Cognitive control and conflict adaptation in youth with high-functioning autism

Michael J. Larson,1,2* Mikle South,1,2* Peter E. Clayson,1 and Ann Clawson1

1Departments of Psychology; 2Neuroscience Center, Brigham Young University, Provo, UT, USA

Introduction

Autism spectrum disorders (ASD) are developmental disorders marked by impairments in social interaction and communication as well as restricted and repetitive behaviors. These social and behavioral characteristics are often associated with underlying deficits in executive functioning (EF), a broad definition of higher-order processes that include behavioral modification, decision making, and goal-setting (Ozonoff, South, & Provencal, 2007). Indeed, individuals with ASD typically display a variety of EF deficits that are important for self-regulation, including reduced cognitive flexibility, impaired performance monitoring, and diminished error-correction abilities (Agam, Joseph, Barton, & Manoach, 2010; Ozonoff et al., 2007; Thakkar et al., 2008). Such EF deficits may compound social and behavioral dysfunction as they disrupt normal behavioral adaptation and the ability to carry out goal-directed behaviors.

These EF deficits are evident in examinations of specific cognitive control component processes. Cognitive control refers to the ability to monitor and regulate goal-directed behaviors by evaluating contextual information and using that information to appropriately adjust behavior (Botvinick, Carter, Braver, Barch, & Cohen, 2001). These processes are present during situations where task-irrelevant stimulus information competes with task-relevant information for attentional control, inducing conflict (Botvinick et al., 2001; Yeung & Cohen, 2006). Appropriate completion of day-to-day tasks in the presence of conflict requires an individual to identify the conflict and subsequently allocate greater attentional resources to overcome the conflict and correctly complete the task at hand. Thus, intact cognitive control requires detecting conflict before and after behavioral responses and adjusting cognitive resources to alter subsequent performance (Botvinick et al., 2001).

Conflict monitoring can be tested using tasks such as the Eriksen flanker task (Eriksen & Eriksen, 1974) that induce response conflict between relevant and irrelevant information. During the flanker task, participants are instructed to respond to a central target arrow that is surrounded by irrelevant flanker stimuli. On low-conflict trials (congruent), flanker and target stimuli are compatible, similarly cueing an appropriate response. On high-conflict trials (incongruent), flanker stimuli cue a response opposite the target stimulus, creating conflict between cued correct and incorrect responses. Participants respond more slowly and less accurately to incongruent trials relative to congruent trials.

Although conflict may lead to slowed responses and decreased accuracy, levels of conflict in previous trials may improve performance on current trials by inducing top-down processes that increase cognitive control (Kerns et al., 2004). For example, on
incongruent trials preceded by congruent trials (called cl trials), individuals typically respond more slowly than incongruent–incongruent (il) trials, demonstrating that the presence of increased conflict in the preceding trial results in the allocation of greater cognitive control to improve current-trial performance (Clayson & Larson, 2011a). However, individuals display faster response times (RTs) for congruent–congruent (cC) relative to incongruent–congruent (iC) trials likely due to increased demands associated with switching between incongruent and congruent trials (Egner & Hirsch, 2005). This characteristic pattern of previous-trial congruency affecting current-trial performance is referred to as conflict adaptation.

According to the conflict-monitoring theory, strategic adjustments in cognitive control are signaled by conflict detection processes in the anterior cingulate cortex (ACC), which recruits the dorsolateral prefrontal cortex (dPFC) to minimize conflict activation on the subsequent trial (Botvinick et al., 2001). After high-conflict trials, increases in cognitive control should result in decreased ACC activation on subsequent high-conflict trials, reflecting decreased conflict activation; however, after low-conflict trials, less cognitive control is recruited and should result in greater activation of the ACC. Importantly, measures of conflict adaptation may specify the systematic nature of cognitive dysfunction in individuals with ASD.

Atypical neural development and structural abnormalities in ASD correlate with differential conflict-monitoring processes, suggesting these cognitive and behavioral symptoms may have a neurological basis. Generally, individuals with ASD display altered neural metabolism (Levitt et al., 2003), early brain overgrowth leading to an overall increase in brain volume (Courchesne, Karns, & Davis, 2001; Herbert et al., 2004), and irregular connectivity resulting in inefficient neural signaling (Belmonte et al., 2004; Just, Cherkassky, Keller, Kana, & Minshew, 2007). Several researchers have also reported specific ASD-related abnormalities in brain regions putatively involved in conflict monitoring and cognitive control, such as the ACC and dPFC. For example, disruption of the white matter tracts (Barnea-Goraly, Lotspeich, & Reiss, 2010) and hypometabolism in the ACC (Agam et al., 2010; Haznedar et al., 2000) may be tied to impaired conflict detection. Studies examining the dPFC in ASD, however, are contradictory, indicating no volumetric abnormalities (Griebling et al., 2010) or abnormalities only in youth with low levels of ASD (Salmond, Vargha-Khadem, Gadian, de Haan, & Baldeweg, 2007). Evidence of altered connectivity with surrounding networks in the ACC (Thakkar et al., 2008) may indicate that neural communication necessary for behavioral modification following conflict may be inefficient such that the integration of information between the ACC and dPFC is impaired.

Electrophysiological indices of conflict adaptation can be measured using event-related potentials (ERPs) such as the ACC-mediated N2, an ERP component that occurs 250–350 ms following stimulus presentation (van Veen & Carter, 2002). N2 amplitude is more negative on incongruent trials compared to congruent trials (Yeung & Cohen, 2006). N2 amplitude is less negative in il trials than cl trials due to increases in cognitive control following high conflict (Clayson & Larson, 2011a). The N2 is attenuated on iC trials relative to cC trials because high previous-trial conflict leads to greater cognitive control and attention to relevant information (e.g. target arrow) than irrelevant information (e.g. flanker arrows) in the current trial (Clayson & Larson, 2011a).

We examined behavioral and electrophysiological indices of conflict adaptation in youth with ASD relative to typically developing (TD) controls. Characteristic EF deficits (Ozonoff et al., 2007) along with evidence from electrophysiological (South, Larson, Krauskopf, & Clawson, 2010; Vlamings, Jonkman, Hoeksma, van Engeland, & Kemner, 2008) and behavioral (Bogte, Flamma, van der Meere, & van Engeland, 2007) studies indicate that conflict may be less salient and behavioral performance may be impaired in ASD (South et al., 2010; Vlamings et al., 2008). Thus, we examined conflict detection and adaptation effects as measured by the N2, RTs, and error rates. We hypothesized that individuals with ASD would have reduced N2 amplitudes relative to TD controls and would display similar RTs for incongruent and congruent trials. Identifying the nature of conflict monitoring in ASD may increase understanding of this complex disorder and improve treatment, specifically during development when these deficits are particularly salient.

Method

Participants

Written consent and assent were obtained as approved by the Institutional Review Board. Potential participants in the TD group were excluded if a parent screening questionnaire completed prior to study enrollment noted previous or suspected diagnoses of attention deficit hyperactivity disorder, learning disability, head injury, ASD, or any other psychiatric or developmental difficulties.

Final study enrollment included 28 individuals with ASD (2 female) between the ages of 9 and 17 years and 36 typically developing controls (7 female) 9–18 years old (see Table 1). Groups did not differ in male-to-female ratio, $\chi^2(1) = 1.97, p = .16$. All individuals in the ASD group met full research criteria for ASD based on the Autism Diagnostic Observation Schedules Generic (ADOS-G; Lord et al., 2000; $M = 12.14, SD = 3.89$) and the Social Communication Questionnaire ($M = 17.14, SD = 9.54$). Baseline measures for all participants included the parent-report Screen for Child Anxiety Related Emotional Disorders (SCARED) to screen for childhood anxiety disorders (Birmaher et al., 1999),
and IQ scores from the Wechsler abbreviated scales of intelligence (WASI). Full-Scale IQ was in the normative range (>75) and group means did not differ on FSIQ, VIQ, or PIQ (see Table 1). Groups differed in parent-rated SCARED score (see Table 1).

**Experimental task**

Participants completed a modified version of the Eriksen flanker task (Eriksen & Eriksen, 1974). Each trial consisted of either congruent (<<<<<, >>>>>) or incongruent (<<<<<, >><>) arrow stimuli presented in white on a black background of a 17-inch computer monitor approximately 20 inch from the participant’s head. Participants were instructed to respond as quickly and accurately as possible with a right-hand key press. An index-finger button press was used if the target stimulus (i.e., middle arrow) pointed to the left and a middle-finger button press was used if the target stimulus pointed to the right. Flanker stimuli were presented 100 ms prior to the onset of the target stimulus, which remained on the screen for 600 ms. If the participant responded after 1,600 ms, the trial was counted as an error of omission. The ITI varied randomly between 800 ms, 1,000 ms, and 1,200 ms, with a mean of 1,000 ms. Two blocks of 200 trials (400 total trials) were presented, with 160 congruent trials (40%) and 240 incongruent trials (60%). Participants completed 24 practice trials prior to beginning the experimental task to ensure adequate task understanding.

**Electrophysiological data recording**

Electroencephalogram (EEG) was recorded from 128 scalp sites using a geodesic sensor net and Electrical Geodesics, Inc. (EGI; Eugene, OR, USA) amplifier system (20K nominal gain, bandpass = 0.10–100 Hz). EEG was referenced to the vertex electrode and digitized continuously at 250 Hz with a 24-bit analog-to-digital converter. Impedances were maintained below 50 kΩ consistent with recommendations of the manufacturer. Data were high-pass filtered at 0.1 Hz and low-pass filtered at 30 Hz.

**ERP measurement**

Individual subject correct-trial N2 data were segmented spanning 250 ms prior to stimulus presentation to 1,000 ms after stimulus presentation. Eye blinks were removed from the segmented waveforms using independent components analysis (ICA) in the ERP PCA Toolkit (Dien, 2010), that uses EEGLAB (Delorme & Makeig, 2004). The ICA components that correlated at .9 with the scalp topography of two blink templates, one generated based on the current data and another provided by the ERP PCA Toolkit author, were removed from the data (see Dien, Michelson, & Franklin, 2010). Notably, there were no group differences in the number of trials corrected for ocular artifacts (ts < 0.98, ps > .33). Trials were considered bad if more than 15% of channels were marked bad. Channels were marked bad if the fast average amplitude exceeded 100 μV or if the differential average amplitude exceeded 50 μV. Channels were also marked globally bad for the entire session if more than 25% of the trials were deemed bad. To correct bad channels, spline interpolation was performed from good channels. Data were average referenced and used the polar average reference effect (PARE) correction to correct for undersampling of the undersurface of the head (Junghöfer, Elbert, Tucker, & Braun, 1999). Data were baseline corrected using a 150 ms window from −250 ms to −100 ms prior to stimulus presentation.

Electrode sites for analysis were chosen based on the scalp distributions of the current data (see supplemental Figure S1). All error and post-error trials were excluded from analyses (Clayson & Larson, 2011b). Correct-trial congruent and incongruent amplitudes for the N2 were extracted using an adaptive mean procedure. In this procedure the peak amplitude is first identified then an average is taken of 15 ms pre-peak to...
15 ms post-peak negative amplitude. The time window for the N2 was 300 ms and 410 ms. The time window is slightly later than commonly used for the N2 due to the flankers being presented 100 ms before the central conflict stimulus; however, the time window is still consistent with several studies of the N2 in children and adolescents (Henderson, 2010; Ladouceur, Conway, & Dahl, 2010). Amplitudes were averaged across three fronto-central electrode sites [numbers 6 (FCz), 12, and 5; see supplemental Figure S1]. In order to assess electrophysiological indicators of conflict adaptation, individual N2 segments were derived based the four possible current- and previous-trial congruency combinations: cC, iC, cl, and ii. After segmentation and artifact rejection, at least 15 segments per condition were retained for averaging for each participant. Error and post-error trials were excluded due to differences in RTs and potential confounding effects with conflict adaptation (see also Egner & Hirsch, 2005; Larson, Kaufman, & Perlstein, 2009b).

**Data analysis**

A 2-Group (ASD, TD) × 2-Previous-trial Congruency (congruent, incongruent) × 2-Current-trial Congruency (congruent, incongruent) mixed-model analysis of variance (ANOVA) was conducted on mean RTs, error rates, and N2 amplitudes for all trials. Partial-eta² (η²_p) is reported for all ANOVA effect sizes. Separate paired-samples t tests by group were conducted comparing iC and cC trials and cl and ii trials to determine if significant conflict adaptation was present. Previous-trial Congruency × Current-trial Congruency ANOVAs as a function of group were used to decompose significant three-way interactions. If both groups exhibited significant conflict adaptation effects, group comparisons for conflict adaptation effect sizes and task-switching effects were conducted. For behavioral and electrophysiological data the magnitude of conflict adaptation effects was determined by subtracting il from cl trials, and the magnitude of task-switching effects was calculated by subtracting cC from iC trials. Zero-order correlations were also conducted between behavioral and electrophysiological indices of conflict adaptation and measures of IQ, ADOS and age. Consistent with previous research (Nieuwenhuis et al., 2006), mean conflict adaptation scores for both behavioral and electrophysiological data used in the correlational analyses were calculated using the following formula: \((cC - iC) - (ii - ic)\). For ease of interpretation, the inverse value of N2 amplitude data (multiplied by negative one) was used in equations of conflict adaptation (i.e. in order to make conflict adaptation presentation positive similar to that for RT and error-rate analyses). Thus, similar to RTs and error rates, larger (i.e. more positive) magnitudes of conflict adaptation effects are indicative of improved recruitment of cognitive control and improved functioning.

**Results**

**Behavioral**

**Response times.** Mean RT data for conflict adaptation effects as a function of group are presented in Table 1 and Figure 1. A Group × Previous-trial Congruency × Current-trial Congruency ANOVA on RTs revealed a significant main effect of current-trial congruency, \(F(1,62) = 458.03, p < .001, \eta^2_p = .88\). Participants responded more quickly to congruent trials compared to trials following incongruent trials. The main effect of previous-trial congruency was also significant, \(F(1,62) = 23.59, p < .001, \eta^2_p = .28\). Participants generally responded more slowly on trials following congruent trials compared to trials following incongruent trials. The main effect of group was not significant, \(F(1,62) = 0.00, p = .99, \eta^2_p < .001\). The Group × Previous-trial Congruency interaction was not significant, \(F(1,62) = 0.25, p = .62, \eta^2_p = .004\). The Previous-trial Congruency × Current-trial Congruency interaction was significant indicating reliable conflict adaptation effects, \(^1 F(1,62) = 106.48, p < .001, \eta^2_p = .63\). RTs were shorter for il relative to cl.

To investigate the facilitative effects of repetition priming on the behavioral data, RTs and error rates were examined after excluding stimulus-response repetitions (Mayr, Awh, & Laurey, 2003). The Previous-trial Congruency × Current-trial Congruency interaction remained significant for RTs, \(F(1,63) = 25.11, p < .001, \eta^2_p = .29\), but not for error rates, \(F(1,63) = 0.11, p = .75, \eta^2_p = .002\). There was not an adequate signal-to-noise ratio for reliable N2 waveforms to examine conflict adaptation effects after excluding stimulus-response repetitions; however, previous work demonstrates that conflict adaptation effects remain intact for the N2 ERP when using a similar flanker paradigm (see Clayson & Larson, 2011a, 2011b).

---

1. To investigate the facilitative effects of repetition priming on the behavioral data, RTs and error rates were examined after excluding stimulus-response repetitions (Mayr, Awh, & Laurey, 2003). The Previous-trial Congruency × Current-trial Congruency interaction remained significant for RTs, \(F(1,63) = 25.11, p < .001, \eta^2_p = .29\), but not for error rates, \(F(1,63) = 0.11, p = .75, \eta^2_p = .002\). There was not an adequate signal-to-noise ratio for reliable N2 waveforms to examine conflict adaptation effects after excluding stimulus-response repetitions; however, previous work demonstrates that conflict adaptation effects remain intact for the N2 ERP when using a similar flanker paradigm (see Clayson & Larson, 2011a, 2011b).
trials collapsed across groups, t(63) = 3.90, p < .001. Participants responded more slowly for iC compared to cC trials, t(63) = -9.19, p < .001. The Group × Current-trial interaction was not significant, F(1,62) = 0.01, p = .91, $\eta^2_p < .001$. The Group × Previous-trial Congruency × Current-trial Congruency interaction was significant, F(1,62) = 5.96, p = .02, $\eta^2_p = .09$.

Decomposition of this effect revealed that both groups demonstrated reliable conflict adaptation effects as indicated by significant Previous-trial Congruency × Current-trial Congruency ANOVAs, ASD: F(1,27) = 46.67, p < .001, $\eta^2_p = .63$; TD: F(1,35) = 61.64, p < .001, $\eta^2_p = .64$. Individuals with ASD showed generally larger task-switching magnitudes; however, the comparison was not significant, t(62) = 1.83, p = .07. Groups did not differ in the magnitude of RT conflict adaptation effects, t(62) = 1.41, p = .17.

**Error rates.** Mean error-rate data for conflict adaptation effects are presented in Table 1 and Figure 1. A Group × Previous-trial Congruency × Current-trial Congruency ANOVA on error rates showed a significant main effect of previous-trial congruency with more errors committed following congruent trials relative to following incongruent trials, F(1,62) = 9.37, p = .003, $\eta^2_p = .13$. More errors were also committed for incongruent trials compared to congruent trials as indicated by a main effect of current-trial congruency, F(1,62) = 167.69, p < .001, $\eta^2_p = .73$. Individuals with ASD showed higher error rates than controls, F(1,62) = 7.67, p = .01, $\eta^2_p = .11$. The Previous-trial Congruency × Current-trial Congruency interaction was significant, F(1,62) = 43.68, p < .001, $\eta^2_p = .41$. Participants committed more errors for cI relative to iI trials and for iC compared to cC trials, t(63) = 6.57, p < .001; t(63) = -3.26, p = .002, respectively. The Group × Previous-trial Congruency interaction was not significant, F(1,62) = 3.69, p = .06, $\eta^2_p = .06$. The Group × Current-trial Congruency interaction was significant, F(1,62) = 4.15, p = .046, $\eta^2_p = .06$. Individuals with ASD showed larger congruency effects for pooled error rates than controls, t(62) = 2.04, p = .046. The Group × Previous-trial Congruency × Current-trial Congruency interaction was not statistically significant, F(1,62) = 0.11, p = .74, $\eta^2_p = .002$, indicating groups did not differ in conflict adaptation effects for error rates.

**N2 amplitude**

Mean N2 component amplitude data for conflict adaptation effects are presented in Table 1 and Figure 1. Grand averaged congruent and incongruent N2 waveforms as a function of group are presented in Figure 2. Grand averaged N2 waveforms for conflict adaptation effects for TD controls and youth with ASD are presented in Figure 2. For ERP data, we first examined the number of trials retained for each condition to test for group differences in signal-to-noise ratio. For individuals with ASD, ERPs contained an average ± standard deviation of 45 ± 17 trials for cC trials, 49 ± 20 for cI trials, 46 ± 19 for iC trials, and 58 ± 28 for il trials. For controls, ERPs contained an average of 54 ± 14 for cC trials, 62 ± 19 for cI trials, 60 ± 20 for iC trials, and 79 ± 29 for il trials.

The Group × Previous-trial Congruency × Current-trial Congruency ANOVA on N2 amplitudes indicated a significant main effect of previous-trial congruency, F(1,62) = 13.78, p < .001, $\eta^2_p = .18$; N2 amplitudes were more negative following congruent trials relative to following incongruent trials. N2 amplitudes were also more negative for incongruent trials compared to congruent trials, which was supported by a main effect of current-trial congruency, F(1,62) = 16.32, p < .001, $\eta^2_p = .21$. The main effect of group was not significant, F(1,62) = 0.05, p = .82, $\eta^2_p = .001$. Neither the Group × Previous-trial Congruency interaction nor Group × Current-trial Congruency interaction were significant, F(1,62) = 1.67, p = .20, $\eta^2_p = .03$; F(1,62) = 2.45, p = .12, $\eta^2_p = .04$, respectively. The Previous-trial Congruency × Current-trial Congruency interaction was significant,
F(1,62) = 4.69, p = .03, $\eta^2_p = .07$. N2 amplitude was more negative for cI trials compared to iI trials, t(63) = -4.52, p < .001; no differences were shown between iC and cC trials, t(63) = -1.41, p = .16. Most importantly, the Group × Previous-trial Congruency × Current-trial Congruency interaction was significant, F(1,62) = 4.71, p = .03, $\eta^2_p = .07$.

For individuals with ASD, a Previous-trial Congruency × Current-trial Congruency interaction revealed non-significant main effects of previous- and current-trial congruencies, F(1,27) = 1.96, p = .17, $\eta^2_p = .07$; F(1,27) = 1.99, p = .17, $\eta^2_p = .07$, respectively. Notably, the Previous-trial Congruency × Current-trial Congruency interaction was significant, F(1,20) < .001, p = .998, $\eta^2_p < .001$. For controls, the main effect of previous-trial congruency was significant with more negative N2 amplitudes for trials following congruent trials compared to trials following incongruent trials, F(1,35) = 19.22, p < .001, $\eta^2_p = .35$. N2 amplitudes were more negative for incongruent trials than for congruent trials as supported by a main effect of current-trial congruency, F(1,35) = 25.06, p < .001, $\eta^2_p = .42$. The Previous-trial Congruency × Current-trial Congruency interaction was significant indicating reliable conflict adaptation effects for the control group, F(1,35) = 11.05, p = .002, $\eta^2_p = .24$ (see Figure 2). N2 amplitudes were more negative for cI trials compared to iI trials, t(35) = -5.47, p < .001, consistent with conflict adaptation. No differences were shown between iC and cC trials, t(35) = -0.89, p = .38.

Correlational analyses
For individuals with ASD, higher ADOS-G scores were significantly associated with lower magnitudes of N2 conflict adaptation effects, r(26) = -0.45, p = .02

*(A Group × Previous-trial Congruency × Current-trial Congruency ANOVA on N2 amplitudes for two separate age groups based on a median split of age was conducted to ensure that group differences were not primarily the result of the large age range. In the younger group (ASD, n = 14, TD, n = 18), the 2-Group (ASD, TD) × 2-Previous-trial Congruency × 2-Current-trial interaction was significant, F(1,30) = 4.30, p = .047, $\eta^2_p = .13$. In the older group (ASD, n = 14, TD, n = 18), the Group × Previous-trial Congruency × Current-trial Congruency interaction was not significant, F(1,31) = 0.59, p = .55, $\eta^2_p = .02$. Neither younger or older individuals in the ASD groups showed significant Previous-trial Congruency × Current-trial Congruency interactions, Fs < 0.62, p > .44. The younger TD group showed a significant Previous-trial Congruency × Current-trial Congruency interaction, F(1,17) = 7.64, p = .01, $\eta^2_p = .31$. The Previous-trial Congruency × Current-trial Congruency interaction approached significance in the older TD group, F(1,17) = 3.57, p = .08, $\eta^2_p = .17$. Considering the small sample sizes in these analyses, we are confident that non-significant findings are attributed to insufficient power. Considering that age was significantly correlated with conflict adaptation effects, it would be expected that more older youth would be needed to detect significant effects. Thus, it does not seem that overall findings of ASD and TD group differences are accounted for by any relation to age.*

**Discussion**

The primary aim of this study was to examine cognitive control functioning – specifically conflict detection and conflict adaptation – in individuals with ASD. Consistent with expectations, individuals in both the ASD and control groups exhibited longer RTs and committed more errors on incongruent trials than congruent trials. Both groups also showed significant behavioral conflict adaptation effects with slower RTs and higher error rates occurring on cI trials then decreased on iI, iC, and cC trials, respectively. Most importantly, the individuals with ASD showed a relative absence of electrophysiological conflict adaptation; whereas control youth showed the expected conflict adaptation patterns – indicating alterations in the neural processes associated with the detection and resolution of conflict in the individuals with ASD. This study is, to the best of our knowledge, the first study of electrophysiological conflict adaptation in youth with an ASD diagnosis.

Current findings are consistent with previous research suggesting that ASD is associated with broader deficits in cognitive control functioning (Agam et al., 2010; Solomon, Ozonoff, Cummings, & Carter, 2008; Solomon et al., 2009). For example, Solomon et al. (2008) found that adolescents with ASD showed impaired ability to inhibit prepotent motor responses relative to controls. Response inhibition deficits in individuals with ASD during saccade tasks are coupled with reduced ACC activation relative to controls (Agam et al., 2010). In the present study, individuals with ASD exhibited non-significant neuroelectric conflict adaptation as well as non-significant differentiation between incongruent and congruent trials. Moreover, higher ADOS-G scores were associated with lower magnitudes of N2 conflict adaptation effects indicating that more

severe autism is associated with worse cognitive control abilities. These findings also suggest impaired ACC-mediated processes relative to TD controls, which likely subsume impairments in implementing the top-down biasing of cognitive control (Botvinick et al., 2001; Kerns et al., 2004). Considering the roles of the ACC and dIPFC in detecting conflict and implementing top-down cognitive control, present findings further support the notion that ASD is associated with impaired dIPFC and ACC functioning which likely contributes to social (Haznedar et al., 2000; Henderson et al., 2006) and communication impairments characteristic of ASD (Haznedar et al., 2000).

Typically developing controls exhibited significant conflict adaptation effects as indexed by the N2. These findings indicate that cognitive control processes are generally intact in neurotypical development, although these processes seem to be impaired in individuals with ASD. According to the conflict-monitoring theory, the ACC signals for increased cognitive control following high-conflict trials, which in turn minimizes the conflict-associated activation on the subsequent trial (Botvinick et al., 2001). The role of the ACC in signaling for increased control was coupled with strategic adjustments in behavior in TD controls; however, individuals with ASD demonstrated behavioral conflict adaptation effects in the absence of electrophysiological modulations of cognitive control.

The dissociation between behavioral and electrophysiological conflict adaptation is consistent with previous research in neurologic disorders. For example, intact behavioral conflict adaptation has been shown in individuals with mild (Larson, Farrer, & Clayson, 2011) and severe traumatic brain injury when electrophysiological conflict adaptation effects were impaired (Larson, Kaufman, & Perlstein, 2009a). The present findings seem to indicate that neurologic abnormalities in ASD are associated with irregular neuroelectric indices of conflict adaptation. It may be that ERPs provide a more sensitive measure of conflict adaptation processes. Alternatively, it is possible that the ERPs represent some compensatory neural processes to achieve similar behavioral effects between the ASD and control groups. Several studies suggest that irregular neural processes are present with similar behavioral performance between individuals with an ASD and controls, but the precise reason for these discrepancies remains unclear (e.g. Courchesne, Lincoln, Yeung-Courchesne, Elmasian, & Grillon, 1989; Ferri et al., 2003). One possible contributing factor is the ratio of incongruent to congruent trials in the current study. Several studies of conflict adaptation use a 70% congruent 30% incongruent or 50% congruent 50% incongruent ratio (e.g. Larson et al., 2009b). The 60% versus 40% ratio used in the current study may, therefore, represent a limitation and possible alternative explanation for these findings.

The current study provides some evidence that neural substrates of conflict adaptation may be associated with improved cognitive development. Specifically, the magnitude of N2 conflict adaptation effects reduced as age increased in the TD controls, suggesting that as individuals mature they may show more efficient electrophysiological conflict adaptation processes than youth. Notably, however, there was no correlation in the youth with an ASD, suggesting alterations in conflict-related developmental processes. Such changes may not be specific to only conflict adaptation, as it appears early aspects of the conflict-related ERP are also attenuated in individuals with ASD (see Figure 2). Future longitudinal studies are needed to clarify these age-related changes across cognitive control functions.

There are some additional limitations of the current manuscript. First, we were unable to remove repetition priming trials from the N2 ERPs due to insufficient signal-to-noise ratio. That said, two recent studies using an identical flanker task with 900 trials suggests repetition priming does not significantly affect conflict adaptation in the N2 (see Clayson & Larson, 2011a, 2011b) and methods were identical for both groups. Thus, we do not feel repetition priming differentially affected the between-groups findings. Second, the age range is quite large across groups (see Footnote 2). Finally, it is likely that differences in neural structure between those with ASD and controls could have contributed to the current findings.

In sum, the present study indicates that youth with ASD showed impaired electrophysiological conflict monitoring and conflict adaptation. HigherADOS-G scores were associated with poorer post-conflict recruitment of cognitive control. Both individuals with ASD and TD controls displayed behavioral conflict adaptation effects. Future studies directly examining trial-by-trial ACC and dIPFC activation are requisite to clarify how ACC abnormalities may impair augmentation of cognitive control in ASD.

Supporting information

Additional Supporting Information may be found in the online version of this article:

**Figure S1** Sensor layout of the 128-channel geodesic sensor net and voltage maps for the N2 difference wave (incongruent minus congruent) for Autism and Controls. The solid-line circle indicates fronto-central recording sites averaged for N2 activity

**Figure S2** Correlation between autism diagnostic observation schedules generic (ADOS-G) scores and inverse magnitude of N2 conflict adaptation effects for youth with an autism spectrum disorders (ASD)

Please note: Wiley-Blackwell are not responsible for the content or functionality of any supporting materials supplied by the authors. Any queries (other than missing material) should be directed to the corresponding author for the article.
Acknowledgements

We gratefully acknowledge the assistance of Annahir Cariello, Oliver Johnston, Jaime Ballard, Adrian Rockwell, Ryan Hunsaker, Sarah Van Tassell, Kyle Jamison, and Tiffani Newton. This study was supported by funds from the Brigham Young University College of Family, Home, and Social Sciences and a Brigham Young University Mentored Environment Grant.

Correspondence to

Michael J. Larson, PhD, Department of Psychology and Neuroscience Center, Brigham Young University, 244 TLRB, Provo, UT 84602, USA; Tel: 801-422-6125; Email: michael_larson@byu.edu

References


Accepted for publication: 26 September 2011
Published online: 16 December 2011