Conflict Adaptation within but not across NoGo Decision Criteria: Event-Related-Potential Evidence of Specificity in the Contextual Modulation of Cognitive Control

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Abstract

From the standpoint of conflict-monitoring theory (Botvinick et al., 2001), detecting an incident of information-processing conflict should attenuate the disruptive influence of information-processing conflicts encountered subsequently, by which time cognitive-control operations will have been engaged. To examine the generality of this *conflict-adaptation* process across task dimensions, the present research analyzed event-related potentials in a Go/NoGo task that randomly varied the NoGo decision criterion applied across trials. Sequential analyses revealed reduced-amplitude fronto-central N2 and NoGo P3 responses on the second of two consecutive NoGo trials. Importantly, both of these conflict-adaptation effects were present only when the same NoGo decision criterion was applied across trials *n* and *n*-1. These findings support the theory that encountering information-processing conflict focuses attention on specific stimulus-response contingencies (Verguts & Notebaert, 2009) rather than engages general cognitive-control mechanisms (Freitas & Clark, 2015). Further implications for the generality of cognitive control are discussed.

Keywords: Go/NoGo; N2; NoGo P3; Cognitive control; Response mapping; Conflict adaptation; Congruence-sequence effects

Conflict Adaptation within but not across NoGo Decision Criteria: Event-Related-Potential Evidence of Specificity in the Contextual Modulation of Cognitive Control

Alternating between controlled and automatic responding is a central feature of human cognition. According to Botvinick, Braver, Barch, Carter, and Cohen's (2001) conflict monitoring theory, detecting an incident of information-processing conflict should attenuate the disruptive influence of information-processing conflicts encountered subsequently, by which time appropriate cognitive-control operations will have been engaged. One issue of substantial debate is the degree of specificity versus generality of conflict adaptation across task dimensions (e.g., Freitas & Clark, 2015; Funes, Lupiañez, & Humphreys, 2010; Kunde & Wuhr, 2006; Li et al., 2014; Notebaert & Verguts, 2007; Wendt, Kluwe, & Peters, 2006; for review, see Braem, Abrahamse, Duthoo, & Notebaert, 2014). The present study examined whether or not conflict-adaptation effects on event-related potentials (ERPs) would transcend different stimulus-response contingencies in a Go/NoGo task with NoGo criteria varying randomly across trials. As elaborated below, this question is motivated by contrasting theoretical perspectives.

Conflict Adaptation through Associative Learning

From the standpoint of an associative-learning model of conflict adaptation (Verguts & Notebaert, 2009), encountering information-processing conflict generates arousal, which serves to increase attention to task-specific stimulus and response dimensions. This model thus stipulates a specific phenomenological signal linking emotional and cognitive processes. The model further specifies the release of noradrenaline throughout the brain facilitating binding between task relevant cortical areas (Verguts & Notebaert, 2009). According to this model, the binding system accounts for how learning occurs in response to specific stimulus features. The

process begins in the medial prefrontal cortex as information-processing conflict is encountered, conveying information to the anterior cingulate cortex (ACC). The ventral and dorsal ACC then projects to the brainstem nuclei potentiating the autonomic nervous system, thus resulting in an autonomic, bottom up response. Following this model, encountering information-processing conflict should lead to a re-focus on the stimulus-response contingency that is specific to the trial with which one is engaged presently, such that conflict-adaption effects should emerge only if the same stimulus-response contingencies are encountered on the subsequent trial. Although studies of pupil dilation have not provided evidence that the binding is mediated by phasic arousal (Brown, Steenbergen, Kedar & Nieuwenhuis, 2014), there is considerable behavioral support for the specificity of conflict adaptation (Verbruggen et al., 2005; Kiesel et al., 2006; Notebaert & Verguts, 2007).

Within the body of research that supports the associative learning model of conflict adaptation, some studies have used two tasks, the Simon and SNARC tasks, in which both relevant and irrelevant dimensions differ for the two tasks (e.g., Notebaert & Verguts, 2008; Sturmer et al., 2005). Using such a design, conflict-adaptation has been found on task repetitions but not on task switches, suggesting that conflict adaptation is specific to particular stimulusresponse contingencies (Notebaert & Verguts, 2008). Interestingly, many of the studies that found specificity of conflict adaptation within task dimensions have used Simon tasks (e.g., Akçay & Hazeltine, 2011; Notebaert & Verguts, 2008; Schlaghecken, Refaat, & Maylor, 2011; Wendt, Kluwe, & Peters, 2006) or reverse Stroop tasks (Funes et al., 2010) intermixed with selective attention tasks. Importantly, such tasks require two different mechanisms to resolve the two different types of conflict; the former entails resolving stimulus-response conflicts, whereas latter requires resolving stimulus-stimulus conflicts. This could be one reason why previous cross-task findings have been mixed, some finding conflict-adaptation across tasks (e.g., Freitas et al., 2007; Kan et al., 2013; Kleiman et al., 2014; Kunde & Wuhr, 2006) and others finding conflict-adaptation only within single tasks (e.g., Ackay & Hazeltine, 2011; Funes et al., 2010; Wendt et al., 2006).

Conflict Adaptation through the Accessibility of Cognitive-Control Mechanisms

In contrast to the associative learning model, an alternative standpoint can be termed a mechanism-accessibility view (e.g., Egner, 2008; Freitas & Clark, 2015). Following this view, conflict-adaptation effects should be observed across different tasks to the extent that performance at trials *n* and *n*-1 depends on the operation of a single mechanism of cognitive control. To the extent that a mechanism of cognitive control (e.g., selective attention) is needed to resolve information-processing conflict at trial n, engaging that process at trial n-1 should facilitate resolving information-processing conflict at trial n (whether or not same stimulusresponse mapping is applied at trials *n* and *n*-1). A recent pair of experiments (Freitas & Clark, 2015, Experiments 3a and 3b) tested the generality of the conflict adaptation effect across three tasks, two that entailed stimulus-stimulus conflicts (a Stroop-trajectory task and a flanker task, each of which presumably depends on selective attention to resolve stimulus-stimulus conflicts; e.g., Kornblum, 1999) and one that entailed a stimulus-response conflict (a Simon task, which presumably depends on response selection to resolve stimulus-response conflict; Hommel, 1995). The authors hypothesized that across-task conflict adaptation effects would be observed only when the same cognitive-control mechanism was engaged across successive trials. Supporting that prediction, conflict-adaptation effects were observed across the Stroop-trajectory and flanker tasks but not across the Stroop-trajectory and Simon tasks. Because the Strooptrajectory and flanker tasks used different stimulus-response contingencies (depending on

stimulus location in the former case but stimulus meaning in the latter case) but depended on a common cognitive-control mechanism (selective attention), results from those studies provided strong support for the mechanism-accessibility view.

As elaborated below, the present study used two different stimulus-response mappings within a single task. This design allows contrasting the mechanism-accessibility and associativelearning models by holding constant the type of information-processing conflict while varying the stimulus-response mapping randomly across trials. If conflict adaptation effects emerge only when the same stimulus-response mapping is applied across successive trials, that would support the associative learning view. If conflict adaptation effects transcend stimulus-response mappings across successive trials, that would support the mechanism-accessibility view. We tested these hypotheses by examining potential ERP correlates of cognitive control in a Go/NoGo task.

Go/NoGo Tasks and the Fronto-Central N2 and NoGo P3

The Go/NoGo task is suitable to investigating cognitive control, given the competition it engenders between generating and withholding responses. A Go/NoGo task requires participants to respond to specific stimuli on Go trials and to withhold responding to specific stimuli on NoGo trials. Effectively withholding responding on a NoGo trial requires response inhibition, whereas the failure to withhold a response on a NoGo trial, termed a commission error, reflects the failure of response inhibition. Researchers have used this task in both clinical (Thomas, Gonsalvez & Johnstone, 2014) and developmental (Inoue et al., 2010) applications, adapting it appropriately for the different purposes.

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Previous research has found the fronto-central N2, a negative-going deflection in the ongoing electroencephalogram (EEG) maximal at fronto-central electrodes and peaking approximately 300 msec following the NoGo cue, to be a good measure of the recruitment of cognitive control (Nieuwenhuis et al., 2003; Smith et al., 2010). Based on the dipole method, the likely generator of the N2 appears to be the ACC, which supports its involvement in the recruitment of cognitive control (Yeung & Cohen, 2006). Several studies indicate that the N2 is a better measure of the recruitment of cognitive control than of inhibition per se. By manipulating NoGo frequency across blocks of trials to investigate implicit expectancy, Nieuwenhuis and colleagues (2003) found support for interpreting the N2 as a measure of conflict rather than inhibition. When manipulating the ratio of Go/NoGo trials and setting NoGo as the dominant trial type, they found relatively increased N2 amplitude during infrequent Go trials, implying a cognitive-control interpretation of N2 amplitude rather than an inhibition interpretation. Interestingly, Nieuwenhuis and colleagues (2003) found higher N2 amplitude on equiprobable NoGo than Go stimuli; the authors attributed this result to a general bias people have to respond to task stimuli. Another study compared results from a go/GO task, where "GO" trials required a response with maximal force, with a Go/NoGo task (Donkers & Von Boxtel, 2004). The N2 was found to be larger for any infrequent trials, and this pattern was found even when "GO" was infrequent when inhibition is not required, thus supporting the conflict-monitoring interpretation of the N2 (Donkers & Von Boxtel, 2004). Prior research has found higher-amplitude N2 responses when infrequent NoGo trials follow Go trials than NoGo trials, presumably reflecting a reduced need to engage cognitive control anew (Clayson & Larson, 2011).

Previous research also has examined effects of demands for cognitive control on the NoGo P3, a late-positive deflection in the ongoing EEG maximal at fronto-central electrodes on NoGo relative to Go trials (Pfefferbaum, Ford, Weller, & Kopell, 1985; Roberts, Rau, Lutzenberger, & Birbaumer, 1994). Supporting interpreting the NoGo P3 as relating generally to cognitive control, NoGo P3 amplitude is diminished in populations understood to have low response control, such as boys with attentional deficit hyperactive disorder (Fallgatter et al., 2004), children who have high levels of impulsivity (Jonkman, Lansbergen, & Stauder, 2003) children of alcoholics (Kamarajan et al., 2005), and individuals with Parkinson's disease (Bokura, Yamaguchi, & Kobayashi, 2005). On the other hand, NoGo P3 amplitude also has been found to reflect the presence in the ongoing EEG of variability relating to activity in the primary motor cortex on Go but not NoGo trials (e.g., Salisbury, Griggs, Shenton, & McCarley, 2004), indicating that processes independent of cognitive control also relate to NoGo P3 amplitude. Perhaps the clearest evidence for a cognitive-control-related interpretation of this component comes from studies finding that NoGo P3 amplitude is greatest when successfully withholding responding is particularly difficult, as when one must withhold responding to a stimulus to which another person contemporaneously responds (Sebanz et al., 2006) or to which one recently responded (Freitas et al., 2007). Following that approach, the present work will examine whether NoGo P3 amplitude is attenuated on the second of two consecutive NoGo trials, a conflictadaptation prediction that would support a cognitive-control-related interpretation of the NoGo P3.

Current Research

As described above, the present study contrasted the associative-learning and mechanism-accessibility models of conflict adaptation by examining ERPs to sequential manipulations of information-processing conflict while varying randomly the Go/NoGo rule participants used across trials. Participants responded to visual stimuli from alternate categories, using different Go/NoGo criteria for stimuli presented at different spatial locations. If encountering information-processing conflict serves to increase attention to task-specific stimulus and response dimensions, attenuated behavioral and neural indicators of conflict detection on the second of two NoGo trials should emerge *only* when the same Go/NoGo criterion is applied across consecutive trials. More specifically, the associative learning model suggests that encountering a NoGo cue at trial *n*-1 would lead to significantly fewer errors on NoGo trials and attenuated N2 and NoGo P3 amplitudes only when the same Go/NoGo rule is applied across successive trials, whereas the mechanism-accessibility model suggests that these conflict-adaptation effects should be observed irrespective of the consistency of the specific Go/NoGo rule applied across successive trials.

The present study also included methodological features that allow it to address alternatives to conflict-monitoring theory that have been proposed to account for putative conflict-adaptation effects. Feature-integration accounts emphasize the necessity of considering repetition priming in any sequential analyses (Mayr et al., 2003). Contingency learning accounts emphasize that sequentially analyzed cognitive-control experiments with more than two responses and half congruent / half incongruent trials can inadvertently create stimuluscontingency confounds, in which frequently occurring stimulus-response pairings become easier to carry out (Mordkoff, 2012; Schmidt & DeHouwer, 2012). Recent behavioral research has developed methods to remove exact repetitions without creating stimulus-contingency confounds, yielding results supporting conflict-monitoring theory (Duthoo, Abrahamse, Braem, Boehler & Notebaert, 2014; Freitas & Clark, 2015, Experiment 1; Kim & Cho, 2014; Weissman, Jiang, & Egner, 2014). The present study used non-repeating specific exemplars of two alternate categories, presented with equivalent frequency, thereby precluding exact stimulus repetitions

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without generating stimulus-contingency confounds. Evidence of attenuated N2 and NoGo P3 amplitude on the second of two consecutive NoGo trials thus would provide support for conflict-monitoring theory independent of feature-integration and contingency-learning accounts.

Methods

Participants

Twenty undergraduates (11 males), aged 17 - 36 (M = 19.71), participated in exchange for course credit.

Procedure

In a darkened, sound-attenuating chamber, participants sat in a large cushioned chair approximately 90 cm from the CRT monitor (running at 75 MHz refresh rate, with 1200 x 800 pixel resolution) on which experimental stimuli were presented. Holding a two-button response device in their laps, participants responded using the left and right thumb.

Stimuli and Task

Experimental stimuli consisted of six color images of fruit (an orange, a plum, a peach, and three varieties of apple) and six color images of pastries (three varieties of donut and three varieties of cookie), each approximately 73 pixels in height and 76 pixels in width, shown over a gray background. Trials began with a 240 msec fixation symbol ("+") centered at the monitor's vertical and horizontal midpoints. Experimental stimuli next were presented for 80 msec, centered at the monitor's horizontal midpoint and either 5% above or below the monitor's vertical midpoint.

Participants used two separate Go/NoGo rules across trials, depending on where stimuli appeared. For stimuli presented above the monitor's vertical midpoint, half of participants withheld responses to fruit; in contrast, they withheld responses to pastries presented below the monitor's vertical midpoint. The remainder of participants used opposite Go/NoGo rules, withholding responses to pastries shown above the monitor's vertical midpoint but to fruit shown below the monitor's vertical midpoint. On Go trials, half of participants pressed the left button for fruit and the right button for pastries, whereas the remainder pressed left for pastries and right for fruit. NoGo trials ended 1200 msec after stimulus presentation; any response recorded therein (and any erroneous response recorded on Go trials) triggered presentation of a brief auditory signal and of a visual reminder of task rules (displayed until participants pressed a response key to resume the task). There was no response deadline on Go trials. The inter-trial interval varied randomly between 700 – 900 msec. Participants acclimated to the task via a 48-trial practice block that concluded with verbal review of the task rules with an experimenter. Next, there were 848 experimental trials, with the first block again containing 48 trials¹ (as had the practice block) and the subsequent eight blocks containing 100 trials each. Brief rests (to allow blinking) were provided after each 25 trials within blocks. Performance feedback (on accuracy and latency) was provided after each block.

The four trial types (Go, upper-screen Go/NoGo rule; NoGo, upper-screen Go/NoGo rule; Go, lower-screen Go/NoGo rule; NoGo, lower-screen Go/NoGo rule) were selected for presentation randomly with replacement, such that Go and NoGo trials were equiprobable (occurring on 49.82% and 50.18% of trials, respectively), as were trials applying the two

¹ Conceived initially as an opportunity for additional practice, this block yielded accuracy rates and response times no different statistically (ts<1) from those recorded on the remaining blocks, warranting their inclusion to increase the signal to noise ratio.

Go/NoGo rules (occurring on 49.51% and 50.49% of trials, respectively). Within stimulus categories (i.e., fruit, pastries), particular items (e.g., plum, peach) were selected for presentation randomly with replacement, with the exception that no single item be displayed across successive trials, thereby eliminating from this experiment exact stimulus repetitions (cf. Mayr et al., 2003). After the experiment concluded, data were re-coded as a function of characteristics of trials *n* and *n*-1, along three orthogonal dimensions reflecting the consistency (Same vs. Change) of the Go/NoGo rule applied across trials *n* and *n*-1 (see examples in Figure 1, upper row of trial labels) and the Go/NoGo status of trials *n* and *n*-1 (see examples in Figure 1, lower row of trial labels). Accordingly, there were eight orthogonal combinations, reflecting the 2 (Consistency of Go/NoGo rule across successive trials) x 2 (Go/NoGo status of trial *n*-1) x 2 (Go/NoGo status of trial *n*) possible relations between trials *n*-1 and *n*.

Electrophysiological Recording

The EEG was recorded continuously via a 32-channel electrode cap (Neuroscan Inc.), using a fronto-central electrode as ground and electronically linked mastoid electrodes as reference. The horizontal electrooculogram (EOG) was monitored from electrodes at the outer canthi of the eyes, and the vertical EOG was monitored from electrodes above and below the orbital region of the left eye. Impedances for all electrodes were kept below 10 K Ω . The EEG and EOG signals were digitized at 500 Hz and amplified with a gain of 1000. The filter bandpass was .01-30 Hz.

ERP Analysis

Results are drawn from epochs beginning 100 msec before each stimulus was presented and concluding 900 msec thereafter. Baseline mean amplitude during the first 100 msec of each

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epoch was subtracted from remaining time points. To address EEG artifact, independent component analysis (ICA), accomplished via the Runica function of EEGlab (Delorme & Makeig, 2004), was used in two steps. First, through visual inspection and an initial ICA, epochs containing extreme non-stereotypic artifacts were identified and removed (6.17% of all trials). Via a second ICA, components reflecting vertical and horizontal eye movements, muscle-related activity, and channel-specific line noise were identified and subtracted. Following ICA-based corrections, any epochs with EEG voltages exceeding $+/-75 \,\mu$ V were removed, resulting in exclusion of 1.42% of remaining trials. The first trial of each block and trials following the brief rests provided within each block necessarily were not included in averages of the eight possible trial *n*-1 x trial *n* combinations, nor were trials on which errors were committed or trials immediately following error trials. The final waveforms for the eight possible trial n-1 x trial ncombinations (as displayed in Figures 3 and 4) were based on an average of 88.43 epochs each (range = 83.70 - 95.20 average epochs per waveform). Given extensive evidence that N2 and P3 effects are most prominent at midline electrodes, ERP analyses reported below are based on amplitude measurements from electrodes Fz, FCz, Cz, CPz, and Pz. Based on visual inspection of the overall Go/NoGo waveforms (collapsed across all other variables; see Figure 2), N2 amplitude was defined as mean amplitude between 300 and 400 msec and P3 amplitude was defined as mean amplitude between 450 and 700 msec. Greenhouse-Geisser-corrected p-values are reported for all comparisons with more than two within-subjects levels (Greenhouse & Geisser, 1959). To assess any potential artifactual influence of activity related to the LRP on the NoGo P3, we used the same data collected in the NoGo P3 window (between 450 and 700 msec). During that window, the LRP was computed as the averaged mean amplitude of C3 minus C4 during left button presses and C4 minus C3 during right button presses.

Results

Behavioral Results

Accuracy. Go and NoGo trials immediately following error trials (3.00% of total) were not analyzed. Average proportions of correct responses were analyzed in a 2 (Consistency of Go/NoGo rule across successive trials) x 2 (Go/NoGo status of trial n-1) x 2 (Go/NoGo status of trial n) repeated-measures analysis of variance (ANOVA). This analysis revealed significant main effects of Go/NoGo Rule Consistency, F(1, 19) = 18.59, p < .001, $\eta_p^2 = .49$, Go/NoGo status of trial *n*-1, *F* (1, 19) = 6.78, p < .05, $\eta_p^2 = .26$, and Go/NoGo status of trial *n*, *F* (1, 19) = 35.25, p < .0001, $\eta_p^2 = .65$. Most relevant to this investigation, there also was an interaction among the three variables, F(1, 19) = 7.49, p < .05, $\eta_p^2 = .28$. Clarifying the nature of this threeway interaction, the two-way interaction between Go/NoGo status at trials n and n-1 was significant when the Go/NoGo rule was the same across successive trials, F(1, 19) = 22.31, p < 100.0001, $\eta_p^2 = .54$, but not when it changed across successive trials, F = 0.12. As reported in Table 1, encountering a NoGo cue at trial n-1 significantly increased accuracy on NoGo trials only when the same Go/NoGo rule was applied across successive trials. Also noteworthy, a change in the Go/NoGo rule applied across successive trials significantly decreased accuracy only when NoGo trials directly followed NoGo trials (see Table 1).

Latency. On Go trials, response times less than 250 msec or greater than 1250 msec (2.08% of total) were not analyzed, nor were response times on error trials or on trials immediately following errors (3.54% of total). Average response times on Go trials were analyzed in a 2 (Consistency of Go/NoGo rule across successive trials) x 2 (Go/NoGo status of trial *n*-1) repeated-measures ANOVA. This analysis revealed significant main effects of

Go/NoGo Rule Consistency, F(1, 19) = 5.14, p < .05, $\eta_p^2 = .21$, and of Go/NoGo status of trial n-1, F(1, 19) = 9.62, p < .01, $\eta_p^2 = .34$. Most relevant to this investigation, there also was an interaction among the two variables, F(1, 19) = 33.20, p < .0001, $\eta_p^2 = .64$. Encountering a Go cue at trial n-1 significantly decreased response times on Go trials only when the same Go/NoGo rule was applied across successive trials (see Table 1). Also noteworthy, a change in the Go/NoGo rule applied across successive trials significantly increased response times only when Go trials directly followed Go trials (see Table 1).

N2 Amplitude

Mean amplitudes during the N2 measurement window were analyzed in a 2 (Consistency of Go/NoGo rule across successive trials) x 2 (Go/NoGo status of trial n-1) x 2 (Go/NoGo status of trial n) x 5 (Electrode Location) ANOVA. This analysis revealed a main effect of trial nGo/NoGo status, F(1, 19) = 12.46, p < .01, $\eta_p^2 = .40$, reflecting a negative deflection on NoGo relative to Go trials that was significant at all electrode locations ($Fs \ge 8.22$, ps < .01). Apart from a main effect of Electrode Location, F(4, 76) = 49.84, p < .0001, $\eta_p^2 = .72$, no other main effects were significant. There also was a significant Go/NoGo Rule Consistency x Electrode Location interaction, F(1, 19) = 21.88, p < .0001, $\eta_p^2 = .54$, reflecting a negative deflection on Rule-Change relative to Rule-Same trials that was significant at electrode FCz, F(1, 19) = 8.42, p < .01, $\eta_p^2 = .31$, (see Figure 3) and at electrode Fz, F(1, 19) = 11.87, p < .01, $\eta_p^2 = .38$, but not at any other electrodes ($Fs \le 1.96$, ps > .17). Most important to this investigation, the three-way interaction between Go/NoGo Rule Consistency, Go/NoGo status of trial n-1, and Go/NoGo status of trial *n* also was significant, F(1, 19) = 24.57, p < .0001, $\eta_p^2 = .56$, and was moderated further (i.e., in a four-way interaction) by Electrode Location, F(4, 76) = 4.41, p < .05, $\eta_p^2 = .19$. Clarifying the nature of the aforementioned three-way interaction, the two-way interaction

between Go/NoGo status at trials *n* and *n*-1 was significant when the Go/NoGo rule was the same across successive trials, F(1, 19) = 29.59, p < .0001, $\eta_p^2 = .61$ (see Figure 3), but not when the Go/NoGo rule changed across successive trials, F = 0.59 (see Figure 4). Clarifying the roles of particular electrode locations in driving the aforementioned four-way interaction, encountering a NoGo cue at trial *n*-1 significantly decreased the negativity of N2 amplitude on NoGo trials at all electrode locations, but most prominently at fronto-central electrodes Fz, FCz, and Cz, again only when the same Go/NoGo rule was applied across successive trials (see Table 2). Also noteworthy, as highlighted in the difference waveforms presented in Figure 5, a change in the Go/NoGo rule applied across successive trials increased the negativity of N2 amplitude when NoGo trials directly followed NoGo trials (at fronto-central electrodes Fz, FCz, and Cz; see Table 2) and when Go trials directly followed Go trials (at all electrode locations; see Table 2).

P3 Amplitude

Mean amplitudes during the P3 measurement window were analyzed in a 2 (Consistency of Go/NoGo rule across successive trials) x 2 (Go/NoGo status of trial *n*-1) x 2 (Go/NoGo status of trial *n*) x 5 (Electrode Location) ANOVA. This analysis revealed significant main effects of Electrode Location, F(4, 76) = 23.08, p < .0001, $\eta_p^2 = .55$, and of trial *n* Go/NoGo status, F(1, 19) = 12.10, p < .01, $\eta_p^2 = .39$. Neither the main effect of Go/NoGo Rule Consistency nor its interaction with Electrode Location was significant ($Fs \le 1.65$, ps > .21). Consistent with previous findings of a relatively frontal topographical distribution of P3 effects on NoGo relative to Go trials, there was a Go/NoGo x Electrode Location interaction, F(1, 19) = 19.54, p < .0001, $\eta_p^2 = .51$, reflecting the fact that P3 amplitude was significantly higher on Go than on NoGo trials only at centro-parietal electrodes Cz, CPz, and Pz ($Fs \ge 15.38$, ps < .001) but not at frontal

electrodes Fz (F = 0.03) and FCz (F(1, 19) = 2.86, p > .10). Most relevant to this investigation, there also was a three-way interaction between Go/NoGo Rule Consistency, Go/NoGo status of trial *n*-1, and Go/NoGo status of trial *n*, $F(1, 19) = 13.62, p < .01, \eta_p^2 = .42$, which was moderated further (i.e., in a four-way interaction) by Electrode Location, $F(4, 76) = 25.71, p < .0001, \eta_p^2 = .58$. Clarifying the nature of the aforementioned three-way interaction, the two-way interaction between Go/NoGo status at trials *n* and *n*-1 was significant when the Go/NoGo rule was the same across successive trials, $F(1, 19) = 33.00, p < .0001, \eta_p^2 = .63$, (see Figure 3), but not when the Go/NoGo rule changed across successive trials, F = 0.99 (see Figure 4). Clarifying the roles of particular electrode locations in driving the aforementioned four-way interaction, encountering a NoGo cue at trial *n*-1 significantly decreased the positivity of P3 amplitude on Go trials. Encountering a Go cue at trial *n*-1 significantly decreased the positivity of P3 amplitude on Go trials (at centro-parietal electrodes Cz, CPz, and Pz; see Table

3) only when the same Go/NoGo rule was applied across successive trials. Also noteworthy, as highlighted in the difference waveforms presented in Figure 5, a change in the Go/NoGo rule applied across successive trials increased the positivity of P3 amplitude when NoGo trials directly followed NoGo trials (at all electrode locations) and when Go trials directly followed Go trials (at centro-parietal electrodes Cz, CPz, and Pz; see Table 3).

Discussion

These findings make several contributions to current understandings of cognitive control and its neuroelectric correlates. Turning first to effects present when collapsing across preceding trial types, there was a pronounced negative deflection 300 – 400 msec following stimulus presentation on NoGo relative to Go trials. This finding replicates previous observations of

NoGo N2 effects even when, as in the present work, Go and NoGo trials are equiprobable (e.g., Nieuwenhuis et al., 2003; Falkenstein, Hoormann, & Hohnsbein, 1999). Drawing on the logic of earlier authors (Jodo & Kayama, 1992; Nieuwenhuis et al., 2003), we assume that the present study's general methods, including its use of brief stimulus presentations and inter-trial intervals, facilitated a general impetus toward rapid responding on Go trials, thereby requiring a substantial degree of cognitive control to withhold responses on NoGo trials and yielding robust NoGo N2 effects.

A clear NoGo P3 pattern also emerged, whereby a positive deflection 450 – 700 msec following stimulus presentation displayed a relatively frontal topographical distribution on NoGo relative to Go trials. Although consistent with a cognitive-control interpretation of the NoGo P3, this main effect also could partly reflect the presence of motor-related activity on Go trials but not NoGo trials (Salisbury et al., 2004). For example, Verleger and colleagues (2006) observed higher NoGo P3 amplitude in blocks entailing generating and inhibiting hand movements than in blocks entailing generating or inhibiting eye movements. That interesting result suggests that motor potentials on Go trials contributed to the observed NoGo P3 effects, given the relatively frontal topographical location of the primary motor cortex, which controls hand movements but not eye movements (Verleger et al., 2006). In this vein, it is important to note that the present work yielded evidence that sequential manipulations of Go/NoGo trial presentations modulated NoGo P3 amplitude. More specifically, as illustrated in Figure 3, P3 amplitude at electrode FCz was higher on NoGo trials preceded by Go trials than on NoGo trial preceded by NoGo trials. This evidence of variability in NoGo P3 amplitude on different types of NoGo trials (during blocks that all entailed the same kinds of motor responses) indicates that NoGo P3 amplitude was highest on trials for which demands cognitive control presumably were greatest,

independent of any role of motor potentials on Go trials. Accordingly, the present sequential effects on NoGo P3 amplitude indicate that this component relates at least partly to current demands for cognitive control, although further work is needed to clarify the nature of task manipulations that do (e.g., Freitas et al., 2007; Sebanz et al., 2006) or do not (Kopp et al., 1996) modulate NoGo P3 amplitude.

Most relevant to our investigation of the generality of contextual adjustments in cognitive control, we found conflict-adaptation effects only when the same Go/NoGo rule was applied across successive trials. Behavioral results revealed that encountering a NoGo cue at trial *n*-1 increased accuracy and decreased response time on trial *n* only when the same Go/NoGo rule was applied across successive trials. Furthermore, electrophysiological data revealed similar results, finding attenuated N2 amplitudes when the previous trial was a NoGo trial only when the same rule was applied across successive trials. Similarly, NoGo P3 amplitude decreased when trial *n*-1 was a NoGo cue only when the same Go/NoGo rule was applied across successive trials. Accordingly, our results strongly supported the associative learning view (Verguts & Notebaert, 2009) rather than the mechanism-accessibility view (Freitas & Clark, 2015), given that conflict-adaptation effects were found only when a consistent NoGo criterion was applied across consecutive trials.

Because conflict-adaptation effects have been observed behaviorally even when qualitatively distinct stimulus-response contingencies are engaged across trials (e.g., Freitas et al., 2007; Kan et al., 2013; Kleiman et al., 2014), it is important for future work to examine whether certain contexts may be more amendable than others to observing specificity relative to generality in the contextual modulation of cognitive control. For example, the present experiment may have facilitated associative learning effects due to how taxing it is to maintain two sets of

rules. Perhaps, once a participant encountered information-processing conflict, the participant had to recall or clarify the specific rule to be used. Some other studies that have found relative specificity of conflict adaptation also appear to have made use of relatively complex rules that may have required participants to think back to them after making an error (e.g., Braem, Verguts, & Notebaert, 2011). In addition, by involving Go/NoGo responses to a single set of stimuli across fluctuating NoGo criteria, the present experiment may have incurred partial-repetition costs, which arise when a task involves repetitions of only some stimulus and response elements across trials (Hommel, 2004). Partial repetitions may have impeded across-rule adaptation effects by making the task more difficult, a possibility consistent with the recent theorizing of Braem, Abrahamse, Duthoo, and Notebaert (2014). Follow-up research can test for conflict adaptation effects across task rules with more dissimilar stimuli (such as food items for one set of rules and tool items for another set of rules).² Also, as previously mentioned, task switching paradigms inevitably result in switch costs that arise when cognitive resources are being exerted (cf. Egner, 2008). Although the present study utilized the same task across trials, the rule-switch itself may have been taxing (e.g., see Figure 5). Future research may include rule-switch

warnings to examine whether conflict adaptation effects can generalize across rules, as we had predicted based on the mechanism-accessibility view, when rule-switch effects are minimized.

² We thank Senne Braem for this suggestion.

More generally, the present study observed clear conflict-adaptation effects, supporting conflict-monitoring theory. Previous examinations of sequential Go/NoGo effects have modified the Go/NoGo task by adding a cue prior to the response stimulus to vary the probability of informative and non-informative cueing, thus manipulating the expectancy of conflict (e.g., Randall & Smith, 2011; Fleming & Bartholow, 2014) or have modified the task by using paired stimuli (e.g., Kropotov, Ponomarev, Hollup, & Mueller, 2011; Smith, Johnstone, & Barry, 2007). In addition, using runs of Go before NoGo trials (e.g., Durston, Thomas, Worden, Yang, & Casey, 2002; Thomas, Gonsalvez & Johnstone, 2014) also can complicate interpretations of sequential analyses. For example, placing Go trials always 1, 3 or 5 trials before a NoGo trial (Durston et al., 2002; Zamorano et al., 2014) can enable participants to learn that a sequence of 5 Go trials must be followed by a NoGo trial, while also precluding presentation of two consecutive NoGo trials, eliminating any analysis of NoGo repetitions, which are crucial to the investigation of conflict-adaptation effects. For these reasons, random (with replacement) selection of Go and NoGo trials would appear to allow clearest interpretation of Go/NoGo sequential effects. Smith, Smith, Provost & Heathcote (2010) utilized equiprobable trains in a random sequence, but the overall ratio of the Go and NoGo trials were 69%:31%, which again makes sequential effects difficult to interpret. In summary, by holding constant overall expectancies and averting incidental learning of stimulus-presentation contingencies, our results provide clear support for conflict-monitoring theory.

Conclusion

We sequentially manipulated the presentation of Go/NoGo cues, such that participants responded to randomly varying NoGo criteria across trials. Analyses of response times, response accuracy, and event-related potentials (the fronto-central N2 and the NoGo P3) all yielded results

indicating that participants adapted to information-processing conflict *only* when the same NoGo criterion was applied across consecutive NoGo trials. These findings support an associative learning view of conflict adaptation, which states that encountering information-processing conflict prompts attention to the specific stimulus-response contingencies inherent in one's ongoing task (Verguts & Notebaert, 2009).

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Authors' Note

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Table 1. Average latency (in msec) and accuracy (proportion correct) of behavioral responses on NoGo and Go trials, reported as a function of trial *n*-1 trial type (pNoGo and pGo) and Go/NoGo ("G/NG") rule consistency across trials *n* and *n*-1 (Same and Change). Difference columns report effects of previous trial type (pGo minus pNoGO) and difference rows report effects of Go/NoGo rule consistency (Change minus Same). Standard errors of the mean in parentheses.

	G/NG Rule Consistency	<u>NoGo Trials</u>			<u>Go Trials</u>		
<u>Variable</u>		pNoGo	pGo	Difference	<u>pNoGo</u>	pGo	Difference
	Same				577.27 (15.62)	516.82 (13.98)	60.46 (9.79)****
Latency	Change				556.49 (18.88)	569.08 (18.20)	-12.59 (10.18)
	Difference				-20.78 (8.79)*	52.26 (9.97)****	
Accuracy	Same	.979 (.004)	.930 (.013)	.050 (.010)****	.995 (.002)	.997 (.002)	003 (.002)
	Change	.935 (.012)	.933 (.011)	.002 (.012)	.991 (.004)	.994 (.003)	002 (.004)
	Difference	044 (.010)****	.003 (.008)		003 (.003)	004 (.003)	

Note: *****p* < .0001; **p* < .05.

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Table 2. Mean N2 amplitude on NoGo and Go trials, reported as a function of trial *n*-1 trial type (pNoGo and pGo) and Go/NoGo ("G/NG") rule consistency across trials *n* and *n*-1 (Same and Change). Difference columns report effects of previous trial type (pGo minus pNoGO). Difference rows report effects of Go/NoGo rule consistency (Change minus Same). Standard errors of the mean in parentheses.

		<u>NoGo Trials</u>			<u>Go Trials</u>		
<u>Electrode</u>	G/NG Rule Consistency	<u>pNoGo</u>	pGo	Difference	<u>pNoGo</u>	pGo	Difference
	Same	-0.30 (0.48)	-2.83 (0.89)	2.53 (0.61)***	-0.43 (0.55)	-0.12 (0.81)	-0.30 (0.48)
Fz	Change	-2.16 (0.80)	-2.26 (0.76)	0.10 (0.60)	-0.72 (0.74)	-1.64 (0.89)	0.92 (0.51)
	Difference	-1.86 (0.42)***	0.57 (0.46)		-0.29 (0.39)	-1.51 (0.40)**	
FCz	Same	0.94 (0.61)	-1.65 (0.99)	2.59 (0.61)***	0.83 (0.63)	1.32 (0.99)	-0.49 (0.56)
	Change	-0.95 (0.88)	-1.01 (0.91)	0.06 (0.75)	0.62 (0.88)	-0.10 (1.00)	0.72 (0.50)
	Difference	-1.89 (0.44)***	0.64 (0.47)		-0.21 (0.41)	-1.42 (0.38)**	
Cz	Same	2.94 (0.66)	0.66 (1.08)	2.28 (0.62)**	3.12 (0.74)	3.85 (1.22)	-0.72 (0.67)
	Change	1.56 (0.84)	1.54 (1.16)	0.01 (0.82)	3.33 (0.99)	2.70 (1.17)	0.62 (0.51)
	Difference	-1.39 (0.35)***	0.88 (0.50)		0.20 (0.44)	-1.14 (0.34)**	
CPz	Same	4.59 (0.67)	2.99 (1.02)	1.60 (0.58)*	5.19 (0.82)	6.21 (1.25)	-1.02 (0.66)
	Change	4.07 (0.74)	3.81 (1.18)	0.26 (0.79)	5.66 (1.02)	5.37 (1.16)	0.29 (0.48)
	Difference	-0.52 (0.35)	0.82 (0.49)		0.47 (0.41)	-0.84 (0.32)*	
Pz	Same	5.32 (0.76)	4.09 (1.05)	1.23 (0.58)*	6.31 (0.87)	7.31 (1.26)	-0.99 (0.64)
	Change	5.54 (0.74)	4.88 (1.21)	0.65 (0.78)	6.88 (1.03)	6.58 (1.17)	0.30 (0.48)
	Difference	0.21 (0.38)	0.79 (0.49)		0.57 (0.38)	-0.72 (0.30)*	

Note: ****p* < .001; ** *p* < .01; **p* < .05.

Table 3. Mean P3 amplitude on NoGo and Go trials, reported as a function of trial n-1 trial type (pNoGo and pGo) and Go/NoGo ("G/NG") rule consistency across trials n and n-1 (Same and Change). Difference columns report effects of previous trial type (pGo minus pNoGO). Difference rows report effects of Go/NoGo rule consistency (Change minus Same). Standard errors of the mean in parentheses.

<u>Go Trials</u>		
rence		
(0.56)		
(0.64)		
(0.60)		
0.64)		
(0.59)*		
(0.56)		
(0.51)***		
(0.48)		
. ,		
(0.42)****		
(0.51)		
. /		

Note: ****p < .0001; ***p < .001; **p < .01; *p < .05.