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Performance monitoring following conflict: Internal adjustments in cognitive control?

Michael J. Larson[a,b,*], Peter E. Clayson[a], Scott A. Baldwin[a]

[a] Department of Psychology, Brigham Young University, Provo, UT, United States
[b] Neuroscience Center, Brigham Young University, Provo, UT, United States

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ABSTRACT

The purpose of this study was to investigate the effects of strategic conflict-related adjustments in cognitive control processes on indices of performance monitoring. Previous research has examined the ability of parametric task-related manipulations to bias attention to errors; however, the present study sought to elucidate the effects of internal adjustments in control mediated by the anterior cingulate cortex on error-related conflict processing. High-density event-related potentials (ERPs) were obtained from 124 healthy individuals (68 female, 66 male) during a modified Eriksen flanker task. Behavioral measures (i.e., error rates, response times [RTs]) and N2 amplitudes showed significant conflict adaptation (i.e., previous-trial congruencies influenced current-trial measures). For error trials, the error-related negativity (ERN) was more negative for errors on high-conflict (i.e., incongruent) trials following high-conflict trials relative to errors on high-conflict trials following low-conflict (i.e., congruent) trials. These findings indicate that error-related conflict-monitoring processes adjust according to the post-conflict recruitment of strategic cognitive control and suggest an ongoing interplay between conflict and internal adjustments in control resources. Interpretations from the perspective of the conflict monitoring theory of cognitive control, the reinforcement learning theory, and the response–outcome theory of the ERN are discussed.

1. Introduction

Cognitive control refers to the ability to adapt to a changing environment and bias information processing to guide behavioral performance. The conflict monitoring theory of cognitive control posits that increased cognitive control is recruited after the detection of high conflict, when errors are likely (Botvinick, Carter, Braver, Barch, & Cohen, 2001; Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999). According to this model, conflict is detected by the anterior cingulate cortex (ACC), which in turn signals for the recruitment of strategic control to diminish conflict and improve subsequent performance (Botvinick et al., 2001; Cohen, Botvinick, & Carter, 2000; Hanslmayr et al., 2008; Kerns et al., 2004). This theory is supported by findings that the ACC is involved in evaluative processes and is activated by response conflict (Botvinick et al., 1999; Carter et al., 1998). Cognitive control theory also indicates that the dorsolateral prefrontal cortex (dlPFC) receives signals from the ACC to augment cognitive control and reduce subsequent conflict activation (Botvinick et al., 2001; Carter & van Veen, 2007; Durston et al., 2003; Egner & Hirsch, 2005a, 2005b; Kerns et al., 2004). The dlPFC minimizes conflict by providing top-down biasing of frontal and posterior systems that consequently reduce conflict and increase strategic focus (Corbetta & Shulman, 2002; Desimone & Duncan, 1995; Egner & Hirsch, 2005a; Rainer, Asaad, & Miller, 1998). Notably, recent data indicate that the right ventrolateral prefrontal cortex (vlPFC) is involved in the recruitment of cognitive control resources following high conflict, whereas the dlPFC is utilized following poor performance (Egner, 2011). Regardless, as a result of enhanced control, subsequent conflict-related activation is decreased. This link between the detection of conflict and the subsequent enhancement of cognitive control resources is known as the conflict-control loop (Carter & van Veen, 2007).

Studies of trial-by-trial adjustments in behavior support the idea of a conflict-control loop. For example, on conflict-laden tasks such as the flanker, current-trial performance is influenced by previous-trial congruency (Botvinick et al., 1999; Egner & Hirsch, 2005a, 2005b; Kerns et al., 2004; Stürmer, Soetens, Leuthold, Schröter, & Sommer, 2002). That is, individuals respond more quickly and accurately to an incongruent trial preceded by another incongruent trial (iI) relative to an incongruent trial preceded by a congruent trial (cI; e.g., Gratton, Coles, & Donchin, 1992; Stürmer et al., 2002; Ullsperger, Bylsma, & Botvinick, 2005). If the idea of a

* Corresponding author at: Department of Psychology and Neuroscience Center, Brigham Young University, 244 TLRB, Provo, UT 84602, United States.
Tel.: +1 801 422 6125; fax: +1 801 422 0163.
E-mail address: michael_larson@byu.edu (M.J. Larson).

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conflict-control loop is correct, ACC activation should be decreased on an iI trial compared to a cI trial, indicating top-down biasing of control by the pre-frontal cortex (PFC) following a higher-conflict incongruent trial (Botvinick et al., 2001; Egner & Hirsch, 2005a, 2005b). This notion is supported by neuroimaging studies showing greater activation of the ACC to cI trials relative to iI trials and greater activation of the lateral PFC on iI compared to cI trials (Egner & Hirsch, 2005b; Kerns, 2006; Kerns et al., 2004). Further support for the conflict-control loop comes from evidence showing decreased conflict-related event-related potential (ERP) and fMRI activity across consecutive incongruent trials (Clayson & Larson, 2011a; Durston et al., 2003). These trial-by-trial adjustments associated with the recruitment of cognitive control following conflict are frequently referred to as conflict adaptation Gratton, or sequential-trial effects.

Yeung, Botvinick, and Cohen (2004) provided an extension of the conflict monitoring theory and how it may pertain to ACC activation on error trials shown by various fMRI studies (e.g., Carter et al., 1998). Larger ACC activation following erroneous responses relative to correct responses putatively reflects response conflict generated by the activation of a fast erroneous response and a subsequent corrective response. Thus, ACC activation is contingent upon continued processing of the target stimulus after an erroneous response, with increased target-stimulus processing associated with increased post-response conflict following errors (Yeung & Cohen, 2006).

The idea of a conflict-control loop would indicate that error-related ACC activity should be influenced by the amount of control present on a current trial. Considering that error-related ACC activity is dependent on target-stimulus processing following errors (Danielmeier, Wessel, Steinhauser, & Ullsperger, 2009; Yeung & Cohen, 2006; Yeung, Ralph, & Nieuwenhuis, 2007), processing should be heightened on high-conflict trials following high-conflict trials (e.g., incongruent trials following incongruent trials; iI) when more cognitive control is present relative to high-conflict trials following low-conflict trials (e.g., incongruent trials following congruent trials; cI). One way to test this possibility is looking at error activation on incongruent trials following either a congruent or incongruent trial (cI or iI). In line with the reasoning above, we expected increased putative ACC activity on errors for iI trials when control is heightened relative to cI trials.

In regards to attention, previous research indicates that ACC activation on error trials is significantly associated with neuropsychological measures of attention, such that increased attention is associated with enhanced ACC activation on error trials (Larson & Clayson, 2011). However, the examination of error-related conflict monitoring following the recruitment of cognitive control provides insight into the effects of modulations in adaptive strategic cognitive control based on individual task performance, rather than from specific manipulations of task structure. Without using task-related manipulations to alter levels of conflict, the present study will examine whether internal adjustments in cognitive control following high-conflict trials enhances post-response error monitoring processes.

To examine error-trial conflict monitoring, we investigated the effects of conflict adaptation on neural correlates of cognitive control using scalp-recorded ERPs. The error-related negativity (ERN) is a response-locked negative deflection in the ERP with fronto-central scalp distribution that putatively reflects conflict activation of the ACC due to competing error and correct responses (e.g., Danielmeier et al., 2009; Yeung et al., 2004) and peaks within 100 ms after an erroneous response (Falkenstein, Hohnsbein, Hoormann, & Banke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). The conflict N2 is a negative deflection in the stimulus-locked ERP with a fronto-central scalp distribution that peaks approximately 250–350 ms after stimulus presentation and represents conflict detection (Folstein & Van Petten, 2008; Nieuwenhuis, Yeung, Van Den Wildenberg, & Ridderinkhof, 2003; Yeung & Cohen, 2006; Yeung et al., 2004). This role in conflict detection is supported by studies demonstrating that N2 amplitude is more negative (larger) on incongruent trials relative to congruent trials (Clayson & Larson, 2011a; Danielmeier et al., 2009; Forster, Carter, Cohen, & Cho, 2011). Source localization studies, including some using in vivo depth electrodes, have implicated the ACC in both ERN (Brazdil, Roman, Daniel, & Rektor, 2005; Stemmer, Segalowitz, Witzke, & Schonle, 2004; van Veen & Carter, 2002) and N2 generation (Ladouceur, Dahl, & Carter, 2007; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Yeung et al., 2004). Taken together, the ERN and N2 reflect similar conflict monitoring process in the ACC with the ERN being associated with conflict between a correct response representation and an executed erroneous response and the N2 reflective of conflict between target-stimulus and flanker processing (Yeung et al., 2004).

By investigating the effects of the top-down biasing of strategic cognitive control on ACC-mediated performance-monitoring processes, we hope to elucidate the effects of modulations of cognitive control on error-trial conflict monitoring (ERN) and stimulus-related conflict monitoring (conflict N2) processes. We hypothesized that ERN amplitudes would be more negative on errors to iI trials relative to cI trials, indicative of enhanced cognitive control associated with the top-down biasing of cognitive control to facilitate enhanced target-stimulus processing (Botvinick et al., 2001). Second, considering that conflict N2 amplitudes are more negative for task-irrelevant information processing (Danielmeier et al., 2009; Yeung & Cohen, 2006; Yeung et al., 2007), we expected conflict N2 amplitudes to be less negative on iI compared to cI trials, indicative of enhanced cognitive control.

2. Materials and methods

2.1. Participants

All participants provided written informed consent as approved by the Brigham Young University Institutional Review Board. Participants were recruited from undergraduate psychology courses. Exclusion criteria included current or previous diagnosis of a psychiatric disorder, psychoactive medication use, substance use or dependence, neurological disorders, head injury, left-handedness, or uncorrected visual impairment. As noted above, the primary purpose of this study was to examine the neural response to errors. Thus, participants that committed fewer than 8 errors on each of the iI and cI trial combinations were omitted from data analysis in order to maintain adequate signal-to-noise ratio (Olvet & Hajcak, 2009). Thus, we analyzed data from a final sample of 124 neurologically and psychiatrically healthy participants who each committed at least 8 errors across study conditions (63 female, 61 male; 17–27 years of age, $M = 20.4$, $SD = 2.2$).

2.2. 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 in. computer monitor approximately 20 in. from the participant's head. Participants were instructed to respond as quickly and accurately as possible with a right-hand key press to the central arrow of a five-arrow array. An index-finger button press was used if the central arrow pointed to the left and a middle-finger button press was used if the central arrow pointed to the right. Flanker stimuli were presented for 100 ms prior to the onset of the central arrow, which remained on the screen for 600 ms. The response window was 1600 ms. If the participant responded after 1600 ms, the trial was counted as an error of omission. The inter-trial interval (ITI) varied randomly between 800 ms, 1000 ms, and 1200 ms, with a mean ITI of 1000 ms. Three blocks of 300 trials (900 total trials) were presented; the task included 405 congruent trials (45%) and 495 incongruent trials (55%). Participants completed 24 practice trials prior to beginning the experimental task.

2.3. Electrophysiological data recording and reduction

Electroencephalogram (EEG) was recorded from 128 scalp sites using a geodesic sensor net and Electrical Geodesics, Inc. (EGI; Eugene, OR) amplifier system (20 K nominal gain, bandpass = .10–100 Hz). Electroencephalogram was

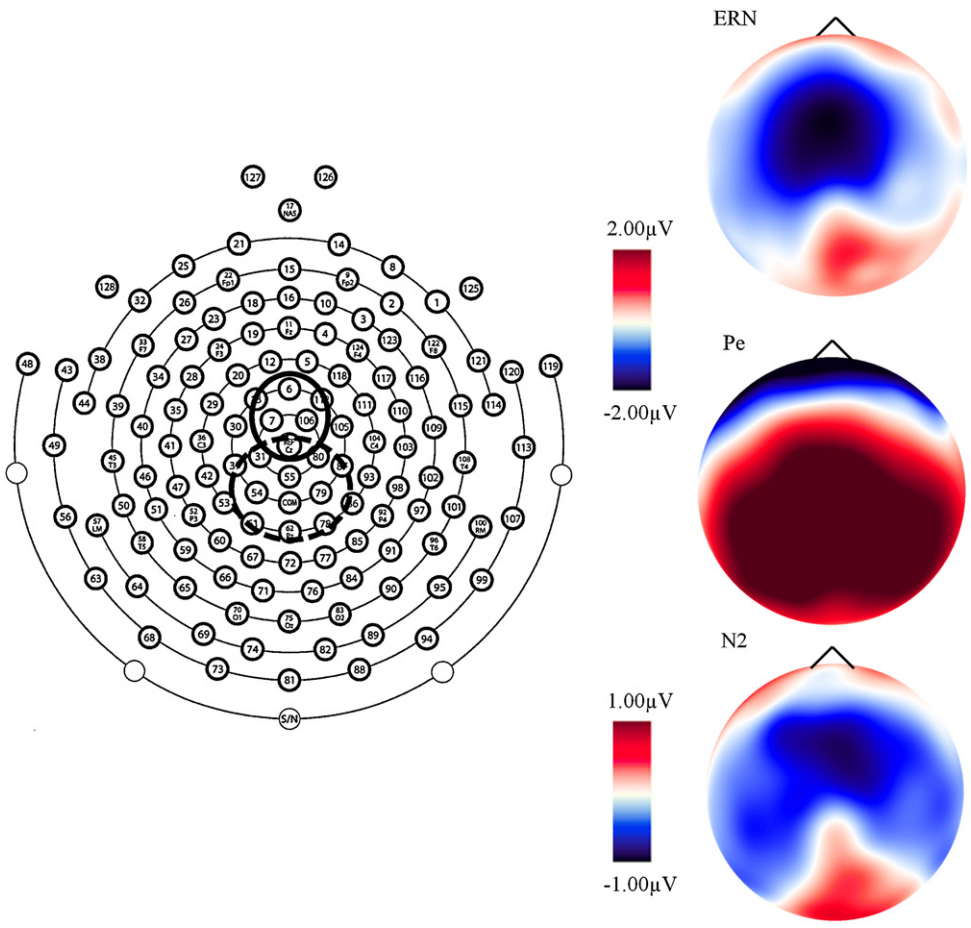

Fig. 1. Sensor layout of the 128-channel geodesic sensor net and voltage maps for the response-locked error minus correct difference for the error-related negativity (ERN) and post-error positivity (Pe) and the stimulus-locked incongruent minus congruent N2. The solid-line circle indicates fronto-central recording sites averaged for ERN and N2 activity, the dotted-line circle indicates centro-parietal recording sites averaged for Pe amplitudes.

initially referenced to the vertex electrode and digitized continuously at 250 Hz with a 24-bit analog-to-digital converter. Consistent with guidelines recommended by the manufacturer, impedances were maintained below 50 kΩ. Data were average-referenced and digitally low-pass filtered at 30 Hz. Eye movement and blink artifacts were corrected using the algorithm described by Gratton, Coles, and Donchin (1983).

For the ERN and post-error positivity (Pe), individual-subject response-locked averages were calculated using a window from 400 ms prior to participant response to 800 ms following participant response. We used a 200 ms time window from 400 ms to 200 ms before the response for baseline correction. Trials containing errors of omission were excluded from averages. Individual-subject, correct-trial N2 data were segmented spanning 150 ms prior to stimulus presentation to 500 ms after stimulus presentation. Epochs were baseline corrected using a 150 ms window from 150 ms before presentation to presentation of the target stimulus. Electrode sites for analysis were chosen based on the scalp distribution of the ERP components of interest (see Fig. 1; e.g., Clayson & Larson, 2011a; Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Gehring et al., 1993; Nieuwenhuis et al., 2003). Error-related negativity and N2 amplitudes were averaged across four fronto-central electrode sites (numbers 6 [FCz], 7, 106, and Ref [Cz]; see Clayson, Clawson, & Larson, 2011). Correct-trial and error-trial ERN amplitudes were extracted as the average of 15 ms pre-peak to 15 ms post-peak negative amplitude within 100 ms of the response. Correct-trial congruent and incongruent amplitudes for the N2 were extracted as the average of 15 ms pre-peak to 15 ms post-peak negative amplitude between 270 ms and 380 ms. Considering previous findings that the Pe is found at centro-parietal electrode locations (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005), error-trial and correct-trial Pe amplitudes were extracted as the individuals-subject mean amplitude from 200 ms to 400 ms post-response across five centro-parietal electrode sites (31, 54, 62 [Pz], 79 and 80).

In order to assess conflict adaptation, individual correct-trial and error-trial ERN and Pe segments were derived based on two possible previous-trial congruencies: iI and cI. Considering that few errors are committed on congruent trials, the ERN investigation only examined current incongruent trials as a function of previous-trial congruency. N2 segments were derived based on four possible current- and previous-trial congruency combinations: a congruent trial following another congruent trial (cC), a cI trial, a congruent trial following an incongruent trial (iC), and an iI trial. For N2 analysis, error and post-error trials were excluded (see also Egner & Hirsch, 2005b; Larson, Kaufman, & Perlstein, 2009).

Table 1
Mean response time (RT; ms), error rates, and ERP component amplitude (μV) data for each previous-trial and current-trial pair (n = 124).

	Mean	*SD*
cC RT	365	34
iC RT	387	36
iI RT	446	35
cI RT	452	36
cC error rates (%)	5	8
iC error rates (%)	6	8
iI error rates (%)	12	9
cI error rates (%)	19	10
cC N2 amplitude	0.44	1.41
iC N2 amplitude	0.49	1.43
iI N2 amplitude	−.02	1.68
cI N2 amplitude	−.46	1.70
cI CRN amplitude	−.57	0.70
cI ERN amplitude	−3.36	1.68
iI CRN amplitude	−.57	0.72
iI ERN amplitude	−3.63	2.04

Note. ERP, event-related potential; cC, congruent trial preceded by a congruent trial; iC, congruent trial preceded by an incongruent trial; iI, an incongruent trial preceded by an incongruent trial; cI, incongruent trial preceded by a congruent trial; CRN, correct-related negativity; ERN, error-related negativity.

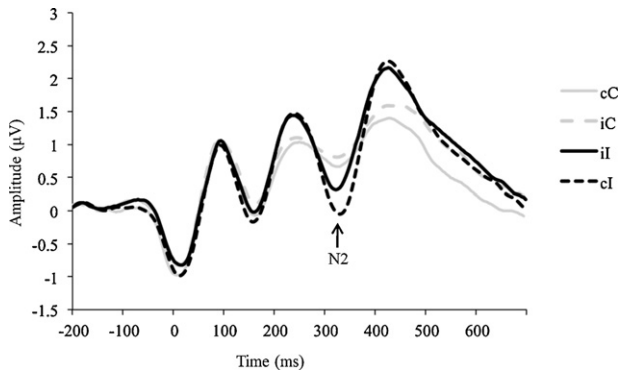

Fig. 2. Grand averaged stimulus-locked ERP waveforms ERP activity averaged across front-central electrode locations for the N2 for each previous-trial and current-trial pair.

2.4. Data analysis

First, conflict adaptation effects were established in behavioral and electrophysiological data using a 2-previous-trial congruency (congruent, incongruent) × 2-current trial congruency (congruent, incongruent) mixed-model analysis of variance (ANOVA) on mean RTs, error rates, and N2 amplitudes. *Partial-eta*^{2} (η_p^2) is reported for all ANOVA effect sizes. Significant previous-trial congruency × current-trial congruency interactions were decomposed using paired samples *t* tests on behavioral and electrophysiological data for cI and iI trials as well as cC and iC trials. After demonstrating conflict adaptation effects and ERN and Pe amplitudes, a previous-trial congruency (cI, iI) × accuracy (correct, error) ANOVA on ERN and Pe amplitudes was conducted. Significant interactions were decomposed using paired samples *t* tests.

3. Results

3.1. Response times and error rates

Response time and error rate data for conflict adaptation effects are presented in Table 1. A previous-trial congruency × current-trial congruency ANOVA on mean RTs revealed a significant main effect of previous-trial congruency, $F(1,123) = 190.87$, $p < .001$, $\eta_p^2 = .61$. Participants responded more slowly after congruent compared to incongruent trials. Participants also responded more slowly to incongruent trials relative to congruent trials as indicated by a main effect of current-trial congruency, $F(1,123) = 2463.65$, $p < .001$, $\eta_p^2 = .95$. The previous-trial congruency × current-trial congruency interaction[1] was significant indicating reliable conflict adaptation effects, $F(1,123) = 507.71$, $p < .001$, $\eta_p^2 = .81$. Follow-up contrasts indicated that RTs were shorter for iI trials than cI trials, $t(123) = 7.96$, $p < .001$, and shorter for cC trials than iC trials, $t(123) = -23.75$, $p < .001$.

The previous-trial congruency × current-trial congruent interaction for error rates showed a significant main effect of previous-trial congruency, $F(1,123) = 159.90$, $p < .001$, $\eta_p^2 = .57$. Participants responded less accurately after congruent trials relative to incongruent trials. The main effect of current-trial congruency was also significant with participants responding less accurately to incongruent trials compared to congruent trials, $F(1,123) = 324.03$, $p < .001$, $\eta_p^2 = .73$. The previous-trial congruency × current-trial congruency interaction was significant, suggesting reliable conflict adaptation effects, $F(1,123) = 311.28$, $p < .001$, $\eta_p^2 = .72$ (see Footnote 1). Importantly, error rates were higher for cI trials relative to iI trials, $t(123) = 16.90$, $p < .001$, and for iC trials compared to cC, $t(123) = -3.93$, $p < .001$.

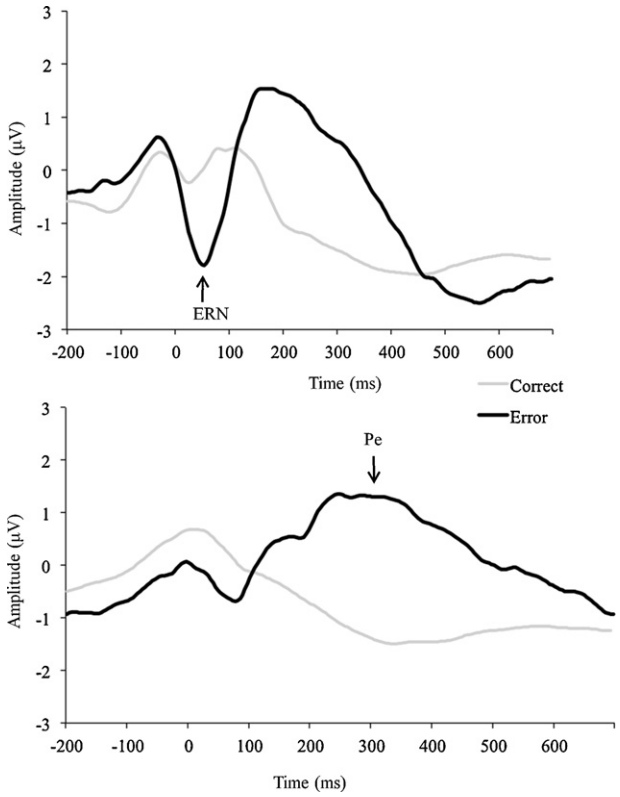

Fig. 3. Grand averaged response-locked ERP activity averaged across fronto-central electrode locations for the error-related negativity (ERN) and averaged across centro-parietal locations for the post-error positivity (Pe).

3.2. Event-related potentials

Grand averaged ERP waveforms for the stimulus-locked ERPs are presented in Fig. 2 and response-locked ERPs are presented in Fig. 3. The grand averaged waveforms for correct response negativity (CRN) and ERN difference waves (iI minus cI) and the corresponding mean values are presented in Fig. 4. Mean ERP component amplitude data are presented in Table 1 and Figs. 1 and 4. The average ± *SD* number of segments contained for stimulus-locked ERP trials were 152 ± 33 for cC trials, 161 ± 32 for cI trials, 158 ± 29 for iC trials, and 177 ± 40 for iI trials. Response-locked waveforms contained an average ± *SD* of 34 ± 27 for error trials and 691 ± 162 for correct trials. Response-locked error trials contained 20 ± 12 cI trials and 11 ± 5 iI trials.

For the N2 component, the previous-trial congruency × current-trial congruency interaction showed significant main effects of previous-trial congruency and current-trial congruency, $F(1,123) = 23.95$, $p < .001$, $\eta_p^2 = .16$; $F(1,123) = 64.77$, $p < .001$, $\eta_p^2 = .35$, respectively. N2 amplitudes were more negative following congruent trials than following incongruent trials, and N2 amplitudes were more negative for incongruent trials relative to congruent trials. The previous-trial congruency × current-trial

[1] After excluding stimulus–response repetitions, the Previous-trial congruency × current-trial congruent interactions remained significant for RTs, $F(1,123) = 120.99$, $p < .001$, $\eta_p^2 = .50$, and error rates, $F(1,123) = 53.66$, $p < .001$, $\eta_p^2 = .30$. We note, however, that we cannot rule out the possible contribution of feature integration effects (e.g., Hommel, Proctor, & Vu, 2004; Notebaert, Soetens, & Melis, 2001) because it is impossible to disentangle the different stimulus and response combinations from congruency effects using a flanker task with two possible stimulus-arrow directions (i.e., left and right) and two possible flanker-arrow directions. We note this as a limitation and possible alternative explanation of the current findings.

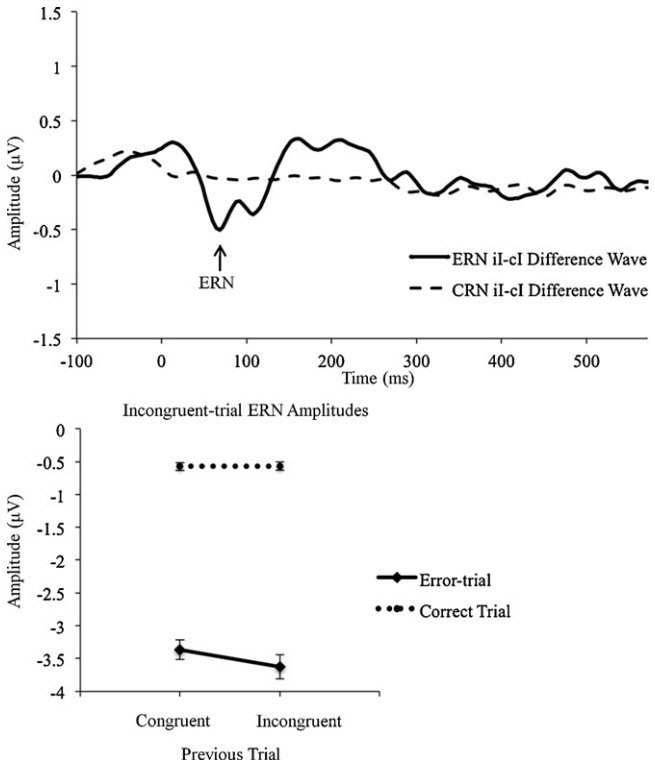

Fig. 4. Top: Grand averaged correct-related negativity (CRN) and error-related negativity (ERN) difference waves (incongruent trial preceded by incongruent trial [iI] minus incongruent trial preceded by congruent trial [cI]). Bottom: Mean CRN and ERN amplitudes for current incongruent trials as a function of previous-trial congruency. Error bars represent the standard error.

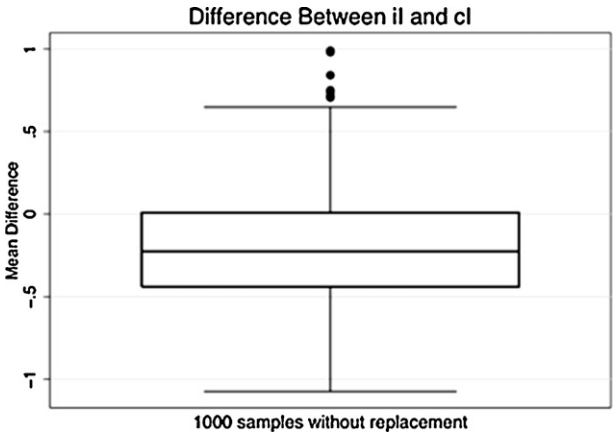

Fig. 5. Mean ERN amplitude differences for errors committed on iI trials minus errors committed on cI trials randomly sampled from eight iI errors and eight cI errors from each participant 1000 times without replacement. Negative values indicate more negative ERN amplitudes to errors on iI trials relative to cI trials.

congruent interaction[2] was significant, showing that significant conflict adaptation effects were elicited, $F(1,123) = 18.45$, $p < .001$, $\eta_p^2 = .13$ (see Figs. 1 and 2). N2 amplitudes were more negative for cI trials compared to iI trials, $t(123) = -6.67$, $p < .001$; no differences were shown between cC and iC trials, $t(123) = -.70$, $p = .48$.

A previous-trial congruency × accuracy ANOVA on ERN amplitudes indicated the main effect of previous-trial congruency, with more negative amplitudes for iI trial compared to cI trials, $F(1,123) = 78.16$, $p < .001$, $\eta_p^2 = .38$. Amplitudes were also more negative on error trials relative to correct trials as supported by a main effect of accuracy, $F(1,123) = 305.24$, $p < .001$, $\eta_p^2 = .71$. The previous-trial congruency × accuracy interaction (see Footnote 2) was also significant, $F(1,123) = 6.03$, $p = .02$, $\eta_p^2 = .05$.

The primary analysis of interest was significant. For errors, iI trials were associated with more negative ERN amplitudes than cI trials, $t(123) = 2.38$, $p = .02$ (see Fig. 4). No differences were demonstrated for correct-trial amplitudes between iI and cI trials, $t(123) = 0.38$, $p = .70$. It is possible that the differences in the number of error trials between iI and cI trials contributed to these findings. As noted above, all participants had at least eight error trials in each condition. To further examine the impact of number of trials on the current findings, we randomly sampled 8 iI errors and 8 cI errors from each participant 1000 times without replacement. We then calculated the iI minus cI difference for each of these random samples. The distribution of these samples is presented in Fig. 5 (negative values indicate more negative ERN amplitudes to errors on iI trials relative to cI trials). As can be seen in the figure, the mean differences remain in the expected direction even with the inclusion of several extreme positive values.

The previous-trial congruency × accuracy ANOVA on Pe amplitudes revealed the main effect of previous-trial congruency, $F(1,123) = 2.32$, $p = .03$, $\eta_p^2 = .04$. Pe amplitudes were more positive for iI trials compared to cI trials. Amplitudes were also more positive for error trials than for correct trials as indicated by the main effect of accuracy $F(1,123) = 76.65$, $p < .001$, $\eta_p^2 = .38$. The previous-trial congruency × accuracy interaction was not significant, $F(1,123) = 0.68$, $p = .41$, $\eta_p^2 = .005$.

4. Discussion

Consistent with the role of the ACC in the conflict-control loop, we hypothesized that error-trial conflict activation would be increased following an incongruent trial relative to following a congruent trial due to enhancements in the processing of task-relevant information associated with the top-down biasing of control. This was evidenced by increased ERN amplitude for error trials following incongruent trials compared to error trials following congruent trials. Furthermore, mean differences on cI and iI trials remained in the expected direction following random samples of eight trials per conflict adaptation condition—thus, findings are not the result of different numbers of trials per conflict adaptation combination (e.g., iI versus cI). Importantly, CRN amplitudes were similar following congruent and incongruent trials. Such a pattern of responses indicates that changes are specific to conflict monitoring activity and not a generalized finding across all response-related ERPs. These finding corroborate the proposed role of the ACC in the conflict-control loop by evidencing that adaptive adjustments in cognitive control enhance focus on task-relevant information.

Current findings of the role of selective control in error-related conflict processing converge well with previous research contending that the ERN reflects conflict monitoring processes. Decreased error-trial conflict monitoring has been shown in an individuals with a left-ACC lesion (Swick & Turken, 2002), after alcohol

[2] The previous-trial congruency × current-trial congruency interaction was not significant for N2 amplitudes after excluding stimulus–response repetitions, $F(1,123) = 1.65$, $p = .20$, $\eta_p^2 = .01$. However, the means followed the expected pattern of less negative N2 amplitudes for iC trials ($M = 0.2$, $SD = 1.6$) relative to cC trials ($M = 0.1$, $SD = 1.7$) and less negative N2 amplitudes for iI trials ($M = -.4$, $SD = 1.9$) compared to cI trials ($M = -.7$, $SD = 1.6$). Previous research investigating N2 conflict adaptation effects indicates that after omitting stimulus–response repetitions in larger samples of 181 and 210 participants N2 conflict adaptation effects remain significant (see Clayson & Larson, 2011a,b). Thus, the current sample is likely underpowered to detect subtle differences in N2 amplitude associated with conflict adaptation after removing stimulus–response repetitions. Considering that repetitions priming effects are not the primary aim of the current examination, we refer readers elsewhere for a more thorough discussion of the effects of repetition priming on N2 conflict adaptation effects (see Clayson & Larson, 2011b).

consumption (Ridderinkhof et al., 2002), for masked trials relative to unmasked trials (Hughes & Yeung, 2011), for trials with a dim compared to a bright target-stimulus (Yeung et al., 2007), and for trials with flankers close in proximity to the target stimulus relative to far away from the target stimulus (Danielmeier et al., 2009). Moreover, conflict activation to errors is greater for individuals scoring higher on neuropsychological measures of attention (Larson & Clayson, 2011). The present study extends these findings by demonstrating that error-trial conflict monitoring is affected by changes in strategic control within an individual related to internal allocation of cognitive resources following conflict.

Current findings also add to previous studies of the ACC and conflict-driven processes. For example, seminal studies by Botvinick et al. (1999) and Kerns et al. (2004), showed increased ACC activation to cI trials relative to iI trials, similar to the increased N2-related activity on cI relative to iI trials in the current study. We note that in the Botvinick et al. (1999) paper ACC activity on iI trials did not significantly differ from that on the congruent trials. Several subsequent studies, including that of Kerns et al. (2004), however, show the expected differentiation between congruent and incongruent trials as a function of previous-trial congruency. Our finding of subsequently decreased error-related putative ACC-activity on cI trials relative to iI trials further suggest that cognitive control adjustments are made on the basis of the degree of conflict that is influenced by both the previous and current trial. Also consonant with our results, Bartholow et al. (2005) showed increased N2 amplitude on trials with higher levels of conflict (e.g., cI trials in the current study); however, in contrast to current results the Bartholow et al. study showed more negative incongruent-trial ERN amplitudes in a congruent-probable condition relative to incongruent-probable condition—leading to the suggestion that expectation of stimulus congruency can influence the ACC-mediated response to errors. Conflict adaptation effects, as assessed in the current paradigm, occurred on a trial-to-trial basis, thus no overall level of expectation was established. Further, Bartholow et al. urged caution in interpreting their results given the low number of error trials produced in their sample. Future studies are requisite to corroborate the present findings and determine the role or expectancy effects on internal adjustments in control.

Despite the seemingly natural fit of the current findings with the conflict monitoring theory of cognitive control, the current data do not rule out other theories of ERN generation. For example, some theorists hypothesize that the ERN represents an affective distress signal when performance is less than ideal (e.g.,Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). It is possible that there is an increased affective response to errors committed during conditions of heightened cognitive control (i.e., iI trials) relative to decreased control leading to ERN differences between trial conditions. Future studies directly examining the aversive nature of errors as a function of the degree of control provided to the stimulus would be needed to investigate this possibility.

The present findings could also be explained by another prominent model of ERN generation, the reinforcement learning theory (RL-ERN; Holroyd & Coles, 2002), as well as with predictions of the response–outcome theory (PRO; Alexander & Brown, 2010). The RL-ERN theory is based upon the temporal-difference hypothesis of dopaminergic functioning and posits that the basal ganglia act as an "adaptive critic", by signaling the ACC when performance outcomes are better or worse than anticipated (Holroyd & Coles, 2002; Holroyd, Yeung, Coles, & Cohen, 2005). Activation of the ACC is dependent upon reinforcement learning properties of the mesencephalic dopaminergic system and is greater when deviations from temporal difference predictions are noted based upon prior learning (i.e., when events are worse than expected). The ACC integrates previous response–outcome predictions over time to guide performance (Holroyd & Coles, 2008; Kennerley, Walton, Behrens, Buckley, & Rushworth, 2006). Greater error-trial ACC activation is shown when error expectancy is low, which is associated with a greater deviation from learned response–outcome predictions (Holroyd, Krigolson, Baker, Lee, & Gibson, 2009). Thus, considering that iI trials were associated with decreased error rates relative to cI trials, it may be considered than an error on an iI trial may associated with a larger violation of error expectancy and subsequent greater ACC activation compared to a cI trial.

The PRO model similarly posits that error-trial ACC activation is dependent upon error expectancy (Alexander & Brown, 2010; Brown & Braver, 2008). However, according the PRO model the medial PFC plays the role of adaptive critic and influences response preparation and execution (Alexander & Brown, 2010). The basal ganglia are involved in the selection of already planned responses to determine which to execute based on previous learning (Brown, Bullock, & Grossberg, 2004). The PRO model predicts that ACC activation will be greatest for unexpected erroneous outcomes as well as unexpected correct outcomes (Alexander & Brown, 2010; Jessup, Busemeyer, & Brown, 2010); the expectedness of an outcome is learned from experience. Thus in this case, the PRO model makes similar predictions regarding ACC activation on iI compared to cI trials; iI trials should be associated with greater ACC activation as it is a greater violation of error expectedness than a cI error. As such, the present investigation does not dissociate between the current models of ERN generation.

According to the error awareness hypothesis, the Pe reflects conscious recognition of erroneous responses (Overbeek et al., 2005). Thus, when control is heightened individuals should attend more to errors as reflected in Pe amplitude (Nieuwenhuis et al., 2001). The current results indicate that neural activation is decreased for cI relative to iI trials for both correct and error trials. These findings may indicate that less neural activation is requisite to register whether a response was incorrect following high-conflict trials when cognitive control is recruited.

Consistent with previous research, we anticipated that N2 amplitudes would be modulated by the recruitment of cognitive control associated with conflict. Current-trial conflict monitoring was sensitive to previous-trial congruency, suggesting that ACC-mediated processes are sensitive to the recruitment of cognitive control. Although dlPFC activity was not directly measured in the present study, previous findings indicate that enhancement of cognitive control by the dlPFC after high-conflict trials serves to minimize subsequent conflict activation (see also Nieuwenhuis, Schweizer, Mars, Botvinick, & Hajcak, 2007). Conflict activation was larger for cI relative to iI trials as indexed by more negative conflict N2 amplitudes for cI trials than for iI trials. These findings suggest that greater strategic control resources were allocated following high-conflict compared to low-conflict trials and minimized conflict activation on high-conflict trials. This notion is further corroborated by adaptive RT and error-rate adjustments. On iI trials, RTs were shorter and error rates were decreased compared to cI trials. Notably, the current ERP results suggest alterations in frontally mediated strategic control processes, but only indirectly assess more sensory attentional control processes. It is possible that the current findings are directly related to strategic control adjustments, although attentional processes may also play a significant role. Taken as a whole, however, current results show reliable conflict adaptation effects in the data for RTs, error rates, and N2 amplitudes.

In sum, the present study demonstrated that indices of performance monitoring, such as RTs, error rates, the conflict N2, and the ERN, are sensitive to strategic adjustments in cognitive control. Notably, for iI relative to cI trials ACC-mediated conflict activation, error rates, and RTs were decreased, suggesting that both behavioral and electrophysiological indices of performance monitoring are influenced by conflict adaptation effects. These findings indicate

an interplay between the detection of conflict, internal adjustments in cognitive control, and subsequent performance.

Conflict of interest

The authors report no conflicts of interest.

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