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When Cognitive Control Is Calibrated: Event-Related Potential Correlates of Adapting to Information-Processing Conflict despite Erroneous Response Preparation

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Abstract

To examine when in the perception-action cycle resolving information-processing conflict modulates signals of the current need for cognitive control, the present work examined event-related potential correlates of response preparation (lateralized readiness potentials; LRPs) and of information-processing conflict (fronto-central N2 responses) on trial *n* flanker trials, as a function of whether trial *n*-1 entailed a congruent flanker, an incongruent flanker, or a NoGo cue. Although LRP-indexed erroneous response preparation was substantial on incongruent trials across all levels of trial *n*-1, N2 amplitudes, and behavioral interference effects, were attenuated on incongruent trials following NoGo and incongruent (relative to congruent) trials. Even after initial attentional and motor-preparation processes have transpired, then, relatively later control mechanisms appear sufficient to signal a reduced need to engage cognitive control anew.

Because demands for cognitive control can fluctuate rapidly across time, calibrating context-appropriate levels of cognitive control is integral to the flexibility of goal-directed thought and behavior. To explain how this calibration is accomplished, an influential conflict-monitoring theory proposes that the anterior cingulate cortex (ACC) detects information-processing conflict and engages structures within the dorsolateral prefrontal cortex that mediate cognitive control; as the amount of competition within processing streams then is reduced, so too is the subsequent level of activation of the ACC (Botvinick et al., 2001). Reduced ACC activation (sometimes termed *conflict adaptation*) indeed often is observed on the second of two high-conflict events, as when Stroop stimuli mismatch in color and word meaning on consecutive trials (Kerns et al., 2004). However, traversing from perception to action can entail confronting an assortment of information-processing conflicts, from perceptual ambiguity (Koechlin & Summerfield, 2007) to incompatibility between ongoing responses and temporally distal goals (Fuster, 2004), addressed via mechanisms apparently distributed functionally across the prefrontal cortex (for reviews, see Badre, 2008; Botvinick, 2008). Understanding the role of conflict monitoring in calibrating cognitive control requires specifying how the outputs of these heterogeneous mechanisms elicit conflict adaptation in the ACC.

For example, information-processing conflict on trial *n*-1 has been found to impact neural correlates of perceptual adjustment (e.g., Egner & Hirsch, 2005a) observable within 70 msec of trial *n* stimulus presentation (Scerif et al., 2006). Although links between these effects and ACC activity have not yet been examined, modulating perceptual processes, such as by enhancing cortical representations of task-relevant stimuli (Egner & Hirsch, 2005b), appears a potent means of facilitating initiating task-appropriate responses and averting subsequent information-processing conflicts. On the other hand, control processes also often need to be applied even

after a task-inappropriate response has been initiated, as when a diner remembers his New Year's dieting resolution after beginning to reach for the desert tray (cf. Fuster, 2004) or when a descending pilot receives a new order to desist landing an aircraft (cf. Logan, Schachar, & Tannock, 1997). It is presently unknown whether conflict adaptation in the ACC is responsive to the output of only relatively early control processes that resolve information-processing conflict before response preparation or also to relatively later control processes operating after response preparation has begun. Addressing this question can help indicate whether cognitive control depends on a monitoring process sensitive to the degree of conflict initially appraised in an event or also to fluctuations in conflict as single events unfold.

The temporal precision of event-related potentials (ERPs) is well suited to examining when conflict adaptation can occur during the perception-action cycle. Events high in information-processing conflict elicit enhanced negative deflections at fronto-central scalp electrodes often traced to a medial-frontal neural generator proximal to the ACC (for a meta-analysis of pertinent source analyses, see Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). The time courses of these negative deflections reflect the tasks and stimuli used, with peaks at around 300 msec for flanker and Go/NoGo tasks using simple visual stimuli (the fronto-central N2), peaks at around 450 msec for Stroop studies using verbal stimuli (the N450), and peaks at around the time of erroneous responses for error-detection studies (the ERN; for an integrative review of these components, see Folstein & Van Petten, 2008). For example, prominent fronto-central N2 effects emerge in forced-choice tasks when participants' non-targets closely resemble their targets, such that two incompatible responses are implicated and information-processing conflict is high (Azizian, Freitas, Parvaz, & Squires, 2006; Nieuwenhuis, Yeung, & Cohen, 2004; Folstein & Van Petten, 2004).

A well-studied neural correlate of motor-response preparation, on the other hand, is the lateralized readiness potential (LRP), a negative deflection measured at scalp electrodes proximal to contralateral relative to ipsilateral primary motor cortex just before left- and right-sided motor responses (e.g., Kutas & Donchin, 1980). For example, subliminal semantic priming has been assessed via transient LRP effects elicited by extremely rapid presentation of verbal cues for left- and right-handed responses, with functional magnetic resonance imaging (fMRI) substantiating involvement of the primary motor cortex in the same task (Dehaene et al., 1998). LRPs time-locked to stimulus presentation rather than to response completion often are interpreted to reflect neural processes relating to stimulus comprehension rather than response execution, respectively (e.g., Coles, 1989; Gladwin, 't Hart, & De Jong, 2008). Accordingly, stimulus-locked LRP latencies can be viewed to reflect the upper boundary of the time required to complete stimulus evaluation prior to response generation (e.g., Töllner, Gramman, Müller, Kiss, & Eimer, 2008), and stimulus-locked LRP amplitudes can be viewed to reflect the magnitude of response activation primed by the stimulus (e.g., Leuthold & Kopp, 1998).

The present research examined potential conflict-adaptation effects in amplitude of the fronto-central N2 and the stimulus-locked LRP. Using a delayed flanker task, in which irrelevant flanking stimuli (e.g., "<< <<") appear just before congruent (e.g., "<") or incongruent (e.g., ">") central cues for left- or right-handed responses, we expected to replicate previous findings of prominent N2 responses on incongruent trials (where information-processing conflict is high) relative to congruent trials (where information-processing conflict is low; e.g., Botvinick et al., 2004; Kopp et al., 1996; Van Veen & Carter, 2002; Wendt, Heldemann, Münte, & Kluwe, 2007) and of LRP-amplitude reversals on incongruent trials (in which flanking stimuli prime motor responses different from those later primed by central stimuli) but not congruent trials (in which

flanking and central stimuli prime the same motor response; Kopp et al., 1996; Mattler, 2003). Of key importance to the present investigation are potential changes in these effects as a function of the local context. If conflict adaptation in the ACC is triggered primarily by the output of early perceptual adjustments, then not only N2 but also LRP effects should attenuate on the second of two high-conflict trials, because decreased priming of erroneous motor responses (as reflected in LRP amplitude) would be the presumed mechanism by which preventing information-processing conflict attenuates ACC activation (as reflected in N2 amplitude). In contrast, if N2 amplitudes attenuate on the second of two high-conflict trials despite prominent LRP-amplitude reversals on those trials, conflict adaptation in the ACC would appear sensitive to the outcomes of relatively later control processes occurring even after erroneous motor responses have been initiated.

Testing these hypotheses, we drew on past evidence that conflict adaptation occurs most reliably when task parameters maximize general information-processing conflict and minimize episodic-retrieval-based stimulus-specific priming effects, as when combining brief stimulus presentations with variable durations between trials and staggered presentation of flanking and central stimuli (Ullsperger, Bylsma, Botvinick, 2005). In the only other examination of electrophysiological correlates of conflict adaptation in the ACC of which we are aware, different task parameters were used, and neither response times nor N2 amplitudes varied as a function of trial *n*-1 flanker congruence (Wendt et al., 2007). Accordingly, extant research has not yet allowed examining electrophysiological correlates of conflict adaptation in the ACC under conditions in which behavioral evidence of conflict adaptation has been present.

Addressing this issue, the present research also varied how cognitive control was engaged. On a minority of trials, following presentation of flanking stimuli, a NoGo cue

indicated that motor responses were to be withheld (for other combined selective attention/NoGo tasks, see, e.g., Kopp et al., 1996; Freitas, Azizian, Leung, & Squires, 2007; Scerif et al., 2006). Particularly when encountered infrequently (e,g., Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003), cues to suppress responses entail significant information-processing conflict (e.g., Falkenstein, Hoormann, & Hohnsbein, 1999). Behavioral indicators of conflict adaptation appear sensitive to general levels of information-processing conflict despite qualitative differences between responses across trials (Freitas, Bahar, Yang, & Banai, 2007). Accordingly, whether in the service of response generation (on incongruent flanker trials) or suppression (on NoGo trials), engaging cognitive control may modulate how subsequent information-processing conflicts are addressed. The present investigation of potential changes in LRP and N2 amplitudes on trial *n* flanker trials, as a function of whether trial *n*-1 entailed a congruent flanker, an incongruent flanker, or a NoGo cue, can clarify when in the perception-action cycle these modulations occur.

Methods

Participants were 23 undergraduates (11 males), aged 18 – 23 (M = 19.91), compensated with either course credit or cash payment (US $20). In a sound-attenuating chamber, participants were seated on a padded chair approximately 90 cm from the CRT monitor (with 75 Mhz refresh rate) on which experimental stimulus arrays (6 x 1 cm in black on a white background) were presented. Participants responded via a handheld two-button response device, using the left and right thumb to respond to left and right arrows, respectively.

Each trial began with a fixation cue (“.”) displayed for either 400, 506.67, 600, or 706.67 msec (determined randomly). Lateral flanker stimuli next were displayed alone for 93.33 msec,

followed by the central stimulus, which was displayed (along with lateral flanker stimuli) for 106.67 msec. On congruent and incongruent trials (each occurring with .40 probability), the central stimulus was an arrow that matched or mismatched the flanking arrows, respectively. On NoGo trials (occurring with .20 probability), the central stimulus was an arrow fragment (either the top or bottom of a left or right arrow). Participants were instructed to press the response button indicating the direction of central arrows and to withhold responses to arrow fragments, with speed and accuracy stressed as equally important. Incorrect responses (including any response within 1000 msec of presentation of NoGo cues) elicited a 200 msec tone. After each response on flanker trials, and 1000 msec after presentation of NoGo cues, the screen remained blank for either 800, 906.67, 1000, or 1106.67 msec. Trials that began with longer-duration pre-stimulus fixation cues ended with shorter-duration post-response blank screens, with the sum of these two durations always equaling 1506.67 msec. There were 800 trials, divided into 20 blocks, between which participants took brief, self-paced breaks. After the experiment, data were recoded to indicate combined trial *n* and *n*–1 pairs. To isolate potential effects of conflict adaptation from effects of repetition priming (Mayr et al., 2003), trials preceded by exact stimulus repetitions (13.95% of all trials) were excluded from analysis.

Electrophysiological Recording

The electroencephalogram (EEG) was recorded continuously via a 32-channel electrode cap (Neuroscan Inc., Sterling USA), 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 KΩ. The EEG and EOG signals were digitized at 500 Hz and were amplified with a gain of 1000. The filter bandpass was .01-30 Hz.

ERP Analysis and Statistics

Epochs began 194 msec before presentation of the critical central stimulus (and, hence, 100.67 msec before presentation of the flanker stimuli) and ended 806 msec thereafter. The first 100 msec of each epoch (corresponding to presentation of the fixation cue) served as the pre-stimulus baseline. Extreme non-stereotypic artifacts were identified visually and removed from the continuous EEG file (17.20% of all trials). Next, using independent component analysis (ICA) via the Runica function of EEGlab (Delorme & Makeig, 2004), components reflecting eye movements, other muscle-related activity, and channel-specific line noise were identified and subtracted. Following ICA-based corrections, any remaining epochs with EEG voltages exceeding +/- 75 μV at any channel were rejected from the average (resulting in exclusion of 4.95% of remaining trials). Based on visual inspection of grand-average waveforms combined on the basis of trial *n* stimulus types (collapsed across trial *n*-1 stimulus types, as shown in Figure 2), the flanker N2 response was defined as mean amplitude between 248 and 338 msec, which corresponds approximately to the time points at which the waveforms associated with congruent and incongruent flanker trials intersected, as the latter fell and then rose in the N2 response, at FCZ (see shaded region of Figure 2, top panel).[1] Also based on visual inspection of grand-average waveforms combined on the basis of trial *n* stimulus types (collapsed across trial *n*-1 stimulus types), the LRP (computed as the averaged mean amplitude of C3 minus C4 during left button presses and C4 minus C3 during right button presses) was assessed between 154 and 354 msec for congruent flanker trials and between 144 and 282 and between 282 and 500 msec for

the first and second measurement windows on incongruent flanker trials, which correspond approximately to the time points at which the trial-*n*-averaged difference waveforms departed from and then returned to baseline (of 0 μV). Prior to statistical analysis, data from each of the three electrodes nearest the midline were placed into groups centered at FZ (FZ, F3, F4), FCZ (FCZ, FC3, FC4), CZ (CZ, C3, C4), CPZ (CPZ, CP3, CP4), and PZ (PZ, P3, P4). In cases of a bad electrode (total $N = 6$ bad electrodes across the 23 participants), data from the participant's remaining electrodes in each grouping were used. Greenhouse-Geisser-corrected *p*-values are reported for all comparisons with more than two within-subjects levels (Greenhouse & Geisser, 1959).

Results

Behavioral Results

Behavioral and electrophysiological responses on or immediately following error trials (8.34 % of total) were not analyzed. Response times below 200 or above 800 msec (0.62% of remaining trials) also were discarded. Response times and accuracy rates on flanker trials were analyzed in 2 (Trial *n*: congruent or incongruent flanker) x 3 (Trial *n*-1: NoGo, congruent or incongruent flanker) repeated-measures ANOVAs (see Figure 1). For response times, there was a significant effect of trial *n* flanker congruence, $F\ (1, 22) = 293.87$, partial $\eta^2 = .93$, $p < .0001$, a significant effect of trial *n*-1 trial type, $F\ (2, 22) = 9.81$, partial $\eta^2 = .31$, $p < .0005$, and, most importantly, a significant interaction between the two factors, $F\ (2, 22) = 21.50$, partial $\eta^2 = .49$, $p < .0001$. Response times on incongruent trials were slower when preceded by congruent flanker trials ($M = 462.09$ msec, $SD = 28.64$) than when preceded by incongruent trials ($M = 451.10$ msec, $SD = 32.44$), $t\ (22) = 3.71$, $p < .005$, or by NoGo trials ($M = 456.17$ msec, $SD = 31.83$), *t*

(22) = 2.66, $p < .02$. In contrast, response times on congruent trials were faster when preceded by congruent trials (M = 362.15 msec, SD = 33.06) than when preceded by incongruent trials (M = 374.77 msec, SD = 28.60), t (22) = 4.08, $p < .0005$, or by NoGo trials (M = 384.40 msec, SD = 34.40), t (22) = 6.35, $p < .0001$. For error rates, there was a significant effect of trial n flanker congruence, F (1, 22) = 12.99, partial η^2=.37, $p < .005$, a significant effect of trial n-1 trial type, F (2, 22) = 13.63, partial η^2=.38, $p < .0001$, and, again, a significant interaction between the two factors, F (2, 22) = 11.74, partial η^2=.35, $p < .0005$. Error rates on incongruent trials were higher when preceded by congruent trials (M = 11.28%, SD = 12.59) than when preceded by incongruent trials (M = 4.98%, SD = 7.44%), t (22) = 3.71, $p < .005$, or by NoGo trials (M = 5.56%, SD = 8.24), t (22) = 5.04, $p < .0001$. In contrast, congruent-trial error rates (which were lower than 0.60% in all cases), did not vary as a function of trial n-1 trial type (ts < 1).

Conflict Adaptation in N2 Amplitude

Grand-average waveforms associated with trial n presentation of congruent and incongruent flanker cues and NoGo cues, collapsed across cue type at trial n-1, are presented in Figure 2. Consistent with extensive previous evidence of pronounced impacts of NoGo cues on fronto-central N2 amplitude (e.g., Falkenstein et al., 1999; Nieuwenhuis et al., 2003) and anterior P3 amplitude (Fallgatter & Strik, 1999; Salisbury, Griggs, Shenton, & McCarley, 2004), waveforms associated with infrequent NoGo cues in the present experiment can be seen to demonstrate prominent N2 and P3 effects maximal fronto-centrally relative to centro-parietally. Also replicating much past research (e.g., Kopp et al., 1996), waveforms associated with incongruent (but not congruent) flanker cues can be seen to demonstrate a clear fronto-central N2 effect, as highlighted in the shaded region of Figure 2.

To examine potential contextual modulation of the N2 flanker effect,[2] amplitude of N2 responses was analyzed in a 2 (trial *n*: congruent or incongruent flanker) x 3 (trial *n*-1: NoGo, congruent or incongruent flanker) x 5 (Electrode Group: F, FC, C, CP, or P) repeated-measures ANOVA (see Table 1). There was a significant effect of trial *n* flanker congruence, F (1, 22) = 10.58, partial η^2=.32, $p < .005$, that was moderated by Electrode Group, F (1, 22) = 4.63, partial η^2=.17, $p < .05$, reflecting the typical fronto-central distribution of the N2 flanker effect (e.g., Kopp et al., 1996). Most importantly, as illustrated in Figure 3, there was a significant interaction between Trial *n* flanker congruence and Trial *n*-1 trial type, F (2, 44) = 3.22, partial η^2=.13, $p <$ .05, which was not further moderated by Electrode Group, F (8, 176) = 1.47. On incongruent trials, N2 amplitudes were more negative when preceded by congruent trials than when preceded by incongruent trials, with this difference statistically significant at electrode groups centered at FCZ, CZ, and CPZ (ts $\geq$ 2.95, ps < .01) and of marginal significance at the electrode groups centered at FZ and PZ (ts $\geq$ 1.92, ps < .07). Similarly, N2 responses on incongruent trials were more negative when preceded by congruent trials than when preceded by NoGo trials, with this difference statistically significant at electrode groups centered at FZ, FCZ, CZ, CPZ, and PZ (ts $\geq$ 2.19, ps < .05). In contrast, on congruent trials, where no clear N2 peak emerged, amplitude during the N2 measurement window did not vary at any electrode locations as a function of whether the previous trial was a congruent trial relative to an incongruent trial (ts $\leq$ 1.53, ps > .13) or a congruent trial relative to a NoGo trial (ts $\leq$ 0.79, ps > .44).

(Lack of) Conflict Adaptation in LRP Amplitude

Amplitude of the LRP on incongruent trials were analyzed in a 2 (Measurement Window: first versus second) x 3 (trial *n*-1: NoGo, congruent or incongruent) repeated-measures ANOVA

(see Table 2). Consistent with previous evidence of LRP amplitude reversals on incongruent delayed-flanker trials (e.g., Kopp et al., 1996, Mattler, 2003), there was a significant effect of Measurement Window, F (1, 22) = 46.06, partial η^2=.68, $p < .0001$; moreover, unlike the N2 results reported above, this effect was not moderated by trial n-1 trial type, F (2, 44) = 1.15, $p > .32$. As shown in Figure 2, LRP amplitude on incongruent trials was consistently negative during the first measurement window (all ts $\geq$ 2.99, ps < .01) and consistently positive during the second measurement window (all ts $\geq$ 4.51, ps < .001), irrespective of trial n -1 trial type. We also compared LRP amplitude in the first measurement window of incongruent trials to LRP amplitude on congruent trials, in a 2 (Trial n: congruent or incongruent flanker) x 3 (Trial n-1: NoGo, congruent or incongruent flanker) ANOVA. Consistent with previous evidence that congruent and incongruent flanker stimuli prime different motor responses (e.g., Kopp et al., 1996, Mattler, 2003), there was a significant effect of trial n flanker congruence, F (1, 22) = 23.35, partial η^2=.51, $p < .0001$, which again was not moderated by trial n-1 trial type, F (2, 44) = 0.03. As shown in Figure 2, amplitude of the LRP was consistently positive during congruent trials (all ts $\geq$ 2.64, ps < .02), irrespective of trial n -1 trial type.

Discussion

Understanding adaptation to contextual demands for cognitive control requires clarifying when in the perception-action cycle resolving information-processing conflict modulates signals of the current need for cognitive control. Oriented toward the distinction between control processes transpiring before and after motor-response initiation, the present work found attenuated electrophysiological responses (reflected in N2 amplitude) and behavioral responses (reflected in response-time and accuracy interference effects) to information-processing conflict

on the second of two high-conflict events, despite erroneous motor-response preparation during those events (reflected in LRP amplitude). These findings indicate that information-processing conflict remained unresolved during the time corresponding to the first LRP measurement window of incongruent flanker trials, irrespective of whether a NoGo cue, an incongruent flanker array, or a congruent flanker array had appeared on trial *n*-1. By the time corresponding to the N2 measurement window, however, the reduced-amplitude N2 responses observed on incongruent flanker trials following NoGo and incongruent flanker (relative to congruent flanker) trials indicate detection of a reduction in information-processing conflict, given extensive previous evidence (reviewed above) that fronto-central N2 responses are sensitive to the amount of information-processing conflict currently encountered. These results suggest that adaptation to information-processing conflict can be accomplished through cognitive-control mechanisms operating even after erroneous responses have begun to be initiated.

By demonstrating conflict adaptation in the fronto-central N2, the present data contribute to a growing research literature examining potential contextual modulation of electrophysiological correlates of cognitive control. An earlier study in this vein varied the proportion of incongruent flankers from .2 to .5 to .8 across blocks (Bartholow et al., 2005). In that study's analysis of stimulus-locked N2 amplitude, the flanker effect at electrode FZ was most pronounced when incongruent flankers were *highly* frequent, and no impact of the frequency manipulation emerged at electrode CZ, findings which may appear at odds with the present trial-specific results and with evidence from previous block-design studies in which infrequently encountered stimulus categories elicit enhanced fronto-central N2 effects in tasks involving cognitive control (Nieuwenhuis et al., 2003) and categorization more generally (for review, see Folstein & Van Petten, 2008). Because they resulted from re-analysis of data from an

experiment assessing the effects of alcohol consumption on cognition (Bartholow et al, 2003), however, the fronto-central N2 data reported in Bartholow et al. (2005) may partly reflect processes specific to the relationship between alcohol consumption and cognitive control. Consistent with this possibility, as reported in the initial description of those data, alcohol dosage significantly impacted the amplitude of the stimulus-locked fronto-central N2 and the amplitude of a response-locked fronto-central negativity observed on error trials (see Figures 2 and 5, respectively, in Bartholow et al., 2003). For this reason, and because they resulted from a block design that did not allow eliminating exact stimulus repetitions across trials (cf. Mayr et al. 2003), the N2 findings from Bartholow et al (2005) may reflect quite different processes from those studied here. Accordingly, further investigation of interactions among alcohol consumption, repetition priming, and cognitive control may prove a fertile area for future research.

As noted above, in the most closely related precursor of the present study of which we are aware, when eliminating from analysis any exact stimulus repetitions, an earlier report found no impact of trial *n*-1 flanker congruence on trial *n* flanker effects, as reflected either in response-time interference or N2 amplitude (Wendt et al., 2007). The differing results between that study and the present one may reflect their differing methods, in that the current investigation, but not the previous one, combined brief stimulus presentations with variable durations between trials and staggered presentation of flanking and central stimuli, which collectively can be hypothesized to minimize stimulus-specific priming effects and maximize general information-processing conflict, thus potentially increasing the likelihood of observing conflict-adaptation effects. Consistent with this possibility, the amount of information-processing conflict (as reflected in response-time differences on incongruent relative to congruent flanker

trials) observed in the earlier report (Wendt et al., 2007) was about a third the size of that observed in an otherwise quite similar study that used staggered flanker-stimulus presentation and variable intervals between trials (Van Veen & Carter, 2002). It also is important to note that conflict-adaptation studies, even those using relatively impoverished stimulus sets, can differ in many other ways (e.g., in whether or not fixation cues and/or error signals are presented) that could impact research participants' task engagement and strategic orientations, thereby potentially contributing to heterogeneity in conflict-adaptation findings across studies (cf. Mayr et al., 2003; Nieuwenhuis et al., 2005; Ullsperger et al., 2005). Finally, the use of stimuli that map meaningfully rather than arbitrarily onto responses (e.g., responding with a left button-press to "><><>" rather than to "HHSHH," respectively), also may increase the propensity of delayed-flanker studies, such as the present one, to facilitate erroneous responses and thereby amplify information-processing conflict. In summary, because they inherently reflect fluctuations in the exertion of cognitive control across time, conflict-adaptation effects may depend to a significant degree on methodological factors that affect the intensity of information-processing conflict encountered across time, as well as the intensity of research participants' task engagement across time.

The present findings also speak to the nature of cognitive-control adjustments that follow conflict detection. Computational work on a range of phenomena, including Stroop-task, flanker-task, and post-error performance, implicates reduced response priming on trial *n* as a primary means by which cognitive control is implemented following conflict detection on trial *n*-1 (Botvinick et al., 2001). Regarding the presently studied flanker task in particular, an enduring assumption has been that contextual adjustments in cognitive control predominantly entail biasing attention toward the central stimulus and away from flanking stimuli, thereby decreasing

the propensity of flanking stimuli to activate a potentially erroneous response (Gratton et al., 1992; Botvinick et al., 2001; Scerif et al., 2006). Whereas reductions in response priming traditionally have been assessed via response latency (e.g., Gratton et al., 1992), however, the currently measured LRPs, with two separately observable peaks on incongruent trials, provide measures of response priming as trials unfold. As reported herein, LRP amplitude during the first measurement window of incongruent trials was substantial across all levels of trial *n*-1, indicating that erroneous response priming in this experiment was not reduced appreciably as a function of recently encountering information-processing conflict.

These results thus highlight the role in conflict adaptation of relatively later control mechanisms, particularly those entailing suppressing or forestalling already-primed responses, which past work suggests should be indexed by fronto-central N2 amplitude (e.g., Falkenstein et al., 1999; Kopp et al., 1996; Nieuwenhuis et al., 2003). Although selecting from among action alternatives ultimately yields categorical decisions (e.g., Nosofsky & Palmeri, 1997), evidence from the current and previous LRP studies shows that response preparation begins before reaching a decision threshold. Particularly when encountering conflicting information, then, incipient motor responses will need to be forestalled until a decision is reached. In the present research, cognitive-control processes acting to suppress these initial responses to flanking stimuli, by supporting the same imperative later signaled by a central cue incongruent with its flankers, may have reduced the amount of information-processing conflict incongruent flanker arrays ultimately elicited, thereby reducing the activation level of the ACC.

Future research might further explore this issue by combining with cognitive-control tasks general manipulations of response suppression, such as by interspersing among flanker

trials "wait" cues to respond immediately or only after some delay (cf. Swainson, Cunnington, Jackson, & Rorden, 2003). Given our attribution of the present results to conflict adaptation despite obligatory erroneous response priming, it will be interesting to test whether incongruent flanker trials preceded by "wait" cues elicit attenuated N2 effects, but equally substantial LRP effects, relative to incongruent flanker trials preceded by "respond immediately" cues. Future research also will need to use additional imaging methods, such as fMRI, to substantiate the hypothesized involvement in these phenomena of particular brain structures, such as the ACC and the primary motor cortex. Such work also could help address an important limitation of the present work. Because they are computed as difference waveforms, the LRPs presented here provide evidence pertaining only to the *relative* preparation of target-cued versus flanker-cued motor responses. Independently assessing impacts of conflict detection on preparation of task-relevant and task-irrelevant responses could be accomplished through independent measurement of variability in hemodynamic activity in primary motor cortex contralateral and ipsilateral to left- and right-handed task performance across time (cf. Dehaene et al., 1998).

In summary, while providing the first evidence of which we are aware of conflict adaptation in an ERP component widely established to reflect the extent of information-processing conflict currently encountered (the fronto-central N2), this investigation helped clarify when in the perception-action cycle this adaptation arises, implicating a monitoring process sensitive to fluctuations in conflict not only across but also within events. These findings suggest that engaging cognitive-control mechanisms oriented toward not only *preventing* (e.g., Allport, 1980) but also *addressing* information-processing conflict once it has arisen (e.g., Burgess & Shallice, 1993) can modulate the amount of conflict elicited by stimuli subsequently encountered, thereby impacting the likelihood of engaging cognitive control anew.

References

Allport, A. (1980). Patterns and actions. In G. Claxton (Ed.), *Cognitive psychology: New directions* (pp. 26–64). London: Routledge & Kegan Paul.

Azizian, A., Freitas, A. L., Parvaz, M., & Squires, N.K. (2006). Beware misleading cues: Perceptual overlap modulates the N2/P3 complex. *Psychophysiology, 253 - 260*.

Badre, D. (2008). Cognitive control, hierarchy, and the rostro–caudal organization of the frontal lobes. *Trends in Cognitive Sciences, 12*, 193-200.

Botvinick, M.M., Braver, T.S., Barch, D.M., Carter, C.S., & Cohen, J.D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108,* 624-652.

Botvinick, M.M. (2008). Hierarchical models of behavior and prefrontal function. *Trends in Cognitive Sciences, 12*, 201-208.

Burgess, P. W., & Shallice, T. (1996). Response suppression, initiation and strategy use following frontal lobe lesions. *Neuropsychologia*, *34*, 263–272.

Coles, M. G. H. (1989). Modern mind-brain reading: Psychophysiology, physiology, and cognition. *Psychophysiology*, *26*, 251–269.

Dehaene, S., Naccache, L., Le Clec'H, G., Koechlin, E., Mueller, M., & Dehaene-Lambertz, G. (1998, October8). Imaging unconscious semantic priming. *Nature*, *395*, 597–600.

Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics. *Journal of Neuroscience Methods, 134*, 9-21.

Egner, T., & Hirsch, J. (2005a). The neural correlates and functional integration of cognitive control in a Stroop task. *NeuroImage, 24*, 539 – 547.

Egner, T., & Hirsch, J. (2005b). Cognitive control mechanisms resolve conflict through cortical amplification of task-relevant information. *Nature Neuroscience*, *8*, 1784–1790.

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (1999). ERP components in go/nogo tasks and their relation to inhibition. *Acta Psychologica*, *101*, 267–291.

Fallgatter, A.J. & Strik, W.K. (1999). The NoGo-anteriorization as a neurophysiological standard-index for cognitive response control. *International Journal of Psychophysiology,* 32, 233–238.

Folstein, J. R. & Van Petten, C. V. (2004). Multidimensional rule, unidimensional rule, and similarity strategies in categorization: Event-related brain potential correlates. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *30*, 1026-1044.

Folstein, J.R., & Van Petten, C. (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: A review. *Psychophysiology, 45*, 152-170.

Freitas, A.L., Azizian, A., Leung, H.-C., & Squires, N.K. (2007). Resisting recently acted-on cues: Compatibility of Go/No-Go responses to response history modulates (anterior P3) event-related potentials. *Psychophysiology, 44*, 2 – 10.

Freitas, A.L., Bahar, M., Yang, S ., & Banai, R . (2007). Contextual adjustments in cognitive control across tasks. *Psychological Science, 18*, 1040 – 1043.

Fuster, J.M. (2004) Upper processing stages of the perception-action cycle. *Trends in Cognitive Sciences, 8,* 143–145.

Gladwin, T.E., 't Hart, B.M., & De Jong, R (2008). Dissociations between motor-related EEG measures in a cued movement sequence task, *Cortex 44*, 521–536.

Gratton, G., Coles, M.G.H., & Donchin, E. (1992). Optimizing the use of information: Strategic control of activation of responses. *Journal of Experimental Psychology: General, 121,* 480-506.

Greenhouse, W.W., & Geisser, S. (1959). On methods in the analysis of profile data. *Psychometrika*, 24, 95-112.

Kerns, J.G., Cohen, J.D., MacDonald, A.W., Cho, R.Y., Stenger, V.A., & Carter, C.S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science 303*, 1023–1026.

Koechlin, E., & Summerfield, C. (2007). An information theoretical approach to prefrontal executive function. *Trends in Cognitive Sciences, 11*, 229–235.

Kopp, B., Mattler, U., Goertz, R., & Rist, F. (1996). N2, P3, and the lateralized readiness potential in a nogo task involving selective response priming. *Electroencephalography Clinical Neurophysiology*, 99, 19 – 27.

Kutas, M., & Donchin, E. (1980). Preparation to respond as manifested by movement-related brain potentials. *Brain Research*, *202*, 95–115.

Leuthold, H., & Kopp, B. (1998). Mechanisms of priming by masked stimuli: Inferences from event-related brain potentials. *Psychological Science, 9*, 263–269.

Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological Science*, *8*, 60–64.

Mattler, U. (2003). Delayed flanker effects on lateralized readiness potentials. *Experimental Brain Research*, *151*, 272–288.

Mayr, U., Awh, E., & Laurey, P. (2003). Conflict adaptation effects in the absence of executive control. *Nature Neuroscience, 6,* 450-452.

Nieuwenhuis, S., Yeung, N., Cohen, J. D. (2004). Stimulus modality, perceptual overlap, and the go/no-go N2. *Psychophysiology, 41*, 157-60.

Nieuwenhuis, S., Yeung, N., van den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a Go/NoGo task: Effects of response conflict and trial-type frequency. *Cognitive, Affective, and Behavioral Neuroscience*, *3*, 17–26.

Nosofsky, R. M., Palmeri, T. J. (1997). An exemplar-based random walk model of speeded classification. *Psychological Review, 104*, 266-300.

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S.(2004). The role of the medial frontal cortex in cognitive control. *Science, 306*, 443–447.

Salisbury D.F., Griggs C.B., Shenton M.E., & McCarley, R.W. (2004). The NoGo P300 'anteriorization' effect and response inhibition. *Clinical Neurophysiology, 115*, 1550-1558.

Scerif, G., Worden, M. S., Davidson, M., Seiger, L., & Casey, B. J. (2006). Context modulates early stimulus processing when resolving stimulus–response conflict. *Journal of Cognitive Neuroscience, 18*, 781-792.

Swainson, R., Cunnington, R., Jackson, G.M., Rorden, C., Peters, A.M., Morris, P.G., & Jackson, S.R. (2003). Cognitive control mechanisms revealed by ERP and fMRI: Evidence from repeated task-switching. *Journal of Cognitive Neuroscience, 15*, 785–799.

Töllner, T., Gramann, K., Müller, H. J., Kiss, M., & Eimer, M. (2008). Electrophysiological markers of visual dimension changes and response changes. *Journal of Experimental Psychology: Human Perception and Performance, 34*, 531-542.

Ullsperger, M., Bylsma, L.M., Botvinick, & M. (2005). The conflict-adaptation effect: It's not just priming. *Cognitive, Affective, and Behavioral Neuroscience, 5,* 467-472.

Van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience*, *14*, 593–602.

Wendt, M., Heldmann, M., Munte, T. F., and Kluwe, R. H. (2007). Disentangling sequential effects of stimulus- and response-related conflict and stimulus-response repetition using brain potentials. *Journal of Cognitive Neuroscience, 19*, 1104–1112.

Authors' Note

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Footnotes

1. Because the difference in fronto-central N2 amplitude on incongruent relative to congruent flanker trials was clearly apparent through visual inspection of the vast majority (20/23) of individual participants' trial *n*-averaged waveforms (collapsed across trial *n*-1), we also were able to define the N2 ideographically, as mean amplitude between the two time points at which the two waveforms intersected (as the waveform associated with incongruent flanker presentation fell and then rose in the N2 response) at electrode FCZ for each individual subject. In analyses of N2 amplitude using these ideographically determined measurement windows (with the grand-average-based measurement window used for the remaining 3 participants), all N2 conflict-adaptation effects reported in this paper increased slightly in effect size and in statistical significance. For example, the interaction between trial *n* flanker congruence and trial *n*-1 trial type increased slightly to F (2, 44) = 4.30, partial η^2=.16, $p < .05$. The most substantial difference between analyses using the grand-average-based and ideographically determined N2 measurement windows was that the latter yielded a considerably larger main effect of flanker congruence, F (2, 44) = 19.20, partial η^2=.47, $p < .0002$. This increase is not surprising, given that the ideographically determined measurement windows were tailored to the particular time points when the incongruent-flanker N2 response occurred for individual subjects. Cumulatively, these ancillary analyses provide additional evidence of contextual modulation of the N2, in that the conflict-adaptation effect was significant under conditions shown to be optimally sensitive to the presence of that which was hypothesized to be modulated (the N2 flanker effect).

2. As reflected in NoGo cues' relatively infrequent (.2) presentation in this study, examining all possible impacts of trial *n*-1 on trial *n* NoGo responses was not an aim of the

present design (i.e., only .2 × .2 = .04 of trial pairs comprised consecutive NoGo cues).

Table 1. Average N2 amplitude (standard error of the mean in parentheses) as a function of trial *n* congruence and trial *n*-1 trial type, at electrode groupings centered at FZ (frontal), FCZ (fronto-central), CZ (central), CPZ (centro-parietal), and PZ (parietal) electrode sites.

	Incongruent trial *n*				Congruent trial *n*		
	Congruent	Incongruent	NoGo	←trial *n*-1→	Congruent	Incongruent	NoGo
Frontal	1.52	2.23	2.23		2.42	2.94	2.25
	(0.74)	(0.86)	(0.75)		(0.87)	(0.87)	(0.86)
Fronto-	2.38	3.42	3.27		4.01	4.40	3.93
Central	(0.85)	(0.93)	(0.90)		(1.00)	(0.99)	(0.98)
Central	4.53	5.64	5.57		6.72	6.79	6.41
	(0.80)	(0.89)	(0.89)		(1.01)	(0.98)	(1.01)
Centro-	5.90	7.05	7.19		8.20	8.07	7.96
Parietal	(0.76)	(0.85)	(0.86)		(0.96)	(0.92)	(0.97)
Parietal	5.35	6.01	6.38		7.29	6.96	7.24
	(0.65)	(0.69)	(0.73)		(0.87)	(0.76)	(0.81)

Table 2. Average stimulus-locked lateralized readiness potential amplitudes (standard error of the mean in parentheses) as a function of trial *n* flanker congruence and trial *n*-1 trial type, during a single measurement window for congruent trials (between 154 and 354 msec following presentation of central response cue) and during two separate measurement windows for incongruent trials (between 144 and 282 and between 282 and 500 msec, respectively, following presentation of central response cue).

	Incongruent trial *n*				Congruent trial *n*		
	Congruent	Incongruent	NoGo	←trial *n*-1→	Congruent	Incongruent	NoGo
First	-0.54	-0.67	-0.73		0.55	0.75	0.79
Window	(0.16)	(0.20)	(0.25)		(0.21)	(0.19)	(0.23)
Second	1.48	1.32	1.09				
Window	(0.28)	(0.29)	(0.24)				

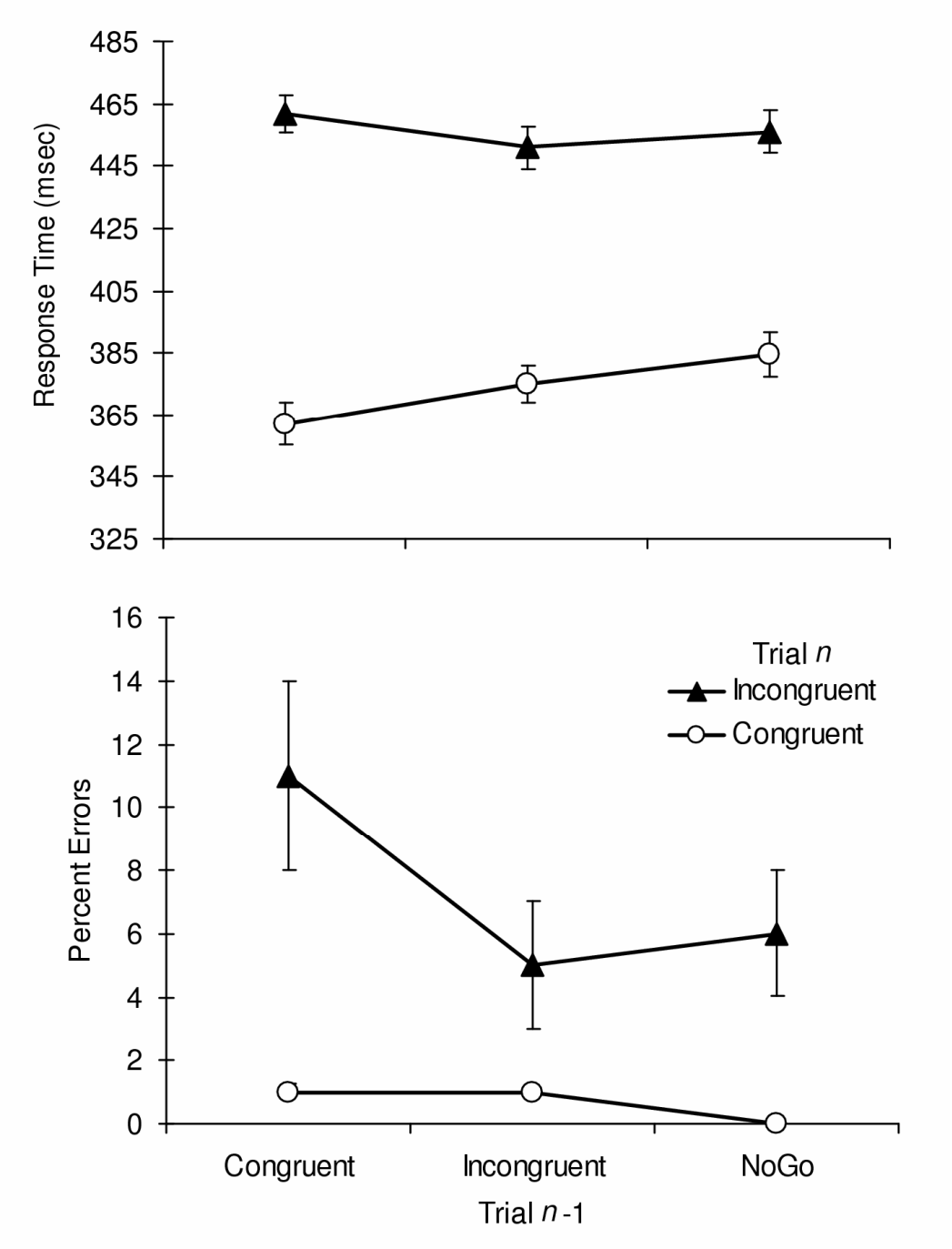
Figure Captions

Figure 1. Response times (top panel) and error rates (bottom panel) on congruent and incongruent flanker trials, reported as a function of whether they followed trial *n*-1 presentation of congruent or incongruent flankers or NoGo cues. Error bars indicate one standard error of the mean (*SEM*; the lack of error bars on congruent-flanker error rates indicates $SEM < 0.25\%$).

Figure 2. Grand-average ERP waveforms at electrodes FCZ and CPZ, time-locked to presentation of the central response cue, with approximate onset of 93.33 msec presentation of lateral flanker stimuli (denoted "F") indicated at -94 msec. Waveforms are averaged on the basis of trial *n* (collapsed across trial *n*-1) trial type (black lines = incongruent flanker; gray lines = congruent flanker; dotted lines = NoGo). The shaded area indicates the N2 measurement window used to test moderation of the flanker N2 effect by trial *n*-1 trial type.

Figure 3. Grand-average ERP waveforms at electrode FCZ, time-locked to presentation of the central response cue, with approximate onset of 93.33 msec presentation of lateral flanker stimuli (denoted "F") indicated at -94 msec. Waveforms are averaged as a function of current flanker congruence (black lines = trial *n* incongruent; gray lines = trial *n* congruent) and previous trial type (dashed lines = trial *n*-1 incongruent; solid lines = trial *n*-1 congruent; dotted lines = trial *n*-1 NoGo), with the arrow in the figure highlighting the prominent N2 response observed on incongruent trials following congruent trials. Voltage maps indicate topographical distribution of amplitude differences between trial *n* incongruent flanker trials that follow trial *n*-1 congruent minus incongruent trials (left inset with dashed border) and between trial *n* incongruent flanker trials that follow trial *n*-1 congruent minus NoGo trials (right inset with dotted border), at 320 msec.

Figure 4. Grand-average lateralized readiness potential waveforms, time-locked to presentation of the central response cue, with approximate onset of 93.33 msec presentation of lateral flanker stimuli (denoted “F”) indicated at -94 msec. Waveforms are averaged as a function of current flanker congruence (black lines = trial *n* incongruent; gray lines = trial *n* congruent) and previous trial type (dashed lines = trial *n*-1 incongruent; solid lines = trial *n*-1 congruent; dotted lines = trial *n*-1 NoGo). These grand-average waveforms were filtered digitally with a low band pass (30 Hz cutoff, 12 db/oct).


Response Time (msec)
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445
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Percent Errors
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Trial n
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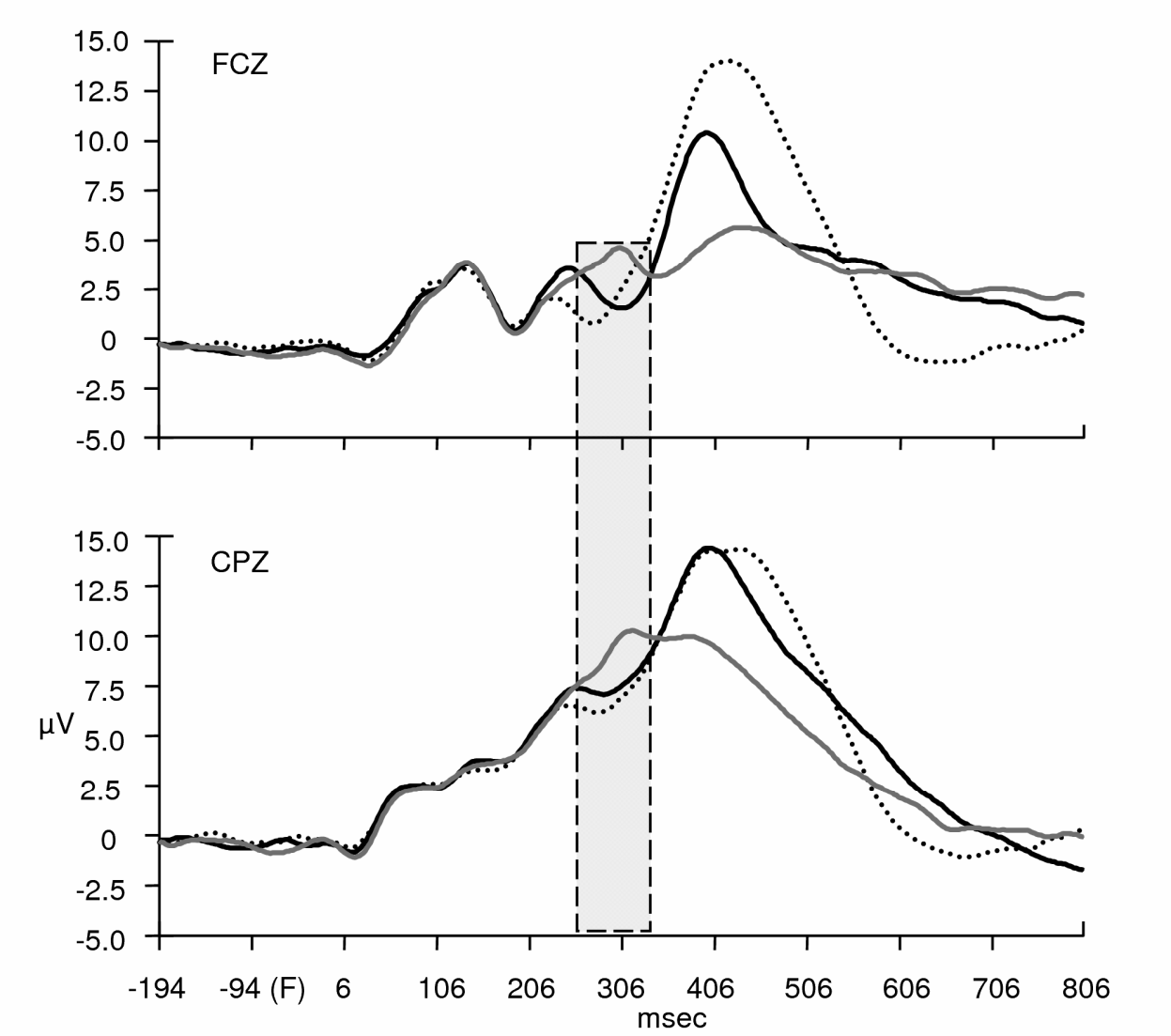
Congruent
Congruent
Incongruent
NoGo
Trial n -1


FCZ
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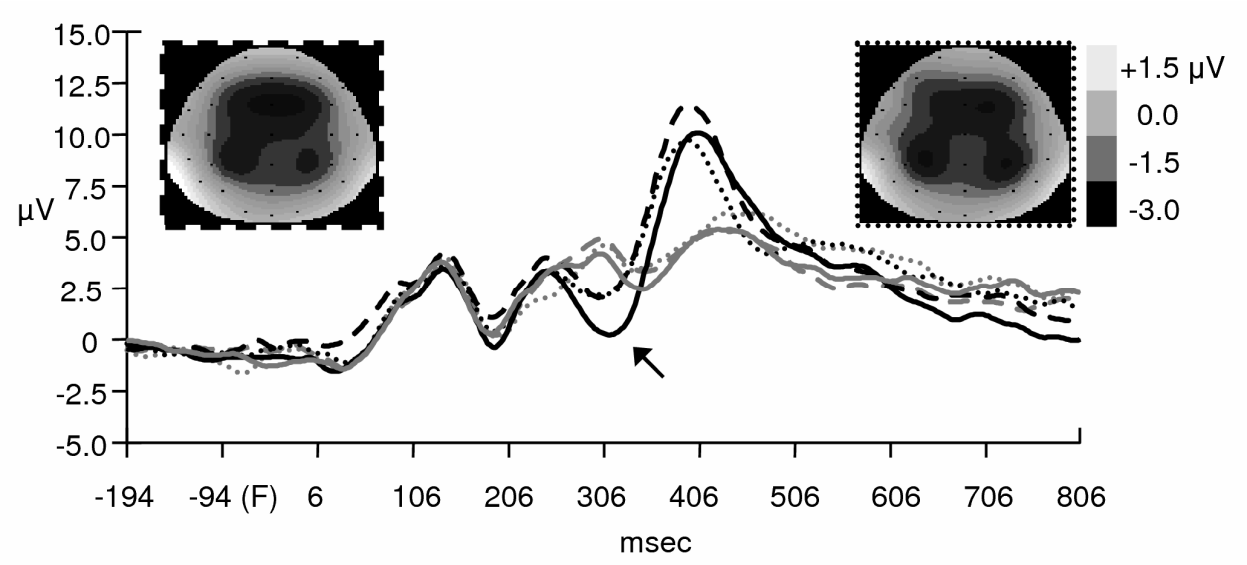
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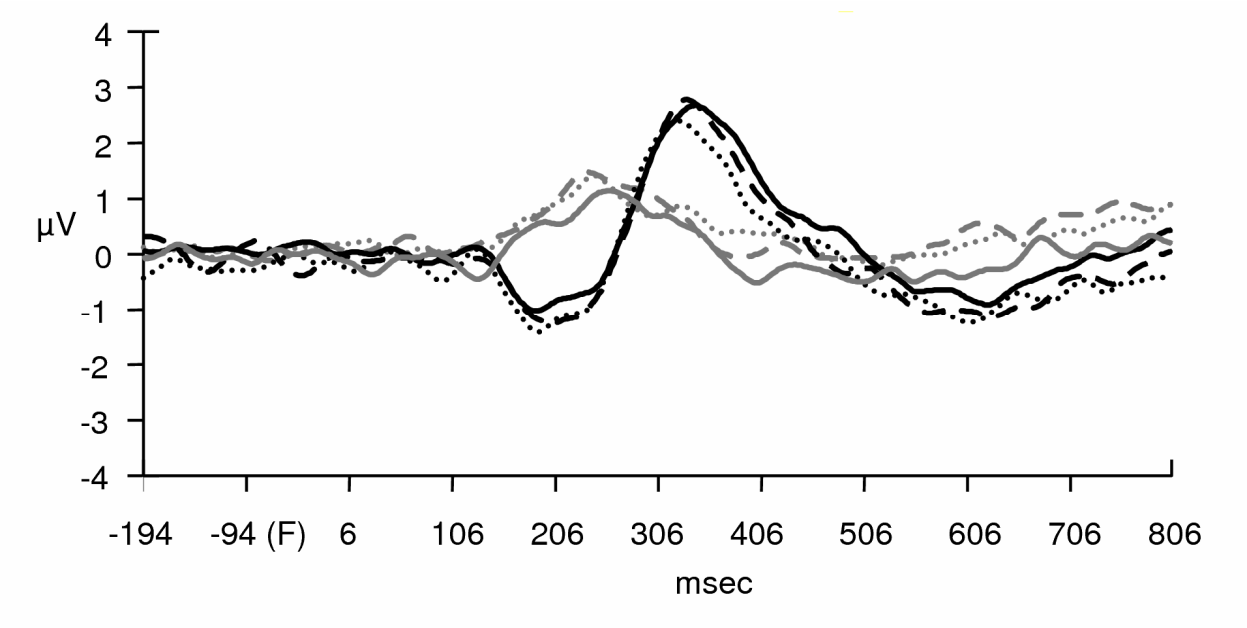
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