### Title

The influence of social motivation on neural correlates of cognitive control in girls




### Journal

Developmental Psychobiology, 63(5)

### ISSN

0012-1630



### Authors

Barker, Tyson V
Buzzell, George A
Troller-Renfree, Sonya V
et al.



### Publication Date

2021-07-01

### DOI

10.1002/dev.22086

Peer reviewed

HHS Public Access

Author manuscript

*Dev Psychobiol*. Author manuscript; available in PMC 2022 May 06.

Published in final edited form as:

*Dev Psychobiol*. 2021 July ; 63(5): 1611–1625. doi:10.1002/dev.22086.

# The influence of social motivation on neural correlates of cognitive control in girls

**Tyson V. Barker**[1], **George A. Buzzell**[2], **Sonya V. Troller-Renfree**[3], **Lindsay C. Bowman**[4], **Daniel S. Pine**[5], **Nathan A. Fox**[6]

[1]Department of Psychology, University of Oregon, Eugene, OR, USA

[2]Department of Psychology, Florida International University, Miami, FL, USA

[3]Department of Biobehavioral Sciences, Teachers College at Columbia University, New York, NY, USA

[4]Department of Psychology, University of California, Davis, CA, USA

[5]Emotion and Development Branch, Intramural Research Program, National Institute of Mental Health (NIMH), Bethesda, MD, USA

[6]Department of Human Development and Quantitative Methodology, University of Maryland, College Park, MD, USA

## Abstract

Motivation influences cognitive control, particularly in childhood and adolescence. Previous work finds that the error-related negativity (ERN), an event-related potential (ERP) linked to cognitive control following errors, is influenced by social motivation. However, it is unclear whether the influences of social motivation on the ERN extend to stimulus-locked neural correlates of cognitive control. This study reexamines how social motivation influences cognitive control in adolescence by exploring motivational influences on two stimulus-locked ERPs; the N2 and P3. Adolescent girls (8–17 years of age) completed a flanker task under two different conditions. In the social condition, girls were led to believe that they were evaluated by a peer during a flanker task. In the nonsocial condition, girls completed a flanker task while evaluated by a computer. Results revealed that all girls exhibited a larger P3 in social as compared to nonsocial contexts, whereas the N2 was not different between contexts. In addition, the largest P3 enhancements were observed among younger girls. These findings suggest that social motivation influences some ERP components related to cognitive control, and such influences change across development. Additionally, findings suggest the importance of including multiple ERPs when interpreting the functional significance of motivation on cognitive control.

## 1 | INTRODUCTION

Adolescence, which is typically defined by the onset of puberty (Peper & Dahl, 2013), is a transition period in development characterized by dramatic hormonal, physical,

**Correspondence:** Tyson V. Barker, Department of Psychology, University of Oregon, 6217 University of Oregon, Eugene, OR 97403-1227, USA. tysonb@uoregon.edu.

and psychological changes (Crone & Dahl, 2012). 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; Ernst et al., 2006; Nelson et al., 2005). Such neural reorganization is theorized to drive heightened social, affective, and reward processing (Dahl & Crone, 2012), as well as the engagement of cognitive control (Breiner et al., 2018; Shulman et al., 2016). As such, it is important to utilize neural markers of cognitive control to explore the influence of social motivation on cognitive control systems across adolescence.

Across a series of studies, we found that the error-related negativity (ERN), an event-related potential (ERP) following error commission (Falkenstein et al., 1991; Gehring et al., 1993), was enhanced by social motivation in both adults (Barker et al., 2015) as well as children and adolescents (Barker et al., 2018; Buzzell, Troller-Renfree, et al., 2017). We also observed changes in the sensitivity of the ERN across development such that the largest ERN enhancements were observed among older children (Barker, Troller-Renfree, et al., 2018). Given the purported link between the ERN, reward processing, and dopamine production in the basal ganglia (Holroyd & Coles, 2002; Holroyd & Yeung, 2012), these findings add to the growing literature of developmental changes in reward processing in the adolescent period both in humans (Blakemore et al., 2010; Ernst & Fudge, 2009) and animal models (Andersen, 2003; Andersen et al., 2000). However, other theories suggest that the ERN is one neural component of a larger cognitive control system associated with a range of other physiologic indices (Carter & Veen, 2007; Cavanagh & Frank, 2014), which can also be modulated by motivational factors (Botvinick & Braver, 2015). Thus, it is critical to examine the full range of ERPs associated with cognitive control to understand comprehensively how social motivation influences cognitive control across development.

### 1.1 | Social motivation and cognitive control across development

There is an increasing interest in understanding how motivation influences cognitive control across development (Braver et al., 2014; Bush et al., 2000; Ernst et al., 2006; Pessoa, 2009). Motivation is typically defined as the modulatory impact of incentives on physiology and behavior (Berridge, 2004; Botvinick & Braver, 2015; Roesch & Olson, 2004). Many studies find that positive incentives affect cognitive control and produce improvements in performance (Boehler et al., 2014; Engelmann et al., 2009; Krebs et al., 2013; Leotti & Wager, 2010; Libby & Lipe, 1992), as well as changes in goal-directed strategies deployed to implement cognitive control (Chiew & Braver, 2016; Krebs et al., 2010). Motivational incentives also increase physiological arousal during the implementation of cognitive control (Chiew & Braver, 2013, 2014), and are associated with increases in related neural activity (Locke & Braver, 2008; Small et al., 2005). Increasing research focuses on the intersection of motivation and cognitive control during childhood and adolescence (Crone & Dahl, 2012), typically finding developmental changes in motivation (Bellis et al., 2001; Lenroot & Giedd, 2010; Luna & Wright, 2016; Shulman et al., 2016), and cognitive control (Luna et al., 2004), during this period. Thus, it is critical to understand how social factors influence cognitive control throughout development.

Less work examines how social-motivational factors influence cognitive control across development (Crone, 2014). The presence of conspecifics reliably increases motivation and arousal in humans (Blascovich & Tomaka, 1996; Triplett, 1898) and animal models (Calcagnetti & Schechter, 1992; Zajonc, 1965). Here, we define aspects of social motivation that allow the mere presence of conspecifics to influence physiology and behavior[1] (Geen, 1991, 1995; Hayden et al., 2007; Kirschbaum et al., 1993). Neuroimaging studies find that processing of social information increases activation in regions implicated in cognitive control, such as the cingulate and prefrontal cortex (Gunther Moor et al., 2010, 2012; Somerville et al., 2006). This overlap in the neural systems supporting social information processing and cognitive control suggests a close link between cognitive control and social motivation (Ninomiya et al., 2018). However, limited research has assessed how social motivation directly influences the cognitive control system.

### 1.2 | Influence of motivation on psychophysiological indicators of cognitive control

Many studies investigate motivational influences on psychophysiological indicators of cognitive control (see Botvinick & Braver, 2015 for a review). One stimulus-locked ERP indexing cognitive control is the N2, which is observed approximately 250–400 ms after stimulus presentation, and is maximal at frontal-central electrodes (Bruno et al., 2007; Folstein & Van Petten, 2008; Kopp et al., 1996; Nieuwenhuis et al., 2003; Yeung et al., 2004). One subtype of the N2, called the *conflict N2* (Folstein & Van Petten, 2008), is typically generated using tasks like the flanker task (Eriksen & Eriksen, 1974). The conflict N2 is larger (i.e., more negative) on incongruent trials, where flanking arrows are incongruent with the target arrow (Bartholow et al., 2005; Donkers & van Box tel, 2004; Folstein & Van Petten, 2008; van Veen et al., 2001). Theoretical accounts of the conflict N2 suggest this component indexes detection of response conflict, an integral aspect of cognitive control (Botvinick et al., 2001; Donkers & van Box tel, 2004; Larson et al., 2014; Ullsperger et al., 2014; Yeung et al., 2004). Localization studies of the N2 suggest the component is primarily generated by the anterior cingulate cortex (ACC; Carter & Veen, 2007; Gruendler et al., 2011; Nieuwenhuis et al., 2003; van Veen et al., 2001), a region known to integrate motivation and cognitive control (Bush et al., 2000; Holroyd & Yeung, 2012).

Another ERP component associated with cognitive control is the P3, which occurs approximately 300–600 ms after stimulus presentation and is maximal at central-parietal electrodes (Polich, 2007; Pritchard, 1981; Sutton et al., 1965). The P3[2] is modulated by numerous factors such as stimulus probability and task relevance (Donchin et al., 1978; Polich, 2007; Squires et al., 1977). Theoretical accounts of the P3 suggest this component reflects an awareness of motivationally significant stimuli (Picton, 1992; Polich, 2007),

[1]Contextual factors, such as the presence of social partners, can lead to increases in both approach motivation and avoidance motivation (Blascovich et al., 1999; Geen, 1991; Gray & McNaughton, 2003), and the balance of approach and avoidance motivation is likely influenced by individual differences (Barker, Buzzell, et al., 2018). Here, we focus on social motivation as a unitary construct which is associated with increases in physiological arousal (Blascovich et al., 1999).

[2] It is important to note that the P3 is typically divided into two subcomponents (Polich, 2007); the P3a, which is elicited in response to a surprising stimulus, and the P3b, also referred to as the "classic P3," which is elicited in response to neural processing of a task-relevant stimulus. The P3 elicited on the flanker task, particularly when incongruent and congruent trials are equiprobable, likely reflects the motivational salience of the flanker stimuli (Nieuwenhuis, Aston-Jones, & Cohen, 2005; Ridderinkhof & Molen, 1995), which most closely resembles the "classic P3." Thus, we refer to this component simply as the P3.

which involves cognitive processes such as context-updating (Donchin & Coles, 1988), and mobilization of cognitive resources in relation to goal-directed actions (Nieuwenhuis et al., 2005). The P3 is also generated during the flanker task and is larger on incongruent trials as compared to congruent trials (Clayson & Larson, 2011; Frühholz et al., 2011; Rosch & Hawk, 2013). The P3 generated on high-conflict trials likely indicates a recognition of greater need for control following response decision (Clayson & Larson, 2011; Nieuwenhuis et al., 2003; Ridderinkhof & Molen, 1995). Localization studies of the P3 suggest that the P3 is generated by a distributed network, including the ACC, anterior insula, and inferior temporal cortex (Bledowski et al., 2004; Linden, 2005; Nee et al., 2007; Nieuwenhuis et al., 2005; Tarkka & Stokic, 1998; Volpe et al., 2007).

The need to further explore the influence of social motivation on the N2 and P3 across development is supported by findings that that N2/P3 complex following stimulus presentation resembles the ERN following an error (Cavanagh & Frank, 2014; Cavanagh et al., 2012; Overbeek et al., 2005; Ridderinkhof et al., 2009; Yeung et al., 2004). In addition, location studies of the N2 and P3 among children and adolescents find that these components share common neural generators with the ERN, namely the anterior and posterior cingulate cortex (Buzzell, Richards, et al., 2017; Herrmann et al., 2004; Ladouceur et al., 2007). These findings suggest that the N2 and P3 may reflect psychological processes related to the ERN. Thus, to functionally interpret the effect of motivation on neural processes across development (Cacioppo & Tassinary, 1990; Richter & Slade, 2017), it is important to examine whether the effects of social motivation shown to influence the ERN also influence the N2 and P3, other ERPs related to cognitive control in youth.

Many studies find that incentives, such as monetary reward, enhance both N2 and P3 magnitude (Amodio et al., 2008; Begleiter et al., 2007; Boksem et al., 2006; Carrillo-de-la-Peña & Cadaveira, 2000; Goldstein et al., 2006; Kleih et al., 2010; Potts, 2011; Ramsey & Finn, 1997). However, relatively little research examines such effects in youth. Among children and adolescents, some find that monetary incentives increase N2 magnitude (Groom et al., 2010) while others find no influence of incentives on N2 magnitude (Rosch & Hawk, 2013). In contrast, there is more consistent evidence that the P3 is enhanced by motivational factors in children and adolescents (Groom et al., 2010; Rosch & Hawk, 2013). To the best of our knowledge, no work examines social-motivational influences on the N2 and P3 in youth.

### 1.3 | Current study

To examine social influences on the neural correlates of cognitive control in children and adolescents, we utilized previously published data (Barker, Troller-Renfree, et al., 2018; Bowers et al., 2018) to explore motivational influences on two stimulus-locked ERPs: the N2 and P3. We recruited girls between 8 and 17 years of age. This age range was chosen to capture variability in pubertal development, thought to influence both motivation and cognitive control (Peper & Dahl, 2013).[3] Specifically, the younger age range was chosen as 8–10 years of age, which marks the onset of puberty in girls (Sun et al., 2002). The upper

[3]Girls also completed the Pubertal Development Scale (PDS; Petersen et al., 1988), a self-report measure of pubertal development. Similar to previous research examining pubertal development across adolescence (Sun et al., 2002), the present sample demonstrated

age range of 17 years was chosen to limit confounds of additional social transitions (e.g., transitioning from high school to college). Only girls participated in the current study due to a number of gender differences in brain and behavior that exist during adolescence, including differences in pubertal development (Sun et al., 2002), differences in brain development (Giedd et al., 1999), difference in social motivation and risk taking (Gullone & Moore, 2000), and differences in the relation between hormones and behavior (Forbes et al., 2010). We created a task in which girls were led to believe that they were being observed and evaluated by peers via a webcam in a virtual chatroom (Barker, Troller-Renfree, et al., 2018; Buzzell, Troller-Renfree, et al., 2017). In the social condition, girls were led to believe that two other adolescents were observing and evaluating their performance during the completion of a flanker task. In the nonsocial condition, girls completed the flanker task while being evaluated by a computer.

Based on our previous work finding that the ERN is enhanced in social contexts (Barker, Troller-Renfree, et al., 2018; Buzzell, Troller-Renfree, et al., 2017), as well as findings that the N2 and P3 are enhanced by motivational contexts (Groom et al., 2010; Rosch & Hawk, 2013), we hypothesized that the N2 and P3 would be enhanced in social contexts as compared to nonsocial contexts. In addition, based on our previous finding that younger girls exhibited larger influences of social context on error monitoring than older girls (Barker, Troller-Renfree, et al., 2018), we hypothesized we would similarly observe developmental changes in the sensitivity of the N2 and P3 to social contexts such that the largest enhancements would be observed among younger girls.

## 2 | METHODS

### 2.1 | Participants

This study was part of a larger project examining social and cognitive development, and the effect of social context on the ERN are reported elsewhere (Barker, Troller-Renfree, et al., 2018). Participants were 76 girls ($M_{age}$ = 11.87 years; $SD$ = 2.2 years; range = 8.7–17.1 years). There were no differences in age between girls who completed the social condition girls ($M_{age}$ = 11.87 years; $SD$ = 2.2 years) and the nonsocial condition ($M_{age}$ = 11.87 years; $SD$ = 2.2 years).

Participants were excluded if they reported that they were not deceived by the social manipulation ($n$ = 4). In addition, 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 analyses, five additional subjects were excluded due to having fewer than 30 artifact-free incongruent trials and 30 artifact-free congruent trials in each condition (Clayson & Larson, 2013). Thus, the final sample for EEG analyses was 63 participants.

wide variability across the age range of the current sample (8–17 years of age) with no floor or ceiling effects ($M$ = 2.20 $SD$ = 0.87; range = 1.10–3.90). Due to high correlations with age, $r$(61) = 0.81, pubertal development was not included in analyses.

## 2.2 | Experimental design

### 2.2.1 | 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 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, Olvet, & Hajcak, 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 was between 75% and 90%, participants received a message that they were doing a good job.

## 2.3 | Procedure

Procedures are described in more detail elsewhere (Barker, Troller-Renfree, et al., 2018). Briefly, girls were fitted with an EEG net and performed the flanker task in one of two conditions, which were counterbalanced across participants. During the nonsocial condition, girls were informed that they would be receiving computer-generated feedback about their performance, and they were asked to adjust their performance based on the feedback (see Figure 1). The feedback received was based on the participant's accuracy on the previous block (block-level feedback; no trial-level feedback was provided). In the social condition, girls were informed that two other similar-aged peers located in another lab would be observing them through a webcam while they played the flanker task, and that the peers located in the other lab would be giving feedback about their performance. However, in actuality, girls were not observed by peers, and all feedback was computer generated.

## 2.4 | EEG collection and data reduction

Continuous EEG was recorded using a 128-channel Hydrocel Geodesic Sensor Net and sampled at 250 Hz using the EGI software (Electrical Geodesic, Inc). Before data collection, all electrode impedances were reduced to below 50 kΩ. All electrodes were referenced online to Cz and re-referenced to an average reference offline. The data were filtered offline using a digital band-pass FIR filter from 0.3 to 30 Hz.

Stimulus-locked epochs on correct trials only were segmented separately for congruent and incongruent trials from 200 ms before stimulus presentation to 800 ms after stimulus presentation (1,000 ms total). At the trial level, 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. Participants needed at least 30 artifact-free incongruent trials and 30 artifact-free congruent trials for each respective condition to be included in analyses (Clayson & Larson, 2013). These artifact criteria were chosen based on an iterative process where different thresholds were tested prior to conducting any

analyses. There were no differences in the number of artifact-free congruent trials between conditions (nonsocial: $M = 135.49$, $SD = 21.59$, social: $M = 136.70$, $SD = 23.01$), $t(62) = 0.64$, $p = .52$, or the number of artifact-free incongruent trials between conditions (nonsocial condition: $M = 112.38$, $SD = 20.14$, social condition: $M = 115.13$, $SD = 21.48$), $t(62) = 1.45$, $p = .15$. Age was unrelated to the number of artifact-free error trials in either condition, $ps > 0.20$.

All waveforms were baseline corrected from 200 to 0 ms before stimulus presentation. Electrode sites for N2 and P3 analysis were determined based on previous research on N2 and P3 topography in developmental populations (Lahat et al., 2014; Lamm et al., 2012) which were also consistent with observed scalp distributions in the current study (see Figure 2 for electrode clusters). Mean amplitude of the N2 was quantified using an adaptive mean approach, centering a 25 ms window on each participant's most negative peak between 200 and 400 ms at the fronto-central electrode cluster (electrode numbers on 128 ch. geodesic net: 5, 6 [FCz], 7, 12, and 106); this process was performed separately for congruent and incongruent trials. Mean amplitude of the P3 was then quantified using an adaptive mean approach, centering a 100 ms window on each participant's the most positive peak between 300 and 700 ms at the centro-parietal electrode cluster (electrode numbers on 128 channel geodesic net: 31, 54, 55, 61, 62, 78 79, 80, 129 [Cz]); again, this process was performed separately for congruent and incongruent trials. Note that the adaptive mean approach retains the benefits of employing a mean amplitude, as opposed to peak amplitude method (i.e., mean amplitudes are less susceptible to noise; Luck & Kappenman, 2012). However, the adaptive mean approach is typically used when there is greater between-subjects variability in latency of ERP components, such as in developmental populations (Clayson et al., 2013).

### 2.5 | Data analysis

To examine social influences on the N2 and P3, multiple linear-mixed models (LMM) were conducted with participant intercept as a random effect, condition as (nonsocial, social), stimulus (incongruent, congruent) as repeated predictors, and age as a continuous predictor. All LMM models utilized an unstructured correlation matrix for the repeated fixed-effects due to differences in variance across repeated measurements. All models were fit using restricted maximum likelihood (REML) criterion. An LMM approach was utilized to allow for the incorporation of both developmental differences and experiment manipulation into the analyses. Similar results were obtained using traditional repeated measures analysis of variance approach. Where applicable, all significant interactions were corrected using Bonferroni method. All statistical analyses were conducted using SPSS (Version 25.0). To graphically display differences in N2/P3 across age, age grouping was computed using a median split (younger girls: $M_{age} = 9.96$ $SD = 0.75$; older girls: $M_{age} = 12.47$ $SD = 1.34$). Interactions were followed up using a region of significance analysis approach utilizing the Johnson–Neyman Procedure available in MEMORE v2.1 (Montoya, 2019). For LMM analyses, effect sizes were estimated using partial $\eta^2$ from similar general mixed model analyses. Cohen's $d$ was calculated as the effect size for correlational analyses. For region of significance analyses, 95% confidence intervals were computed.

# 3 | RESULTS

## 3.1 | Behavior

Table 1 displays the behavioral measures for the social and nonsocial condition. As expected, response times on incongruent trials were significantly slower than response times on congruent trials, $F(1, 61) = 249.66$, $p < .001$, partial $\eta^2 = 0.80$. In addition, there was a main effect of age such that increasing age was associated with faster response times regardless of social context or congruency, $F(1, 61) = 50.06$, $p < .001$, partial $\eta^2 = 0.45$. There was also a main effect of condition such that response times were faster in the social condition as compared to the nonsocial condition, $F(1, 61) = 10.76$, $p < .001$, partial $\eta^2 = 0.15$. However, no interactive effects with condition reached significance ($p$s > 0.07), indicating no effect of social contexts on response conflict.

Analysis of accuracy revealed a main effect of stimulus type such that adolescents were more accurate on congruent trials as compared to incongruent trials, $F(1, 61) = 198.30$, $p < .001$, partial $\eta^2 = 0.77$. This effect was qualified by a significant age x stimulus type interaction, $F(1, 61) = 4.73$, $p = .034$, partial $\eta^2 = 0.07$, indicating that increasing age was associated with better accuracy on congruent trials, for both the social condition, $r = 0.35$, $p = .005$, $d = 0.75$, and the nonsocial condition, $r = 0.48$, $p < .001$, $d = 1.09$. In contrast, age was not associated with accuracy on incongruent trials for either the social condition, $r = 0.04$, $p = .78$, $d = 0.08$, or the nonsocial condition, $r = -0.05$, $p = .71$, $d = 0.10$.

### 3.1.1 | N2—

Figure 3 displays the stimulus-locked waveforms for congruent and incongruent trials for the nonsocial and social condition. Analysis of the N2 on congruent and incongruent trials indicated a main effect of congruency such that the N2 on incongruent trials was larger (i.e., more negative) on incongruent trials as compared to congruent trials across both the social and nonsocial condition, $F(1, 61) = 4.21$, $p = .044$, partial $\eta^2 = 0.07$. In addition, there was a main effect of age such that N2 magnitude decreased across development for both conditions and stimulus types, $F(1, 61) = 6.47$, $p = .012$, partial $\eta^2 = 0.10$. However, there was no effect of social context on N2 magnitude, $F(1, 61) = 0.39$, $p = .54$, partial $\eta^2 = 0.01$, and no interactions with age reached significance ($p$s > 0.10).

### 3.1.2 | P3—

Analysis of the P3 indicated a main effect of stimulus type such that the P3 on incongruent trials was larger (i.e., more positive) than the P3 on congruent trials, $F(1, 61) = 82.75$, $p < .001$, partial $\eta^2 = 0.58$. In addition, there was a main effect of social context such that the P3 was larger in the social compared to the nonsocial condition, $F(1, 61) = 4.48$, $p = .038$, partial $\eta^2 = 0.07$; this was qualified by a significant condition x stimulus type interaction, $F(1, 61) = 10.36$, $p = .002$, partial $\eta^2 = 0.15$. Follow-up tests for each stimulus type revealed that the P3 was enhanced in the social condition as compared to the nonsocial condition for incongruent trials, $F(1, 61) = 8.77$, $p = .004$, partial $\eta^2 = 0.13$, but not for congruent trials, $F(1, 61) = 0.945$, $p = .34$, partial $\eta^2 = 0.02$. The follow-up test for each condition revealed that the P3 was larger on incongruent trials for both the social condition, $F(1, 61) = 79.37$, $p < .001$, partial $\eta^2 = 0.57$, as well as the nonsocial condition, $F(1, 61) = 44.09$, $p < .001$, partial $\eta^2 = 0.42$. Finally, there was also a condition x age interaction, $F(1, 61) = 4.52$, $p = .038$, partial $\eta^2 = 0.07$.[4] No other interactions reached significance.

Figure 4 presents the ERPs across conditions for younger adolescents and older adolescents. Region of significance analysis for incongruent trials indicated that the P3 was significantly larger in the social condition as compared to the nonsocial condition for girls from 8.67 years of age, $t(61) = 2.95$, $p = .004$, 95% CI [0.66, 3.43], through 12.72 years of age, $t(61) = 1.99$, $p = .05$, 95% CI [0, 1.71]. There were no signifcant differeces between conditions for girls above 12.72 years of age ($p$s > 0.05). However, for congruent trials, the P3 was significantly larger in the social condition as compared to the nonsocial condition for girls within a smaller age rage. Specifically, there were signficant differences bewteen conditions for congurent trials from 8.67 years of age, $t(61) = 2.59$, $p = .012$, 95% CI [0.39, 2.98], through 10.67 years of age, $t(61) = 1.99$, $p = .05$, 95% CI [0, 1.63]. There were no signifcant differences between conditions for girls above 10.67 years of age ($p$s > 0.05). Taken together, region of significance analyses suggest that younger adolecent girls, but not older adoelcent girls, exhibited enhancements of the P3 in the social condition as compared to the nonsocial condition.

## 4 | DISCUSSION

The purpose of the present study was to examine whether stimulus-locked neural components associated with cognitive control are sensitive to social-motivational factors across childhood and adolescence (Crone & Dahl, 2012). To this end, we examined the N2 and P3, two stimulus-locked event-related potentials (ERPs) related to cognitive control (Larson et al., 2014; Polich, 2007), that demonstrate developmental changes across adolescence (van Dinteren et al., 2014; Hoyniak, 2017). We tested whether these ERPs were enhanced in a social versus nonsocial context among adolescents. We found such an effect only for the P3, particularly on trials with high response conflict (incongruent trials). We also explored whether age influenced the effect of social context on the N2 and P3. In line with our hypotheses, the degree of P3 enhancement within the social context was influenced by age: the greatest P3 enhancements manifested in younger girls. Contrary to our hypothesis, we did not find the N2 to be influenced by social context or age.

We observed that the P3 was sensitive to both congruency and social-motivational influences and that such effects differed as a function of age. Such findings extend prior work in youth (Groom et al., 2010; Rosch & Hawk, 2013) and adults (Amodio et al., 2008; Begleiter et al., 2007; Boksem et al., 2006; Carrillo-dela-Peña & Cadaveira, 2000; Goldstein et al., 2006; Kleih et al., 2010; Potts, 2011; Ramsey & Finn, 1997). While the P3 is thought to index processing stages that *follow* stimulus evaluation and categorization (Kutas et al., 1977; Magliero et al., 1984; McCarthy & Donchin, 1981), the effects of social context on P3 magnitude could reflect the downstream results of motivation enhancing stimulus processing and categorization (Ridderinkhof & Molen, 1995) and/or increased attentional demands of completing the task within a social context (Polich, 2007). Such changes in general stimulus processing may be more generally associated with motivational changes in cognitive control tasks across adolescence.

[4]We also conducted analyses using residualized scores of the P3 by regressing the congruent P3 on the incongruent P3 for both conditions (Meyer et al., 2017). Results revealed extremely high correlations between subtraction score variables (e.g., incongruent P3 minus Congruent P3) and residualized score variables for both conditions, $r$s > 0.98. Accordingly, similar results were observed.

In addition, the degree of P3 enhancement in social contexts was largest in the youngest girls. One possibility for these findings is that children and younger adolescents are less able to initiate control processes automatically when needed as compared to older adolescents (Luna et al., 2004; Williams et al., 1999), and instead more strongly rely on conscious processes to guide goal-directed behavior based on contextual factors. However, it is important to note that we observed developmental changes in P3 sensitivity to social contexts for both congruent and incongruent trials. That is, while social context was shown to exhibit a selective enhancement of conflict-related neural processing for all participants (described above), developmental changes in the P3 to social contexts were not specific to conflict-related processing and instead must index neural activity that is similarly engaged on both high- and low-conflict trials.

It is interesting to note that the largest P3 effects of social context were observed among children and younger adolescents, which is in contrast to much functional neuroimaging work (i.e., functional magnetic reponse imaging; fMRI), which typically finds greater neural sensitivity to social contexts in mid-to-late adolescence (Chein et al., 2011; Smith et al., 2018), and greater cognitive control in the presence of peers among adolescents (Breiner et al., 2018). Changes in adolescent sensitivity to social contexts are theorized to be driven by the reorganization of neural circuits critical for social motivation and reward processing (Crone & Dahl, 2012; Ernst & Fudge, 2009; Nelson et al., 2016), and different development patterns between reward-related and cognitive-related regions of the brain (Shulman et al., 2016). However, there is debate about the exact timing of changes in reward processing and social motivation in adolescence (Pfeifer & Allen, 2012, 2016), and many brain and behavior changes associated with adolescence may occur much earlier in development than previously thought. For example, structural neuroimaging studies find that peak white matter density in the prefrontal cortex (PFC) is reached by 10–12 years of age in girls (Giedd et al., 1999). In addition, a recent meta-analysis found that younger adolescents (11–13 years of age) exhibit more risk-taking behaviors than older adolescents (14–19 years of age; Defoe et al., 2015). Taken together, these findings add support to the present findings that younger adolescents may be more sensitive to social influences than older adolescents. However, it is still an open question why the present findings differ greatly from other functional neuroimaging studies of peer influence in adolescence.

In contrast, no differences in N2 magnitude were observed between contexts among adolescents. A lack of motivational influences on the N2 is consistent with the study by Rosch and Hawk (2013), which found that monetary incentives did not influence N2 magnitude during a flanker task in children and adolescents. In contrast, Groom et al., (2010) found that monetary incentives increased N2 magnitude during a Go/NoGo task. One possibility for a lack of motivational influences on the N2 in the present study and that of Rosch and Hawk (2013) could be that both studies employ a flanker task, as compared to the Go/Nogo task employed by Groom et al., (2010). Along these lines, it is important to note that the N2 generated during a motor inhibition task (i.e., Go/Nogo) may reflect a different functional processes as compared to the N2 generated within a stimulus conflict task like the flanker (Folstein & Van Petten, 2008; Gehring et al., 1992; Larson et al., 2014; Nieuwenhuis et al., 2003).

Another possibility for the lack of impact of social motivation on the N2 is that the flanker task used elicited a relatively small N2 as compared to the P3 within this sample of adolescent females. Although inhibition/conflict tasks such as the go/no-go task typically elicit a robust N2 effect, in which the N2 is larger (i.e., more negative) on trials requiring inhibition (Donkers & van Boxtel, 2004), the flanker tasks typically elicits a small (or nonexistent) "conflict N2" when congruent and incongruent trials are equiprobable (Kałamała et al., 2018). Furthermore, a weak or nonexistent conflict N2 effect is generally consistent with previous reports in developmental populations (Johnstone, Barry, Markovska, Dimoska, & Clarke, 2009; Rueda, Posner, Rothbart, & Davis-Stober, 2004). Thus, it is possible that the N2 was insensitive to the social manipulation because of the relatively small conflict N2 elicited during the flanker task. Future research should explore the influence of social motivation using tasks that elicit a robust N2 (e.g., go/nogo task).

Changes in pubertal hormones may account for some of the observed social effects observed among girls. Sex hormone concentrations during puberty are suggested to lead to changes in adolescent brain function (Forbes & Dahl, 2010; Peper & Dahl, 2013), leading to increased social motivation (Crone & Dahl, 2012). Although the exact age of the beginning of adolescence is debated (Pfeifer & Allen, 2012), some suggest adolescence begins at the onset of puberty (Peper & Dahl, 2013), which is between 8 and 10 years of age for girls (Sun et al., 2002). In addition, peak white matter density in the PFC is reached by 10–12 years of age in girls (Giedd et al., 1999), similar to the age range of the current study with the largest neural effects of social context. However, it is unclear if we would observe the same findings with boys, given boys enter puberty later than girls (Sun et al., 2002), and exhibit vastly different sex hormone patterns (Peper & Dahl, 2013). Recent work finds that among girls, pubertal hormones in predict neural function on reward and risk tasks (Ladouceur et al., 2019; Op de Macks et al., 2016), and that the degree that hormones predict neural function differences between genders (Forbes et al., 2010). In addition, studies of animal models find different patterns of development of dopamine receptors between sexes (Andersen et al., 2002).

The current findings raise questions about the functional interpretation of motivational influences on the error-related negativity (ERN), an event-related potential observed following error commission (Falkenstein et al., 1990; Gehring et al., 1993). We previously found that the ERN is enhanced by social-motivational factors (Barker, Troller-Renfree, et al., 2018; Buzzell, Troller-Renfree, et al., 2017), which we interpreted as increasing error significance (Hajcak et al., 2005; Weinberg et al., 2016). However, many suggest the ERN represents a component of a larger cognitive control system (Carter & Veen, 2007; Cavanagh et al., 2012). In line with theories that the N2/P3 complex shares functional similarities with the ERN (Carter & Veen, 2007; Cavanagh et al., 2012), the present findings of an enhanced P3 in social contexts suggest that social motivation may lead to a more general enhancement of neural components related to cognitive control, as opposed to error monitoring more specifically.

Given the limited inference about psychological states based on physiological indices (Cacioppo et al., 2000; Cacioppo & Tassinary, 1990), questionable inferences follow about the specificity of motivational influences on the ERN (Richter & Slade, 2017). Thus, in the

absence of analyses of other ERP components, it is difficult to interpret motivational effects. Such issues of interpretation complicate many forms of neuroimaging data (Poldrack, 2006). Thus, future work on the intersection of social motivation and cognitive control could benefit from broad perspectives encompassing findings for multiple subprocesses.

A few limitations of the present study should be noted. First, we only examined social motivational influences in girls during adolescence. Thus, it is unknown if similar patterns would be observed for girls beyond the adolescent period and during adulthood, as well as whether boys would exhibit similar developmental changes in P3 sensitivity to social contexts. Another limitation is the somewhat small sample size, which warrants cautious interpretation of the observed three-way interactions. Future studies should continue to examine the interaction between social motivation and cognitive control with larger samples.

In summary, the present study examined the influence of social motivation on the neural correlates of cognitive control in girls across childhood and adolescence. Having girls complete the same flanker task in social and nonsocial contexts, we found that girls exhibited a larger P3 in social contexts, particularly on high-conflict trials, and the degree of P3 enhancement (regardless of congruency) was largest among children and younger adolescents. However, no differences in the N2 between contexts were observed. These findings suggest that social factors which are known to increase motivation also enhance neural correlates of cognitive control. In addition, the specific influence(s) of motivation on cognitive control appears to change across development, at least for girls.

## ACKNOWLEDGMENTS

This research was partially supported by the National Institutes of Health (grant Nos. F32HD097921 to T.V.B., HDR3717899 to N.A.F., and 5T32HD007542 to Melanie Killen supporting T. V. B). S.V.T. is supported by a National Science Foundation Graduate Research Fellowship (DGE1322106). D.S.P. is supported by the NIMH Intramural Research Program (ZIAMH-002781). The authors declare no competing financial interests.

### Funding information

National Institutes of Health, Grant/Award Number: F32HD097921, HDR3717899 and 5T32HD007542; National Science Foundation Graduate Research Fellowship, Grant/Award Number: DGE1322106; NIMH Intramural Research Program, Grant/Award Number: ZIAMH-002781

### DATA AVAILABILITY STATEMENT

Data for the experiment reported here are available at https://osf.io/bx35p/. The current experiment was not formally registered.

## REFERENCES

Amodio DM, Master SL, Yee CM, & Taylor SE (2008). Neurocognitive components of the behavioral inhibition and activation systems: Implications for theories of self-regulation. Psychophysiology, 45(1), 11–19. 10.1111/j.1469-8986.2007.00609.x [PubMed: 17910730]

Andersen SL (2003). Trajectories of brain development: Point of vulnerability or window of opportunity? Neuroscience & Biobehavioral Reviews, 27(1), 3–18. 10.1016/S0149-7634(03)00005-8 [PubMed: 12732219]

Andersen SL, Thompson AP, Krenzel E, & Teicher MH (2002). Pubertal changes in gonadal hormones do not underlie adolescent dopamine receptor overproduction. Psychoneuroendocrinology, 27(6), 683–691. 10.1016/S0306-4530(01)00069-5 [PubMed: 12084661]

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

Barker TV, Buzzell GA, & Fox NA (2018). Approach, avoidance, and the detection of conflict in the development of behavioral inhibition. New Ideas in Psychology, 10.1016/j.newideapsych.2018.07.001

Barker TV, Troller-Renfree SV, Bowman LC, Pine DS, & Fox NA (2018). Social influences of error monitoring in adolescent girls. Psychophysiology, 55(9), e13089. 10.1111/psyp.13089 [PubMed: 29682751]

Barker TV, Troller-Renfree S, Pine DS, & Fox NA (2015). Individual differences in social anxiety affect the salience of errors in social contexts. Cognitive, Affective, & Behavioral Neuroscience, 1–13, 10.3758/s13415-015-0360-9

Bartholow BD, Pearson MA, Dickter CL, Sher KJ, Monica F, & Gabriele G (2005). Strategic control and medial frontal negativity: Beyond errors and response conflict. Psychophysiology, 42(1), 33–42. 10.1111/j.1469-8986.2005.00258.x [PubMed: 15720579]

Begleiter H, Porjesz B, Chou CL, & Aunon JI (2007). P3 and stimulus incentive value. Psychophysiology, 20(1), 95–101. 10.1111/j.1469-8986.1983.tb00909.x

Bellis MDD, Keshavan MS, Beers SR, Hall J, Frustaci K, Masalehdan A, Noll J, & Boring AM (2001). Sex differences in brain maturation during childhood and adolescence. Cerebral Cortex, 11(6), 552–557. 10.1093/cercor/11.6.552 [PubMed: 11375916]

Berridge KC (2004). Motivation concepts in behavioral neuroscience. Physiology & Behavior, 81(2), 179–209. 10.1016/j.physbeh.2004.02.004 [PubMed: 15159167]

Blakemore S-J, Burnett S, & Dahl RE (2010). The role of puberty in the developing adolescent brain. Human Brain Mapping, 31(6), 926–933. 10.1002/hbm.21052 [PubMed: 20496383]

Blascovich J, Mendes WB, Hunter SB, & Salomon K (1999). Social “facilitation” as challenge and threat. Journal of Personality and Social Psychology, 77(1), 68–77. 10.1037/0022-3514.77.1.68 [PubMed: 10434409]

Blascovich J, & Tomaka J (1996). The biopsychosocial model of arousal regulation. In Zanna MP (Ed.), Advances in experimental social psychology, Vol. 28 (pp. 1–51). Academic Press. 10.1016/S0065-2601(08)60235-X

Bledowski C, Prvulovic D, Hoechstetter K, Scherg M, Wibral M, Goebel R, & Linden DE (2004). Localizing P300 generators in visual target and distractor processing: A combined event-related potential and functional magnetic resonance imaging study. The Journal of Neuroscience, 24(42), 9353–9360. 10.1523/JNEUROSCI.1897-04.2004 [PubMed: 15496671]

Boehler CN, Schevernels H, Hopf J-M, Stoppel CM, & Krebs RM (2014). Reward prospect rapidly speeds up response inhibition via reactive control. Cognitive, Affective, & Behavioral Neuroscience, 14(2), 593–609. 10.3758/s13415-014-0251-5

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

Botvinick M, & Braver T (2015). Motivation and cognitive control: From behavior to neural mechanism. Annual Review of Psychology, 66(1), 83–113. 10.1146/annurev-psych-010814-015044

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

Bowers ME, Buzzell GA, Bernat EM, Fox NA, & Barker TV (2018). Time-frequency approaches to investigating changes in feedback processing during childhood and adolescence. Psychophysiology, 55(10), e13208. 10.1111/psyp.13208 [PubMed: 30112814]

Braver TS, Krug MK, Chiew KS, Kool W, Westbrook JA, Clement NJ, Adcock RA, Barch DM, Botvinick MM, Carver CS, Cools R, Custers R, Dickinson A, Dweck CS, Fishbach A, Gollwitzer PM, Hess TM, Isaacowitz DM, Mather M, … Somerville LH (2014). Mechanisms of motivation-cognition interaction: Challenges and opportunities. Cognitive, Affective & Behavioral Neuroscience, 14(2), 443–472. 10.3758/s13415-014-0300-0

Breiner K, Li A, Cohen AO, Steinberg L, Bonnie RJ, Scott ES, Taylor-Thompson K, Rudolph MD, Chein J, Richeson JA, Dellarco DV, Fair DA, Casey BJ, & Galván A (2018). Combined effects of peer presence, social cues, and rewards on cognitive control in adolescents. Developmental Psychobiology, 60(3), 292–302. 10.1002/dev.21599 [PubMed: 29388187]

Bruno K, Fred R, & Uwe M (2007). N200 in the flanker task as a neurobehavioral tool for investigating executive control. Psychophysiology, 33(3), 282–294. 10.1111/j.1469-8986.1996.tb00425.x

Bush G, Luu P, & Posner MI (2000). Cognitive and emotional influences in anterior cingulate cortex. Trends in Cognitive Sciences, 4(6), 215–222. 10.1016/S1364-6613(00)01483-2 [PubMed: 10827444]

Buzzell GA, Richards JE, White LK, Barker TV, Pine DS, & Fox NA (2017). Development of the error-monitoring system from ages 9–35: Unique insight provided by MRI-constrained source localization of EEG. NeuroImage, 157(Supplement C), 13–26. 10.1016/j.neuroimage.2017.05.045 [PubMed: 28549796]

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

Cacioppo JT, Berntson GG, Larsen JT, Poehlmann KM, & Ito TA (2000). The psychophysiology of emotion. Handbook of Emotions, 2, 173–191.

Cacioppo JT, & Tassinary LG (1990). Inferring psychological significance from physiological signals. American Psychologist, 45(1), 16. 10.1037/0003-066X.45.1.16

Calcagnetti DJ, & Schechter MD (1992). Place conditioning reveals the rewarding aspect of social interaction in juvenile rats. Physiology & Behavior, 51(4), 667–672. 10.1016/0031-9384(92)90101-7 [PubMed: 1594664]

Carrillo-de-la-Peña MT, & Cadaveira F (2000). The effect of motivational instructions on P300 amplitude. Neurophysiologie Clinique/Clinical Neurophysiology, 30(4), 232–239. 10.1016/S0987-7053(00)00220-3 [PubMed: 11013896]

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

Casey B, Jones RM, Levita L, Libby V, Pattwell S, Ruberry E, Soliman F, & Somerville LH (2010). The storm and stress of adolescence: Insights from human imaging and mouse genetics. Developmental Psychobiology, 52(3), 225–235. 10.1002/dev.20447 [PubMed: 20222060]

Cavanagh JF, & Frank MJ (2014). Frontal theta as a mechanism for cognitive control. Trends in Cognitive Sciences, 18(8), 414–421. 10.1016/j.tics.2014.04.012 [PubMed: 24835663]

Cavanagh JF, Zambrano-Vazquez L, & Allen JJB (2012). Theta lingua franca: A common mid-frontal substrate for action monitoring processes. Psychophysiology, 49(2), 220–238. 10.1111/j.1469-8986.2011.01293.x [PubMed: 22091878]

Chein J, Albert D, O'Brien L, Uckert K, & Steinberg L (2011). Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. Developmental Science, 14(2). 10.1111/j.1467-7687.2010.01035.x

Chiew KS, & Braver TS (2013). Temporal dynamics of motivation-cognitive control interactions revealed by high-resolution pupillometry. Frontiers in Psychology, 4, 10.3389/fpsyg.2013.00015

Chiew KS, & Braver TS (2014). Dissociable influences of reward motivation and positive emotion on cognitive control. Cognitive, Affective, & Behavioral Neuroscience, 14(2), 509–529. 10.3758/s13415-014-0280-0

Chiew KS, & Braver TS (2016). Reward favors the prepared: Incentive and task-informative cues interact to enhance attentional control. Journal of Experimental Psychology: Human Perception and Performance, 42(1), 52. [PubMed: 26322689]

Clayson PE, Baldwin SA, & Larson MJ (2013). How does noise affect amplitude and latency measurement of event-related potentials (ERPs)? A methodological critique and simulation study. Psychophysiology, 50(2), 174–186. 10.1111/psyp.12001 [PubMed: 23216521]

Clayson PE, & Larson MJ (2011). Conflict adaptation and sequential trial effects: Support for the conflict monitoring theory. Neuropsychologia, 49(7), 1953–1961. 10.1016/j.neuropsychologia.2011.03.023 [PubMed: 21435347]

Clayson PE, & Larson MJ (2013). Psychometric properties of conflict monitoring and conflict adaptation indices: Response time and conflict N2 event-related potentials. Psychophysiology, 50(12), 1209–1219. 10.1111/psyp.12138 [PubMed: 23992600]

Crone EA (2014). The role of the medial frontal cortex in the development of cognitive and social-affective performance monitoring. Psychophysiology, 51(10), 943–950. 10.1111/psyp.12252 [PubMed: 24942498]

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

Defoe IN, Dubas JS, Figner B, & Van Aken MA (2015). A meta-analysis on age differences in risky decision making: Adolescents versus children and adults. Psychological Bulletin, 141(1), 48. 10.1037/a0038088 [PubMed: 25365761]

Donchin E, & Coles MGH (1988). Is the P300 component a manifestation of context updating? Behavioral and Brain Sciences, 11(3), 357–374. 10.1017/S0140525X00058027

Donchin E, Ritter W, & McCallum WC (1978). Cognitive psychophysiology: The endogenous components of the ERP. In Callaway P, Tueting P, & Koslow S (Eds.), Event-related brain potentials in man (pp 349–411) New York: Academic Press.

Donkers FCL, & van Boxtel GJM (2004). The N2 in go/no-go tasks reflects conflict monitoring not response inhibition. Brain and Cognition, 56(2), 165–176. 10.1016/j.bandc.2004.04.005 [PubMed: 15518933]

Engelmann JB, Damaraju E, Padmala S, & Pessoa L (2009). Combined effects of attention and motivation on visual task performance: Transient and sustained motivational effects. Frontiers in Human Neuroscience, 3, 4. 10.3389/neuro.09.004.2009 [PubMed: 19434242]

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

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

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

Falkenstein M, Hohnsbein J, Hoormann J, & Blanke L (1990). Effects of errors in choice reaction tasks on the ERP under focused and divided attention. Psychophysiological Brain Research, 1, 192–195.

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

Folstein JR, & Van Petten C (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: A review. Psychophysiology, 45(1), 152–170. 10.1111/j.1469-8986.2007.00602.x [PubMed: 17850238]

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

Forbes EE, Ryan ND, Phillips ML, Manuck SB, Worthman CM, Moyles DL, Tarr JA, Sciarrillo SR, & Dahl RE (2010). Healthy adolescents' neural response to reward: Associations with puberty, positive affect, and depressive symptoms. Journal of the American Academy of Child and Adolescent Psychiatry, 49(2), 162–172.e1–5. 10.1097/00004583-201002000-00010 [PubMed: 20215938]

Frühholz S, Godde B, Finke M, & Herrmann M (2011). Spatio-temporal brain dynamics in a combined stimulus–stimulus and stimulus–response conflict task. NeuroImage, 54(1), 622–634. 10.1016/j.neuroimage.2010.07.071 [PubMed: 20691791]

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

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

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

Gehring WJ, Gratton G, Coles MG, & Donchin E (1992). Probability effects on stimulus evaluation and response processes. Journal of Experimental Psychology. Human Perception and Performance, 18(1), 198–216. 10.1037/0096-1523.18.1.198 [PubMed: 1532188]

Giedd JN, Blumenthal J, Jeffries NO, Castellanos FX, Liu H, Zijdenbos A, Paus T, Evans AC, & Rapoport JL (1999). Brain development during childhood and adolescence: A longitudinal MRI study. Nature Neuroscience, 2(10), 861–863. 10.1038/13158 [PubMed: 10491603]

Goldstein RZ, Cottone LA, Jia Z, Maloney T, Volkow ND, & Squires NK (2006). The effect of graded monetary reward on cognitive event-related potentials and behavior in young healthy adults. International Journal of Psychophysiology, 62(2), 272–279. 10.1016/j.ijpsycho.2006.05.006 [PubMed: 16876894]

Gray JA, & McNaughton N (2003). The neuropsychology of anxiety: An enquiry into the function of the septo-hippocampal system. Oxford University Press. 10.1093/acprof:oso/9780198522713.001.0001/acprof-9780198522713

Groom MJ, Scerif G, Liddle PF, Batty MJ, Liddle EB, Roberts KL, Cahill JD, Liotti M, & Hollis C (2010). Effects of motivation and medication on electrophysiological markers of response inhibition in children with attention-deficit/hyperactivity disorder. Biological Psychiatry, 67(7), 624–631. 10.1016/j.biopsych.2009.09.029 [PubMed: 19914599]

Gruendler TOJ, Ullsperger M, & Huster RJ (2011). Event-related potential correlates of performance-monitoring in a lateralized time-estimation task. PLoS One, 6(10), e25591. 10.1371/journal.pone.0025591 [PubMed: 22039418]

Gullone E, & Moore S (2000). Adolescent risk-taking and the five-factor model of personality. Journal of Adolescence, 23(4), 393–407. 10.1006/jado.2000.0327 [PubMed: 10936013]

Gunther Moor B, Güroğlu B, Op de Macks ZA, Rombouts SA, Van der Molen MW, & Crone EA (2012). Social exclusion and punishment of excluders: Neural correlates and developmental trajectories. NeuroImage, 59(1), 708–717. 10.1016/j.neuroimage.2011.07.028 [PubMed: 21791248]

Gunther Moor B, van Leijenhorst L, Rombouts SA, Crone EA, & Van der Molen MW (2010). Do you like me? Neural correlates of social evaluation and developmental trajectories. Social Neuroscience, 5(5–6), 461–482. 10.1080/17470910903526155 [PubMed: 20721813]

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

Hayden BY, Parikh PC, Deaner RO, & Platt ML (2007). Economic principles motivating social attention in humans. Proceedings of the Royal Society of London B: Biological Sciences, 274(1619), 1751–1756. 10.1098/rspb.2007.0368

Herrmann MJ, Römmler J, Ehlis A-C, Heidrich A, & Fallgatter AJ (2004). Source localization (LORETA) of the error-related-negativity (ERN/Ne) and positivity (Pe). Cognitive Brain Research, 20(2), 294–299. 10.1016/j.cogbrainres.2004.02.013 [PubMed: 15183400]

Holroyd CB, & Coles MGH (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. Psychological Review, 109(4), 679–709. 10.1037/0033-295X.109.4.679 [PubMed: 12374324]

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

Hoyniak C (2017). Changes in the NoGo N2 event-related potential component across childhood: A systematic review and meta-analysis. Developmental Neuropsychology, 42(1), 1–24. 10.1080/87565641.2016.1247162 [PubMed: 28059561]

Kałamała P, Szewczyk J, Senderecka M, & Wodniecka Z (2018). Flanker task with equiprobable congruent and incongruent conditions does not elicit the conflict N2. Psychophysiology, 55(2), e12980. 10.1111/psyp.12980

Kirschbaum C, Pirke K-M, & Hellhammer DH (1993). The 'trier social stress test' – A tool for investigating psychobiological stress responses in a laboratory setting. Neuropsychobiology, 28(1–2), 76–81. 10.1159/000119004 [PubMed: 8255414]

Kleih SC, Nijboer F, Halder S, & Kübler A (2010). Motivation modulates the P300 amplitude during brain–computer interface use. Clinical Neurophysiology, 121(7), 1023–1031. 10.1016/j.clinph.2010.01.034 [PubMed: 20188627]

Kopp B, Rist F, & Mattler U (1996). N200 in the flanker task as a neurobehavioral tool for investigating executive control. Psychophysiology, 33(3), 282–294. 10.1111/j.1469-8986.1996.tb00425.x [PubMed: 8936397]

Krebs RM, Boehler CN, Appelbaum LG, & Woldorff MG (2013). Reward associations reduce behavioral interference by changing the temporal dynamics of conflict processing. PLoS One, 8(1), e53894. 10.1371/journal.pone.0053894 [PubMed: 23326530]

Krebs RM, Boehler CN, & Woldorff MG (2010). The influence of reward associations on conflict processing in the Stroop task. Cognition, 117(3), 341–347. 10.1016/j.cognition.2010.08.018 [PubMed: 20864094]

Kutas M, McCarthy G, & Donchin E (1977). Augmenting mental chronometry: The P300 as a measure of stimulus evaluation time. Science, 197(4305), 792–795. [PubMed: 887923]

Johnstone SJ, Barry RJ, Markovska V, Dimoska A, & Clarke AR (2009). Response inhibition and interference control in children with AD/HD: A visual ERP investigation. International Journal Psychophysiology, 72, 145–153.

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

Ladouceur CD, Kerestes R, Schlund MW, Shirtcliff EA, Lee Y, & Dahl RE (2019). Neural systems underlying reward cue processing in early adolescence: The role of puberty and pubertal hormones. Psychoneuroendocrinology, 102, 281–291. 10.1016/j.psyneuen.2018.12.016 [PubMed: 30639923]

Lahat A, Walker OL, Lamm C, Degnan KA, Henderson HA, & Fox NA (2014). Cognitive conflict links behavioural inhibition and social problem solving during social exclusion in childhood. Infant and Child Development, 23(3), 273–282. [PubMed: 25705132]

Lamm C, White LK, McDermott JM, & Fox NA (2012). Neural activation underlying cognitive control in the context of neutral and affectively charged pictures in children. Brain and Cognition, 79(3), 181–187. 10.1016/j.bandc.2012.02.013 [PubMed: 22542842]

Larson MJ, Clayson PE, & Clawson A (2014). Making sense of all the conflict: A theoretical review and critique of conflict-related ERPs. International Journal of Psychophysiology, 93(3), 283–297. 10.1016/j.ijpsycho.2014.06.007 [PubMed: 24950132]

Lenroot RK, & Giedd JN (2010). Sex differences in the adolescent brain. Brain and Cognition, 72(1), 46. 10.1016/j.bandc.2009.10.008 [PubMed: 19913969]

Leotti LA, & Wager TD (2010). Motivational influences on response inhibition measures. Journal of Experimental Psychology: Human Perception and Performance, 36(2), 430. [PubMed: 20364928]

Libby R, & Lipe MG (1992). Incentives, effort, and the cognitive processes involved in accounting-related judgments. Journal of Accounting Research, 249–273. 10.2307/2491126

Linden DEJ (2005). The P300: Where in the brain is it produced and what does it tell us? The Neuroscientist, 11(6), 563–576. 10.1177/1073858405280524 [PubMed: 16282597]

Locke HS, & Braver TS (2008). Motivational influences on cognitive control: Behavior, brain activation, and individual differences. Cognitive, Affective, & Behavioral Neuroscience, 8(1), 99–112. 10.3758/CABN.8.1.99

Luck SJ, & Kappenman ES (eds). (2012). The Oxford Handbook of (Event)-Related Potential Components. Oxford University Press.

Luna B, Garver KE, Urban TA, Lazar NA, & Sweeney JA (2004). Maturation of cognitive processes from late childhood to adulthood. Child Development, 75(5), 1357–1372. 10.1111/j.1467-8624.2004.00745.x [PubMed: 15369519]

Luna B, & Wright C (2016). Adolescent brain development: Implications for the juvenile criminal justice system. In Heilbrun K, DeMatteo D, & Goldstein NES (Eds.), APA handbooks in psychology®. APA handbook of psychology and juvenile justice (pp. 91–116). American Psychological Association. 10.1037/14643-005

Magliero A, Bashore TR, Coles MG, & Donchin E (1984). On the dependence of P300 latency on stimulus evaluation processes. Psychophysiology, 21(2), 171–186. 10.1111/j.1469-8986.1984.tb00201.x [PubMed: 6728983]

McCarthy G, & Donchin E (1981). A metric for thought: A comparison of P300 latency and reaction time. Science, 211(4477), 77–80. [PubMed: 7444452]

Meyer A, Lerner MD, Reyes ADL, Laird RD, & Hajcak G (2017). Considering ERP difference scores as individual difference measures: Issues with subtraction and alternative approaches. Psychophysiology, 54(1), 114–122. 10.1111/psyp.12664 [PubMed: 28000251]

Montoya AK (2019). Moderation analysis in two-instance repeated measures designs: Probing methods and multiple moderator models. Behavior Research Methods, 51(1), 61–82. 10.3758/s13428-018-1088-6 [PubMed: 30306409]

Nee DE, Wager TD, & Jonides J (2007). Interference resolution: Insights from a meta-analysis of neuroimaging tasks. Cognitive, Affective, & Behavioral Neuroscience, 7(1), 1–17. 10.3758/CABN.7.1.1

Nelson EE, Jarcho JM, & Guyer AE (2016). Social re-orientation and brain development: An expanded and updated view. Developmental Cognitive Neuroscience, 17, 118–127. 10.1016/j.dcn.2015.12.008 [PubMed: 26777136]

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

Nieuwenhuis S, Aston-Jones G, & Cohen JD (2005). Decision making, the P3, and the locus coeruleus–norepinephrine system. Psychological Bulletin, 131(4), 510. 10.1037/0033-2909.131.4.510 [PubMed: 16060800]

Nieuwenhuis S, Yeung N, van den Wildenberg W, & Ridderinkhof KR (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency. Cognitive, Affective, & Behavioral Neuroscience, 3(1), 17–26. 10.3758/CABN.3.1.17

Ninomiya T, Noritake A, Ullsperger M, & Isoda M (2018). Performance monitoring in the medial frontal cortex and related neural networks: From monitoring self actions to understanding others' actions. Neuroscience Research, 10.1016/j.neures.2018.04.004

Op de Macks ZA, Bunge SA, Bell ON, Wilbrecht L, Kriegsfeld LJ, Kayser AS, & Dahl RE (2016). Risky decision-making in adolescent girls: The role of pubertal hormones and reward circuitry. Psychoneuroendocrinology, 74, 77–91. 10.1016/j.psyneuen.2016.08.013 [PubMed: 27591399]

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

Peper JS, & Dahl RE (2013). The teenage brain surging hormones—brain-behavior interactions during puberty. Current Directions in Psychological Science, 22(2), 134–139. 10.1177/0963721412473755 [PubMed: 26290625]

Pessoa L (2009). How do emotion and motivation direct executive control? Trends in Cognitive Sciences, 13(4), 160–166. 10.1016/j.tics.2009.01.006 [PubMed: 19285913]

Petersen AC, Crockett L, Richards M, & Boxer A (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. Journal of Youth and Adolescence, 17(2), 117–133. 10.1007/BF01537962 [PubMed: 24277579]

Pfeifer JH, & Allen NB (2012). Arrested development? Reconsidering dual-systems models of brain function in adolescence and disorders. Trends in Cognitive Sciences, 16(6), 322–329. 10.1016/j.tics.2012.04.011 [PubMed: 22613872]

Pfeifer JH, & Allen NB (2016). The audacity of specificity: Moving adolescent developmental neuroscience towards more powerful scientific paradigms and translatable models. Developmental Cognitive Neuroscience, 17, 131–137. 10.1016/j.dcn.2015.12.012 [PubMed: 26754460]

Picton TW (1992). The P300 wave of the human event-related potential. Journal of Clinical Neurophysiology, 9(4), 456–479. 10.1097/00004691-199210000-00002 [PubMed: 1464675]

Poldrack RA (2006). Can cognitive processes be inferred from neuroimaging data? Trends in Cognitive Sciences, 10(2), 59–63. 10.1016/j.tics.2005.12.004 [PubMed: 16406760]

Polich J (2007). Updating P300: An integrative theory of P3a and P3b. Clinical Neurophysiology, 118(10), 2128–2148. 10.1016/j.clinph.2007.04.019 [PubMed: 17573239]

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

Pritchard WS (1981). Psychophysiology of P300. Psychological Bulletin, 89(3), 506. 10.1037/0033-2909.89.3.506 [PubMed: 7255627]

Ramsey SE, & Finn PR (1997). P300 from men with a family history of alcoholism under different incentive conditions. Journal of Studies on Alcohol, 58(6), 606–616. 10.15288/jsa.1997.58.606 [PubMed: 9391920]

Richter M, & Slade K (2017). Interpretation of physiological indicators of motivation: Caveats and recommendations. International Journal of Psychophysiology, 119, 4–10. 10.1016/j.ijpsycho.2017.04.007 [PubMed: 28442270]

Ridderinkhof KR, & Molen MW (1995). A psychophysiological analysis of developmental differences in the ability to resist interference. Child Development, 66(4), 1040–1056. 10.2307/1131797

Ridderinkhof KR, Ramautar JR, & Wijnen JG (2009). To PE or not to PE: A P3-like ERP component reflecting the processing of response errors. Psychophysiology, 46(3), 531–538. 10.1111/j.1469-8986.2009.00790.x [PubMed: 19226310]

Roesch MR, & Olson CR (2004). Neuronal activity related to reward value and motivation in primate frontal cortex. Science, 304(5668), 307–310. 10.1126/science.1093223 [PubMed: 15073380]

Rosch KS, & Hawk LW (2013). The effects of performance-based rewards on neurophysiological correlates of stimulus, error, and feedback processing in children with ADHD. Psychophysiology, 50(11), 1157–1173. 10.1111/psyp.12127 [PubMed: 24033316]

Rueda MR, Posner MI, Rothbart MK, & Davis-Stober CP (2004). Development of the time course for processing conflict: An event-related potentials study with 4 year olds and adults. BMC Neuroscience, 5(1), 39. 10.1186/1471-2202-5-39 [PubMed: 15500693]

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

Small DM, Gitelman D, Simmons K, Bloise SM, Parrish T, & Mesulam M-M (2005). Monetary incentives enhance processing in brain regions mediating top-down control of attention. Cerebral Cortex, 15(12), 1855–1865. 10.1093/cercor/bhi063 [PubMed: 15746002]

Smith AR, Rosenbaum GM, Botdorf MA, Steinberg L, & Chein JM (2018). Peers influence adolescent reward processing, but not response inhibition. Cognitive, Affective, & Behavioral Neuroscience, 18(2), 284–295. 10.3758/s13415-018-0569-5

Somerville LH, Heatherton TF, & Kelley WM (2006). Anterior cingulate cortex responds differentially to expectancy violation and social rejection. Nature Neuroscience, 9(8), 1007. 10.1038/nn1728 [PubMed: 16819523]

Squires KC, Donchin E, Herning RI, & McCarthy G (1977). On the influence of task relevance and stimulus probability on event-related-potential components. Electroencephalography and Clinical Neurophysiology, 42(1), 1–14. 10.1016/0013-4694(77)90146-8 [PubMed: 64341]

Sun SS, Schubert CM, Chumlea WC, Roche AF, Kulin HE, Lee PA, Himes JH, & Ryan AS (2002). National estimates of the timing of sexual maturation and racial differences among US children. Pediatrics, 110(5), 911–919. 10.1542/peds.110.5.911 [PubMed: 12415029]

Sutton S, Braren M, Zubin J, & John ER (1965). Evoked-potential correlates of stimulus uncertainty. Science, 150(3700), 1187–1188. [PubMed: 5852977]

Tarkka IM, & Stokic DS (1998). Source localization of P300 from oddball, single stimulus, and omitted-stimulus paradigms. Brain Topography, 11(2), 141–151. 10.1023/A:1022258606418 [PubMed: 9880172]

Triplett N (1898). The dynamogenic factors in pacemaking and competition. The American Journal of Psychology, 9(4), 507–533. 10.2307/1412188

Ullsperger M, Danielmeier C, & Jocham G (2014). Neurophysiology of performance monitoring and adaptive behavior. Physiological Reviews, 94(1), 35–79. 10.1152/physrev.00041.2012 [PubMed: 24382883]

van Dinteren R, Arns M, Jongsma MLA, & Kessels RPC (2014). P300 development across the lifespan: A systematic review and meta-analysis. PLoS One, 9(2), e87347. 10.1371/journal.pone.0087347 [PubMed: 24551055]

van Veen V, Cohen JD, Botvinick MM, Stenger VA, & Carter CS (2001). Anterior cingulate cortex, conflict monitoring, and levels of processing. NeuroImage, 14(6), 1302–1308. 10.1006/nimg.2001.0923 [PubMed: 11707086]

Volpe U, Mucci A, Bucci P, Merlotti E, Galderisi S, & Maj M (2007). The cortical generators of P3a and P3b: A LORETA study. Brain Research Bulletin, 73(4–6), 220–230. 10.1016/j.brainresbull.2007.03.003 [PubMed: 17562387]

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

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

Williams BR, Ponesse JS, Schachar RJ, Logan GD, & Tannock R (1999). Development of inhibitory control across the life span. Developmental Psychology, 35(1), 205. 10.1037/0012-1649.35.1.205 [PubMed: 9923475]

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

Zajonc RB (1965). Social facilitation. Science, 149(Whole No. 3681), 269–274. 10.1126/science.149.3681.269 [PubMed: 14300526]

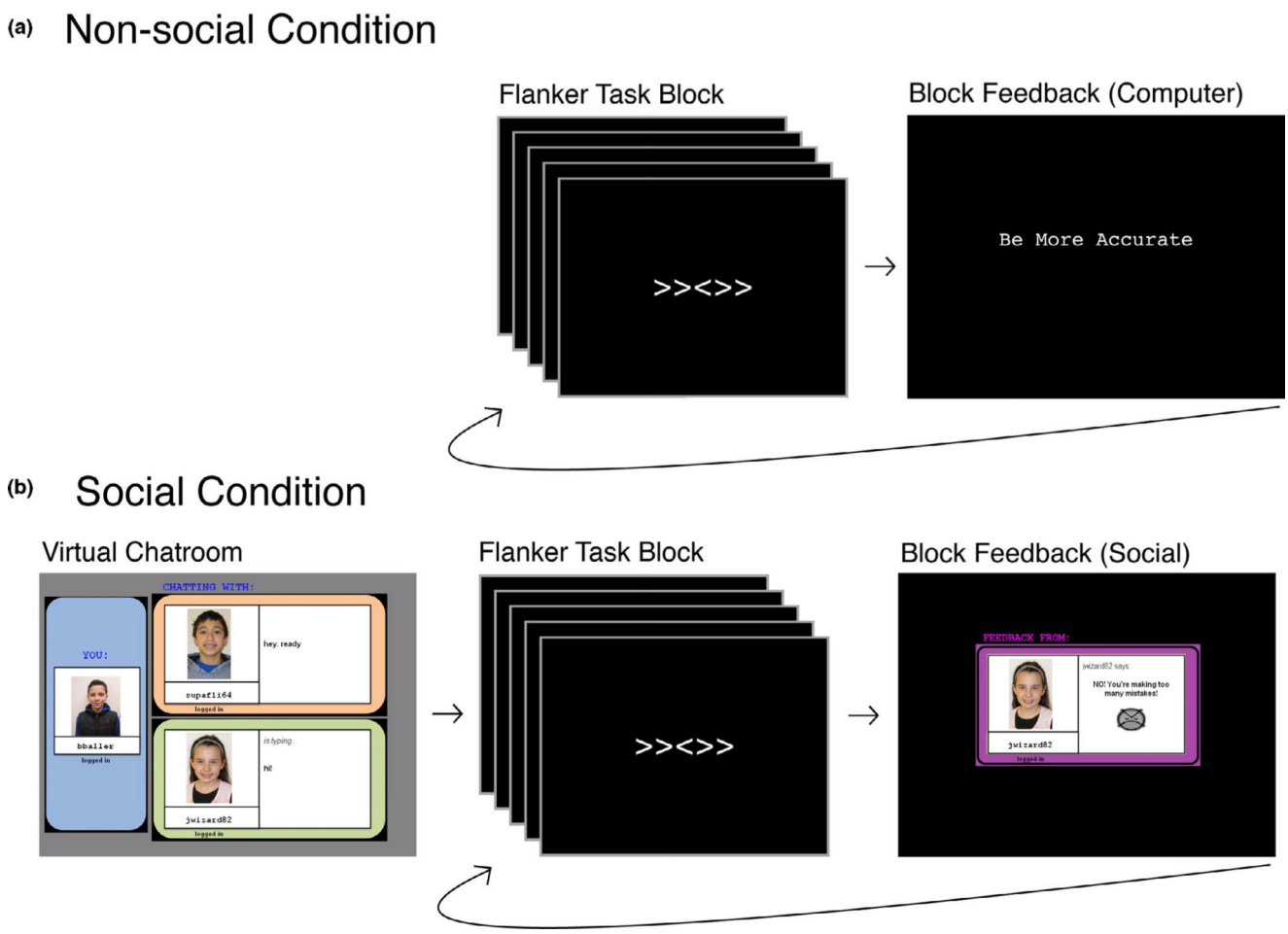


**FIGURE 1.**

Experimental paradigm. Adolescents received 10 feedback breaks (e.g., after each block of 32 trials) for each of 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. Figure adapted from Barker, Troller-Renfree, et al., 2018

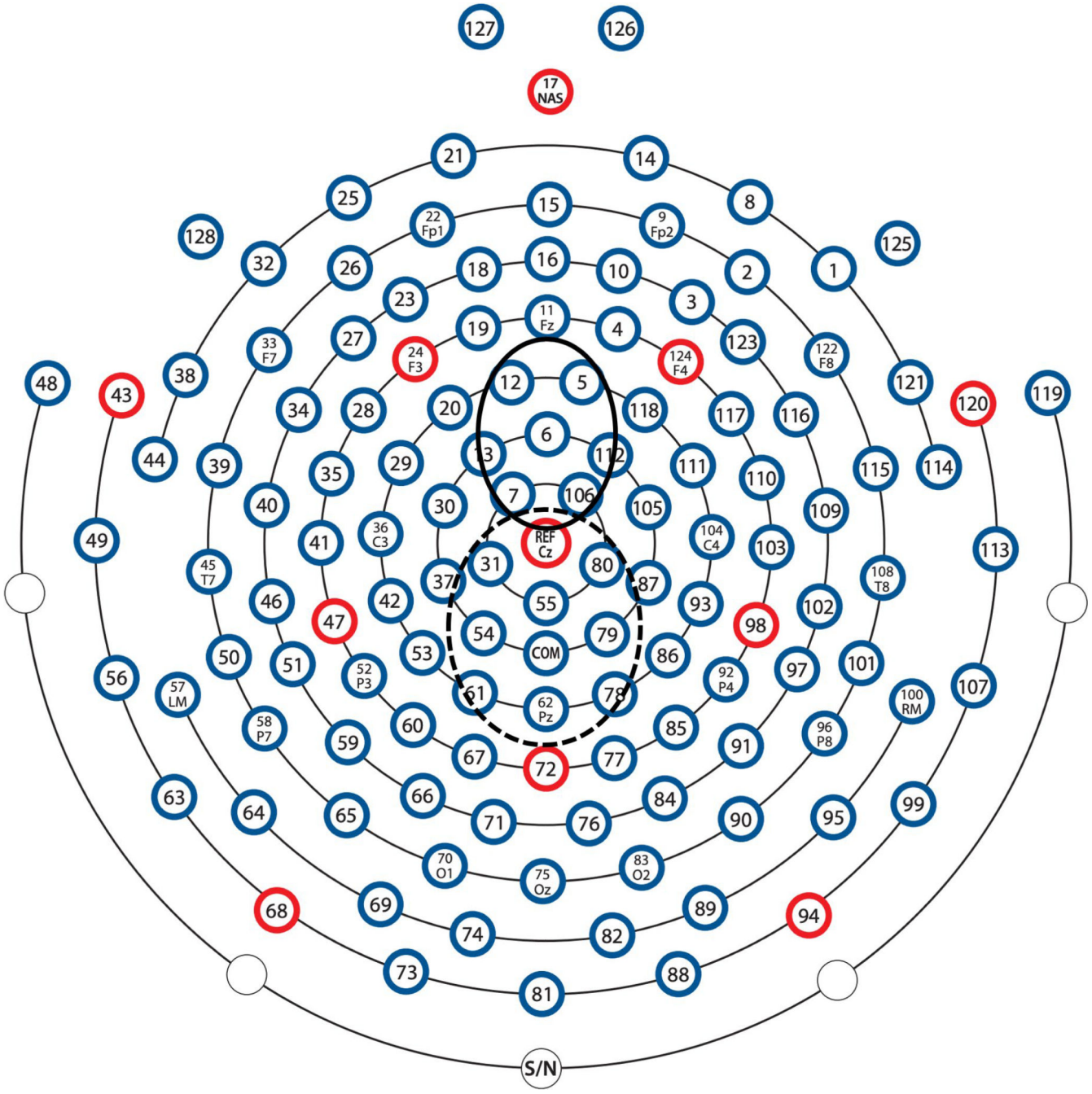


**FIGURE 2.**

Sensor layout for the Electrical Geodesics Inc. (EGI) 128-channel hydrocel sensor net. Electrode locations averaged for measurement of the N2 are in the solid circle and for the P3 in the dashed circle

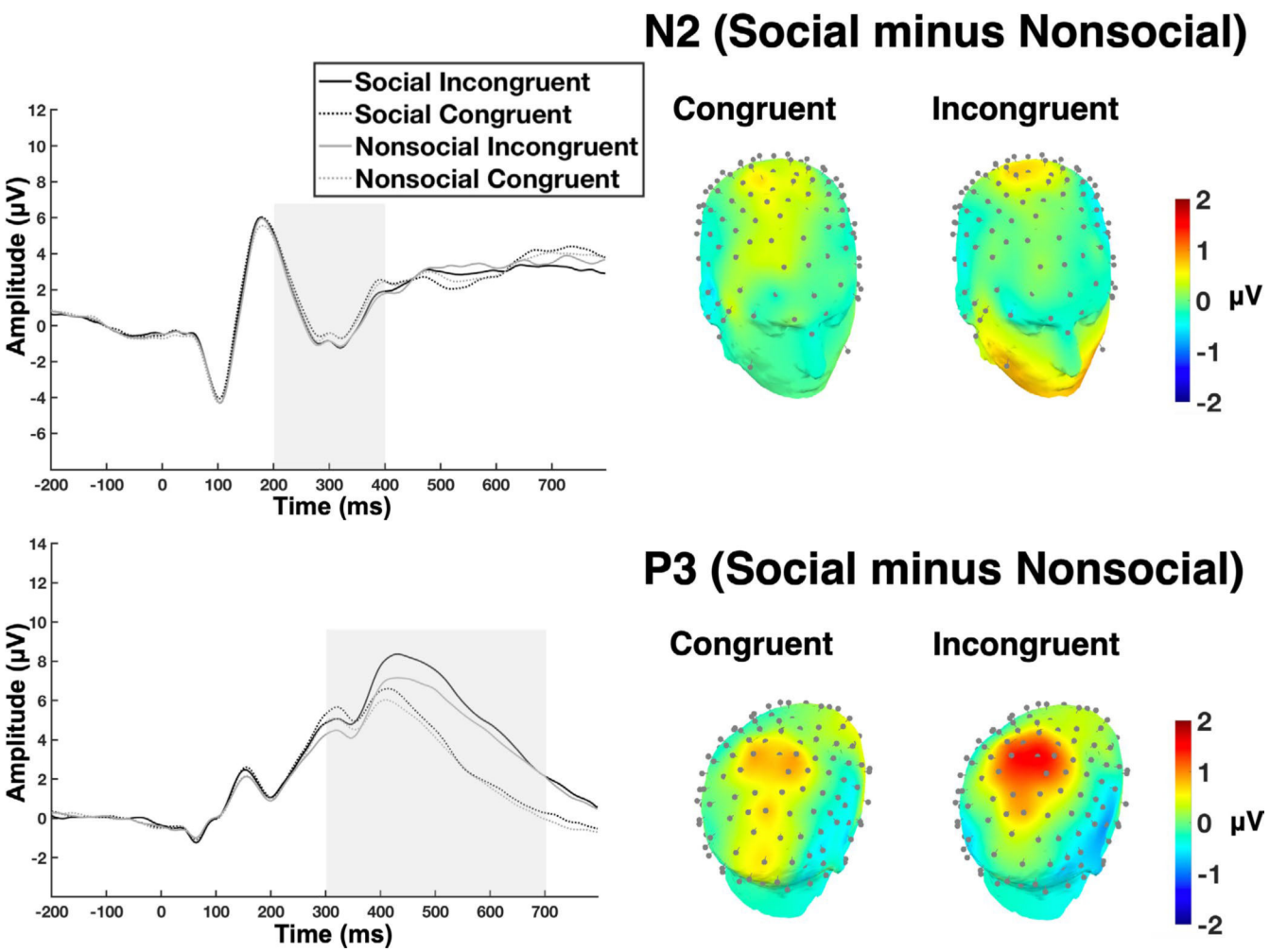


**FIGURE 3.**

Left: Stimulus-locked event-related potential waveforms for incongruent (solid lines) and incongruent trials (dotted lines) for the social condition (black lines) and the nonsocial condition (grey lines) for a fronto-central electrode cluster where N2 was maximal (top) and a central-partial electrode cluster where the P3 was maximal (bottom). Shaded region represents moving average window. Right: Scalp topographies depicting the effect of social context (i.e., social condition minus nonsocial condition) on the N2 at 280 ms post-stimulus (top) and P3 at 440 ms post-stimulus (bottom)

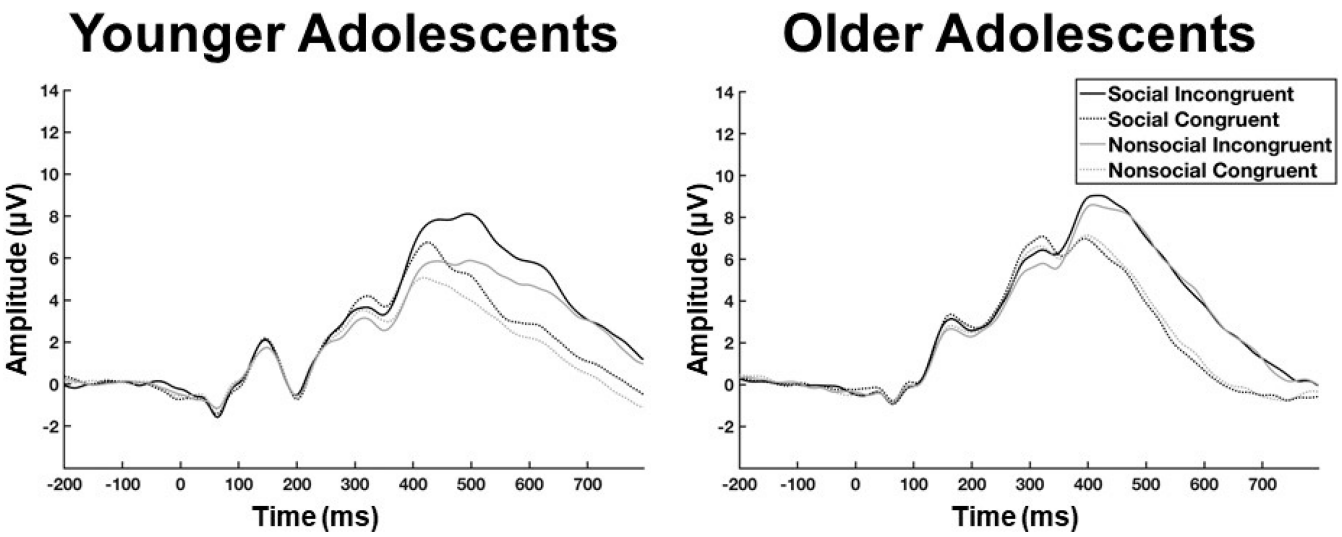


**FIGURE 4.**

Waveforms for the P3 for younger adolescents (left) and older adolescents (right). A median split was used to create age groupings for visualization purposes.

## TABLE 1

Raw means for behavioral performance and ERP measures for the nonsocial and social condition (standard deviations)

| Behavior measures | Nonsocial | Social |
|---|---|---|
| Incongruent response time (ms) | 540.98 (107.74) | 516.00 (87.55) |
| Congruent response time (ms) | 458.68 (96.02) | 439.06 (76.76) |
| Accuracy on incongruent trials (%) | 79.29 (8.67) | 78.81 (9.17) |
| Accuracy on congruent trials (%) | 93.40 (5.44) | 94.22 (5.45) |
| ERPs (μV) | | |
| Incongruent N2 | −0.09 (2.77) | −0.05 (3.03) |
| Congruent N2 | 0.13 (2.89) | 0.34 (2.84) |
| Incongruent P3 | 7.66 (4.45) | 8.80 (4.72) |
| Congruent P3 | 6.16 (4.08) | 6.51 (4.35) |