

# Temporal dynamics of reactive cognitive control as revealed by event-related brain potentials

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## Abstract

Reactive cognitive control refers to a complementary set of cognitive operations by which individuals monitor for and detect the presence of goal-interfering conflict (i.e., conflict monitoring/evaluation) and, subsequently, initiate attention-focusing and response selection processes to bolster goal-directed action in the face of such conflict (regulative control). The purpose of the current study was to characterize the nature of conflict adaptation in both components of this dynamic process across sequences of trials and, more broadly, across time as participants complete a cognitive control task. Fifty-two young adults completed a standard arrow flanker task while behavioral and ERP data were recorded. Multilevel modeling of sequences of compatible and incompatible trials over time showed that, whereas response time data demonstrated a typical conflict adaptation effect throughout the task, N2 and frontal slow wave (FSW) indices of conflict monitoring and regulative control, respectively, demonstrated significant conflict adaptation only during the early part of the task. Moreover, although differential change in N2 and FSW over time suggested that conflict monitoring and regulative control were dissociable, a reciprocal relation between them was maintained throughout the task and was not present in a component theoretically unrelated to conflict adaptation (visual attention-related N1). Findings are discussed in terms of compensatory processes that help to maintain goal-directed performance even as control-related neural responses become fatigued.

## KEYWORDS

cognitive control, conflict adaptation, conflict monitoring, ERPs, multilevel modeling

## 1 | INTRODUCTION

Humans' ability to flexibly adjust attention and responses according to current goals, particularly overcoming prepotent or habitual responses, reflects a property of the information-processing system known as *cognitive control* (Alexander & Brown, 2010; Miller & Cohen, 2001; Shenhav, Cohen, & Botvinick, 2016). In particular, when a given context contains stimuli that have the potential to interfere with goal-directed action, cognitive control is needed to perform a number of complementary functions, including maintaining current goals in working memory, focusing attention on goal-relevant stimulus features so that goal-irrelevant features can be ignored, and biasing motor responding in favor

of goal-appropriate responses (e.g., Botvinick, & Cohen, 2014; Braver, 2012; Jacoby, Jennings, & Hay, 1996; Meyer & Kieras, 1997).

The operation of these processes in the laboratory is commonly examined using speeded response time (RT) tasks in which visual targets are presented amid goal-challenging distracters. For example, in the flanker task (Eriksen & Eriksen, 1974), participants must identify a target flanked on both sides by distracters associated either with the same response as the target (i.e., compatible or congruent trials) or an opposing response (i.e., incompatible or incongruent trials). The commonly observed compatibility effect (or congruency effect) reflects participants' superior performance on compatible relative to incompatible trials, and is taken as

evidence that response activation is influenced by flanker stimuli (see Coles, Gratton, Bashore, Eriksen, & Donchin, 1985; Coles, Gratton, & Donchin, 1988; Gratton, Coles, Sirvaag, Eriksen, & Donchin, 1988). That is, incompatible flankers elicit response conflict.

The malleability of cognitive control is evident in the fact that the compatibility effect varies as a function of context. Gratton, Coles, and Donchin (1992) first reported that the compatibility effect is smaller when the previous flanker trial is incompatible versus compatible, a finding that since has been replicated many times (for reviews, see Clayson & Larson, 2013; Verguts & Notebaert, 2008). The dominant explanation for this phenomenon is that the experience of conflict—particularly when conflict is aversive (see Desender, Van Opstal, & Van den Bussche, 2014; Driesbach & Fischer, 2015; Inzlicht, Bartholow, & Hirsch, 2015; Steenbergen, Band, & Hommel, 2009)—elicits adjustments in cognitive control so that subsequent conflict can be better managed—a so-called *conflict adaptation effect*.<sup>1</sup>

This explanation accords with dominant theories emphasizing the dynamic interplay of conflict monitoring, attention control, and response implementation. In particular, the conflict monitoring theory (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Botvinick & Cohen, 2014; Botvinick, Cohen, & Carter, 2004; Yeung, 2013) posits complementary processes responsible for adjusting control when performance is threatened by interference. Specifically, the theory posits an evaluative component that monitors performance for instances of conflict; when conflict is detected, a second, regulative component is responsible for increasing control to ensure that intended responses are implemented. Evidence from ERP and fMRI studies has associated the evaluative, cognitive control-monitoring process with activity in the medial prefrontal cortex (mPFC), particularly dorsal anterior cingulate cortex (dACC; Carter & van Veen, 2007; Sheth et al., 2012; Van Veen & Carter, 2002; Yeung, Botvinick, & Cohen, 2004), whereas the regulative control process has been associated with activity in dorsolateral areas of PFC (dlPFC) responsible for goal representation and response selection (Kerns et al., 2004; MacDonald, Cohen, Stenger, & Carter, 2000; West, Bailey, Tiernan, Boonsuk, & Gilbert, 2012). According to the dual mechanisms of control model (DMC; Braver, 2012), the interplay of these two components describes a reactive control process, whereby control fluctuates moment to moment in reaction to the presence of conflict.

Several studies have produced evidence consistent with the basic premise that the detection of conflict by dACC is

associated with increased activation dlPFC (i.e., regulative control) shortly thereafter (e.g., Durston et al., 2003; Kerns, 2006; Kerns et al., 2004). Other work has shown that, whereas dACC is sensitive to the occurrence of conflict, dlPFC responds to information signaling upcoming conflict (MacDonald et al., 2000). Critically, however, no research to date has tested a reciprocal premise of the conflict monitoring theory that increased regulative control should reduce the extent to which evaluative conflict monitoring is engaged thereafter. To the extent that the experience of conflict instigates greater regulative control (Kerns et al., 2004), and following the logic outlined in the DMC that greater engagement of dlPFC should downregulate mPFC reactivity to conflict “in the moment,” it stands to reason that increased cognitive control regulative control in response to an instance of conflict should predict reduced conflict monitoring-related neural responses thereafter. Indirect evidence supporting this idea has come from studies demonstrating that mPFC responses on incompatible trials are reduced following incompatible relative to compatible trials (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Clayson & Larson, 2011a, 2012; Forster, Carter, Cohen, & Cho, 2010). But the specific premise that neural responses associated with regulative control predict reduced neural conflict-monitoring reactivity has never been tested. One goal of the current research was to test this basic premise, using ERP measures that previous research has associated with conflict monitoring and regulative aspects of control, thereby filling a critical gap in the literature pertaining to the dynamic nature of control variability associated with the reactive control process (Braver, 2012).

More generally, the current research aimed to model the extent to which behavioral and neural indices of conflict adaptation vary across time as participants engage in a typical laboratory control task. Just as control-related processes are hypothesized to fluctuate on a moment-to-moment basis (Botvinick et al., 2001; Braver, 2012), there are a number of reasons to hypothesize that the extent of this fluctuation also should vary over longer time periods. In particular, several theories predict (e.g., Botvinick, & Cohen, 2014; Robinson, Schmeichel, & Inzlicht, 2010; Shenhav et al., 2016) and empirical evidence suggests (Fischer, Dreisbach, & Goschke, 2008) that at least some aspects of control are resource intensive and therefore susceptible to fatigue. For example, research has shown that both time on task (e.g., Barwick, Arnett, & Slobounov, 2012; Boksem, Meijman, & Lorist, 2006; Warm, Matthews, & Finomore, 2008) and previous mental effort (Hagger, Wood, Stiff, & Chatzisarantis, 2010) result in fatigue and impaired performance on laboratory control tasks. Another possibility suggested by recent theories is that flagging motivation leads people to disengage from tasks that require control, which similarly can result in

<sup>1</sup>See online supporting information for a brief discussion of alternative explanations of this effect, known more generally as the congruency sequence effect, along with analyses meant to address some of those alternative explanations.

performance decrements (Braver, Krug, Chiew, Kool, & Westbrook, 2014; Francis & Inzlicht, 2016; Kool, McGuire, Rosen, & Botvinick, 2010; Kouneiher, Charron, & Koechlin, 2009).

To the extent that such effects reflect changes in activation of neural circuits associated with control, measures of such activation should show reductions as time on task increases. Some previous research provides support for this idea. For example, Boksem et al. (2006) found that ERP measures of conflict monitoring decreased from the first 20 min to the final 20 min of a very long (2 hr) cognitive control task, and that the amplitude reduction was evident only for high-conflict trials (also see Lorist, Boksem, & Ridderinkhof, 2005). To date, however, few studies have investigated whether behavioral and neural indices of conflict adaptation vary over the course of a cognitive control task (see Lorist & Jolij, 2012), nor, more specifically, have previous investigations tested the extent to which neural measures reflecting evaluative/conflict monitoring versus regulative aspects of reactive cognitive control vary differentially over the course of task engagement. In other words, the dynamic interplay between these two, theoretically distinct aspects of conflict adaptation hypothesized in dominant neurocognitive theories of control (Botvinick & Cohen, 2014; Braver, 2012) has yet to be fully characterized.

## 1.1 | Current study

The current study had three primary aims: (a) to characterize the nature of conflict adaptation effects in both evaluative/conflict monitoring and regulative aspects of reactive cognitive control; (b) to model the nature of change in these processes over the course of a standard cognitive control task; and (c) to examine the extent to which conflict adaptation-related increases in regulative control predict subsequent change in evaluative, conflict-monitoring processes and behavioral performance, and whether these relationships change over the course of the task. The amplitude of the frontocentral N2, a transient negativity emerging 200–350 ms after stimulus onset and known to be generated in mPFC (van Veen & Carter, 2002), served as a neural index of conflict monitoring. Numerous studies have shown that N2 amplitude is larger on incompatible than compatible trials in cognitive control tasks (see Folstein & Van Petten, 2008; Larson, Clayson, & Clawson, 2014; van Veen & Carter, 2002), suggesting that it reflects the evaluative/conflict monitoring function of control (Yeung et al., 2004). Moreover, neuroimaging and ERP data consistently show that indices of mPFC activation, including the N2, show conflict adaptation effects, being smaller for high-conflict stimuli when preceded by other high-conflict stimuli relative to low-conflict stimuli (e.g., Botvinick et al., 1999; Clayson & Larson,

2011a, 2012; Forster et al., 2010; Larson, Kaufman, & Perlstein, 2009; Sheth et al., 2012).

Another stimulus-locked ERP component sensitive to the presence of conflict is the frontal slow wave (FSW). The FSW is a low-frequency, negative-going voltage deflection arising late in stimulus-locked ERP epochs (generally 600–1,000 ms) over frontal and central scalp locations (West & Alain, 1999, 2000; West & Bailey, 2012) that is larger following high-conflict relative to low-conflict trials in cognitive control tasks (Bailey, West, & Anderson, 2010; Bartholow, Dickter, & Sestir, 2006; Curtin & Fairchild, 2003; West et al., 2012), including the flanker (Bailey, Bartholow, Saults, & Lust, 2014). The FSW is thought to reflect adjustment-related activity in the lateral PFC (West & Bailey, 2012; West et al., 2012), in that FSW amplitude is correlated with behavioral indices of control adjustment (Bailey et al., 2014; Bartholow et al., 2006) and varies along with the amount of conflict in a block of trials (West & Bailey, 2012). Given these findings, coupled with other evidence demonstrating a role for dlPFC in regulative control (De Pisapia & Braver, 2006.; Kerns, 2006; Kerns et al., 2004; MacDonald et al., 2000; Marklund & Persson, 2012), the amplitude of the FSW was used to represent the regulative component of reactive control.<sup>2</sup>

To examine change over time in control-related responses, data from this study were analyzed using mixed-effects multilevel modeling (MLM). Recent reports have emphasized the advantages of MLM over traditional repeated measures analysis of variance (rmANOVA) for psychophysiological data (Kristjansson, Kircher, & Webb, 2007; Tibon & Levy, 2015; Tremblay & Newman, 2015; Vossen, Van Breukelen, Hermens, Van Os, & Lousberg, 2011). Of greatest relevance for the current research, MLM allows the modeling of data from individual trials over time (Tibon & Levy, 2015; Vossen et al., 2011). Virtually all theoretical models of cognitive control posit dynamic temporal relationships between multiple control-related processes—conflict adaptation is an example—but traditional approaches to neurophysiological signal quantification (e.g., signal averaging across all trials within conditions) and analysis (rmANOVA) are not well suited to modeling such dynamics, for several reasons. For example, the typical signal averaging technique assumes that each time-locked ERP consists of a signal (i.e., the neurocognitive process of interest) and random noise, and that the signal remains constant across all trials. This latter assumption is not valid in situations where signal change

<sup>2</sup>Note that the conflict-related slow potential (i.e., conflict SP), frequently observed in ERP studies of the Stroop task (see Larson et al., 2009), also has been posited to reflect conflict adjustment or resolution (i.e., regulative control; see Larson et al., 2014). However, the conflict SP typically is not observed in flanker tasks. Future research should endeavor to determine the extent to which the FSW and conflict SP are related.

is anticipated (e.g., from fatigue or habituation; see Boksem et al., 2006; Kato, Endo, & Kizuka, 2009; Woestenburg, Verbaten, Van Hees, & Slangen, 1983). Moreover, signal averaging necessarily ignores potentially interesting sources of variation related to any such changes, particularly in situations where the signal might vary over time in some conditions more than others.

MLM can handle large numbers of repeated measurements per person, which means data from individual trials can be modeled using trial as a continuous predictor. This approach is arguably preferable to other techniques, such as binning of trials into sequential blocks, which still results in considerable loss of within-person variability (see Vossen et al., 2011). Modeling of individual trials is facilitated by the fact that MLM can account for multiple sources of variability, including that attributable to individual subjects, stimuli, electrodes, and so on, and can accommodate specification of error terms at each level of nesting (e.g., electrodes within subjects). Additionally, missing data need not result in listwise deletion of cases or imputation of missing values, as MLM is robust to imbalanced data structures across individual cases.

The ability to model data from individual trials allowed us to examine a number of issues related to this study's aims. First, we examined whether conflict adaptation effects in neural measures of conflict monitoring and regulative control decrease with time on task. To the extent that conflict adaptation requires cognitive control resources that are susceptible to depletion over time due to fatigue or other influences (e.g., Boksem et al., 2006), it could be expected that conflict adaptation effects will be more evident earlier compared to later in the course of a task. This is consistent with the suggestion that N2-related conflict adaptation effects have been more apparent in studies with fewer trials (see West & Bailey, 2012).

Second, although a number of studies have shown support for the conflict monitoring theory's prediction that mPFC responses to conflict positively predict subsequent dlPFC activation (e.g., Durston et al., 2003; Kerns, 2006; Kerns et al., 2004), no existing studies have examined the complementary prediction that dlPFC activation in response to conflict should have a negative (i.e., downregulating) effect on mPFC responses. Based on predictions derived from conflict monitoring and DMC theories (Botvinick et al., 2001; Braver, 2012), we hypothesized that FSW amplitude elicited on a given trial would be inversely associated with N2 amplitude elicited on the next trial. We similarly predicted that the extent of regulative control elicited by a given stimulus would predict performance on the subsequent trial, such that previous-trial FSW amplitude would be positively associated with current-trial RT (i.e., larger FSW would predict faster RT).

## 2 | METHOD

### 2.1 | Participants

Individuals contributing data for the current report were among 142 young adults (ages 21–32 years) who participated in a study investigating the acute effects of alcohol and caffeine on cognitive control and its neural correlates (see Bailey et al., 2016). The current report includes data from participants who were randomly assigned to the control condition (consumed neither alcohol nor caffeine;  $n = 27$ ) or a placebo condition (consumed a beverage they were told contained alcohol and caffeine but actually contained neither;  $n = 25$ ) of that larger study. Participants were recruited from the Columbia, MO, community using email circulars, advertisements, and posted flyers. Eligible individuals completed lab sessions for which they were paid \$12/hour.<sup>3</sup>

### 2.2 | Materials

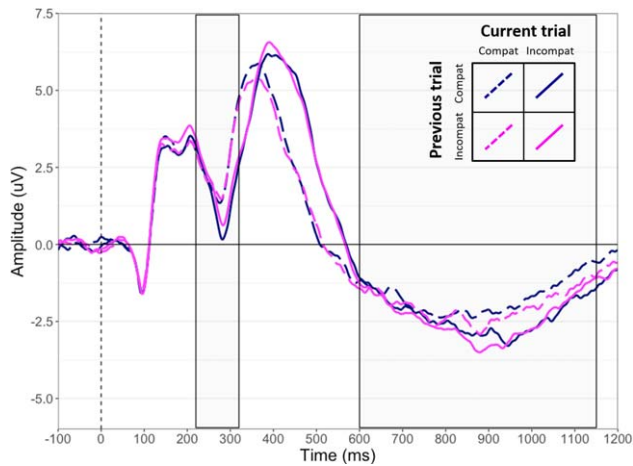
#### 2.2.1 | Flanker task

On each of 800 experimental trials (eight blocks of 100 trials), participants viewed arrow arrays and were to identify the direction of the central arrow (right or left) via button press. On compatible trials, the flanker (peripheral) and target arrows indicated the same response (i.e.,  $\rightarrow\rightarrow\rightarrow\rightarrow$  or  $\leftarrow\leftarrow\leftarrow\leftarrow$ ); on incompatible trials, the flankers and target indicated opposing responses (i.e.,  $\rightarrow\rightarrow\leftarrow\rightarrow$  or  $\leftarrow\leftarrow\rightarrow\leftarrow$ ). Compatible and incompatible arrays were presented pseudorandomly, with the constraints that they occurred with equal probability and left- and right-hand responses occurred equally often. Arrays were presented for 100 ms with an unlimited response window followed by a randomly varying intertrial interval (1,100 or 1,500 ms). Participants completed 100 practice trials prior to the experimental trials.

#### 2.2.2 | Electrophysiological recording

The EEG was sampled at 1000 Hz (filtered online at 0.05–40 Hz) with 32 tin electrodes placed in standard locations (American Encephalographic Society, 1994). Scalp electrodes were referenced online to the right mastoid; an average mastoid reference was derived offline. Impedance was kept below 5 k $\Omega$ . Blinks were removed from the EEG offline using a regression-based procedure. EEG data were segmented into epochs of –100 to 1,200 ms of poststimulus activity and scanned for artifacts (trials containing voltage deflections of  $\pm 75 \mu\text{V}$  [ $<3\%$  of all trials] were discarded). Consistent with previous studies (see Folstein & Van Petten,

<sup>3</sup>Eligibility criteria are detailed in Bailey et al. (2016).



**FIGURE 1** Grand-averaged ERP waveforms averaged across nine electrodes (F3, Fz, F4, FC3, FCz, FC4, C3, Cz, C4) plotted as a function of current and previous trial type (compatible, incompatible). The dotted vertical line indicates stimulus onset, and the boxes indicate the time windows during which the mean amplitude of the N2 and FSW were computed. Compat = compatible trials; incompat = incompatible trials

2008), visual inspection of the grand-averaged waveforms (see Figure 1) indicated that the N2 emerged ~220–320 ms poststimulus and was most prominent at frontal and central scalp sites (electrodes F3, Fz, F4, FC3, FCz, FC4, C3, Cz, C4). Thus, the N2 was measured as the average amplitude 220–320 ms poststimulus at those nine electrodes. The grand averages also show a more sustained negativity emerging around 600 ms poststimulus that is also most prominent at frontal and frontocentral scalp sites, consistent with the FSW observed in previous cognitive control tasks (e.g., Bailey et al., 2014; Bartholow et al., 2006; Curtin & Fairchild, 2003; West & Alain, 1999). Therefore, the FSW was quantified as the average amplitude 600–1,150 ms poststimulus at the same nine-electrode array used to quantify the N2.

Additionally, we quantified the posterior N1, an early-latency component typically elicited in response to visual stimulation. The N1 is moderated by selective attention and thus has been most commonly enlisted to examine attentional processing (Mangun & Hillyard, 1991). As the N1 is theoretically not related to cognitive control, demonstrating that FSW does not significantly predict N1 on the subsequent trial would confirm the discriminant validity of the relationship between the FSW and N2 by implying that any such relationship is not the product of a general similarity in the EEG across trials. The N1 was quantified as the average amplitude 125–220 ms poststimulus at electrodes P3, PZ, P4, O2, and O1.

### 2.3 | Procedure

Upon arrival, participants provided informed consent and completed affidavits assessing compliance with pre-session

protocols (e.g., abstention from alcohol and other drugs for the previous 24 hr). Participants then were escorted to an EEG acquisition suite where experimenters placed and tested the electrodes. Next, participants completed the practice trials followed by a break, during which they consumed a tonic water beverage (according to the goals of the larger study). They then completed the experimental trials of the flanker task, after which they were debriefed and dismissed.

### 2.4 | Data analysis

Data from two participants were completely discarded due to experimenter errors during data collection ( $n = 1$ ) and failure to demonstrate adequate performance in the flanker task ( $< 50\%$  accuracy;  $n = 1$ ). Equipment malfunctions resulted in the loss of 13 additional subjects' EEG data. One additional subject's EEG data were discarded due to a large number of artifacts (only 23.3% usable trials). This resulted in a final sample of 50 individuals for RT analyses and 36 individuals for separate analyses of N2 and FSW amplitude. ERP data were unable to be merged with the behavioral data for three additional subjects, resulting in a sample of 33 individuals for analyses examining relationships between variables. Although this small sample size can adversely affect tests of group-level variances within a multilevel model, regression coefficients and their standard errors are estimated without bias, even with a sample size of 30 (Maas & Hox, 2005).

The R package lme4 (Bates et al., 2015) was used to fit multilevel models for data analysis. To determine the most appropriate random effects structure, we used the model-specification procedures described by Matuscheck, Kliegl, Vasishth, Baayen, and Bates (2017), starting with a maximal model and then removing random slopes based on the magnitude of the correlations between random effects (.80 was used as the cutoff threshold). Satterthwaite approximations were used to estimate degrees of freedom and to obtain two-tailed  $p$  values; in situations where degrees of freedom were  $> 200$ , we report the results as  $z$  statistics. Data and code used for analysis can be found at <https://github.com/hiv8r3/ConfDyn>.

All models utilized single trial-level data rather than individual participant means. Separate MLMs were constructed to predict each outcome measure of interest (RT, N2, FSW) using previous trial type (compatible, incompatible), current trial type (compatible, incompatible), and trial as predictors. The intercept and all slopes were allowed to vary by subject and, where appropriate (N2 and FSW models), the intercept was allowed to vary by electrode nested with subject. For all models, individual trials were included only if correct responses were made on both the trial in question (current trial) and the trial preceding it (previous trial). Additionally, to reduce skew and eliminate fast “guessing” responses, trials

with RTs < 100 ms and > 2,000 ms were excluded from the analyses.

Models examining associations among the outcomes were constructed using the disaggregation procedure described by Curran and Bauer (2011), which permits separation of variability attributable to differences between persons from the variability attributable to processes of interest that differ within persons. The amplitude of the predictor variable on each trial was centered on each participant's mean. To simplify these models, amplitude was averaged over the nine electrodes at which it was measured (i.e., so that only a single mean was used for each subject). The mean of the predictor variable was also added as a Level 2 predictor in each model. This resulted in two predictors, one representing the between-persons variation in the predictor and the other the within-person variation in the predictor. The within-person estimate is more appropriate for examining cognitive control dynamics, since within-person relationships reflect differences across occasions (i.e., trials) rather than differences across individuals. Disaggregating these two sources of variability allows for a clearer inference that changes in the activation of one control-related process are associated with changes in another control-related process within or across trials (Fleeson, 2007). In each model, the intercept was allowed to vary by subject. To test whether the relationship between predictor and criterion variables changed over the course of the task, trial and its interaction with the within-subject effect were added to each of the disaggregation models.

### 3 | RESULTS

Fixed effects derived from models in which RT, N2, and FSW were predicted from current trial type, previous trial

type, trial, and all interactions are given in Table 1. In these models, trial was rescaled and centered (see online supporting information for more details on the logic behind the rescaling procedure). As a result, the effects of current trial type, previous trial type, and their interaction are estimated at the midpoint of the experiment in the main models. Significant Current  $\times$  Previous  $\times$  Trial interactions were probed with additional models in which the trial variable was centered around the first trial (representing trials toward the beginning of the experiment) and the last trial (representing trials toward the end of the experiment), respectively, following procedures outlined by Aiken and West (1991). The average number of trials included in the ERP analyses for each condition for each subject is given in Table 2. MLM is robust to unbalanced data, through a procedure called partial pooling (Gelman & Hill, 2007; Tibon & Levy, 2015), and therefore differing numbers of trials per condition are less problematic than in traditional rmANOVA.

#### 3.1 | RT

As indicated in Table 1, the model predicting RT showed a significant main effect of current trial type, such that participants were faster to correctly identify targets on compatible trials ( $M = 382$  ms;  $SD = 104$  ms) than on incompatible trials ( $M = 430$  ms;  $SD = 106$  ms). This main effect was qualified by a small but significant Current  $\times$  Previous trial type interaction. The means presented in Figure 2b–d show a pattern generally consistent with conflict adaptation: the compatibility effect was smaller following incompatible versus compatible trials. Because trial is centered and included in the model, these effects are interpreted at the midpoint of the task.

**TABLE 1** Fixed effects estimated by multilevel models examining each dependent measure separately

	RT		N2		FSW	
	<i>b</i>	<i>p</i>	<i>b</i>	<i>p</i>	<i>b</i>	<i>p</i>
Current	29.06 (1.29)	<b>.000</b>	-.39 (.08)	<b>.000</b>	-.25 (.08)	<b>.004</b>
Previous	.53 (.48)	.271	.07 (.05)	.163	-.07 (.06)	.288
Current $\times$ Previous	-1.33 (.57)	<b>.023</b>	.08 (.06)	.218	.04 (.06)	.559
Trial	-1.13 (.20)	<b>.000</b>	-.05 (.01)	<b>.000</b>	.33 (.01)	<b>.000</b>
Trial $\times$ Current	-.62 (.20)	<b>.001</b>	-.01 (.01)	.464	.05 (.01)	<b>.000</b>
Trial $\times$ Previous	-.06 (.20)	.745	-.05 (.01)	<b>.000</b>	-.04 (.01)	<b>.000</b>
Trial $\times$ Current $\times$ Previous	.03 (.20)	.867	.02 (.01)	<b>.024</b>	-.04 (.01)	<b>.000</b>

*Note.* Unstandardized betas are presented. Bold values indicate *p* values below .05. Standard errors of estimates are in parentheses. Satterthwaite approximations were used to estimate degrees of freedom to calculate *p* values. For analyses, the trial variable was rescaled and centered to range from -4 to 4. Changing the range of the trial variable in this way increases the betas associated with the effect of trial and its interactions tenfold (to aid in their interpretation), but does not influence significance testing. In the model presented in this table, the effects of current trial, previous trial, and their interaction are interpreted at the midpoint of the task. Categorical variables were effect coded.

**TABLE 2** Average number of trials included in ERP analyses as a function of condition

Previous trial	Current trial	
	Compatible	Incompatible
<b>N2</b>		
Compatible	172.5	158.0
Incompatible	160.6	144.5
<b>FSW</b>		
Compatible	167.8	154.2
Incompatible	156.5	141.4
<b>Disaggregate analyses</b>		
Compatible	140.2	140.8
Incompatible	142.4	129.9

The RT model also showed a main effect of trial (Table 1), indicating that RTs decreased over the course of the task. However, this effect was qualified by a significant Trial  $\times$  Current trial interaction. Examination of the simple slopes (Figure 2a, Table 3) revealed that, whereas RT for incompatible trials decreased over the course of the task, RT for compatible trials did not change. Neither the Trial  $\times$  Previous trial type interaction nor the three-way Trial  $\times$  Previous trial type  $\times$  Current trial type interaction were significant. That is, the magnitude and form of the conflict adaptation effect in RT did not vary over the course of the task.<sup>4</sup>

### 3.2 | N2 Amplitude

The model predicting N2 amplitude showed a significant main effect of current trial type (Table 1). As is evident in Figure 1, incompatible trials ( $M = 1.85$   $\mu$ V;  $SD = 9.79$   $\mu$ V) elicited larger (more negative) N2s than compatible trials ( $M = 2.65$   $\mu$ V;  $SD = 9.79$   $\mu$ V). The pattern of means in the N2 is suggestive of a conflict adaptation effect (Figure 1), in that incompatible trial N2s appeared larger when the previous trial was compatible versus incompatible, consistent with previous studies (e.g., Clayson & Larson, 2011a; Forster et al., 2010). But neither the previous trial type main effect nor the Current  $\times$  Previous trial type interaction were significant at the midpoint of the task.

<sup>4</sup>Some previous research has indicated that the conflict adaptation effect (or congruency sequence effect) is eliminated when trial sequences with repeated features are eliminated (see Mayr, Awh, & Laurey, 2003). Thus, we examined the effects of both target repetitions (e.g., <<><< followed by >>>>) and flanker repetitions (e.g., >>>> followed by ><>>) in our data; these analyses are presented in the supporting information. Summarizing, there was no evidence supporting the response priming view of the congruency sequence effect, and mixed support for both conflict-adaptation and feature integration accounts.

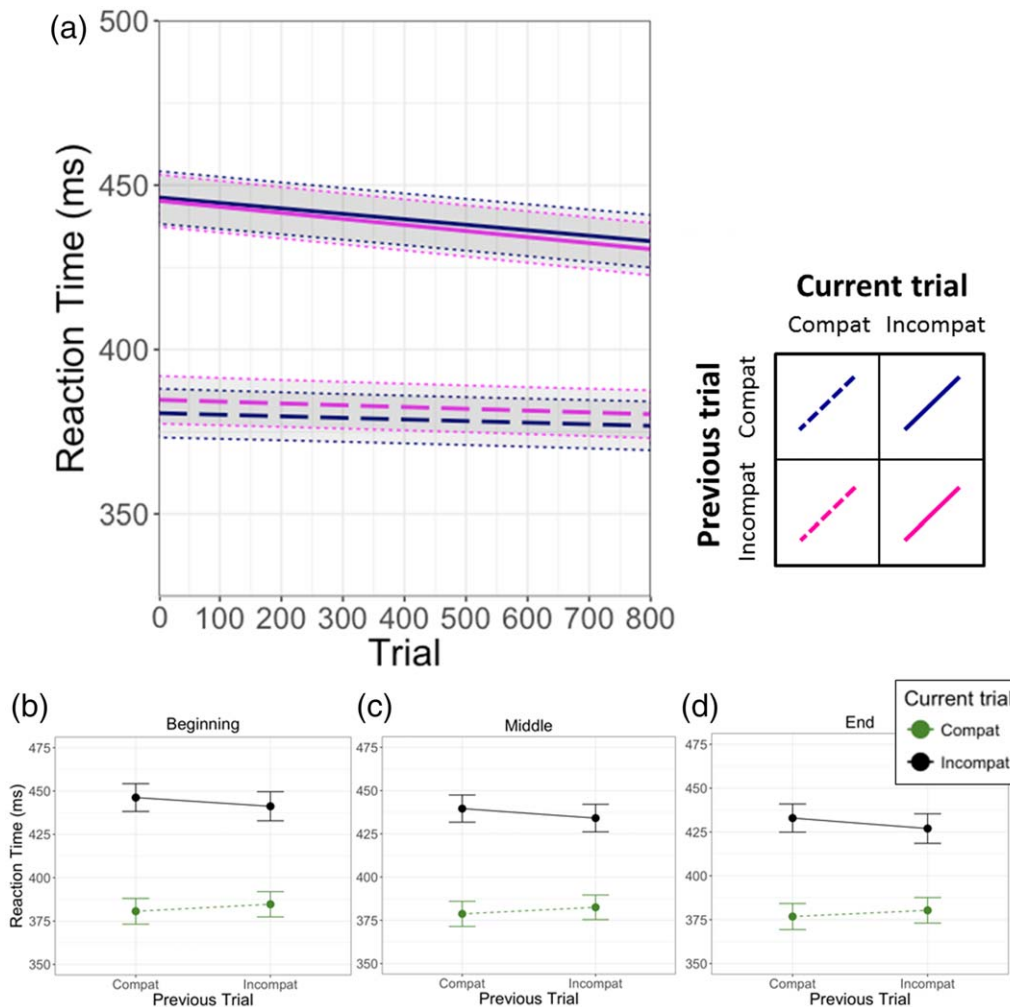
However, the presence of a significant Previous trial type  $\times$  Current trial type  $\times$  Trial interaction suggests a more nuanced picture (see Table 1). This significant interaction was decomposed by fitting additional models with the trial variable centered around the beginning and the end of the task. These additional models revealed a significant conflict adaptation effect (i.e., a significant Current  $\times$  Previous interaction) during the first portion of the trials,  $b = .158$ ,  $t(60.3) = 2.24$ ,  $p = .029$ , but not during the middle,  $b = .08$ ,  $t(34.4) = 1.25$ ,  $p = .218$ , or latter portion of the trials,  $b = -0.004$ ,  $t(63.7) = -0.06$ ,  $p = .95$  (see Figure 3b–d). In other words, conflict adaptation was more evident during approximately the first half of the trials and decreased over time.

Another way of interpreting these relationships is in terms of the difference in slopes representing the change in N2 amplitude over time in the four conditions of interest. Examination of the slopes and 95% confidence intervals in Table 3 indicates that, whereas N2 amplitude elicited on trials following compatible trials remained relatively stable over the course of the task, the N2 elicited on trials following incompatible trials tended to get larger as the task progressed.

### 3.3 | FSW amplitude

A significant main effect of current trial type indicated that incompatible trials elicited larger (more negative) FSW amplitude ( $M = -2.31$   $\mu$ V;  $SD = 10.7$   $\mu$ V) than compatible trials ( $M = -1.82$   $\mu$ V;  $SD = 10.6$   $\mu$ V). Neither the main effect of previous trial type nor the interaction between current and previous trial type were significant at the midpoint of the task. As with the N2, however, the Previous trial type  $\times$  Current trial type  $\times$  Trial interaction was significant (see Table 1). Further exploration of the Previous  $\times$  Current trial interaction at the beginning and end of the task using additional models indicated a significant conflict adaptation effect during the first portion of the trials,  $b = .18$ ,  $t(68.2) = 2.49$ ,  $p = .015$ , but not during the middle,  $b = .04$ ,  $t(33.9) = 0.59$ ,  $p = .56$ , or latter portion of the trials,  $b = -.11$ ,  $t(73.0) = -1.47$ ,  $p = .14$  (see Figure 4b–d).

Another way of interpreting the significant Previous  $\times$  Current  $\times$  Trial interaction is in terms of the difference in slopes representing the change in FSW amplitude over time in the four conditions of interest. As evident by the slope values given in Table 3 (and depicted in Figure 4), FSW amplitude decreased (i.e., became more positive) relatively dramatically over the course of the task in all conditions, but more so in the previous compatible, current incompatible condition than in any of the others. Inspection of the 95% confidence intervals in each condition shows that, whereas the slopes in each of the other conditions did not differ



**FIGURE 2** (a) Change in reaction times as a function of condition over the course of the experiment. Solid and dashed lines represent the simple slopes for the four sequential trial combinations of interest, estimated from the model corresponding to Table 1. (b) (c) (d) Estimated (model-derived) means and standard errors for each condition at the beginning, middle, and end of the task, each estimated using a separate model with the trial variable centered on the first trial, middle trial, and last trial, respectively

significantly (i.e., confidence intervals overlapped), the slope in the previous compatible, current incompatible condition differed from each of the others.

### 3.4 | Associations among control-related processes and performance

#### 3.4.1 | Conflict monitoring predicting regulative control

Conflict monitoring theory predicts that detection of conflict increases the implementation of regulative control (see Kerns et al., 2004). Although not directly related to the aims of the current study, we tested the magnitude of this association in the current data by predicting FSW amplitude from N2 amplitude elicited within the same trial. Consistent with theory, the within-subject effect of N2 was strongly associated with the FSW within the same trial,  $b = .59$ ,  $z = 89.4$ ,  $p < .001$ .

Moreover, when trial was added as a predictor, this association was not moderated by trial,  $b = -.002$  ( $z < 1$ ,  $p = .538$ ), indicating that the size of this association remained relatively constant throughout the task.

#### 3.4.2 | Regulative control predicting conflict monitoring

The prediction that the extent of regulative control (FSW) elicited on one trial would downregulate conflict monitoring (N2) on the next trial was tested using a model in which previous-trial FSW amplitude was used to predict current-trial N2 amplitude. The model results indicated a significant between-subject effect,  $b = -0.11$ ,  $z = -2.0$ ,  $p = .043$ , suggesting that individuals with larger previous-trial FSW also tend to experience smaller current-trial N2 reactivity. More importantly, a significant within-subject effect,  $b = -0.05$ ,  $z = -6.7$ ,  $p < .001$ , indicated that a larger FSW on one trial



**TABLE 3** Simple slopes describing the change in each dependent measure over the course of the experiment

	Current trial	
	Compatible	Incompatible
<b>RT</b>		
Compatible	-0.48 [-1.23, 0.26]	<b>-1.66</b> [-2.44, -0.88]
Incompatible	-0.55 [-1.32, 0.23]	<b>-1.85</b> [-2.66, -1.03]
<b>N2</b>		
Compatible	-0.03 [-0.06, 0.01]	0.03 [-0.01, 0.06]
Incompatible	<b>-0.09</b> [-0.13, -0.06]	<b>-0.12</b> [-0.16, -0.08]
<b>FSW</b>		
Compatible	<b>0.28</b> [0.24, 0.32]	<b>0.45</b> [0.41, 0.49]
Incompatible	<b>0.27</b> [0.23, 0.31]	<b>0.30</b> [0.26, 0.34]

Note. The 95% confidence interval for each estimate is presented in brackets; intervals that do not cross zero are considered statistically significant (estimates in boldface).

predicted a smaller N2 on the following trial regardless of the magnitude of an individual's FSW relative to others in the sample. When trial was added as a predictor, there was no interaction between the within-subject effect and trial,  $b = 0.00$ ,  $z = .37$ ,  $p = .711$ , suggesting that the sequential relationship between the FSW and N2 did not change over the course of the task.

We also examined whether this relationship varied according to trial type by estimating an additional model with previous trial FSW amplitude, previous trial type, and current trial type predicting current-trial N2 amplitude. This model produced a significant interaction between FSW amplitude, current trial type, and previous trial type,  $b = 0.10$ ,  $z = 3.57$ ,  $p < .001$ . To understand this interaction, we examined the effect of previous-trial FSW on current trial N2 separately for the four trial sequence conditions. There was a significant within-subject effect of FSW in cI (previous compatible, current incompatible) trials,  $b = -0.05$ ,  $z = -4.0$ ,  $p < .001$ , and iC (previous incompatible, current compatible) trials,  $b = -0.08$ ,  $z = -5.9$ ,  $p < .001$ . However, this relationship did not hold for iI trials,  $b = -0.02$ ,  $z = -1.4$ ,  $p = .165$ , or cC trials,  $b = -0.02$ ,  $z = -1.8$ ,  $p = .065$ .

### 3.4.3 | Regulative control predicting RT

Conflict-monitoring theory also predicts that enhanced regulative control should improve performance (e.g., by limiting the influence of subsequent conflict). The disaggregation model examining the relationship between FSW and RT showed a nonsignificant between-subjects effect ( $b = 0.26$ ,  $z = .44$ ,  $p = .663$ ) but a significant within-subject effect ( $b = 0.21$ ,  $z = 3.0$ ,  $p = .002$ ), indicating that larger (more negative) FSW amplitude on one trial predicted better

performance (faster RTs) on the subsequent trial. When trial was added as a predictor, the interaction between the within-subject effect and trial was also nonsignificant,  $b = 0.06$ ,  $z = 1.9$ ,  $p = .056$ , suggesting that the relationship between previous-trial FSW and current-trial RT was maintained throughout the task.

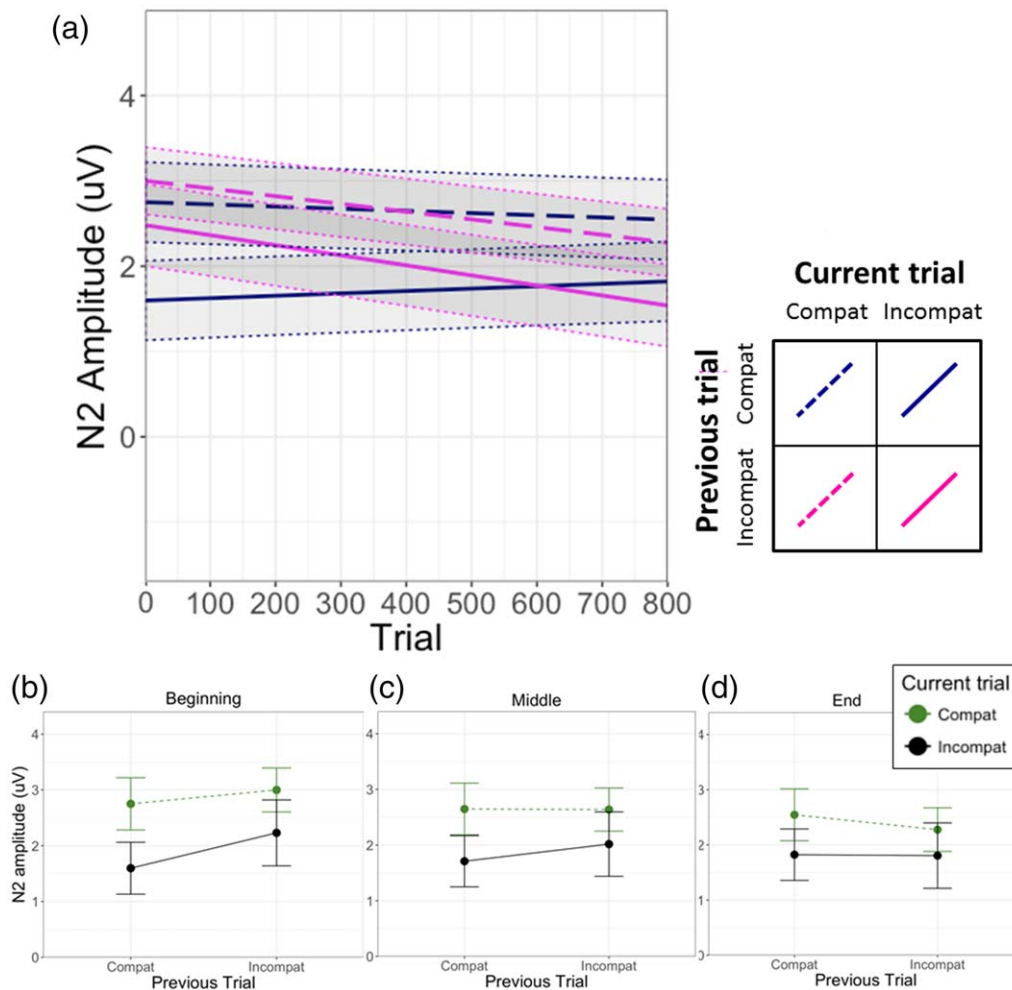
### 3.4.4 | Regulative control predicting visual-attentional processing

To confirm that these relationships are indicative of links between theoretical constructs rather than simply the result of common method variance, we additionally examined the relationship between previous-trial FSW and current-trial N1. The disaggregation model examining the relationship between FSW and N1 on the following trial showed a nonsignificant between-subjects effect ( $b = -0.07$ ,  $z = -1.4$ ,  $p = .149$ ), as well as a nonsignificant within-subject effect ( $b = -.001$ ,  $z = -.19$ ,  $p = .846$ ). Additionally, when trial was added as a predictor, the interaction between the within-subject effect and trial was also nonsignificant,  $b = -.001$ ,  $z = -2.6$ ,  $p = .796$ .

## 4 | DISCUSSION

Dominant theories of cognitive control (Botvinick et al., 2001; Botvinick & Cohen, 2014; Braver, 2012) posit multiple components of control responsible for ensuring acceptable levels of performance when goal-directed action is threatened by distracting or goal-conflicting information. Moreover, these theories posit dynamic, reciprocal associations between the components of control rooted in mPFC (conflict monitoring) and lateral PFC (regulative control) as they react to varying levels of conflict. Although such control dynamics have generated intense interest among researchers (see Clayson & Larson, 2011a,b; Forster et al., 2010; Larson et al., 2009; Verguts & Notebaert, 2008), there is a dearth of evidence concerning how the control components respond to each other and how their sensitivity to changing demands varies over the course of a challenging task.

Several aspects of the current data help to address this gap in understanding. First, although conflict adaptation (Gratton et al., 1992) was present in behavioral performance throughout the task, neurophysiological correlates of conflict monitoring (N2) and regulative control (FSW) showed a more time-limited adaptation effect. Specifically, both the N2 and FSW showed patterns indicative of conflict adaptation during early but not later trials (see Figure 3–4). Whereas previous studies have reported decreased compatibility effects (current trial compatibility) in conflict monitoring-related neural responses from the first to the last

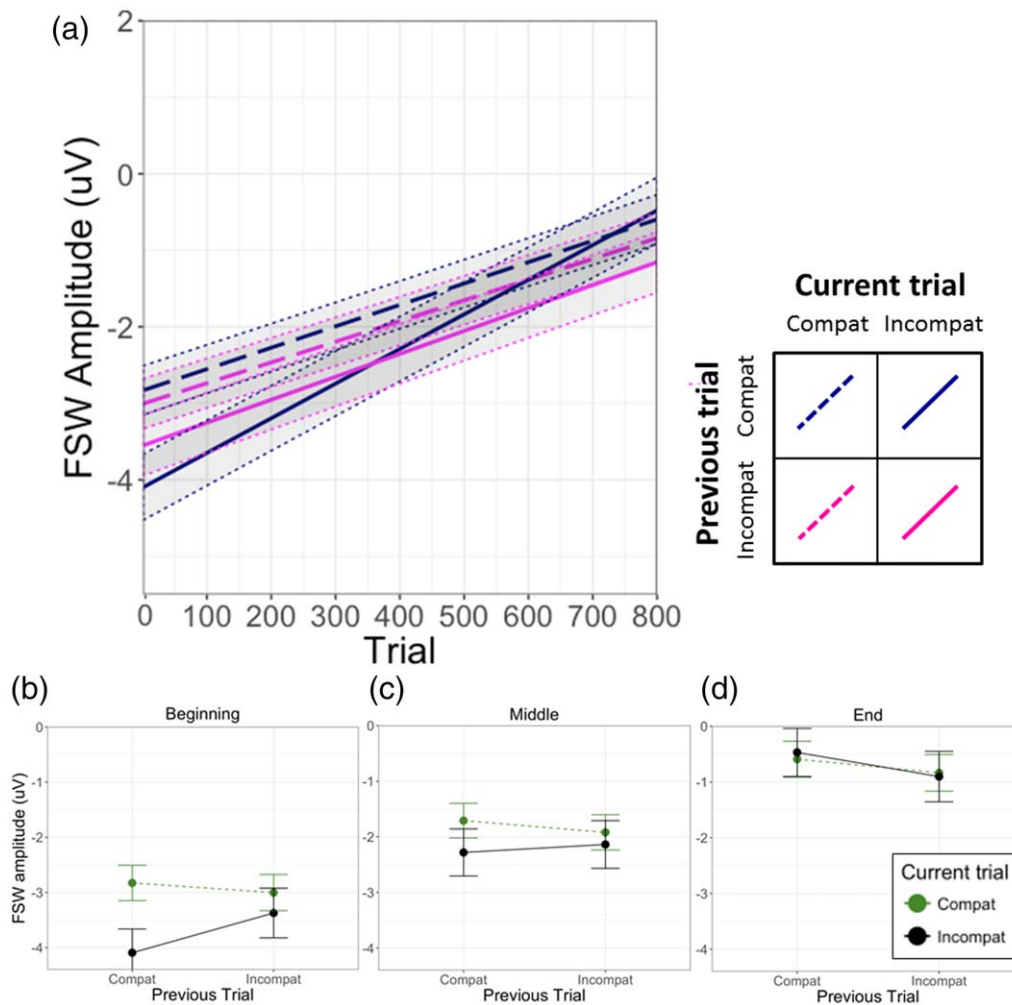


**FIGURE 3** (a) Change in N2 amplitudes as a function of condition over the course of the experiment. Solid and dashed lines represent the simple slopes for the four sequential trial combinations of interest, estimated from the model corresponding to Table 1. (b) (c) (d) Estimated (model-derived) means and standard errors for each condition at the beginning, middle, and end of the task, each estimated using a separate model with the trial variable centered on the first trial, middle trial, and last trial, respectively

block of a lengthy control task (Boksem et al., 2006; Kato et al., 2009), little previous work has examined how the conflict adaptation effect (i.e., Previous  $\times$  Current trial compatibility) in such a measure varies over time (but see Lorist & Jolij, 2012). The fact that the N2 effect diminished as the task progressed is consistent with the observation that conflict adaptation effects in the N2 have been more apparent in studies with fewer trials (see West & Bailey, 2012).

Of greater relevance to testing the full scope of the conflict monitoring theory is whether fluctuation in the instigation of regulative control, which in theory reflects compensatory adjustments in attention and perceptual selection aimed at reducing the influence of subsequent conflict (see Berlyne, 1960; Botvinick et al., 2001), is associated with modulation of subsequent mPFC reactivity. The current results showed mixed evidence in support of this idea, in the form of a negative association between FSW elicited on a given trial and N2 amplitude elicited on the subsequent trial. To our knowledge, this study is the first in either the ERP or

fMRI literatures to demonstrate the posited inverse association between a neural reflection of regulative control elicited on a given trial and a subsequent conflict-monitoring response. The strength of this relationship did not change over time, suggesting that the downregulation of conflict reactivity following a regulative control increase does not become less efficient or decoupled over time, even though the absolute magnitude of regulative control responses might decrease. Importantly, however, this predicted association was evident for only some types of sequences (cI and iC trials) and not for others (iI and cC trials). The strongest form of conflict-monitoring theory would predict that, other factors being equal, the experience of conflict on a given trial would produce a large reactive control response (both conflict-monitoring [N2] and regulative [FSW]) on that trial, followed by a reduced conflict-monitoring response (i.e., N2 amplitude) to conflict experienced on the next trial. The lack of a significant association for iI trials in our data could reflect that regulative control is imperfect and does not



**FIGURE 4** (a) Change in FSW amplitudes as a function of condition over the course of the experiment. Solid and dashed lines represent the simple slopes for the four sequential trial combinations of interest, estimated from the model corresponding to Table 1. (b) (c) (d) Estimated (model-derived) means and standard errors for each condition at the beginning, middle, and end of the task, each estimated using a separate model with the trial variable centered on the first trial, middle trial, and last trial, respectively

consistently downregulate the conflict-monitoring process when two high-conflict stimuli are encountered in a row. Additional research is needed to further test this idea and determine the extent to which the observed association on iC and cI trials in the current data is related to conflict adaptation or some other process.

An additional piece of evidence suggesting that the FSW→N2 association across trials was related in some way to conflict adaptation—and is not merely an artifact of a general similarity in the EEG across trials within the same brain—comes from the analysis showing that FSW amplitude on a given trial was unrelated to the amplitude of the N1 on the next trial. If any observed association across trials was due to psychological processes unrelated to conflict-related processing (e.g., visual attention) or even nonpsychological processes (e.g., volume conduction or signal drift), we would expect the FSW to correlate with an earlier-latency component on subsequent trials. This did not occur, providing some

evidence of discriminant validity for the predicted association between regulative and evaluative reactive control across trials.

Despite the existence of this predicted relationship, the current findings also provide evidence of the dissociation of the conflict-monitoring and regulative components of reactive control. Consideration of the patterns in the trial-level data of responses to incompatible trials is instructive here. The magnitude of responses on incompatible trials following compatible trials (i.e., cI trials) can be said to represent conflict reactivity, whereas responses on incompatible trials following incompatible trials (i.e., iI trials) represent adaptation to conflict. Although the conflict reactivity (cI) response in the N2 was robust and remained stable over the course of the task, the conflict reactivity response in the FSW showed a rather dramatic reduction over time. This dissociation suggests the possibility that regulative control reactivity to conflict is a resource-intensive process subject to fatigue.

Similarly, the conflict adaptation response in the FSW also decreased over the course of the task, but conflict adaptation in the N2 actually increased over time.

These differing patterns further suggest that, although both mPFC (N2) and lateral PFC (FSW) are sensitive to conflict and show a decrease in the conflict adaptation effect (Previous trial  $\times$  Current trial interaction) over time, they might do so for different reasons mainly related to responses on iI trials. Whereas the N2 on cI trials remained stable, the N2 on iI trials increased. In contrast, the dominant change in the regulative control response was that FSW amplitude on both cI and iI trials decreased, but the magnitude of this reduction was much larger for cI trials. Considered together, these patterns suggest a kind of compensatory process whereby control engagement shifts toward a greater reliance on the evaluative/conflict-monitoring component while the influence of the regulative component wanes over time. The DMC (Braver, 2012) similarly posits compensatory changes between reactive and proactive forms of control, particularly as the more resource-intensive proactive form becomes taxed. The current data suggest that this same logic might apply to differing forms of reactive control, and that the regulative process might be more susceptible to depletion than is conflict monitoring.

Such compensation across these two modes of control could explain why, despite the apparent “disappearance” of conflict adaptation in both FSW and N2 as the task progressed, a behavioral conflict adaptation effect remained intact throughout the task. This kind of compensatory hypothesis was suggested by Boksem et al. (2006), who also observed a large reduction in the flanker compatibility effect in the N2 over time—and attributed this effect to fatigue (also see Beste, Kneiphof, & Woitalla, 2015)—but no reduction in the size of the effect in RT. As posited by Boksem and colleagues, “fatigue is more than an effort/reward imbalance and involves adaptive strategies to keep performance at an acceptable level under adverse internal circumstances” (p. 129). In the case of the current study, the adverse internal circumstances would appear to have been the sharp decline in the regulative control response over time, particularly on cI trials, and the adaptive strategy may have involved stronger reliance on conflict monitoring. In their study, Boksem et al. observed an overall RT slowing, which they attributed to participants taking longer to resolve conflict on incompatible trials as the conflict-monitoring response in N2 became exhausted. In contrast to those results, here we observed an overall decrease in RT across the task. It seems plausible that this could have resulted from the fact that the N2 response to conflict did not decrease and, instead, remained stable (cI trials) or increased (iI trials) over time.

Interpretation of the current findings should be considered in light of the study’s limitations. First, the sample was

relatively modest in size compared to some in the conflict adaptation literature (e.g., Clawson, Clayton, Keith, Catron, & Larson, 2017; Clayton & Larson, 2011a, 2011b, 2012); the specific relationships among reactive control components investigated here should be tested in a larger sample. Nevertheless, the task involved a relatively large number of experimental trials (800). This is an important asset of the study given the mixed-model, trial-level approach to analyzing the data, which allowed for an internally reliable estimate of within-person effects. Given the focus of the current study on cognitive control dynamics, this aspect of the design is more important than between-persons variability. Additionally, the participants were taking part in a study on the effects of alcohol and caffeine on cognition. Although none of the participants included in the present analyses actually ingested either substance, some of them (i.e., placebo group) were led to believe that they had, which could have influenced their performance motivation (e.g., Marczinski & Fillmore, 2005). However, statistical comparison of performance across the placebo and control groups showed no significant differences.

Second, the change in congruency sequence effects examined in this study has been framed primarily in terms of conflict adaptation. This account competes with alternative explanations grounded in learning and memory processes (for reviews, see Schmidt, 2013; Weissman, Jiang, & Egner, 2014). In some previous reports, excluding sequences with stimulus or response repetitions has eliminated congruency sequence effects (Akçay & Hazeltine, 2007, 2011; Clayton & Larson, 2011b; Egner & Hirsch, 2005; Funes, Lupiáñez, & Humphreys, 2010; Kunde & Wühr, 2006; Notebaert & Verguts, 2007). Our own test of this hypothesis (see supporting information) provided mixed support for both conflict adaptation and memory-based accounts. Full consideration of the implications of these varying accounts of congruency sequence effects and how they might account for changes across the course of the experiment is beyond the scope of this report.

Finally, although the estimation of change over time in cognitive control processes afforded by the trial-level MLM approach is a considerable strength of the study, effects associated with trial (i.e., time on task) should be interpreted with caution. We have suggested that changes over the course of the experiment might be due to fatigue, or the taxing of a cognitive resource. However, given the large number of trials used in the study, it is possible that practice resulted in alterations in the psychological mechanisms enlisted to perform the task and/or enabled the automatization of the conflict resolution process, thereby reducing the resource demands of conflict adaptation. It is therefore possible that the observed improvement in RT on incompatible trials is unrelated to the amplified conflict monitoring-related activity seen across the task as indexed by the bolstered N2. It is also possible that

the diminished FSW seen as a function of time on task is the result of a more efficient process being employed to perform the task, which countermands the need for increased control recruitment. Moreover, mental fatigue itself is a multifaceted concept (Ackerman, 1990; Francis & Inzlicht, 2016; Kool & Botvinick, 2014; Kurzban, Duckworth, Kable, & Myers, 2013) with several disparate explanations. The current data are agnostic with respect to the processes that underlie behavioral and neural indices of cognitive fatigue, which range from resource depletion (Baumeister, Vohs, & Tice, 2007), emotion and motivation shifting (Francis & Inzlicht, 2016; van der Linden, 2011), and decision-theoretic choices (Kool & Botvinick, 2014). Furthermore, both time on task and the number of trials performed during a task can independently contribute to fatigue (Grinband et al., 2011). The design of the present study does not allow for the separation of these two potential sources of change.

In summary, the findings contribute to the understanding of reactive control processes theorized to underlie conflict adaptation effects. Both evaluative/conflict monitoring (N2) and regulative control (FSW) demonstrated changes in conflict adaptation as the task progressed. Moreover, differential change in N2 and FSW over time suggest that conflict monitoring and regulative control were dissociable. Yet, the critical reciprocal relation between the two, whereby adaptation-related increases in regulative control on a given trial predict subsequent decreases in evaluative, conflict-monitoring processes, was maintained throughout the task. Future research could explore how the distinction between reactive control dynamics as a function of adjacent trial sequences interfaces with the distinction between transient, stimulus-driven control (reactive control) versus sustained, internally driven control (proactive control; see Braver, 2012).

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## SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

### Appendix Figure S1

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