Neural time course of conflict adaptation effects on the Stroop task

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A B S T R A C T

Cognitive control theory suggests conflict effects are reduced following high- relative to low-conflict trials. Such reactive adjustments in control, frequently termed “conflict adaptation effects,” indicate a dynamic interplay between regulative and evaluative components of cognitive control necessary for adaptable goal-directed behavior. The current study examined conflict adaptation effects while 36 neurologically-normal participants performed a single-trial color-naming Stroop task. Trials preceded by incongruent (high conflict) and congruent (low conflict) trials were compared for behavioral (response time [RT] and error rate) and electrophysiological (N450 and conflict SP components of the event-related potential [ERP]) concomitants of cognitive control. A conflict adaptation effect was present for RTs that could not be accounted for by associative or negative priming. ERPs revealed a parietal conflict slow potential (conflict SP) that differentiated incongruent from congruent trials and monotonically differentiated current trial congruency on the basis of previous-trial context (i.e., showed conflict adaptation); the fronto-medial N450 was sensitive to current trial congruency but not to previous-trial context. Direct comparison of normalized conflict SP and N450 amplitudes showed the conflict SP was sensitive to the effects of previous-trial context, while the N450 was so to a lesser extent and in a different pattern. Findings provide clarification on the neural time course of conflict adaptation and raise further questions regarding the relative roles of the parietal conflict SP and fronto-medial N450 in conflict detection and processing.

Goal-directed behavior requires a flexible and adaptive cognitive control system for recognizing appropriate or inappropriate task completion and dynamically adjusting performance when control is misdirected or inadequate. The cognitive control mechanisms required to monitor for performance errors or response conflict (i.e., the simultaneous activation of two competing response options) and to signal for subsequent adjustments are critical to adaptive behavior and efficient task completion (see Botvinick, Carter, Braver, Barch, & Cohen, 2001). Performance adjustments include slowing of response time (RT) following an error to increase accuracy (i.e., the Rabbitt effect; Rabbitt, 1966, 1968) and facilitation of RTs following high- relative to low-conflict trials (i.e., the Gratton or “conflict adaptation” effect; Gratton, Coles, & Donchin, 1992).

Several authors have utilized the Stroop color-naming task to examine such performance adjustments. In the Stroop task (Stroop, 1935), conflict is greater for incongruent (e.g., the word BLUE written in red) than congruent (RED written in red) color-naming trials due to the simultaneous activation of competing representations. Behavioral adjustments in control following high conflict include faster RTs on incongruent trials preceded by incongruent trials (hereafter referred to as iI trials) than on incongruent trials preceded by congruent trials (ci), and slower RTs for congruent trials preceded by incongruent trials (iC) relative to congruent trials preceded by congruent trials (cC) (Kerns et al., 2004). The explanation offered for this pattern of performance adjustments is that high conflict detected on an incongruent trial leads to recruitment of greater cognitive resources than on congruent trials; the cognitive resources are then utilized on the subsequent trial to enhance performance (Botvinick et al., 2001; Egner, 2007; Gratton et al., 1992; Kerns, 2006; Kerns et al., 2004; Ullsperger, Blysma, & Botvinick, 2005). In consequence, RTs on iI trials are faster than ci trials because the preceding incongruent trial results in increased signaling for cognitive control, while when the preceding trial is congruent fewer cognitive resources are allocated for use on the following trial. Response times for iC trials tend to be longer than those for cC trials due to switching between congruencies and because conflict-driven control reduces the facilitating effect of consecutive repetition of congruent trials.
review). These adjustments in RT due to differing levels of conflict are frequently referred to as conflict adaptation effects and have been demonstrated in several studies using conflict-laden tasks (e.g., the Stroop, Simon, and Eriksen Flanker Tasks; Akcay & Hazeltine, 2008; Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Di Pellegrino, Caramelli, & Ladavas, 2007; Egner & Hirsch, 2005; Fischer, Dreisbach, & Goschke, 2008; Gratton et al., 1992; Kerns, 2006; Kerns et al., 2004; Notebaert, Gevers, Verbruggen, & Liefooghe, 2006; Notebaert & Verguts, 2006; Scharf, Worden, Davidson, Seiger, & Casey, 2006; Ullsperger et al., 2005; West & Moore, 2005; Wuhr, 2005; Verbruggen, Notebaert, Liefooghe, & Vandierendonck, 2006).

The neural mechanisms of conflict adaptation effects are primarily understood in the context of the conflict-monitoring hypothesis (Botvinick et al., 2001; Botvinick, Cohen, & Carter, 2004; Carter et al., 1998). This theory indicates neural generators in the anterior cingulate cortex (ACC) monitor on-line performance to detect conflict (including performance errors) and signal for increased implementation of top-down control mediated by the dorsolateral prefrontal cortex (dLPC) to appropriately adjust or bias performance (Botvinick et al., 2001). In this way, cognitive control is a dynamic process implemented in a distributed network in the brain that involves closely interacting, but dissociable components (see MacDonald, Cohen, Stenger, & Carter, 2000). Indeed, recent work supporting this explanation shows increased coupling between the ACC and dLPC on incongruent trials (Hanslmayr et al., 2008).

Event-related potential (ERP) indices allow for examination of the neural time course of conflict monitoring processes with millisecond resolution. Cognitive tasks that require detection of processing conflicts between competing response options (e.g., incongruent condition of the Stroop task) reliably evoke a late frontal-central phasic ERP signature referred to as the N450 (Liotti, Woldorff, Perez, & Mayberg, 2000; Perlstein, Larson, Dotson, & Kelly, 2006; West, 2003; West & Alain, 1999, 2000; West, Jakubek, Wymbs, Perry, & Moore, 2005). The N450 is a negative-going deflection in the ERP waveform peaking approximately 450 ms following the presentation of a stimulus with high levels of response conflict (Grapperon, Vidal, & Leni, 1988; Liotti et al., 2000; West & Alain, 1999). N450 is present following both stimulus and response conflict (van Veen & Carter, 2002; West, Bowry, & McConville, 2004) and shows greater amplitude when the degree of stimulus prepotency is increased by utilizing relatively more congruent than incongruent trials (e.g., 70% congruent vs. 30% incongruent; West & Alain, 2000). Functional magnetic resonance imaging studies (fMRI) (e.g., MacDonald et al., 2000) and ERP source localization efforts implicate the ACC as the neural generator of the N450 component (Liotti et al., 2000; West, 2003).

Another component of the ERP frequently associated with conflict monitoring, novelty detection, and sequential mapping processes is the N2 (Folstein & Van Petten, 2008). The anterior N2 (or N2c) is a negative fronto-central deflection that emerges around 250 ms post-stimulus and shows greater amplitudes for incongruent than congruent trials during interference tasks (Gehring, Gratton, Coles, & Donchin, 1992; Kopp, Mattler, Goertz, & Rist, 1996). Anterior N2 responses have been associated with conflict monitoring, such that the N2 has been proposed to reflect pre-response conflict during correct trials (Yeung, Botvinick, & Cohen, 2004). Similar to the N450, source localization analysis and hemodynamic studies have identified the ACC as the neural generator of conflict-related anterior N2 processing (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; van Veen & Carter, 2002). However, anterior N2 sensitivity to conflict appears to be paradigm-specific (Folstein, Van Petten, & Rose, 2008; West et al., 2005) and appears to be exclusively elicited by response conflict (van Veen & Carter, 2002). In contrast, the N450 is associated with both response and non-response conflict, and is seen primarily during Stroop-related conflict (West et al., 2004, 2005). Due to findings that the N450 reflects a broader form of conflict monitoring (involving both perceptual and response conflict) and is more sensitive to Stroop-related interference (West et al., 2005), the current study’s hypotheses focus on the N450, not N2, component of the ERP.

Researchers have also observed a more tonic, sustained, conflict-sensitive slow potential (conflict SP) that is present following the more phasic conflict-detection N450 (Liotti et al., 2000; West, 2003; West & Alain, 2000). The conflict SP appears as a sustained parietal positivity/lateral frontal negativity beginning approximately 500 ms after stimulus onset that is more positive following correct incongruent trials than congruent trials or errors—perhaps indicating a role in conflict resolution (Liotti et al., 2000; Perlstein et al., 2006; West, 2003; West & Alain, 2000). However, a recent study indicates the conflict SP is correlated with overall RT and accuracy suggesting a role in response selection rather than specifically to conflict processing (West et al., 2005). The neural generator of the conflict SP remains somewhat uncertain, though a source localization study suggests neural contributions from areas of the middle and inferior frontal gyri, as well as the extrastriate cortex (West, 2003).

Few studies have examined the neural time course of conflict adaptation effects in humans with the millisecond resolution afforded by ERP methods. Sturmer, Leuthold, Soetens, Schroter, and Sommer (2002) and Sturmer and Leuthold (2003) used a Simon task and ERPs to show modulations of the lateralized readiness potential (LRP) as a function of previous-trial accuracy and correspondence with the presented stimulus. Scharf et al. (2006) found an enhanced visual P1 component on iI trials relative to all other conditions, leading to the suggestion that conflict adaptation effects are influenced by early feature-based processing. This study did not examine the later N450 and conflict SP components. West and Moore (2005) report data on RT and accuracy conflict adaptation effects in younger and older adults as well as ERPs for congruent and incongruent current trials irrespective of previous-trial congruency. Findings from this study indicate significant RT conflict-sensitive effects for both younger and older adults coincident with a parietal negative slow wave that differentiated incongruent from congruent color-naming trials. These authors, however, did not report a direct examination of ERP concomitants of these conflict adaptation adjustments.

1. Alternatives to conflict adaptation

Several alternatives to the conflict adaptation explanation of repetition effects should also be considered and addressed. Mayr, Awh, and Laurey (2003) suggest that the repetition of the exact stimulus or stimulus attributes (e.g., the color of the word in the Stroop color-priming condition) accounts for the conflict adaptation effect following iI trials relative to cI trials. That is, the conflict adaptation effect may be accounted for by bottom-up associative priming (i.e., feature repetition) mechanisms rather than top-down adjustments in cognitive control processes. To test this hypothesis, Mayr et al. (2003) conducted two separate studies and found that conflict adaptation effects were present only when there was an exact stimulus repetition, rather than following incongruent trial repetitions.

Other studies explain conflict adaptation effects as a form of feature integration (Hommel, Proctor, & Vu, 2004; Notebaert, Soetens, & Melis, 2001; Wendt, Kluwe, & Peters, 2006). Briefly, feature integration refers to the spontaneous integration of stimulus and response features when they co-occur in time (Hommel et al., 2004). There are three major types of stimulus-response pairings...
associated with feature integration: (1) complete repetition (the exact replication of a stimulus); (2) complete alternations (in a Stroop task, neither the color nor the word of the previous-trial stimulus matches the current trial stimulus); and (3) partial repetitions (where one feature, either color or word, repeat, but not both). For RTs, research indicates both complete repetitions and complete alternations are faster than partial repetitions (Hommel, 1998; Notebaert et al., 2001).

Conflict adaptation effects may also be affected through negative priming. Negative priming occurs when the response-related stimulus (i.e., color-naming) corresponds with the irrelevant or inhibited portion of the stimulus (i.e., word-reading) on the previous-trial (Hanslmayr et al., 2008). Such negative priming effects tend to increase Stroop-related interference (i.e., increase RTs on negatively primed incongruent trials; see Hanslmayr et al., 2008) and may serve to increase, rather than decrease, RTs on ill trials that are negatively primed.

An additional potential confound of examining the neural reflections of conflict adaptation effects is the inclusion of error and post-error trials (Egner & Hirsch, 2005). Error trials are frequently associated with faster RTs (see Riddervold, 2002), while post-error trials are associated with reliable RT slowing (Rabbitt, 1966, 1968). Thus, we chose to exclude error and post-error trials from analyses to separate conflict adaptation processes from those associated with error processing.

2. Current study

Given the paucity of information on the neural time course of behavioral adjustments in cognitive control and the relative ambiguity of the role of the conflict SP in conflict processing/response selection, the current study was designed to: (1) replicate previous findings of conflict adaptation effects on RTs (i.e., an interaction between previous and current trial congruency) and determine the role of associative priming in these effects; (2) examine the role of previous-trial context in modulating the N450 and conflict SP components of the ERP; and (3) directly compare conflict adaptation effects as a function of ERP component (N450 and conflict SP).

3. Methods

3.1. Participants

Participants were recruited from undergraduate psychology courses as well as via flyer and advertisement from the local community. Study enrollment included 36 right-handed individuals (20 female) with a mean age of 24.3 years (S.D. = 7.9, range 18–49 years). All participants were screened for potential psychiatric disorders using the Mental Health Screening Form-III (Carroll & McGinley, 2000, 2001). Pre-screening also excluded participants if they endorsed a history of learning disability, attention-deficit hyperactivity disorder, alcohol or substance abuse, acquired brain disorders (e.g., traumatic brain injury, epilepsy, stroke), or color-blindness as measured by the Ishihara pseudo-isochromatic color plates (Clark, 1924).

3.2. Experimental task

Participants performed a modified color-naming version of the single-trial Stroop task. In this task, participants are presented with one of three words (RED, GREEN, BLUE) printed in one of the same three colors at a visual angle of 3.65°. Congruent trials comprised words presented in their same color of ink; incongruent trials comprised color-words printed in a different color of ink (e.g., the word BLUE printed in blue ink); incongruent trials comprised color-words printed in a different color of ink (e.g., the word BLUE printed in red ink). Participants were instructed to respond as quickly and accurately as possible to the color of the word (while ignoring the word itself) with a button press to one of three color-coded response keys using the index, middle, and ring fingers of their right hand. Color-to-key mapping was practiced prior to task performance using 40 presentations of each color-key combination. Stroop trials were 3 s in duration and consisted of a Stroop color-word presented for 1.5 s followed by a 1.5 s fixation cross to allow electrophysiological activity to return to baseline. Six blocks of 100 trials, for a total of 600 trials and approximately 30 min in EEG testing were presented. To increase the potency of the conflict stimulus, 70% of trials were congruent (approximately 420 trials) and 30% were incongruent (approximately 180 trials).

3.3. Electrophysiological data recording

Electroencephalogram (EEG) was recorded from 46 scalp sites using a geodesic sensor net and Electrical Geodesics, Inc. (EGI, Eugene, Oregon) amplifier system (20K gain, nominal bandwidth = 10–100 Hz). Electrode placements enabled recording vertical and horizontal eye movements reflecting electro-oculographic (EOG) activity. EEG was referenced to Cz and digitized continuously at 250 Hz with a 16-bit analog-to-digital converter. A right posterior electrode approximately two inches behind the right mastoid served as ground common. Electrode impedance was maintained below 50 kΩ. Eye movement and blink artifacts were corrected using a spatial filtering method (Berg & Scherg, 1994; Ille, Berg, & Scherg, 1997, 2002) implemented in Brain Electric Source Analysis (BESA) software (Scherg, 1990). Continuous EEG was segmented into condition-related epochs, and single-trial epochs with voltages that exceeded ±150 μV or transitions (sample-to-sample) thresholds of 100 μV were discarded. Single-trial EEG was then digitally re-referenced to an average reference (Bertrand, Perrin, & Perrier, 1985). Prior to analyses, EEG was digitally low-pass filtered at 15 Hz.

3.4. Event-related potential reduction and measurement

Individual-subject stimulus-locked averages were derived for correct trials only with post-error trials excluded for each congruency (congruent, incongruent) and sequential trial repetition possibility (CC, CI, IC, and II). Epochs spanned 100 ms prior to and 900 ms following stimulus presentation. ERPs were baseline corrected using the 100 ms pre-stimulus window. Analyses of electrophysiological data focused on selected electrode sites based on previous findings indicating that the ERP modulations of interest are relatively focal over fronto-medial (N450; Liotti et al., 2000; West & Alain, 1999, 2000) and posterior parietal sites (conflict SP; Liotti et al., 2000), as well as the scalp-distribution maps of the present data which indicated maximal N450 and conflict SP amplitudes over these regions. The phasic fronto-central N450 was quantified as the voltage at the most negative peak between 350 and 500 ms at sites 4 (FCz), 65 (Cz), 5, and 55 (see Perielsen et al., 2006 for figure of the 64-channel geodesic sensor net), while the more tonic conflict SP was quantified as the mean voltage from 650 to 850 ms at electrode sites 34 (Pz), 38, 33 and 41. Measured voltages and latencies for both components of interest were averaged across sites prior to analyses. Latency measurements for the N450 component were indexed as the time of the peak negative-going amplitude within the same 350–500 ms window. No latency measurements are provided for the conflict SP, as it is a slow, tonic component.

3.5. Data analysis

Median correct-trial RTs (Ratcliff, 1993), arc sine transformed error rates (Neter, Wasserman, & Kutner, 1985) excluding non-response trials, and ERP component amplitude and latency data were analyzed using separate repeated-measures analyses of variance (ANOVA). Arc sine-corrected error rates were used to normalize the data due to a positive skew frequently associated with error rate data (Neter, Wasserman, & Kutner, 1985). To isolate conflict adaptation effects, RT and ERP analyses excluded error trials and trials immediately following errors. The Huynh-Feldt epsilon adjustment was applied for ANOVAs with error-rate data (Neter et al., 1985). To isolate conflict adaptation effects, z-score normalized single-trial EEG was then digitally low-pass filtered at 15 Hz.
context strongly influences responses to current-trial context. Error rates indicated a significant main effect of current trial congruency, but the main effect of previous-trial congruency was not significant, indicating conflict adaptation effects were not reliably present for error rates. The main effect of previous-trial congruency, however, was not significant, F(1,35) = .52, p > .05. Most importantly, a significant Previous Trial × Current Trial interaction indicated a reliable conflict adaptation effect, F(1,35) = 11.17, p < .002, $\eta^2 = .24$, wherein RTs were slowest for incongruent trials preceded by congruent trials (cI), followed in order of RT by iI, iC, then cC trials. This pattern of RT effects was supported by a highly significant linear trend over condition, F(1,35) = 132.22, p < .001, $\eta^2 = .79$. The main effect of previous-trial congruency was not significant, F(1,35) = 1.00, p > .32, $\eta^2 = .03$, indicating conflict adaptation effects were not reliably present for error rates.

4. Results

4.1. Behavioral performance

Data for RTs and error rates as a function of previous-trial congruency and current-trial congruency are presented in Table 1. Analyses of correct-trial RTs, excluding post-error trials, revealed the expected main effect of current-trial congruency reflecting Stroop RT interference (i.e., longer RTs to incongruent than congruent current trials), F(1,35) = 132.22, p < .001, $\eta^2 = .79$. The main effect of previous-trial congruency was not significant, F(1,35) = 5.2, p > .79, $\eta^2 = .02$. Most importantly, a significant Previous Trial × Current Trial interaction indicated a reliable conflict adaptation effect, F(1,35) = 11.17, p < .002, $\eta^2 = .24$, wherein RTs were slowest for incongruent trials preceded by congruent trials (cI), followed in order of RT by iI, iC, then cC trials. This pattern of RT effects was supported by a highly significant linear trend over condition, F(1,35) = 139.95, p < .001, and clearly indicates that preceding context strongly influences responses to current-trial context.

The Previous Trial × Current Trial ANOVA on arcsine-corrected error rates indicated a significant main effect of current trial congruency, F(1,35) = 33.37, p < .001, $\eta^2 = .49$, reflecting Stroop error-rate interference. The main effect of previous-trial congruency, however, was not significant, F(1,35) = 7.73, p > .23, $\eta^2 = .04$, nor was the Previous Trial × Current Trial interaction, F(1,35) = 1.00, p > .32, $\eta^2 = .03$, indicating conflict adaptation effects were not reliably present for error rates.

4.2. Potential impact of associative and negative priming

As noted above, recent findings challenge the validity of sequential trial effects, suggesting instead that such effects may be influenced by associative or negative priming (Mayr et al., 2003; Hanslmayr et al., 2008; Hommel et al., 2004). Given this, we re-analyzed RT and error-rate data in two additional ways: (1) excluding trials that repeated the same stimulus color; (2) excluding ‘negative priming’ trials wherein the color of the incongruent current-trial stimulus corresponded with the word of the preceding incongruent trial stimulus.

The pattern of results for the Previous Trial × Current Trial ANOVAs on RTs and error rates excluding color repetitions was consistent with analyses presented above (see Table 1). For RTs, the main effect of current trial congruency remained significant, F(1,35) = 85.26, p < .001, $\eta^2 = .71$, as did the Previous Trial × Current Trial interaction, F(1,35) = 56.49, p < .001, $\eta^2 = .62$, and the linear trend over condition, F(1,35) = 105.16, p < .001. For arcsine-corrected error rates, the main effect of current trial congruency remained significant, F(1,35) = 14.31, p < .002, $\eta^2 = .29$, the Previous Trial × Current Trial interaction remained non-significant, F(1,35) = 1.85, p > .18, $\eta^2 = .05$.

Similarly, the Previous Trial × Current Trial ANOVAs on RTs and error rates excluding negative priming trials remained significant. For RTs, there remained a main effect of current trial congruency, F(1,35) = 131.72, p < .001, $\eta^2 = .79$, a Previous Trial × Current Trial interaction, F(1,35) = 11.57, p < .002, $\eta^2 = .25$, and a robust linear trend over condition, F(1,35) = 154.51, p < .001. Again, for arcsine-corrected error rates, the main effect of congruency remained, F(1,35) = 31.39, p < .001, $\eta^2 = .47$; however, the Previous Trial × Current Trial interaction was significant at only a trend level, F(1,35) = 4.05, p < .06, $\eta^2 = .10$, indicating that error-related conflict adaptation effects were present to a greater degree when negative priming trials were removed relative to unadjusted trials or trials adjusted for associative priming.

Taken together, conflict adaptation effects persisted even after accounting for the potential contributions of associative and negative priming. Since the pattern of behavioral results did not meaningfully change when trials that would lead to associative or negative priming were removed, ERP analyses were conducted on all trials to maximize signal-to-noise ratio.

4.3. Event-related potential data

Current trial congruent waveforms contained an average (±S.D.) of 349.8 ± 32.9 trials (range: 283–403), while incongruent current trial waveforms contained an average of 145.9 ± 16.3 trials (range: 115–173). When broken down to examine potential conflict adaptation effects, the number of trials retained was 236.8 ± 24.0 (range: 182–281) for cC trials, 101.6 ± 12.2 (range: 79–123) for ci trials, 100.4 ± 11.3 (range: 64–120) for iC trials, and 40.3 ± 5.7 (range: 28–60) for iI trials. Stimulus-locked ERP waveforms reflecting the medio-frontal N450 and parietal conflict SP as a function of previous and current trial congruency are shown in Figs. 1 and 2, respectively.

Mean ERP amplitude data for the N450 and conflict SP, as well as latency data for the N450, are presented in Table 2.
For the N450, examination of fronto-central stimulus-locked ERP waveforms indicated a negative-going deflection that is more negative to incongruent than congruent trials—replicating previous findings of a conflict-sensitive N450 (e.g., Perlstein et al., 2006; West, 2003; West & Alain, 2000; Fig. 1a). Confirming this observation, a Previous Trial × Current Trial ANOVA on N450 amplitude yielded only a significant main effect of current trial congruency, F(1,35) = 14.32, p < .001, \( \eta^2 = .25 \). Analyses of orthogonal trend over condition analyses yielded significant linear, F(1,35) = 22.22, p < .001, and quadratic, F(1,35) = 14.29, p < .001, trends over condition.

A Previous Trial × Current Trial ANOVA on N450 peak latencies indicated latencies were similar for both congruencies, as reflected by a non-significant main effect of current trial congruency, F(1,35) = 3.90, p > .06, \( \eta^2 = .11 \). Neither the main effect of previous trial, F(1,35) = .67, p > .42, \( \eta^2 = .02 \), nor the Previous Trial × Current Trial interaction were statistically significant, F(1,35) = .63, p > .44, \( \eta^2 = .02 \).

For conflict SP amplitude, a Previous Trial × Current Trial ANOVA revealed a significant main effect of current trial congruency (Fig. 2a), F(1,35) = 15.94, p < .001, \( \eta^2 = .31 \), replicating previous findings of congruency-related parietal electrophysiological activity (e.g., Liotti et al., 2000; West & Alain, 2000; West & Moore, 2005). More importantly, a significant Previous Trial × Current Trial interaction indicated a conflict adaptation effect that differed as a function of previous-trial congruency (Fig. 2b), F(1,35) = 11.77, p < .002, \( \eta^2 = .25 \). Analyses of orthogonal trend over condition analyses yielded only a significant linear trend over condition, F(1,35) = 22.31, p < .001. As seen in Fig. 2a and Table 2, data show a nearly monotonic increase in conflict SP amplitude as a function of previous-trial congruency (cC < iC < iI < cI), consistent with the hypothesis that the conflict SP amplitude evoked by the degree of current-trial conflict is modulated by the degree of previous-trial conflict.

### Table 2

<table>
<thead>
<tr>
<th>Condition</th>
<th>Amplitude (( \mu V ))</th>
<th>N450 Latency (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N450</td>
<td>Conflict SP</td>
</tr>
<tr>
<td>Current-trial congruent</td>
<td>−.06 (2.2)</td>
<td>−.12 (1.6)</td>
</tr>
<tr>
<td>Current-trial incongruent</td>
<td>−.52 (2.2)</td>
<td>−.78 (1.6)</td>
</tr>
<tr>
<td>iC</td>
<td>−.06 (2.2)</td>
<td>−.19 (1.7)</td>
</tr>
<tr>
<td>iI</td>
<td>−.36 (2.1)</td>
<td>.07 (1.5)</td>
</tr>
<tr>
<td>cI</td>
<td>−.61 (2.4)</td>
<td>.52 (1.7)</td>
</tr>
<tr>
<td>cl</td>
<td>−.53 (2.2)</td>
<td>.94 (1.7)</td>
</tr>
</tbody>
</table>

For the conflict SP amplitude, a Previous Trial × Current Trial ANOVA revealed a significant main effect of current trial congruency (Fig. 2a), F(1,35) = 15.94, p < .001, \( \eta^2 = .31 \), replicating previous findings of congruency-related parietal electrophysiological activity (e.g., Liotti et al., 2000; West & Alain, 2000; West & Moore, 2005). More importantly, a significant Previous Trial × Current Trial interaction indicated a conflict adaptation effect that differed as a function of previous-trial congruency (Fig. 2b), F(1,35) = 11.77, p < .002, \( \eta^2 = .25 \). Analyses of orthogonal trend over condition analyses yielded only a significant linear trend over condition, F(1,35) = 22.31, p < .001. As seen in Fig. 2a and Table 2, data show a nearly monotonic increase in conflict SP amplitude as a function of previous-trial congruency (cC < iC < iI < cI), consistent with the hypothesis that the conflict SP amplitude evoked by the degree of current-trial conflict is modulated by the degree of previous-trial conflict.

4.4. Comparison of conflict adaptation for the N450 and conflict SP

Results of the 2-Component × 4-Condition ANOVA on z-score normalized ERP data yielded a significant main effect of condition, F(3,105) = 14.33, p < .001, and a significant Component × Condition interaction, F(3,105) = 2.96, p < .05. Analyses of orthogonal trend over condition yielded a significant linear trend over condition, F(1,35) = 39.80, p < .001, and a significant interaction of Component × Quadratic Trend over condition, F(1,35) = 8.11, p < .01. Follow-up trend tests on each ERP component separately, conducted to maximize power, revealed a significant linear trend over condition for the conflict SP, F(1,35) = 28.51, p < .001; N450 showed both linear, F(1,35) = 10.01, p < .005, and quadratic trends, F(1,35) = 5.35, p < .03, over condition. These patterns are evident in Fig. 3.

4.5. Correlational analyses

Pearson’s correlation analyses (two-tailed) were conducted between the incongruent minus congruent difference waves for both the N450 and conflict SP components and incongruent minus congruent trial RTs. Incongruent minus congruent RT differences significantly correlated with difference wave amplitudes for the N450, r(35) = −.51, p < .01, but not conflict SP components of the ERP, r(35) = .05, p > .77. To examine conflict adaptation effects, we initially conducted correlations between RTs and ERP difference wave amplitudes for cl minus iI trials. Response-time differences did not significantly relate to either N450, r(35) = −.26, p > .13, or conflict SP amplitude differences, r(35) = .01, p > .95. Additional correlations examining the relationship between cl minus iI ERP difference waves for both the N450 and conflict SP and overall RT-related conflict adaptation effects (iI
minus IC under minus IC minus [cl minus cC]) were also not significant, rs < .16, ps > .35.

Overall, correlations are consistent with the idea that the N450 is associated with conflict monitoring processes, with increased incongruent minus congruent N450 differences associated with higher levels of Stroop-related interference. Conflict SP amplitudes were not related to RT differences. Further, ERP amplitudes were not significantly related to RT manifestations of conflict adaptation.

5. Discussion

In this study we examined the behavioral and electrophysiological correlates of conflict processing and conflict adaptation effects. Behavioral data revealed the anticipated increases in RTs and error rates on incongruent relative to congruent trials (i.e., Stroop interference). Replicating several previous studies using the Stroop task (Egner & Hirsch, 2005; Kerns et al., 2004; Notebaert et al., 2006), a conflict adaptation effect was observed for RT data that remained when color and negative priming repetitions were removed. Error rates were generally not modulated by the congruency of the previous trial—likely due to the low number of errors committed on the task (i.e., ceiling effect).

Event-related potentials were used to examine the time course of neural activity reflecting putative conflict monitoring (N450) and conflict processing (conflict SP) mechanisms (Liotti et al., 2000; Perlstein et al., 2006; West, 2003, 2004; West et al., 2005). Consistent with predictions, a negative deflection in the ERP occurring between approximately 420 and 440 ms (N450) differentiated congruent and incongruent trials (West & Alain, 2000); however, N450 amplitude was not differentially affected by previous-trial context. That is, the N450 did not exhibit a significant conflict adaptation effect.

The absence of differences in N450 amplitude as a function of previous-trial congruency suggests it may reflect more automatic conflict monitoring mechanisms uninfluenced by the implementation of controlled processes following previous trials of different congruencies (i.e., previous-trial context). That is, the N450 may reflect underlying neural processes that are consistent (i.e., more automatic) regardless of the amount of top-down control needed or implemented during a particular trial, while the conflict SP reflects more controlled processes that adapt to the level of control necessary to accurately complete a trial. This finding was somewhat surprising given fMRI evidence of decreased ACC activity on iI relative to cl trials (Kerns et al., 2004) and prior evidence suggesting the N450 is mediated by the ACC (Liotti et al., 2000; West, 2003). While unexpected, the absence of a conflict adaptation effect on N450 could alternatively be due to: (1) the large variability in the measured N450 in the current study; (2) the presence of multiple simultaneously active sources contributing to volume conduction and a consequent washing out of an N450 adaptation effect if present; (3) an absence of conflict adaptation effects on the N450; or (4) increasing ACC activity beginning early after presentation of the conflict stimulus (i.e., during the N450 time window) and escalating on conflict trials during the time window of the conflict SP (see Hanslmayr et al., 2008). Additional research is necessary to adjudicate among these possibilities.

Following the N450, a distinct slow potential, the conflict SP, also differentiated congruent and incongruent trials, but was modulated by previous-trial context. The conflict SP is generally thought to reflect controlled processing, perhaps devoted to the resolution of conflict or signaling for increased implementation of attentional control (Perlstein et al., 2006; West, 2003; West & Alain, 2000). If, as suggested by cognitive control theory, conflict detection signals for the recruitment of controlled cognitive resources toward adaptive resolution of conflict (Botvinick et al., 1999; Kerns et al., 2004; Miller & Cohen, 2001), the conflict SP should monotonically differ as a function of degree of conflict to be resolved. Consistent with this possibility, parietal conflict SP amplitude was greatest on cl trials where the greatest amount of putative conflict is present and monotonically decreased with lesser degrees of conflict (i.e., cl > iI > iC > cC trials). These findings converge with recent research indicating a role of the parietal cortex in the detection (Liston, Matalon, Hare, Davidson, & Casey, 2006) and resolution (Egner, Delano, & Hirsch, 2007) of stimulus-based conflict.

Direct comparison of normalized conflict SP and N450 amplitudes showed the conflict SP was differentially influenced by previous-trial context, while the N450 was influenced to a lesser extent and in a different pattern. Findings indicate that the conflict SP and N450 are distinct components of the ERP reflecting different underlying neural processes. As noted above, this may be due to the N450 being a reflection of more automatic conflict detection processes, while the conflict SP may be the reflection of more controlled conflict resolution or signaling processes that are more strongly influenced by the degree of control resources previously recruited.

Previous studies indicate N450 amplitude covaries with the magnitude of Stroop-related RT interference (West & Alain, 2000). Results from the current study support this finding by showing increased N450 incongruent minus congruent differences associated with increased Stroop interference. Conflict SP amplitude, however, did not significantly relate to RT difference scores. Relationships between electrophysiological (i.e., ERP) and behavioral (i.e., RT) manifestations of conflict adaptation were also nonsignificant. The latter findings suggest the conflict adaptation effects seen in the conflict SP component of the ERP are not directly predictive of behavioral performance. This could be due to several factors, including the possibility that behavioral data reflect intermediate processes not necessarily reflected in short-latency ERP components such as the N450 and conflict SP (see Cacioppo & Tassinary, 1990).

Findings of the current study should be considered within the context of potential limitations. First, while we were able to examine sequential trial conflict adaptation effects, we were unable to examine post-error slowing due to the probability distribution of congruent and incongruent trials (i.e., the preponderance of participant commission errors occurred in the incongruent condition and were followed by congruent trials due to the 70% congruent/30% incongruent proportion of trials employed; thus, post-error slowing was not evaluated as participants traditionally respond faster to congruent than incongruent trials). Second, the current data did not allow for direct examination of feature integration effects because we were unable to remove partial repetitions from il trials (i.e., with only three color-words there are no possible complete alternations on il trials; thus, we could not remove partial repetitions and still have il trials to examine). However, after removing all color repetition trials, remaining cc trials are complete alternations, remaining cl and IC trials consist of some complete alternations and some partial repetitions, while the remaining il trials are all partial repetitions. According to the feature integration model, having all partial repetition trials should slow down RTs on il trials compared to cl trials. Data indicate il trials are considerably faster than cl trials, a finding that can be accounted for by conflict adaptation but not by feature integration effects. Third, due to the complications associated with examining individual-trial ERPs (i.e., low signal-to-noise ratio in the absence of signal averaging), ERP data examining conflict adaptation effects were reduced by binning individual trials into epochs based on previous and current trial congruency. Epochs were then averaged for each participant and statistical values obtained on these averages. Thus, we were unable to examine potentially meaningful associations between individual-trial ERP components. For example, Kerns et al. (2004) in their fMRI exami-
nation of conflict adaptation effects, used ACC activity on previous trials to predict dPFC activity levels on subsequent trials and vice-versa. Our inability to examine single-trial data reliably due to the limitations inherent in the ERP methodology prevented such examination between, for example, individual-trial N450 and conflict SP amplitudes across sequential trials. Lastly, the time window for the conflict SP overlaps with the participant RTs. It is possible, therefore, that response-related processes could be an alternative explanation for the conflict SP findings. Alternatively, since the conflict SP occurs at, and following, response selection, the conflict SP may represent post-response conflict or resolution, rather than response selection or resolution of stimulus-based conflict.

6. Summary and conclusions

Present findings are consistent with previous studies indicating robust behavioral conflict adaptation effects that cannot be accounted for solely by associative or negative priming. The neural time course of conflict adaptation effects, as measured by ERPs, revealed a parietal conflict SP that monotonically differentiated Stroop stimuli on the basis of previous-trial context and a fronto-medial N450 that was sensitive to current-trial congruency but was not differentiated by previous-trial context. Future research is necessary to determine the precise role of the conflict SP and N450 components in conflict detection/processing and the relationship between these ERP components and behavior.

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