previous findings (Davies et al., 2004; Ladouceur et al., 2004, 2007; Santesso & Segalowitz, 2008; Santesso et al., 2006). In contrast, we observed no relation between the ERN and age in social contexts, suggesting that contextual factors that influence motivation diminished the typically observed relation between the ERN and age. One possibility for this finding is that increased motivation among younger adolescents led to concomitant increases in ERN magnitude, thereby reducing the ERN and age association. However, we did not observe an association between self-report of effort and ERN magnitude. Another possibility for this finding is that older adolescents intrinsically evaluate and value their performance more than younger adolescents (Eccles & Wigfield, 2002; Gottfried, Fleming, & Gottfried, 2001), whereas younger adolescents are more likely to be influenced by external evaluation of performance (Shulman et al., 2016; Steinberg, 2007, 2008), particularly by social peers (Ernst et al., 2006, 2009). Recent developmental theories of the ERN have similarly suggested that changes from external to internal valuation of errors may account for some of the observed changes in the ERN in adolescence (Meyer, 2017).

We observed that increases in self-report of effort in social contexts was related to a larger Pe in social contexts, but unrelated to changes in the ERN in social contexts. In addition, enhancements of the ERN in social contexts were unrelated to enhancements of the Pe in social contexts. One possibility for these differences is that increases in self-report of perceived effort in social contexts may reflect a greater awareness of performance and error behavior. The Pe has been theorized to represent conscious awareness of an error (Endrass, Franke, & Kathmann, 2005; Endrass, Reuter, & Kathmann, 2007; Nieuwenhuis et al., 2001), and the motivational salience of an error (Overbeek et al., 2005). Thus, it is possible that enhancements of the Pe in social contexts are related to the effects of perceived motivation on error awareness. In contrast, we found no relation between increased self-report effort in the social condition and ERN. Although there is strong evidence that the ERN is enhanced by motivational factors, less research has examined if ERN magnitude is related to individual differences in subjective report of motivation and arousal (Yeung, Holroyd, & Cohen, 2005). Further work should continue to explore the effect of perceived effort in influencing the ERN and Pe.

Decreased behavioral variability, as measured as the within-individual standard deviation of response times, was observed in the social condition as compared to the nonsocial condition. It has been suggested that the smaller ERN observed in childhood and early adolescence is in part due to increased behavioral variability, causing the ERN to be more variable in relation to the response, which mutes the averaged waveform (DuPuis et al., 2015). Indeed, larger behavioral variability is associated with a smaller ERN (DuPuis et al., 2015; Richardson et al., 2011). Although we observed decreased behavioral variability in social contexts, age did not significantly moderate this effect. In addition, we did not find that behavioral variability mediated developmental changes in the ERN in the social condition, suggesting that enhancements of the ERN in social contexts was independent of changes in behavioral variability. However, future research should continue to explore the influence of behavioral variability on the development of the ERN.

There is evidence that other physiological measures of motivation, such as startle potentiation, pupil dilation, and skin conductance, are associated with error commission (Critchley et al., 2005; Hajcak & Foti, 2008; Hajcak et al., 2003b, 2004; Riesel et al., 2012). However, there is mixed evidence that the ERN is *directly* related to such physiological measures during error commission (Hajcak, McDonald, & Simons, 2003a; Lewis & Pitts, 2015; Lo, Schroder, Moran, Durbin, & Moser, 2015; Moser, Moran, Schroder, Donnellan, & Yeung, 2014; Riesel, Weinberg, Moran, & Hajcak, 2013), raising questions as to whether motivational influences on ERN magnitude reflect increased motivational significance in relation to a specific error, or more general increases in motivation which result in enhanced ERN magnitude. Indeed, measures such as skin conductance and startle potentiation are highly sensitive to contextual factors that increase motivation and arousal (Bradley et al., 2018; Grillon, 2002). Thus, it is possible that contextual influences on the ERN in part represent more general motivational factors unrelated to errors commission. Future research should further examine this point.

Developmental changes in the mesencephalic dopamine system may account for observed changes in the sensitivity to the ERN to social contexts across adolescence. Mesencephalic

Dopamine (DA) neurons are located within the midbrain, which project to regions in the cingulate and prefrontal cortices (i.e., mesocortical pathway; Ernst et al., 2009). DA neurons are thought to play a key role in motivation, reward processing, and error monitoring (Fibiger & Phillips, 2011; Nicola, 2007; Schultz, 2007). The density of DA neurons peak in early adolescence, around the onset of puberty (Andersen, Dumont, & Teicher, 1997; Andersen, Thompson, Rutstein, Hostetter, & Teicher, 2000; Tarazi, Tomasini, & Baldessarini, 1999). It has been argued that changes in DA neuron structure in the mesocortical pathway may explain increased adolescent social motivation (Ernst & Fudge, 2009; Ernst et al., 2006, 2009; Spear, 2000). Motivation and reward learning theories of the ERN suggest that the ERN is produced by changes in phasic DA during error commission (Frank, 2005; Holroyd & Coles, 2002), and developmental changes in the ERN may reflect changes in DA (Davies et al., 2004). Thus, it is possible that the current findings of a larger ERN in social contexts among younger adolescents may reflect increased DA functioning toward social stimuli, resulting in a larger ERN. However, future studies should continue to utilize the ERN to examine developmental changes in reward processing and social motivation in adolescence.

4.1 Limitations and future directions

There are a number of limitations in the present study. First, it should be noted that only adolescent girls participated, making it unknown whether the present findings generalize to adolescent boys. A number of differences have been observed between the ERN and social development between sexes. For example, adolescent girls display a different developmental pattern of the ERN than adolescent boys (Davies et al., 2004), and are more likely to report fear of social evaluation (La Greca & Lopez, 1998). In addition, it is possible that some of the observed age differences of the ERN in social contexts were because older adolescents were less likely believe the social manipulation. However, to limit these possibilities, we excluded all adolescents who indicated that they thought they were not actually being observed and evaluated by other adolescents. Lastly, it is unknown how pubertal factors such sex hormones impact the ERN and social motivation. There is growing evidence that sex hormones influence brain and behavior function in adolescence (Forbes & Dahl, 2010; Herting et al., 2014). However the exact relationship is unclear (Ordaz, Fritz, Forbes, & Luna, 2017).

It is important to note that the presentation of feedback was different across conditions. Specifically, the nonsocial condition contained only text feedback whereas the social condition contained both text feedback was well as emoji/emoticons. Such differences in feedback amount and type between conditions may have changed the salience of the context in the social condition beyond general factors with social observation and evaluation. Future research should continue to explore the specificity of social influences on the ERN.

In the present study, we found that age influenced the degree to which the ERN was enhanced by social contexts such that the largest enhancements were observed among younger adolescents. In addition, we observed the Pe was enhanced in social contexts as compared to nonsocial contexts, and such enhancements were not influenced by age. Lastly, we observed that increasing age was associated with a larger ERN in nonsocial contexts. In

contrast, age was unrelated to the ERN in social contexts. Taken together, these findings suggest that the ERN is also sensitive to social motivational factors in early adolescence, and that developmental differences in the sensitivity of the ERN to social contexts may in part reflect contextual factors that influence motivation.

Acknowledgments

References

Agam Y, Hämäläinen MS, Lee AKC, Dyckman KA, Friedman JS, Isom M, Manoach DS. Multimodal neuroimaging dissociates hemodynamic and electrophysiological correlates of error processing. Proceedings of the National Academy of Sciences. 2011; 108(42):17556–17561. DOI: 10.1073/pnas.1103475108

Andersen SL, Dumont NL, Teicher MH. Developmental differences in dopamine synthesis inhibition by (±)-7-OH-DPAT. Naunyn-Schmiedeberg's Archives of Pharmacology. 1997; 356(2):173–181. DOI: 10.1007/PL00005038

Andersen SL, Thompson AT, Rutstein M, Hostetter JC, Teicher MH. Dopamine receptor pruning in prefrontal cortex during the periadolescent period in rats. Synapse. 2000; 37(2):167–169. [PubMed: 10881038]

Barker TV, Troller-Renfree S, Pine DS, Fox NA. Individual differences in social anxiety affect the salience of errors in social contexts; Cognitive, Affective, & Behavioral Neuroscience. 2015. 1–13.

Blascovich J, Mendes WB, Hunter SB, Salomon K. Social "facilitation" as Challenge and Threat. Journal of Personality and Social Psychology. 1999; 77(1):68–77. [PubMed: 10434409]

Blascovich J, Tomaka J. The Biopsychosocial Model of Arousal Regulation. In: Zanna MP, editorAdvances in Experimental Social Psychology. Vol. 28. Academic Press; 1996. 1–51.

Boksem MAS, Meijman TF, Lorist MM. Mental fatigue, motivation and action monitoring. Biological Psychology. 2006; 72(2):123–132. DOI: 10.1016/j.biopsycho.2005.08.007 [PubMed: 16288951]

Boksem MAS, Tops M, Wester AE, Meijman TF, Lorist MM. Error-related ERP components and individual differences in punishment and reward sensitivity. Brain Research. 2006; 1101(1):92–101. DOI: 10.1016/j.brainres.2006.05.004 [PubMed: 16784728]

Botvinick MM, Braver TS, Barch DM, Carter CS, Cohen JD. Conflict monitoring and cognitive control. Psychological Review. 2001; 108(3):624–652. DOI: 10.1037/0033-295X.108.3.624 [PubMed: 11488380]

Bradley MM, Zlatar ZZ, Lang PJ. Startle reflex modulation during threat of shock and "threat" of reward. Psychophysiology. 2018; 55(2)doi: 10.1111/psyp.12989

Buzzell GA, Richards JE, White LK, Barker TV, Pine DS, Fox NA. Development of the error-monitoring system from ages 9–35 unique insight provided by MRI-constrained source localization of EEG. NeuroImage. 2017.

Buzzell GA, Troller-Renfree SV, Barker TV, Bowman LC, Chronis-Tuscano A, Henderson HA, Fox NA. A neurobehavioral mechanism linking behaviorally inhibited temperament and later adolescent social anxiety. Journal of the American Academy of Child & Adolescent Psychiatry. 2017; 56(12):1097–1105. DOI: 10.1016/j.jaac.2017.10.007 [PubMed: 29173744]

Carter CS, van Veen V. Anterior cingulate cortex and conflict detection: An update of theory and data. Cognitive, Affective, & Behavioral Neuroscience. 2007; 7(4):367–379. DOI: 10.3758/CABN.7.4.367

Casey BJ, Jones RM, Hare TA. The Adolescent Brain. Annals of the New York Academy of Sciences. 2008; 1124:111–126. DOI: 10.1196/annals.1440.010 [PubMed: 18400927]

Chiew KS, Braver TS. Temporal Dynamics of Motivation-Cognitive Control Interactions Revealed by High-Resolution Pupillometry. Frontiers in Psychology. 2013; 4doi: 10.3389/fpsyg.2013.00015

Coles MGH, Scheffers MK, Holroyd CB. Why is there an ERN/Ne on correct trials? Response representations, stimulus-related components, and the theory of error-processing. Biological Psychology. 2001; 56(3):173–189. DOI: 10.1016/S0301-0511(01)00076-X [PubMed: 11399349]

Coles MG, Scheffers MK, Fournier L. Where did you go wrong? Errors, partial errors, and the nature of human information processing. Acta Psychologica. 1995; 90(1–3):129–144. [PubMed: 8525868]

Critchley HD, Tang J, Glaser D, Butterworth B, Dolan RJ. Anterior cingulate activity during error and autonomic response. NeuroImage. 2005; 27(4):885–895. DOI: 10.1016/j.neuroimage.2005.05.047 [PubMed: 15996878]

Crone EA, Dahl RE. Understanding adolescence as a period of social-affective engagement and goal flexibility. Nature Reviews. Neuroscience. 2012; 13(9):636–650. DOI: 10.1038/nrn3313 [PubMed: 22903221]

Dahl RE. Adolescent Brain Development: A period of vulnerabilities and opportunities. Keynote address. Annals of the New York Academy of Sciences. 2004; 1021(1):1–22. DOI: 10.1196/annals.1308.001 [PubMed: 15251869]

Davies PL, Segalowitz SJ, Gavin WJ. Development of Response-Monitoring ERPs in 7- to 25-Year-Olds. Developmental Neuropsychology. 2004; 25(3):355–376. DOI: 10.1207/s15326942dn2503_6 [PubMed: 15148003]

Dehaene S, Posner MI, Tucker DM. Localization of a neural system for error detection and compensation. Psychological Science. 1994; 5(5):303–305. DOI: 10.1111/j.1467-9280.1994.tb00630.x

Di Gregorio F, Maier ME, Steinhauser M. Errors can elicit an error positivity in the absence of an error negativity: Evidence for independent systems of human error monitoring. NeuroImage. 2018; 172:427–436. DOI: 10.1016/j.neuroimage.2018.01.081 [PubMed: 29409999]

Dien J. The ERP PCA Toolkit: An open source program for advanced statistical analysis of event-related potential data. Journal of Neuroscience Methods. 2010; 187(1):138–145. DOI: 10.1016/j.jneumeth.2009.12.009 [PubMed: 20035787]

DuPuis D, Ram N, Willner CJ, Karalunas S, Segalowitz SJ, Gatzke-Kopp LM. Implications of ongoing neural development for the measurement of the error-related negativity in childhood. Developmental Science. 2015; 18(3):452–468. DOI: 10.1111/desc.12229 [PubMed: 25209462]

Eccles JS, Wigfield A. Motivational beliefs, values, and goals. Annual Review of Psychology. 2002; 53(1):109–132. DOI: 10.1146/annurev.psych.53.100901.135153

Elkins-Brown N, Saunders B, Inzlicht M. Error-related electromyographic activity over the corrugator supercilii is associated with neural performance monitoring. Psychophysiology. 2016; 53(2):159–170. DOI: 10.1111/psyp.12556 [PubMed: 26470645]

Endrass T, Franke C, Kathmann N. Error awareness in a saccade countermanding task. Journal of Psychophysiology. 2005; 19(4):275–280.

Endrass T, Reuter B, Kathmann N. ERP correlates of conscious error recognition: aware and unaware errors in an antisaccade task. European Journal of Neuroscience. 2007; 26(6):1714–1720. DOI: 10.1111/j.1460-9568.2007.05785.x [PubMed: 17880402]

Eriksen BA, Eriksen CW. Effects of noise letters upon the identification of a target letter in a nonsearch task. Perception & Psychophysics. 1974; 16(1):143–149. DOI: 10.3758/BF03203267

Ernst M, Fudge JL. A developmental neurobiological model of motivated behavior: anatomy, connectivity and ontogeny of the triadic nodes. Neuroscience and Biobehavioral Reviews. 2009; 33(3):367–382. DOI: 10.1016/j.neubiorev.2008.10.009 [PubMed: 19028521]

Ernst M, Pine DS, Hardin M. Triadic model of the neurobiology of motivated behavior in adolescence. Psychological Medicine. 2006; 36(3):299–312. DOI: 10.1017/S0033291705005891 [PubMed: 16472412]

Ernst M, Romeo RD, Andersen SL. Neurobiology of the development of motivated behaviors in adolescence: A window into a neural systems model. Pharmacology Biochemistry and Behavior. 2009; 93(3):199–211. DOI: 10.1016/j.pbb.2008.12.013

Falkenstein M, Hielscher H, Dziobek I, Schwarzenau P, Hoormann J, Sunderman B, Hohnsbein J. Action monitoring, error detection, and the basal ganglia: an ERP study. Neuroreport. 2001; 12(1): 157–161. [PubMed: 11201078]

Falkenstein M, Hohnsbein J, Hoormann J, Blanke L. Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. Electroencephalography and Clinical Neurophysiology. 1991; 78(6):447–455. DOI: 10.1016/0013-4694(91)90062-9 [PubMed: 1712280]

Falkenstein M, Hoormann J, Christ S, Hohnsbein J. ERP components on reaction errors and their functional significance: a tutorial. Biological Psychology. 2000; 51(2–3):87–107. DOI: 10.1016/S0301-0511(99)00031-9 [PubMed: 10686361]

Fibiger HC, Phillips AG. Reward, Motivation, Cognition: Psychobiology of Mesotelencephalic Dopamine Systems. In: Terjung R, editorComprehensive Physiology. Hoboken, NJ, USA: John Wiley & Sons, Inc; 2011.

Forbes EE, Dahl RE. Pubertal development and behavior: hormonal activation of social and motivational tendencies. Brain and Cognition. 2010; 72(1):66–72. DOI: 10.1016/j.bandc.2009.10.007 [PubMed: 19942334]

Frank M. Dynamic dopamine modulation in the basal ganglia: A neurocomputational account of cognitive deficits in medicated and nonmedicated Parkinsonism. Journal of Cognitive Neuroscience. 2005; 17(1):51–72. DOI: 10.1162/0898929052880093 [PubMed: 15701239]

Frank MJ, Woroch BS, Curran T. Error-related negativity predicts reinforcement learning and conflict biases. Neuron. 2005; 47(4):495–501. DOI: 10.1016/j.neuron.2005.06.020 [PubMed: 16102533]

Geen RG. Social motivation. Annual Review of Psychology. 1991; 42(1):377–399. DOI: 10.1146/annurev.ps.42.020191.002113

Geen RG. Human motivation: A social psychological approach. Thomson Brooks/Cole Publishing Co; 1995.

Geen RG, Bushman BJ. The arousing effects of social presence. In: Wagner H, Manstead A, editorsHandbook of social psychophysiology. Oxford, England: John Wiley & Sons; 1989. 261–281.

Gehring WJ, Goss B, Coles MGH, Meyer DE, Donchin E. A neural system for error detection and compensation. Psychological Science. 1993; 4(6):385–390. DOI: 10.1111/j.1467-9280.1993.tb00586.x

Gentsch A, Ullsperger P, Ullsperger M. Dissociable medial frontal negativities from a common monitoring system for self-and externally caused failure of goal achievement. Neuroimage. 2009; 47(4):2023–2030. [PubMed: 19486945]

Gottfried AE, Fleming JS, Gottfried AW. Continuity of academic intrinsic motivation from childhood through late adolescence: A longitudinal study. Journal of Educational Psychology. 2001; 93(1):3.

Grammer JK, Carrasco M, Gehring WJ, Morrison FJ. Age-related changes in error processing in young children: A school-based investigation. Developmental Cognitive Neuroscience. 2014; 9(Supplement C):93–105. DOI: 10.1016/j.dcn.2014.02.001 [PubMed: 24631799]

Greca AML, Lopez N. Social anxiety among adolescents: Linkages with peer relations and friendships. Journal of Abnormal Child Psychology. 1998; 26(2):83–94. DOI: 10.1023/A:1022684520514 [PubMed: 9634131]

Grillon C. Startle reactivity and anxiety disorders: aversive conditioning, context, and neurobiology. Biological Psychiatry. 2002; 52(10):958–975. DOI: 10.1016/S0006-3223(02)01665-7 [PubMed: 12437937]

Groom MJ, Liddle EB, Scerif G, Liddle PF, Batty MJ, Liotti M, Hollis CP. Motivational incentives and methylphenidate enhance electrophysiological correlates of error monitoring in children with attention deficit/hyperactivity disorder. Journal of Child Psychology and Psychiatry. 2013; 54(8): 836–845. DOI: 10.1111/jcpp.12069 [PubMed: 23662815]

Haenlein M, Caul WF. Attention deficit disorder with hyperactivity: A specific hypothesis of reward dysfunction. Journal of the American Academy of Child & Adolescent Psychiatry. 1987; 26(3): 356–362. DOI: 10.1097/00004583-198705000-00014 [PubMed: 3597291]

Hajcak G, Foti D. Errors are aversive: defensive motivation and the error-related negativity. Psychological Science. 2008; 19(2):103–108. DOI: 10.1111/j.1467-9280.2008.02053.x [PubMed: 18271855]

Hajcak G, McDonald N, Simons RF. Anxiety and error-related brain activity. Biological Psychology. 2003a; 64(1–2):77–90. DOI: 10.1016/S0301-0511(03)00103-0 [PubMed: 14602356]

Hajcak G, McDonald N, Simons RF. To err is autonomic: error-related brain potentials, ANS activity, and post-error compensatory behavior. Psychophysiology. 2003b; 40(6):895–903. [PubMed: 14986842]

Hajcak G, McDonald N, Simons RF. Error-related psychophysiology and negative affect. Brain and Cognition. 2004; 56(2):189–197. DOI: 10.1016/j.bandc.2003.11.001 [PubMed: 15518935]

Hajcak G, Moser JS, Yeung N, Simons RF. On the ERN and the significance of errors. Psychophysiology. 2005; 42(2):151–160. DOI: 10.1111/j.1469-8986.2005.00270.x [PubMed: 15787852]

Hajcak G, Simons RF. Error-related brain activity in obsessive–compulsive undergraduates. Psychiatry Research. 2002; 110(1):63–72. [PubMed: 12007594]

Hayes AF. Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. Communication Monographs. 2009; 76(4):408–420. DOI: 10.1080/03637750903310360

Hayes AF. PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling. University of Kansas; KS: 2012.

Herting MM, Gautam P, Spielberg JM, Kan E, Dahl RE, Sowell ER. The role of testosterone and estradiol in brain volume changes across adolescence: A longitudinal structural MRI study. Human Brain Mapping. 2014; 45(11):5633–5645. DOI: 10.1002/hbm.22575

Hogan AM, Vargha-Khadem F, Kirkham FJ, Baldeweg T. Maturation of action monitoring from adolescence to adulthood: an ERP study. Developmental Science. 2005; 8(6):525–534. DOI: 10.1111/j.1467-7687.2005.00444.x [PubMed: 16246244]

Holroyd CB, Coles MGH. The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. Psychological Review. 2002; 109(4):679–709. [PubMed: 12374324]

Holroyd CB, Dien J, Coles MGH. Error-related scalp potentials elicited by hand and foot movements: evidence for an output-independent error-processing system in humans. Neuroscience Letters. 1998; 242(2):65–68. DOI: 10.1016/S0304-3940(98)00035-4 [PubMed: 9533395]

Holroyd CB, Yeung N. Motivation of extended behaviors by anterior cingulate cortex. Trends in Cognitive Sciences. 2012; 16(2):122–128. DOI: 10.1016/j.tics.2011.12.008 [PubMed: 22226543]

Kim EY, Iwaki N, Uno H, Fujita T. Error-related negativity in children: effect of an observer. Developmental Neuropsychology. 2005; 28(3):871–883. DOI: 10.1207/s15326942dn2803_7 [PubMed: 16266253]

Ladouceur CD, Dahl RE, Carter CS. ERP correlates of action monitoring in adolescence. Annals of the New York Academy of Sciences. 2004; 1021(1):329–336. DOI: 10.1196/annals.1308.040 [PubMed: 15251905]

Ladouceur CD, Dahl RE, Carter CS. Development of action monitoring through adolescence into adulthood: ERP and source localization. Developmental Science. 2007; 10(6):874–891. DOI: 10.1111/j.1467-7687.2007.00639.x [PubMed: 17973802]

Larson MJ, Baldwin SA, Good DA, Fair JE. Brief reports: Temporal stability of the error-related negativity (ERN) and post-error positivity (Pe): The role of number of trials. Psychophysiology. 2010; 47(6):1167–1171. DOI: 10.1111/j.1469-8986.2010.01022.x [PubMed: 20477982]

Lewis M, Pitts M. Replication of Hajcak & Foti (2008, PS, Study 1). 2015. Jul 14, http://doi.org/10.17605/OSF.IO/HSNTD

Liu Y, Hanna GL, Carrasco M, Gehring WJ, Fitzgerald KD. Altered relationship between electrophysiological response to errors and gray matter volumes in an extended network for error-processing in pediatric obsessive-compulsive disorder. Human Brain Mapping. 2013; 35(4):1143–1153. DOI: 10.1002/hbm.22240 [PubMed: 23418104]

Lo SL, Schroder HS, Moran TP, Durbin CE, Moser JS. Neurophysiological evidence of an association between cognitive control and defensive reactivity processes in young children. Developmental

Cognitive Neuroscience. 2015; 15(Supplement C):35–47. DOI: 10.1016/j.dcn.2015.09.001 [PubMed: 26386550]

Maruo Y, Sommer W, Masaki H. The effect of monetary punishment on error evaluation in a Go/No-go task. International Journal of Psychophysiology. 2017; 120(Supplement C):54–59. DOI: 10.1016/j.ijpsycho.2017.07.002 [PubMed: 28711700]

Masaki H, Maruo Y, Meyer A, Hajcak G. Neural Correlates of choking under pressure: Athletes high in sports anxiety monitor errors more when performance is being evaluated. Developmental Neuropsychology. 2017; 42(2):104–112. DOI: 10.1080/87565641.2016.1274314 [PubMed: 28452597]

Meyer A. A biomarker of anxiety in children and adolescents: A review focusing on the error-related negativity (ERN) and anxiety across development. Developmental Cognitive Neuroscience. 2017; 27(Supplement C):58–68. DOI: 10.1016/j.dcn.2017.08.001 [PubMed: 28818707]

Meyer A, Bress JN, Proudfit GH. Psychometric properties of the error-related negativity in children and adolescents. Psychophysiology. 2014; 51(7):602–610. DOI: 10.1111/psyp.12208 [PubMed: 24646380]

Moser J, Moran T, Schroder HS, Donnellan MB, Yeung N. The case for compensatory processes in the relationship between anxiety and error monitoring: a reply to Proudfit, Inzlicht, and Mennin. Frontiers in Human Neuroscience. 2014; 8doi: 10.3389/fnhum.2014.00064

Moser JS, Moran TP, Schroder HS, Donnellan MB, Yeung N. On the relationship between anxiety and error monitoring: a meta-analysis and conceptual framework. Frontiers in Human Neuroscience. 2013; 7doi: 10.3389/fnhum.2013.00466

Muris P, Merckelbach H, Meesters C, van den Brand K. Cognitive development and worry in normal children. Cognitive Therapy and Research. 2002; 26(6):775–787. DOI: 10.1023/A:1021241517274

Nelson EE, Leibenluft E, McClure EB, Pine DS. The social re-orientation of adolescence: a neuroscience perspective on the process and its relation to psychopathology. Psychological Medicine. 2005; 35(2):163–174. DOI: 10.1017/S0033291704003915 [PubMed: 15841674]

Nicola SM. The nucleus accumbens as part of a basal ganglia action selection circuit. Psychopharmacology. 2007; 191(3):521–550. [PubMed: 16983543]

Nieuwenhuis S, Ridderinkhof KR, Blom J, Band GPH, Kok A. Error-related brain potentials are differentially related to awareness of response errors: Evidence from an antisaccade task. Psychophysiology. 2001; 38(5):752–760. DOI: 10.1111/1469-8986.3850752 [PubMed: 11577898]

Olvet DM, Hajcak G. The stability of error-related brain activity with increasing trials. Psychophysiology. 2009; 46(5):957–961. DOI: 10.1111/j.1469-8986.2009.00848.x [PubMed: 19558398]

Ordaz SJ, Fritz BL, Forbes EE, Luna B. The influence of pubertal maturation on antisaccade performance. Developmental Science. 2017

Overbeek T, Nieuwenhuis S, Richard K. Dissociable components of error processing: On the functional significance of the Pe vis-à-vis the ERN/Ne. Journal of Psychophysiology. 2005; 19(4):319–329. DOI: 10.1027/0269-8803.19.4.319

Pailing PE, Segalowitz SJ. The effects of uncertainty in error monitoring on associated ERPs. Brain and Cognition. 2004a; 56(2):215–233. DOI: 10.1016/j.bandc.2004.06.005 [PubMed: 15518937]

Pailing PE, Segalowitz SJ. The error-related negativity as a state and trait measure: Motivation, personality, and ERPs in response to errors. Psychophysiology. 2004b; 41(1):84–95. DOI: 10.1111/1469-8986.00124 [PubMed: 14693003]

Pailing PE, Segalowitz SJ, Dywan J, Davies PL. Error negativity and response control. Psychophysiology. 2002; 39(2):198–206. DOI: 10.1111/1469-8986.3920198 [PubMed: 12212669]

Parker JG, Rubin KH, Erath SA, Wojslawowicz JC, Buskirk AA. Peer Relationships, Child Development, and Adjustment: A Developmental Psychopathology Perspective. In: Cicchetti D, Cohen DJ, editorsDevelopmental Psychopathology. John Wiley & Sons, Inc; 2015. 419–493.

Perrin F, Pernier J, Bertnard O, Giard MH, Echallier JF. Mapping of scalp potentials by surface spline interpolation. Electroencephalography and Clinical Neurophysiology. 1987; 66(1):75–81. DOI: 10.1016/0013-4694(87)90141-6 [PubMed: 2431869]

Perrin F, Pernier J, Bertrand O, Echallier JF. Spherical splines for scalp potential and current density mapping. Electroencephalography and Clinical Neurophysiology. 1989; 72(2):184–187. DOI: 10.1016/0013-4694(89)90180-6 [PubMed: 2464490]

Petersen AC, Crockett L, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. Journal of Youth and Adolescence. 1988; 17(2):117–133. [PubMed: 24277579]

Potts GF. Impact of reward and punishment motivation on behavior monitoring as indexed by the error-related negativity. International Journal of Psychophysiology. 2011; 81(3):324–331. DOI: 10.1016/j.ijpsycho.2011.07.020 [PubMed: 21855583]

Proudfit GH, Inzlicht M, Mennin DS. Anxiety and error monitoring: the importance of motivation and emotion. Frontiers in Human Neuroscience. 2013; 7

Richardson C, Anderson M, Reid CL, Fox AM. Neural indicators of error processing and intraindividual variability in reaction time in 7 and 9 year-olds. Developmental Psychobiology. 2011; 53(3):256–265. DOI: 10.1002/dev.20518 [PubMed: 21400488]

Riesel A, Weinberg A, Endrass T, Kathmann N, Hajcak G. Punishment has a lasting impact on error-related brain activity. Psychophysiology. 2012; 49(2):239–247. DOI: 10.1111/j.1469-8986.2011.01298.x [PubMed: 22092041]

Riesel A, Weinberg A, Moran T, Hajcak G. Time course of error-potentiated startle and its relationship to error-related brain activity. Journal of Psychophysiology. 2013; 27(2):51–59. DOI: 10.1027/0269-8803/a000093

Roger C, Bénar CG, Vidal F, Hasbroucq T, Burle B. Rostral cingulate zone and correct response monitoring: ICA and source localization evidences for the unicity of correct-and error-negativities. Neuroimage. 2010; 51(1):391–403. [PubMed: 20152906]

Rubin KH, Bukowski WM, Parker JG. Handbook of Child Psychology. John Wiley & Sons, Inc; 2007. Peer Interactions, Relationships, and Groups.

Santesso DL, Segalowitz SJ. Developmental differences in error-related ERPs in middle- to late-adolescent males. Developmental Psychology. 2008; 44(1):205–217. DOI: 10.1037/0012-1649.44.1.205 [PubMed: 18194018]

Santesso DL, Segalowitz SJ, Schmidt LA. Error-related electrocortical responses in 10-year-old children and young adults. Developmental Science. 2006; 9(5):473–481. DOI: 10.1111/j.1467-7687.2006.00514.x [PubMed: 16911449]

Scheffers MK, Coles MGH. Performance monitoring in a confusing world: Error-related brain activity, judgments of response accuracy, and types of errors. Journal of Experimental Psychology: Human Perception and Performance. 2000; 26(1):141–151. DOI: 10.1037/0096-1523.26.1.141 [PubMed: 10696610]

Schillinger FL, Smedt BD, Grabner RH. When errors count: an EEG study on numerical error monitoring under performance pressure; ZDM. 2015. 1–13.

Schroder HS, Moran TP, Moser JS, Altmann EM. When the rules are reversed: Action-monitoring consequences of reversing stimulus–response mappings. Cognitive, Affective, & Behavioral Neuroscience. 2012; 12(4):629–643. DOI: 10.3758/s13415-012-0105-y

Schultz W. Multiple dopamine functions at different time courses. Annual Review of Neuroscience. 2007; 30(1):259–288. DOI: 10.1146/annurev.neuro.28.061604.135722

Segalowitz SJ, Dywan J. Individual differences and developmental change in the ERN response: implications for models of ACC function. Psychological Research PRPF. 2009; 73(6):857–870. DOI: 10.1007/s00426-008-0193-z

Sergeant J. The cognitive-energetic model: an empirical approach to Attention-Deficit Hyperactivity Disorder. Neuroscience & Biobehavioral Reviews. 2000; 24(1):7–12. DOI: 10.1016/S0149-7634(99)00060-3 [PubMed: 10654654]

Shulman EP, Smith AR, Silva K, Icenogle G, Duell N, Chein J, Steinberg L. The dual systems model: Review, reappraisal, and reaffirmation. Developmental Cognitive Neuroscience. 2016; 17(Supplement C):103–117. DOI: 10.1016/j.dcn.2015.12.010 [PubMed: 26774291]

Spear LP. The adolescent brain and age-related behavioral manifestations. Neuroscience & Biobehavioral Reviews. 2000; 24(4):417–463. DOI: 10.1016/S0149-7634(00)00014-2 [PubMed: 10817843]

Steiger JH. Tests for comparing elements of a correlation matrix. Psychological Bulletin. 1980; 87(2): 245.

Steinberg L. Risk taking in adolescence new perspectives from brain and behavioral science. Current Directions in Psychological Science. 2007; 16(2):55–59. DOI: 10.1111/j. 1467-8721.2007.00475.x

Steinberg L. A social neuroscience perspective on adolescent risk-taking. Developmental Review. 2008; 28(1):78–106. DOI: 10.1016/j.dr.2007.08.002 [PubMed: 18509515]

Steinberg L, Albert D, Cauffman E, Banich M, Graham S, Woolard J. Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model. Developmental Psychology. 2008; 44(6):1764–1778. DOI: 10.1037/a0012955 [PubMed: 18999337]

Stürmer B, Nigbur R, Schacht A, Sommer W. Reward and punishment effects on error processing and conflict control. Frontiers in Psychology. 2011; 2doi: 10.3389/fpsyg.2011.00335

Tamnes CK, Østby Y, Fjell AM, Westlye LT, Due-Tønnessen P, Walhovd KB. Brain maturation in adolescence and young adulthood: Regional age-related changes in cortical thickness and white matter volume and microstructure. Cerebral Cortex. 2010; 20(3):534–548. DOI: 10.1093/cercor/bhp118 [PubMed: 19520764]

Tamnes CK, Walhovd KB, Torstveit M, Sells VT, Fjell AM. Performance monitoring in children and adolescents: A review of developmental changes in the error-related negativity and brain maturation. Developmental Cognitive Neuroscience. 2013; 6:1–13. DOI: 10.1016/j.dcn. 2013.05.001 [PubMed: 23777674]

Tarazi FI, Tomasini EC, Baldessarini R. Postnatal development of dopamine D1-like receptors in rat cortical and striatolimbic brain regions: an autoradiographic study. Developmental Neuroscience. 1999; 21(1):43–49. [PubMed: 10077701]

Taylor SF, Stern ER, Gehring WJ. Neural systems for error monitoring: Recent findings and theoretical perspectives. The Neuroscientist. 2007; 13(2):160–172. DOI: 10.1177/1073858406298184 [PubMed: 17404376]

Thomas MS, Annaz D, Ansari D, Scerif G, Jarrold C, Karmiloff-Smith A. Using developmental trajectories to understand developmental disorders. Journal of Speech, Language, and Hearing Research. 2009; 52(2):336–358.

Torpey DC, Hajcak G, Klein DN. An examination of error-related brain activity and its modulation by error value in young children. Developmental Neuropsychology. 2009; 34(6):749–761. DOI: 10.1080/87565640903265103 [PubMed: 20183731]

Tripp G, Wickens JR. Research review: Dopamine transfer deficit: a neurobiological theory of altered reinforcement mechanisms in ADHD. Journal of Child Psychology and Psychiatry. 2008; 49(7): 691–704. DOI: 10.1111/j.1469-7610.2007.01851.x [PubMed: 18081766]

Van Meel CS, Van Heijningen CAA. The effect of interpersonal competition on monitoring internal and external error feedback. Psychophysiology. 2010; 47(2):213–222. DOI: 10.1111/j. 1469-8986.2009.00944.x [PubMed: 20074085]

Van Veen V, Carter CS. The timing of action-monitoring processes in the anterior cingulate cortex. Journal of Cognitive Neuroscience. 2002; 14(4):593–602. DOI: 10.1162/08989290260045837 [PubMed: 12126500]

Vidal F, Burle B, Bonnet M, Grapperon J, Hasbroucq T. Error negativity on correct trials: a reexamination of available data. Biological Psychology. 2003; 64(3):265–282. DOI: 10.1016/S0301-0511(03)00097-8 [PubMed: 14630407]

Vidal F, Hasbroucq T, Grapperon J, Bonnet M. Is the 'error negativity' specific to errors? Biological Psychology. 2000; 51(2–3):109–128. DOI: 10.1016/S0301-0511(99)00032-0 [PubMed: 10686362]

Weinberg A, Meyer A, Hale-Rude E, Perlman G, Kotov R, Klein DN, Hajcak G. Error-related negativity (ERN) and sustained threat: Conceptual framework and empirical evaluation in an adolescent sample. Psychophysiology. 2016; 53(3):372–385. DOI: 10.1111/psyp.12538 [PubMed: 26877129]

Weinberg A, Olvet DM, Hajcak G. Increased error-related brain activity in generalized anxiety disorder. Biological Psychology. 2010; 85(3):472–480. DOI: 10.1016/j.biopsycho.2010.09.011 [PubMed: 20883743]

Weinberg A, Riesel A, Hajcak G. Integrating multiple perspectives on error-related brain activity: The ERN as a neural indicator of trait defensive reactivity. Motivation and Emotion. 2012; 36(1):84–100.

Wessel JR, Ullsperger M. Selection of independent components representing event-related brain potentials: A data-driven approach for greater objectivity. NeuroImage. 2011; 54(3):2105–2115. DOI: 10.1016/j.neuroimage.2010.10.033 [PubMed: 20965258]

Westlye LT, Walhovd KB, Dale AM, Bjørnerud A, Due-Tønnessen P, Engvig A, Fjell AM. Life-span changes of the human brain white matter: diffusion tensor imaging (DTI) and volumetry. Cerebral Cortex. 2010; 20(9):2055–2068. DOI: 10.1093/cercor/bhp280 [PubMed: 20032062]

Wiersema JR, van der Meere JJ, Roeyers H. Developmental changes in error monitoring: An event-related potential study. Neuropsychologia. 2007; 45(8):1649–1657. DOI: 10.1016/j.neuropsychologia.2007.01.004 [PubMed: 17303199]

Yeung N, Botvinick MM, Cohen JD. The Neural basis of error detection: conflict monitoring and the error-related negativity. Psychological Review. 2004; 111(4):931–959. DOI: 10.1037/0033-295X.111.4.931 [PubMed: 15482068]

Yeung N, Holroyd CB, Cohen JD. ERP correlates of feedback and reward processing in the presence and absence of response choice. Cerebral Cortex. 2005; 15(5):535–544. DOI: 10.1093/cercor/bhh153 [PubMed: 15319308]

Zajonc RB. Social facilitation. Science. 1965; 149:269–274. DOI: 10.1126/science.149.3681.269 [PubMed: 14300526]

Zajonc RB, Paulus PB. Psychology of group influence. Hillsdale, NJ: Erlbaum; 1980.
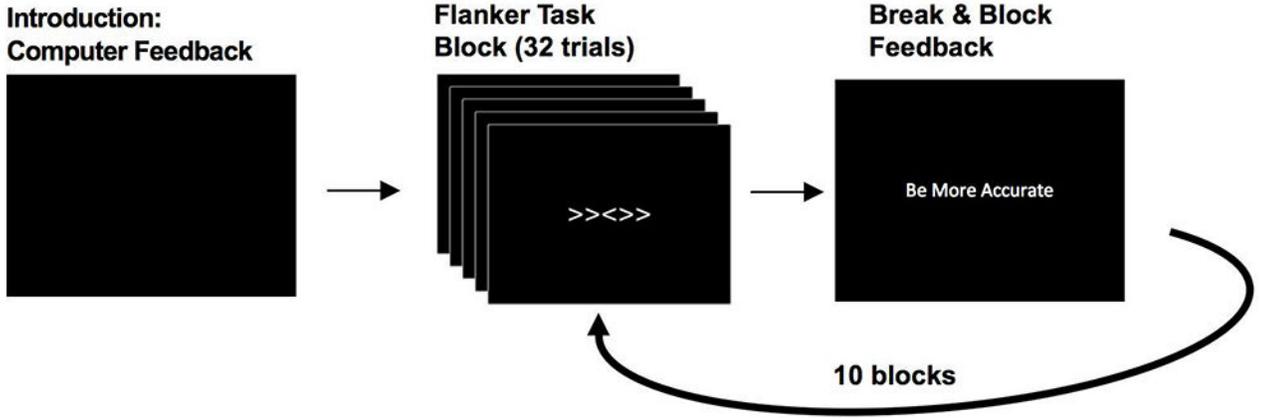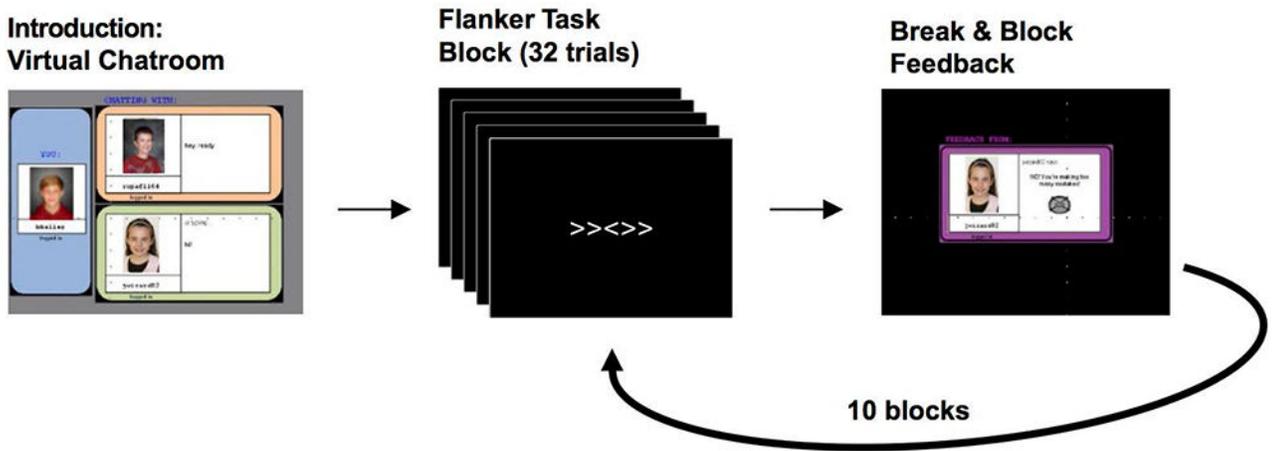
Figure 1.
Experimental paradigm. Adolescents received 10 feedback breaks (e.g., after each block of 32 trials) for each the social and nonsocial condition. A) Depiction of trial sequence for the flanker task in the nonsocial condition. Adolescents were told that they would receive computer-based feedback. Feedback that adolescents received was dependent on accuracy on the prior block. B) Depiction of the flanker task in the social condition. Adolescents were told that two other adolescents would be observing and monitoring their performance (i.e., accuracy, response times) during the flanker task. Like the nonsocial condition, feedback that adolescents received in the social condition was dependent on accuracy on the previous block.

Error-Related Negativity (ERN)

Error Positivity (Pe)
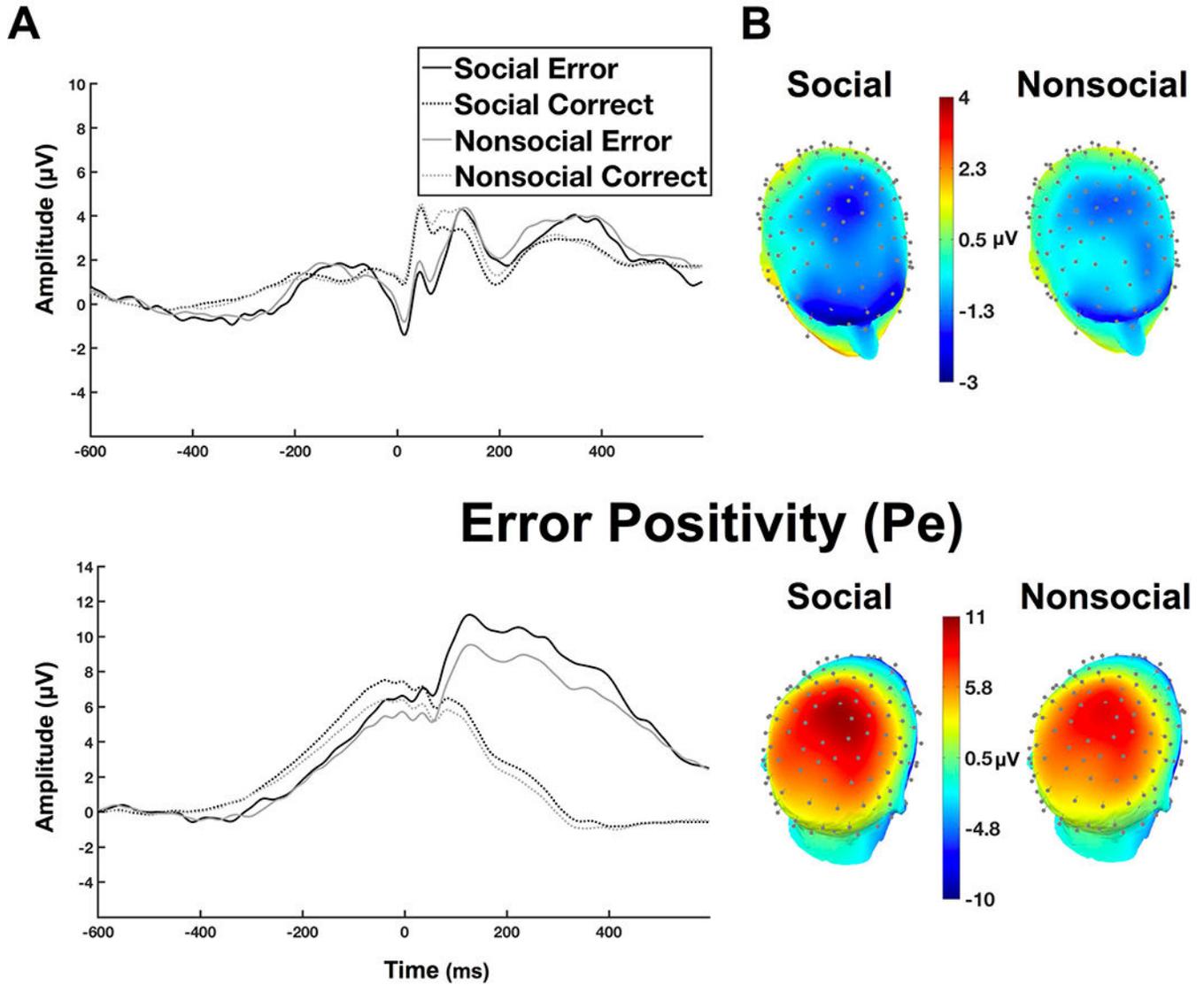
Figure 2.
A) Response-locked event-related potential (ERP) waveforms for correct and error responses in the nonsocial condition and social condition. The top row is the fronto-central electrode grouping where the ERN was maximal. The bottom row is the centro-parietal electrode grouping, where Pe was maximal. B) Topographic plots of the ERN 20 ms post-response (top) and Pe 250 ms post-response (bottom).

Younger Adolescents
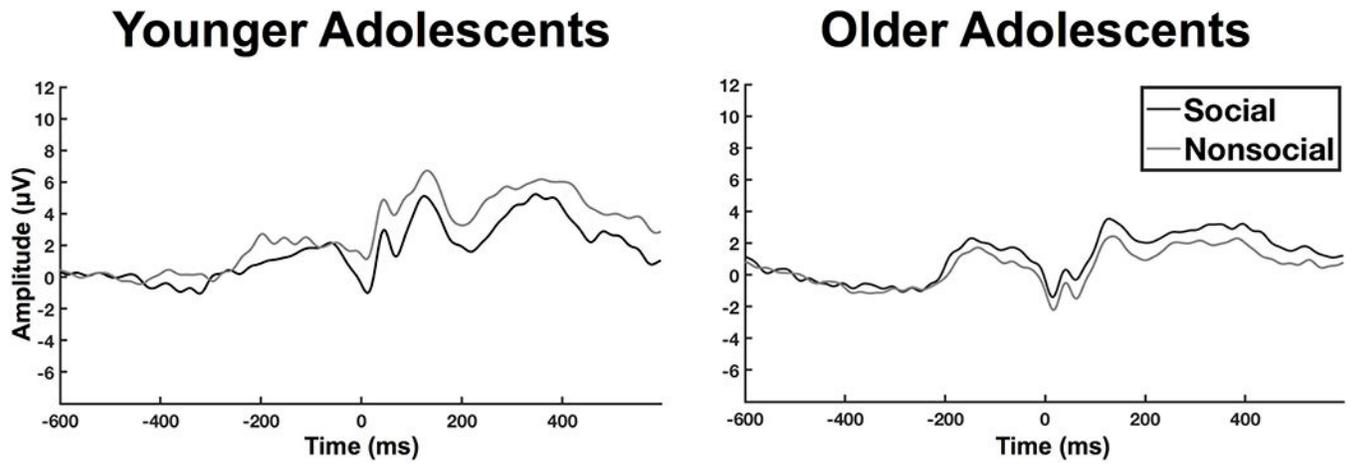
Older Adolescents

Figure 3.
Waveforms for the ERN among older adolescents (right) and younger adolescents (left) for the nonsocial condition (grey) and social condition (black). The ERN was significantly larger in the social condition as compared to the nonsocial condition for younger adolescents, $p < .001$, but not for older adolescents, $p > .20$.
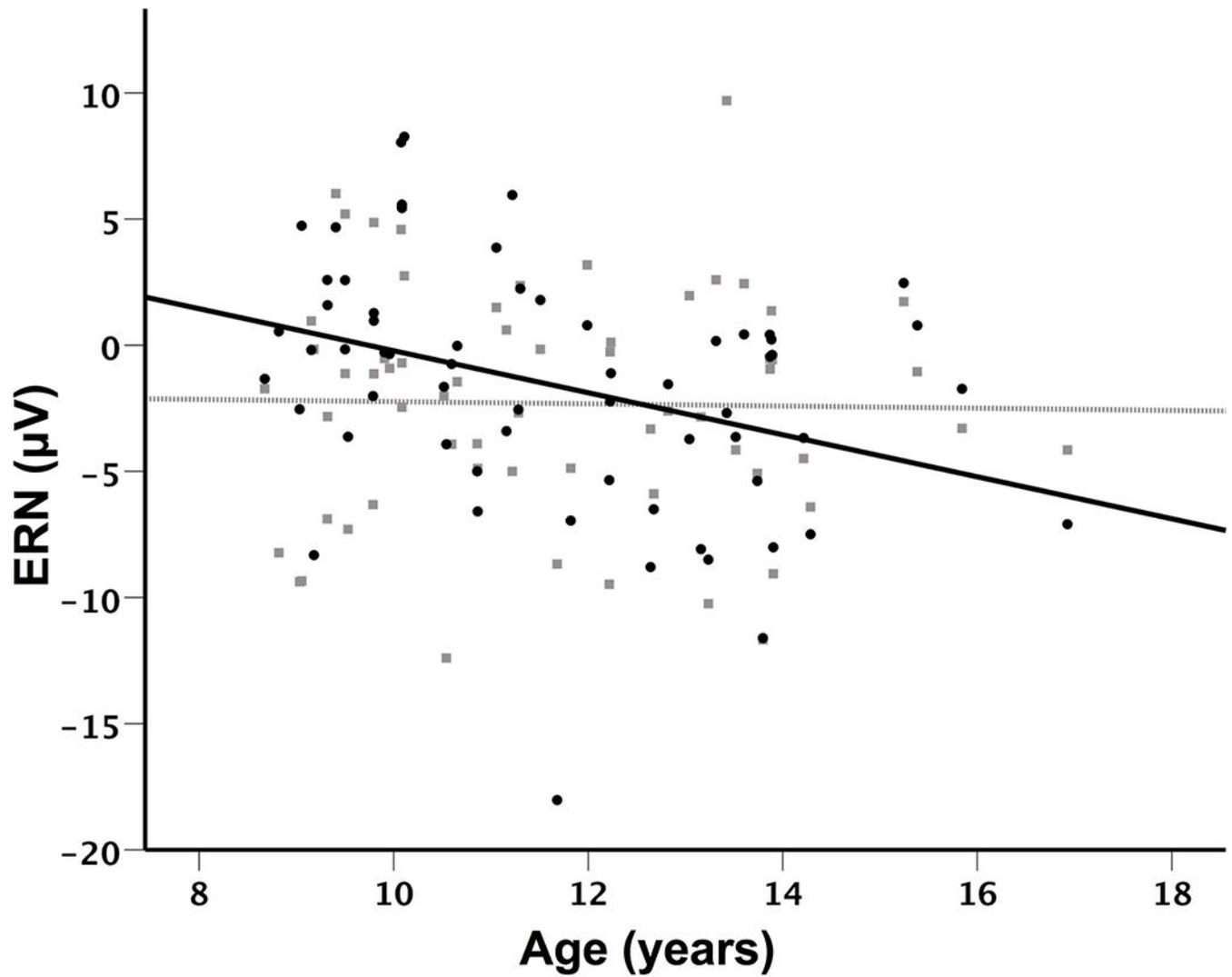
Figure 4.
Scatterplot depicting the relation between the ERN in the nonsocial condition (black circles, black line) and the ERN in the social condition (grey squares, grey line) with age. The relation between the nonsocial ERN and age reached significance, $p = .005$. In contrast, the ERN in the social condition was unrelated to age, $p = .88$.

Table 1

Raw means for behavioral performance and event-related potential (ERP) measures for the nonsocial and social condition (standard deviations).

	Nonsocial	Social	*F*
Behavior Measures			
Effort (au)	7.40 (2.04)	8.25 (1.77)	17.20[*]
Error response time (ms)	410.65 (92.10)	389.11 (74.98)	10.11[*]
Correct response time (ms)	498.50 (100.13)	475.88 (83.88)	10.11[*]
Accuracy (%)	86.20 (5.84)	86.66 (5.92)	0.66
Error response variability	122.47 (66.86)	110.11 (58.79)	6.50[*]
Correct response variability	130.37 (46.05)	118.73 (34.44)	6.50[*]
Post-error slowing (ms)	4.78 (41.67)	8.41 (33.99)	0.41
ERP's (μV)			
ERN	−1.62 (4.81)	−2.31 (4.58)	1.06
CRN	0.43 (3.15)	0.42 (3.36)	1.06
ERN	−2.05 (3.83)	−2.74 (3.68)	1.41
Pe	7.70 (4.83)	9.25 (5.43)	7.65[*]
Correct Pe	0.27 (2.65)	0.72 (3.09)	7.65[*]
Pe	7.44 (4.70)	8.53 (4.38)	4.81[*]

Note: au: arbitrary units;

[*] Indicates $p < .05$; ERN refers to error minus correct trials in the time window of the ERN. Pe refers to error minus correct trials in the time window of the Pe. Identical F-values represent a main effect across response type.