Sequence effects in cued task-switching modulate response preparedness and repetition priming processes.

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Abstract

In task-switching paradigms, reaction time (RT) switch cost is eliminated on trials after a no-go trial (no-go/go sequence effect). We examined the locus of no-go interference on task-switching performance by comparing the ERP timecourse of go/go and no-go/go sequences from cue onset to response execution. We also examined whether non-informative trials (i.e., delayed reconfiguration, no response inhibition) produce similar sequence effects. Participants switched using informative and non-informative cues (Expt.2) intermixed with no-go trials (Expt.1). Repeat RT was slower for both no-go/informative (pNG/I) and non-informative/informative (pNI/I) than informative/informative sequences. ERPs linked to anticipatory preparation showed no effect of trial sequence. ERPs indicated that pNG/I sequences reduce response readiness whereas pNI/I sequences reduce repetition benefit for repeat trials. Implications for task-switching models are discussed.

Task-switching paradigms require rapid alternation between simple tasks that are defined on the basis of distinct or partially overlapping stimulus–response (S-R) contingencies or *task-sets* (ie., letter: vowel or consonant; number: odd or even). The task active on any particular trial is defined either by a cue presented prior to each trial (*cued trials paradigm*; Meiran, 1996) or by a predictable repeating task sequence (ie., AABB; *alternating runs paradigm*; Rogers & Monsell, 1995). Typically, reaction time (RT) is longer on switch as compared to repeat trials within a switch block (*RT switch cost*). Task-switching paradigms manipulate the *preparation interval* to examine changes in RT switch cost. In cued trials paradigms, this corresponds to the interval between the informative cue and the stimulus (ie., cue-to-stimulus interval, CSI) whereas in alternative runs paradigms, it corresponds to the interval between the response to the preceding stimulus and onset of the next stimulus (ie., response-to-stimulus interval, RSI). Increasing the preparation interval from 150 to 600 ms significantly reduces RT switch cost (e.g. Karayanidis, Coltheart, Michie & Murphy, 2003; Meiran, 2000; Nicholson, Karayanidis, Poboka, Heathcote & Michie, 2005; Rogers & Monsell, 1995). However, a significant '*residual*' RT switch cost remains even at preparation intervals exceeding 1000ms (e.g., Rogers & Monsell, 1995; Karayanidis et al., 2003; Meiran, 2000; Nicholson et al., 2005).

Theoretical models differ in the degree to which they invoke active control processes or passive stimulus-triggered interference processes to account for RT switch cost. Rogers and Monsell (1995) argue that switching between tasks involves an obligatory process of *task-set reconfiguration* that entails shifting from a readiness to perform one task to a readiness to perform a different task. The reduction in RT switch cost with increasing preparation interval is said to reflect an endogenous component of task-set reconfiguration, an anticipatory process that can be voluntarily activated and, given information about the upcoming task and an adequate preparation interval, can be initiated prior to stimulus onset. Although the reduction in switch cost with increasing RSI or CSI has been extensively replicated and most authors concede that there is some type of preparation process, the nature of this preparation process is still an issue of contention (e.g. Arrington, Logan & Schneider, 2007; Allport & Wylie, 2000; Karayanidis et al., 2003; Koch & Allport, 2006; Koch & Philipp, 2005; Monsell, 2003; Nicholson et al., 2005; Rubinstein,

Meyer & Evans, 2001; Sinai, Goffaux & Phillips, 2007; Schuch & Koch, 2003; Waszak, Hommel & Allport, 2003; Wylie & Allport, 2000).

A number of competing models have been developed to explain the residual switch cost. Rogers and Monsell (1995) attribute the residual switch cost to a second 'stimulus-triggered' component of the task-set reconfiguration process, an exogenous control process that cannot be initiated until stimulus onset. De Jong (2000) suggests that it is an artifact of trial by trial variation in preparation. Other models argue that the residual switch cost can be accounted for by stimulus-response (S-R) interference processes resulting from persistent activation of the previously active task-set and persistent inhibition of the currently active task-set (e.g. Allport, Styles & Hseih, 1994; Mayr & Keele, 2000; Wylie & Allport, 2000). In these models, the level of activation and/or inhibition of task-sets passively dissipates over time and carries over into the following trial. On repeat trials, persistent activation benefits task performance. However on switch trials, persistent activation and inhibition results in interference between task-sets, resulting in longer RT. Residual switch cost is therefore due to the combined effects of increased interference between task-sets on switch trials and facilitation of task-set activation on repeat trials. Other models invoke both active anticipatory and passive interference-related processes (e.g. Meiran, 2000) since increasing preparation interval and increasing time for passive dissipation of interference both reduce RT switch cost (Meiran, 2000; Nicholson et al., 2005).

Task-set reconfiguration may be conceptualized as a set of processes that are necessary only on switch trials (Rogers & Monsell, 1995) or a set of processes that, depending on task parameters and transient fluctuations in attention, may be activated on both switch and repeat trials but that would, under most circumstances, require greater cognitive effort for switch trials (Monsell & Mizon, 2006; Nicholson et al., 2005). Rubinstein et al. (2001) propose that control processes involved in task-switching include goal shifting and rule activation. Goal shifting keeps track of the sequence of tasks (i.e. 'do the letter task') by actively inserting or deleting task goals and can occur either before or after stimulus onset depending on the context (e.g. length of preparatory interval). Rule activation is placed after stimulus identification and before response selection and is argued to load the relevant task rules and disable the now irrelevant task rules. While goal shifting and rule activation appear to loosely map onto Roger and

Monsell's anticipatory and stimulus-triggered task-set reconfiguration processes, anticipatory reconfiguration could also encompass both goal shifting and rule activation, as the cue defines the relevant task rules.

ERP indices of cognitive processes contributing to task-switching

Event-related potentials (ERP) provide a high temporal resolution index of neural processes associated with cue processing, stimulus processing and response execution and can help characterize the timeline of cognitive processes that contribute to the behavioral end-product (Rugg & Coles, 1995). Task-switching studies have shown differential modulation of ERP components for switch as compared to repeat trials. ERP components activated within the CSI are associated with cue processing and preparation processes, whereas ERP components activated after stimulus onset in long CSI conditions are associated with post-stimulus processes related to stimulus identification, decision making and execution of the response.

Within the preparation interval, a larger centroparietally-maximal positive component is elicited for switch relative to repeat trials (e.g. Karayanidis et al., 2003; Kieffaber & Hetrick, 2005; Miniussi, Marzi & Nobre, 2005; Nicholson et al., 2005; Nicholson, Karayanidis, Bumak, Poboka & Michie, 2006; Nicholson, Karayanidis, Davies & Michie, 2006; Poulsen, Luu, Davey & Tucker, 2005; Rushworth, Passingham & Nobre, 2002; 2005). This cue-locked *differential switch-positivity* emerges as early as 150ms after cue onset (Nicholson et al., 2005; Nicholson, Karayanidis, Davies & Michie, 2006) and varies in duration depending on task parameters. At long preparation intervals (>600ms), the switch-positivity fully resolves before stimulus onset, whereas at short preparation intervals, it peaks after stimulus onset (Karayanidis et al., 2003; Nicholson et al., 2005). When cues differing in task informativeness are employed, this component is observed only after task-relevant information becomes available (Nicholson et al., 2005; Nicholson, Karayanidis, Davies & Michie, 2006). This pattern of results suggests that the switch-positivity may be an index of anticipatory preparation, and some evidence suggests that the amplitude of the switch-positivity is related to behavioural indices of preparation (Kieffaber & Hetrick, 1995; Lavric, Mizon, & Monsell, in press; but see Swainson, Jackson & Jackson, 2006). It is important to note that a positive deflection can often be seen in both switch and repeat cue-

locked waveforms, albeit larger for switch trials (Goffaux, Phillips, Sinai & Pushkar, 2006; Karayanidis et al., 2003; Kieffaber & Hetrick, 2005; Nicholson et al., 2005), suggesting that the processes associated with this positivity may be activated for both trial types albeit to a different degree[1]. Although other electrophysiological components, i.e., frontal cue-locked switch-negativity, have also been found to vary within the CSI as a function of task-switching, they are not observed as consistently (e.g. Astle, Jackson & Swainson, 2006; Miniussi et al., 2005; Poulsen et al., 2005; Rushworth et al., 2002; 2005; Swainson, Cunnington, Jackson, Rorden, Peters, et al., 2003; Swainson et al., 2006).

Stimulus-locked switch trials show a *smaller* positivity than repeat trials especially over the central and parietal scalp (e.g, Karayanidis et al., 2003; Kieffaber & Hetrick, 2005; Miniussi et al., 2005; Nicholson et al., 2005; Poulsen et al., 2005) We will refer to this as the stimulus-locked *differential switch-negativity*. When preceded by a long CSI, the differential switch-negativity emerges as early as 150ms and peaks around 400-800ms after stimulus onset (e.g., Karayanidis et al., 2003; Nicholson et al., 2005). When there is little or no preparation interval (Karayanidis et al., 2003; Nicholson et al., 2005), the switch-negativity is delayed by more than 300ms and is often preceded by a differential switch-positivity. This suggests that the processes indexed by this switch-negativity are not initiated until the processes indexed by the preceding switch-positivity are initiated or completed. The differential switch-negativity is likely to reflect processes affected by greater difficulty of rule implementation and more S-R interference for switch as compared to repeat trials (Nicholson et al., 2005). The finding that the differential switch-negativity is smaller for stimulus sets that are specific to a single task (univalent set) as compared to

[1] Although this larger centro-parietal positivity for switch as compared to repeat trials has been consistently found, it has been inconsistently labelled in existing task-switching literature. Some authors refer to it as an increased P3b (Goffaux et al., 2006; Kieffaber & Hetrick, 2005; Poulsen et al., 2005), others as a parietal switch-positivity (Astle et al. 2006; Karayanidis et al., 2003; Nicholson et al., 2005; Nicholson et al., 2006a;b; Swainson et al., 2006). Similar issues plague the stimulus-locked ERP waveforms comparing switch and repeat trials and the labelling or definition of the relative reduction in positivity for switch as compared to repeat trials. Given that the identity, number, and cortical source of ERP components involved in task-switching and the underlying cognitive processes are still being defined, we believe that there is no advantage to using existing ERP component labels to identify these effects until such correspondence has been empirically established. Therefore the term 'cue-locked positivity' is used to define the P3b-like component seen in cue-locked waveforms for both switch and repeat trials and the term 'differential switch-positivity' is used for the relative increase in this positivity for cue-locked switch trials that is emphasized in switch-repeat difference waveforms. These terms are merely descriptive and provide bookmarks for phenomena that still need to be defined.

bivalent sets that possess features relevant for both tasks (Karayanidis et al., 2003; Poulsen et al., 2005) is consistent with the above interpretation.

In summary, ERPs support multi-component models of task-switching. The differential switch-positivity is consistent with anticipatory task-set reconfiguration processes that are triggered when information about an impending change in task-set becomes available. The differential switch-negativity is consistent with post-stimulus processes that emerge after task-set reconfiguration and may be affected by stimulus-triggered interference.

Sequence effects in task-switching

Within any task context, performance on the current trial is affected not only by current stimulus and response parameters, but also by the parameters and outcomes of preceding trials. These sequence effects may reflect passive processes associated with the carry-over of recent stimulus and response activations as well as active processes involved in overcoming any interference from such carry-over effects. Sequence effects are an inherent component of task-switching performance. For tasks *A* and *B*, a switch trial is defined as a *BA* or *AB* trial sequence whereas a repeat trial is an *AA* or *BB* trial sequence. Sequence effects also are also an important parameter in theoretical accounts of task-switching phenomena. For example, the task-set inertia hypothesis attributes switch cost partly to interference arising from carryover of task-set activation and/or inhibition from the preceding trial (Allport et al., 1994). In a similar vein, the finding that RT shows little decline over successive repeat trials has been used as evidence for the all-or-none nature of task-set reconfiguration (Rogers &Monsell, 1995).

Sequence effects that persist over more than two consecutive trials have been used to help define the processes that result in differential modulation of RT on switch vs. repeat trials. Backward inhibition – the finding that RT is faster on the third trial in a *CBA* sequence as compared to an *ABA* sequence (Mayr & Keele, 2000; Schuch & Koch, 2003) - indicates persistent interference resulting from having shifted away from task *A* two trials back. This suggests that inhibition of the previously active task-set has lasting effects on the processing of subsequent trials. Depending on the locus and character of this inhibition, this finding can have different implications for models of task-switching. Schuch and Koch (2003) included no-go trials in a cued-trials task-switching paradigm to examine whether inhibition of the previously

active task-set operates at the level of reconfiguration or response selection. As no-go trials were identical to go trials in the cue-stimulus interval, they evoked the same preparation processes. However, on no-go trials, the imperative stimulus was accompanied by a no-go tone and required either no response or an indiscriminate response (e.g., press both keys simultaneously). Schuch and Koch found that go switch trials that followed a no-go trial (*no-go/go sequence*) showed no RT switch cost and no backward inhibition effect and concluded that inhibition of the previously active task-set does not interfere with processes occurring during the preparation interval. They argued that, on go/go switch sequences, there is persistent interference between competing task-sets. This is resolved only when the relevant category-response (C-R) rule is implemented and response selection occurs, resulting in inhibition of the previous task-set. As no-go trials do not involve response selection, the previously active C-R rule is not inhibited. Consequently, on the following go switch trial, there is no inhibition carried over from the preceding trial, and therefore no switch cost. The proposal that inhibition of the previously active task-set occurs at the level of response selection is supported by behavioral data showing no preparation benefit for semi-specific cues which indicate that a task will not be repeated (Dreisbach, Haider & Kluwe, 2002; Nicholson, Karayanidis, Davies & Michie, 2006).

In contrast, ERP findings suggest that inhibition of the irrelevant task-set may occur during the preparation period (Nicholson, Karayanidis, Davies & Michie, 2006; Sinai et al., 2007). The discrepancy between the conclusions drawn from behavioral and ERP data may reflect the fact that RT is the cumulative end-product of a number of partially overlapping processes. Karayanidis, Mansfield, Galloway, Smith, Provost and Heathcote (in revision) found that partially informative cues that signaled that a switch in task was required but did not specify the task to switch to produced a behavioral benefit relative to non-informative cues on the non-decision component of RT. This was not reflected in mean RT because partially informative and non-informative cues also differed in speed-accuracy tradeoff. Note also that Schuch and Koch's (2003) behavioral results do not unambiguously support the conclusion that inhibition of the previously active task-set is a by-product of response selection. If abolition of residual switch cost in no-go/go sequences is due to the absence of response selection on the preceding no-go trial, this effect should be attributable to the reduction in switch trial RT. Yet, the reduction in RT switch cost

occurred because repeat trial RT was significantly longer on no-go/go sequences as compared to go/go sequences (Schuch & Koch, 2003). Koch and Philipp (2005) proposed instead that response selection triggers an activation bias that favors task repetition. This activation bias dissipates quickly and needs to be refreshed on subsequent trials. Hence, if response selection does not occur on the following trial - as in the case of a no-go trial - there is no activation bias in favor of the current task, i.e., no repetition benefit on the subsequent trial, and resulting in longer repeat trial RT and no residual switch cost.

Despite different accounts for the no-go/go sequence effects on RT switch cost, Schuch and Koch (2003) and Koch and Philipp (2005) both argue that the effect is not due to inhibition of the prepared task-set but to the absence of response selection on no-go trials. However, Astle et al. (2006) showed that no-go/go sequences differed from go/go sequences in both cue- and stimulus-locked ERP components, suggesting that sequence effects affect both preparation to switch and implementation of a switch.

Furthermore, the contention that no-go trials do not involve response selection has not been empirically tested. In most experiments presented by Koch and colleagues, the no-go signal was a tone that accompanied a valid task stimulus. Since the stimulus was mapped to a response, it is possible that response selection occurred on some, if not all, no-go trials. A notable exception was Schuch and Koch's Experiment 1B which used a neutral visual no-go stimulus that was not mapped to a response and found that the pattern of sequence effects on RT switch cost was very similar to that with validly mapped no-go stimuli (see also Astle et al., 2006). However, when compared to unmapped no-go/go sequences (Experiment 1B), mapped sequences (e.g., Experiment 1A) had slower RT overall and disproportionately so at the long, presumably prepared, CSI (an increase in RT of 100-150ms). This overall increase in RT on no-go/go trials could reflect a change in response criterion resulting from the need to withhold a response on a certain proportion of trials (Astle et al., 2006; Ivanoff & Klein, 2001). The effect of suppressing a prepared response set would be larger on trials that involve greater response preparation, that is, trials with a long CSI. Indeed, Koch and Philipp (2005) note that their results do not rule out the possibility that no-go trials involve the active suppression of a motor response that would have been correct if executed in a go trial. Thus, the question remains whether no-go trials eliminate RT switch cost on the following go trial because they don't result in response selection (and consequently, do not trigger

inhibition of the previously active task rules) or through some inhibitory mechanism, such as inhibition of the prepared task-set or motor response.

Koch and colleagues also argue that the finding that no-go/go sequences eliminate switch cost for both long and short CSI conditions suggests that the effect is independent of preparation (Koch & Philipp, 2005; Schuch & Koch, 2003). Yet, Astle et al. (2006) argue that sequence effects can affect preparation processes, as indicated by the finding that at least some cue-locked ERP components are differentially modulated for switch as compared to repeat cues for go/go but not no-go/go sequences. Therefore, it remains unclear if sequence effects elicited by no-go trials occur because these no-go trials do not involve response selection (Schuch & Koch, 2003), do not trigger an activation bias for repeat trials (Koch & Philipp, 2005), disrupt preparation processes (Astle et al., 2006), and/or produce inhibition of the prepared task-set or the preparedness to respond.

In summary, although the no-go/go sequence effects clearly indicate that the preceding trial can modulate the size, or indeed the presence, of residual switch cost on the current trial, the processes that mediate these sequence effects have not been clearly defined and this has implications for the processes that underlie task-switching performance. ERPs provide detailed temporal information over the course of processing of go/go and no-go/go sequences from cue onset to response execution. By examining the timecourse of differences between go/go and no-go/go sequences, ERPs can inform us regarding the locus of interference processes arising from no-go trials.

Experiment 1

The aims of the current experiment are two-fold. Firstly, we use ERP measures associated with processing of the cue, stimulus and response to examine the locus of the interference created by no-go/go trial sequences. Secondly, we examine whether another type of trial sequence that does not involve withholding of a response also affects residual switch cost on the subsequent go trial, and whether this effect has the same locus as no-go/go sequence effects. In cued-trials paradigms with blocked long CSI, each informative cue trial involves a structured series of processes, i.e., *cue-prepare-stimulus-respond*, which is repeated throughout the block of trials. No-go trials introduce a new trial type that consists of a somewhat different series of processes i.e., *cue-prepare-stimulus-withhold response*. It is possible that the

behavioral difference between go/go and no-go/go trials occurs because the preceding trial is different to the current one rather than the absence of response selection per se. In this case, other trial types would also be expected to affect switching performance on the subsequent go trial even if they do not involve absence of response selection. In the present study, we tested this by including another trial type that involves the same processes as informative cue go trials, but differs in the order in which these processes are completed. Non-informative cues inform of an upcoming go trial but the identity of the relevant task is delayed until stimulus onset. So while informative and non-informative trials involve the same processes, the order of these processes differs because, on non-informative trials, task-set reconfiguration cannot be initiated until after stimulus onset (*cue-stimulus-prepare-respond)*.

We investigated sequence effects on the processing of both switch and repeat trials using a cued-trials paradigm with randomized presentation of three trial types. *Informative (I) trials* included an informative cue that allowed for task-specific preparation followed by a stimulus that was validly mapped to a response. *Non-informative (NI) trials* included a non-informative cue that did not carry task-specific information followed by a stimulus that indicated which task was relevant and was validly mapped to a response. *No-go (NG) trials* included an informative cue followed by a stimulus that was not mapped to any response. We compared behavioral and ERP effects on informative/informative (pI/I; note p refers to *previous trial*) trial sequences to both no-go/informative (pNG/I) and non-informative/informative (pNI/I) sequences. These comparisons targeted two types of sequence effects on informative switch and repeat trials – sequence effects arising from whether a response was (pI/I) or was not (pNG/I) selected on the previous trial as well as sequence effects arising from whether anticipatory preparation could (pI/I) or could not (pNI/I) be completed during the CSI on the previous trial. ~~The inclusion of non-informative trials also allowed us to examine whether no-go/go sequence effects are evidenced on these unprepared go trials.~~ As Koch and colleagues argue that the no-go/go sequence effects are independent of preparation, we also examined no-go/go sequence effects (pNI/NI vs. pNG/NI) and sequence effects arising from the absence of an opportunity for anticipatory preparation (pNI/NI vs. pI/NI) on these unprepared trials. A non-informative cue ensures that no anticipatory preparation is possible, as opposed

to a short CSI condition where some anticipatory preparation is possible and provides an RT benefit (Nicholson et al., 2005).

ERP components elicited at different levels of the processing continuum and lateralized readiness potentials (LRP) were analyzed to determine the locus of sequence-induced interference effects and whether these are similar for pNG/I and pNI/I sequences. Analysis of ERP components at different levels of the processing continuum can define the level at which sequence effects modulate processing on the current trial. Cue-locked ERP waveforms tap into both general preparation (for I, NI and NG trials) and task-specific preparation (for I and NG trials only) processes initiated by the cue. If pNG/I and/or pNI/I sequences affect anticipatory task-set reconfiguration on the current trial, these sequences should show a smaller differential switch-positivity as compared to pI/I sequences. Stimulus-locked ERPs index processes initiated at stimulus onset. If pNG/I and/or pNI/I sequences affect rule implementation and/or S-R interference on the current trial, these sequences should show reduced a smaller differential switch-negativity as compared to pI/I sequences.

Stimulus-locked ERPs can also test the argument that the no-go/go sequence effect is not due to inhibition of the prepared task-set but to the absence of response selection on no-go trials (Schuch & Koch, 2003; Koch & Philipp, 2005). In typical go/no-go paradigms, no-go trials elicit a large frontocentral positivity that is associated with inhibition of a prepotent motor response (e.g. Falkenstein, Hoorman & Hohnsbein, 2002, Smith, Johnstone & Barry, 2006). This positivity, which has been labeled the *no-go P3*, occurs across a variety of no-go paradigms regardless of stimulus or response modality and has even been found with inhibition of covert activity (such as inhibition of an imagined response; Burle, Vidal & Bonnet, 2004). The fMRI response inhibition literature suggests that no-go trials involve suppression of response selection (Rubia, Russell, Overmeyer, Brammer, Bullmore, Sharma, et al., 2001). If no-go stimuli elicit a frontocentral P3, that would suggest that, contrary to Schuch & Koch's (2003) argument, no-go/go sequence effects may affect residual switch cost because they involve inhibition of the prepared task-set.

The lateralized readiness potential (LRP) reflects differential activation over the motor cortex contralateral to the side of responding and provides an index of the processes leading from the stimulus to

the overt response (Coles, 1989). Premotor and motor processes can be dissociated by locking the LRP to either the stimulus or the response. The interval between stimulus onset and the onset of the stimulus-locked LRP (sLRP) indexes the duration of premotor processes, such as response selection (Miller & Hackley, 1992; Mordkoff & Gianaros, 2000), whereas the interval between the onset of the response-locked LRP (rLRP) and the response indexes the preparation and activation of the appropriate response (Miller & Hackley, 1992; Mordkoff & Gianaros, 2000). Thus, manipulations that affect only premotor processes will result in a delay in the onset of the sLRP, but no change in the onset of the rLRP. Conversely, any manipulation that affects motoric but not premotor processes will result in a delay in the onset of the rLRP but not the sLRP (Mordkoff & Gianaros, 2000). If, as argued by Koch and colleagues, no-go/go sequence effects arise specifically because the absence of response selection on the preceding no-go trial removes response repetition bias on the current go trial, response selection as indexed by sLRP onset or response activation as indexed by rLRP onset should be later for pNG/I repeat trials as compared to pI/I repeat trials, with no difference between pNI/I and pI/I sequences.

Conversely, Astle et al. (2006) argue that no-go stimuli lead to inhibition of the readiness to respond upon stimulus onset and that it is this inhibition of response readiness that must be overcome on the subsequent go trial. Response-locked ERP waveforms are suitable to address this possibility. ERPs time-locked to a response and with a long pre-response interval index more generic response readiness processes and are characterized by a frontocentral slow negative wave (see Sinai et al., 2007). If no-go stimuli inhibit response readiness, then recovery from inhibition should be evident as a difference in response-locked ERPs between pNG/I as compared to pI/I sequences, but no difference would be expected between pNI/I vs. pI/I sequences. Using the logic of Sinai et al. (2007), if the difference between pNG/I and pI/I sequences emerges before r-LRP onset, recovery from inhibition occurs before or during the response selection stage, whereas if it emerges after r-LRP onset, recovery from inhibition emerges at the response preparation stage.

Methods

Participants

Twenty-four young adults (mean age 25+/- 6 years, 9 male, right handed) with no prior exposure to the current paradigm participated in this experiment.

Stimuli and Tasks

The task was programmed in Presentation (Neurobehavioral Systems). A square box was outlined in grey (100 x 100 pixels) and presented against a black background on a computer monitor for the duration of the experiment. Each trial began with the presentation of an informative or non-informative cue (fixation cross 40 x 40 pixels), which remained on the screen for the entire cue-stimulus interval of 700 ms and was replaced by the stimulus. Non-informative cues were presented in grey and informative cues were presented in one of four 'hot' (red, pink, orange, burgundy) and four 'cold' (dark blue, green, sky blue, turquoise) colors that were mapped to one of the two tasks. Cue color was never repeated on successive trials to eliminate any confounding effect of cue identity repetition (Logan & Bundesen, 2003; note, however, that mediator cue effects could still be operating i.e., hot/cold or letter/digit). On informative and no-go trials, an informative cue was presented in one of the above colors, and the stimulus was white. As the cue information was removed prior to stimulus onset, participants were required to prepare on every trial where an informative cue was presented (Verbruggen et al., 2007). On non-informative trials, the non-informative cue was replaced by a stimulus in one of the above colors.

On informative and non-informative trials, the stimulus was a letter-digit pair (such as 'A4') presented in 12pt SimSun font (viewed from approximately 90cm; visual angle 1º). The position of the letter and digit in the pair was randomized between trials (i.e. the stimulus could be 'A4' or '4A'). On each trial, participants responded to either the letter or the digit. For the letter task, participants classified the letter as vowel (A, E, I, U) or consonant (G, K, M, R). For the digit task, they classified the digit as odd (3, 5, 7, 9) or even (2, 4, 6, 8). Participants used their left and right index fingers to respond. Stimulus-response mapping and cue-task mapping were counterbalanced across participants. Only incongruent character pairs (letter-digit combinations) were presented, that is the task-irrelevant character was always mapped to a response with the other hand (e.g., A4: even and vowel mapped to the different hands). On no-go trials, the stimulus was two non-alphanumeric characters that were not mapped to either

task and were selected from a set of four exemplars (#, $, %, &). Overt responses were required on informative and non-informative trials, but not on no-go trials.

The stimulus onset asynchrony (SOA; cue to cue) varied between 2.5 and 7 sec in an exponential distribution, with a mean of 3.5 sec. Each trial began with the cue that was presented for 700 ms and replaced by the stimulus. On informative and non-informative trials, the stimulus was removed at response onset. The response was followed 300 ms later by one of two feedback tones. Correct responses were followed by a 1000 Hz 'correct' tone, and incorrect responses were followed by an 'incorrect' (Microsoft Windows 'dong') tone. The response-cue interval was determined by the SOA on that particular trial after subtracting the CSI, RT and feedback interval. On no-go trials, a response led to an 'incorrect' feedback tone. When a response was correctly withheld, a 'correct' feedback tone was presented 300 ms prior to the cue for the subsequent trial.

The three trial types (informative, non-informative and no-go) were equiprobable and were presented with overall switch trial probability of 33% in a pseudorandom sequence that included no more than three consecutive switch or repeat trials and no more than four consecutive trials of the same condition. Each participant completed a total 120 switch and 240 repeat trials within each of the three conditions (total 1080 trials; after artifact rejection average 35 switch and 70 repeat trials per condition for each participant. Average SOA = 3.5 sec; approx. 60min recording time). The sequence was divided into four blocks of 270 trials. Each block was followed by a brief rest.

Procedure

Participants completed three sessions. The first session was a task practice session, and included training initially on each task alone and then in switching between the tasks with the informative cue. The other two sessions included testing with either ERP or fMRI[2] data acquisition. All except five participants completed the ERP testing in the second session that was scheduled no more than a week after session one. Session two began with further practice on the informative condition, and training on

[2] Jamadar, Karayanidis, Hughes and Michie (in preparation). The temporal and spatial dynamics of anticipatory preparation and response inhibition.

the non-informative and no-go conditions. Across both practice sessions, participants completed a total of 750 training trials before testing.

Participants were instructed to respond as quickly as possible whilst maintaining a high level of accuracy. Following each block, RT and percent accuracy feedback was displayed and participants were encouraged to monitor and improve their performance.

Data Analysis

The first two trials of every run, trials associated with an incorrect response, trials immediately following an incorrect response, and trials with a response occurring outside a 200-2000ms window after stimulus onset were excluded from behavioral and ERP analysis. The EEG data of three participants were excluded from further analysis due to equipment malfunction. For both behavioral and ERP analyses, degrees of freedom for factors with more than two levels were adjusted using Greenhouse-Geisser correction for the violation of the assumption of sphericity (Vasey & Thayer, 1987).

Behavioural Data Analysis

RT was averaged separately for switch and repeat trials on informative and non-informative trials. RT and proportion error data sequence effects were analyzed for both informative and non-informative trial types by averaging RT separately according to trial type of the preceding trial (approx. 40 trials per sequence). Switch cost was calculated by subtracting the repeat trial from switch trial scores. Analysis for proportion error data were run on arc sine transformed scores to normalize the distribution.

EEG Recording and Data Analysis

EEG was recorded using a Quik-cap from 62 scalp electrodes referenced to the nose electrode. Vertical and horizontal EOG were recorded via electrodes positioned above and below the left eye, and on the outer canthi of each eye, respectively. EEG and EOG were continuously sampled at 500Hz/channel on a Synamps 1 system (Neuroscan) with a bandpass of 0.01-30Hz using a 50Hz notch filter.

Vertical eyeblink artifact was corrected in the continuous EEG files using the algorithm developed by Semslitch, Anderer, Schuster and Presslich (1986) as implemented by Neuroscan software. These files were visually inspected and sections of EEG contaminated with channel saturation or noise

were excluded from further analysis. Continuous EEG files were re-referenced to the average of left and right mastoids to be consistent with previous methods in our laboratory (e.g. Karayanidis et al. 2003). Cue- and stimulus-locked averages were created by extracting 1400ms epochs around the onset of the cue or stimulus, respectively. Cue- and stimulus-locked averages were created by extracting 1400ms epochs around the onset of the cue/stimulus (-200 to 1200ms~~; -50 to 50ms baseline~~) for switch and repeat trials for each sequence. A -50 to 50ms baseline was employed to account for the shifting pre-cue and pre-stimulus baseline apparent in these waveforms (see also Karayanidis et al., 2003). Response-locked averages were created by extracting 1400ms epochs around the onset of the response (-1000ms to 400ms). Response-locked waveforms were baseline corrected over -1000 to -700ms, and were averaged separately for switch and repeat trials for each sequence. Mean amplitude windows were extracted using EEGDisplay software (Fulham, 2005). Although ERPs are shown at midline sites only, recording from a large montage allows us to make inferences regarding differences in scalp topographies between experimental conditions. An ERP component that shows a similar amplitude and duration but different scalp topography between conditions, is likely to have been generated by distinct neuronal populations (Otten & Rugg, 2005).

LRPs were extracted from C3 and C4 electrodes using the averaging method according to Coles (1989). C4 minus C3 waveforms were derived for left hand responses and C3 minus C4 for right hand responses. These were averaged to create separate LRP waveforms for each sequence and trial type. sLRPs were locked to stimulus onset (-200 to 1200ms; prestimulus baseline -200 to 0ms). rLRPs were locked to response onset (-1000 to 400ms; -700 to -400ms baseline). Onset latency of sLRP and rLRP was measured on jackknifed LRPs (Ulrich and Miller, 2001) using the segmented regression method with 1 degree of freedom (Schwarzenau, Falkenstein, Hoorman & Hohnsbein, 1998).

In each analysis, current trial effects were analyzed by simple comparisons of informative switch vs. repeat trials and informative (averaged over switch and repeat) vs. non-informative and were corrected for multiple comparisons (α=.025). Previous trial effects were analyzed using comparisons of informative vs. non-informative and informative vs. no-go trials (α=.025). Significant effects of electrode were analyzed at each electrode separately (α=.016).

Results

Behavioural Results

RT and error data were analyzed with separate 2 current cue (informative cue, non-informative cue) x 3 previous trial (informative, non-informative, no-go) x 2 trial type (switch, repeat) x 2 task (letter, digit) within subjects ANOVA. There was no main effect or interaction with task. As shown in Figure 1A, RT was longer for non-informative relative to informative cue trials ($F(1,20)=49.36$, $p<.001$) and for switch as compared to repeat trials ($F(1,20)=12.45$, $p=.002$). Switch cost was significantly larger for non-informative as compared to informative cue trials ($F(1,20)=42.17$, $p<.001$).

RT was affected by previous trial ($F(1,20)=27.47$, $p<.001$), and this effect was moderated by interactions with current trial ($F(1,20)=4.10$, $p=.03$) and trial type ($F(1,20)=19.37$, $p<.001$). Overall, RT was slower for trials preceded by a no-go trial ($F(1,20)=34.07$, $p<.001$). For informative trials, repeat trial RT was fastest for pI/I sequences, slower for pNI/I sequences ($F(1,20)=15.14$, $p=.001$) and slowest for pNG/I sequences ($F(1,20)=37.68$, $p<.001$), but switch trial RT was not modulated by preceding trial type ($p>.10$). For non-informative trials, repeat trial RT did not differ as a function of whether the previous trial was informative or non-informative, but was significantly longer when preceded by a no-go trial (pNG/I; $F(1,20)=45.93$, $p<.001$). Switch trial RT also showed a small but significant RT increase when preceded by a no-go trial ($F(1,20)=6.16$, $p=.022$).

As RT switch cost differed significantly between conditions, switch costs (Figure 1) were analyzed using one way ANOVA for each combination of current trial and previous trial. Informative trials showed significant positive switch cost when preceded by an informative trial (pI/I sequence: $F(1,20)=6.78$, $p=.017$), whereas non-informative trials showed a significant positive switch cost when preceded by either informative or non-informative trials, (pI/NI sequence: $F(1,20)=46.28$, $p=.003$; pNI/NI: $F(1,20)=23.55$, $p<.001$, respectively). All other conditions showed either no RT switch cost or a *negative* switch cost (pNG/I sequence: $F(1,20)=11.62$, $p=.003$). These findings replicate no-go/go sequence effects on repeat trial RT (Schuch & Koch, 2003; Koch & Phillip, 2005) and also show that no-go effects occur for both prepared (pNG/I) and unprepared (pNG/NI) go trials. Importantly, a similar though smaller increase in repeat trial RT and loss of RT switch cost was found with pNI/I sequences.

Error data are shown in Figure 1B. Overall, errors rate was higher on non-informative relative to informative trials ($F(1,20)=6.62$, $p=.018$) and on switch as compared to repeat trials ($F(1,20)=8.39$, $p=.009$). The significant previous trial x trial type ($F(2,40)=13.29$, $p<.001$) and current trial x previous trial x trial type ($F(2,40)=3.37$, $p=.048$) interactions reflected that error switch cost was reduced when the current and previous cues differed (pNI/I and pI/NI sequences), and reversed when the previous trial was a no-go trial. These results are largely consistent with those observed on the RT switch cost.

ERP Results

Cue-Locked Waveforms

Figure 2 shows cue-locked waveforms superimposed for informative switch, informative repeat and non-informative cue trials, plotted separately according to previous trial. Cue-locked difference waveforms for informative minus non-informative cues (informative effect), and informative switch minus informative repeat cues (switch effect) were analyzed using point-by-point t-tests to identify points of significant difference at midline sites (Fz, Cz, Pz). Type 1 error was controlled at $\alpha=.05$ using an autocorrelation coefficient of 0.9 (Guthrie & Buchwald, 1991). In the current context, an interval was considered significant if there were at least 12 consecutive points (24ms) of significant difference. At midline sites, intervals resulted in a minimum of 21 and a maximum of 100 consecutive points of significance. These analyses suggested two areas of maximal difference: 300-400ms for differences between informative and non-informative cues and 450-550ms for differences between switch and repeat cues. Mean amplitude measures of the cue-locked waveforms over these intervals were analyzed using a 3 current trial (informative switch, informative repeat, non-informative) x 3 previous trial (informative, non-informative, no-go) x 3 electrode (Fz, Cz, Pz) ANOVA.

Both analysis windows showed significant main effects of current trial (300-400ms: $F(2,40)=7.98$, $p=.001$; 450-550ms: $F(2,40)=6.19$, $p=.005$) and interaction between current trial and electrode (300-400ms: $F(4,80)=8.77$, $p<.001$; 450-550ms: $F(4,80)=9.74$, $p=.001$). Parietally, informative cues produced a significantly larger positivity than non-informative cues over 300-400ms ($F(1,20)=36.15$, $p<.001$) but this was not sustained in the later time window. Rather, over 450-550ms, informative switch cues produced a larger positivity than informative repeat cues at both central and parietal sites (central:

F(1,20)=13.92, p=.001, parietal: F(1,20)=33.87, p<.01). The parietal distribution of the early informative-positivity and centro-parietal distribution of the later switch-positivity are shown in scalp distribution maps (Figure 2, bottom).

In both analysis windows, the significant main effect of previous trial (300-400ms: F(2,40)=14.85, p<.001; 450-550ms: F(2,40)=11.34, p=.001) and interaction between previous trial and electrode (300-400ms: F(4,80)=23.17, p<.001; 450-550ms: F(4,80)=34.85, p<.001) reflected reduced overall positivity when the current trial was preceded by a no-go as compared to an informative trial at both Cz (300-400ms: F(1,20)=10.82, p=.004; 450-550ms: F(1,20)=8.91, p=.007) and Pz (300-400ms:, F(1,20)=59.27, p<.001; 450-550ms: F(1,20)=51.41, p<.001). This overall difference in morphology between trials preceded by a no-go trial and other trial types occurred because no-go trials concluded with a 'correct' feedback tone that always occurred 300ms prior to cue onset and resulted in a negative-going baseline for all trials following a no-go trial. A similar but much smaller reduction in parietal positivity was obtained when the preceding trial was non-informative (300-400ms: F(1,20)=9.05, p=.007; 450-550ms: F(1,20)=9.64, p=.006). Importantly, previous trial did not interact with current trial type, indicating that neither the early informative-positivity nor the later switch-positivity were modulated by trial sequence effects.

In summary, cue-locked waveforms showed an early differential positivity for informative as compared to non-informative cues and a later differential positivity for informative switch as compared to informative repeat cues. No sequence effects were found on either positivity, indicating that neither the pNG/I nor the pNI/I effect on RT switch cost reflects a disruption of anticipatory preparation processes.

Stimulus-Locked Waveforms

Informative and non-informative stimulus-locked waveforms were characterized by a large frontocentral P2 (200ms) followed by a posterior late positive component (LPC, 300-700ms; Figure 3A). Point-by-point analysis of switch-repeat difference waveforms revealed no significant switch negativity for either informative or non-informative trials. We therefore derived two mean amplitude windows based on inspection of the grand average waveforms so as to capture the early LPC and early frontal positivity (300-400ms) and the late LPC and late frontal positivity (550-650ms). These were analyzed using a 2

current trial (informative, non-informative) x 2 trial type (switch, repeat) x 3 previous trial (informative, non-informative, no-go) x 3 electrode (Fz, Cz, Pz) repeated measures ANOVA.

Overall, stimulus-locked waveforms were more positive for informative as compared to non-informative trials (300-400ms: $F(1,20)=29.64$, $p<.001$; 550-650ms: $F(1,20)=38.11$, $p<.001$). Significant interactions between trial type and electrode (300-400ms: $F(2,40)=11.24$, $p<.001$; 550-650ms: $F(2,40)=26.02$, $p<.001$) as well as trial type, current trial and electrode were evident for both time windows (300-400ms: $F(2,40)=11.14$, $p=.001$; 550-650ms: $F(2,40)=5.44$, $p=.024$). For informative trials, both early and late frontal positivities were larger for switch than repeat trials (300-400ms: $F(1,20)=5.44$, $p=.030$; 550-650ms: $F(1,20)=15.42$, $p=.001$). The expected parietal switch negativity was only marginally significant (300-400ms: $F(1,20)=3.94$, $p=.069$; 550-650ms: $F(1,20)=3.70$, $p=.069$). Stimulus-ERP showed a large frontal differential switch positivity, but little evidence of the parietal differential switch-negativity reported previously (e.g. Karayanidis et al., 2003; Nicholson et al., 2005).

Over 300-400ms, previous trial type exhibited a significant main effect ($F(2,40)=5.93$, $p=.017$) and interaction with current trial ($F(2,40)=6.41$, $p=.006$). For informative trials, pNG/I produced a larger positivity than pI/I sequences ($F(1,20)=9.21$, $p=.007$). Previous trial type did not modulate switch vs. repeat differences on the current trial ($F<1.0$). So, while previous trial type affected the amplitude of the early positivity for informative trials (pNG/I > pNI/I sequences), there were no sequence effects for switch vs. repeat trials.

Stimulus-locked waveforms for no-go trials were compared to informative trials as they involved identical preparation (Figure 4) in order to examine whether these no-go trials elicited ERP components associated with inhibition, even though the stimuli were not mapped to any response. At Fz, both no-go and informative trials elicited an N2 (250-350ms; $F<1.0$). No-go trials also elicited a larger frontal P3 (350-450ms; $F(1,20)=40.91$, $p<.001$) suggesting inhibition of the prepared task-set or of the readiness to respond.

Response-Related Effects

Stimulus-locked LRPs (sLRP) emerged around 300-400ms (Figure 3B) and were less well defined for non-informative as compared to informative trials, possibly reflecting the longer and more variable RT. Response-locked LRPs (rLRP; Figure 5A) emerged around 200ms pre-response (185+/-18ms), peaking 100 ms before response. Onset latency of sLRP (informative: 298ms, non-informative 299ms) and rLRP (informative:-185ms, non-informative: -185ms) were not affected by current trial, previous trial or trial type (all $F<1$). Thus, trial sequence had no effect on sLRP and rLRP onset latency.

Response-locked ERP waveforms (Figure 5B) were characterized by a sharp positivity that peaked at response onset and was preceded by a pre-response complex characterised by a broad centro-parietal negativity emerging around 800 ms before the response. Mean amplitude was measured in three time windows. The early window (-600 to -500ms) measured the peak of the centro-parietal negativity. The second window (-400 to -300ms) targeted activity preceding rLRP onset by more than 2 standard deviations (mean onset latency = -185ms, maximum standard deviation across all trial types = 18ms). The final window (-250 to -150ms) captured activity around rLRP onset. Mean amplitude was analyzed using a 2 current trial x 3 previous trial x 2 trial type x 3 electrode repeated measures ANOVA.

The early negativity was larger for informative than non-informative trials ($F(1,20)=6.99$, $p=.016$) especially at centro-parietal sites ($F(2,40)=15.74$, $p<.001$). This early negativity showed a large effect of previous trial ($F(2,40)=8.80$, $p=.002$) as well as interactions with current trial and trial type (previous x current: $F(2,40)=6.46$, $p=.004$; previous x current x type: $F(2,40)=3.29$, $p=.048$; current x type x electrode interactions ($F(2,40)=11.05$, $p=.001$). For informative trials, the negativity was smaller for pNG/I as compared to pI/I sequences ($F(1,20)=15.74$, $p=.001$), and this effect was larger for repeat trials, resulting in a reversal of the switch/repeat difference ($F(1,20)=6.05$, $p=.023$). There was no difference between pNI/I and pI/I sequences in this or any other window (all $F<1.74$). Non-informative trials showed no effect of trial type or previous trial ($F<1.0$).

In the interval immediately preceding rLRP onset (-400 to-300ms), there was again a significant effect of previous trial ($F(2,40)=9.88$, $p=.001$) and interactions with current trial and trial type (current x previous: $F(2,40)=4.06$, $p=.029$; current x electrode: $F(2,40)=5.96$, $p=.018$; current x type x electrode: $F(2,40)=7.59$, $p=.005$). Again, this negativity was smaller for pNG/I as compared to pI/I sequences

(F(1,20)=12.74, p=.002) and as well as for pNG/NI as compared to pNI/NI sequences (F(1,20)=4.39, p=.049). Parietally, pNG/I repeat trials continued to show the largest reduction in negativity (F(1,20)=6.33, p=.021). Again, non-informative trials were not affected by trial type (F<1.0).

Finally, during rLRP onset (-250 to -150ms), both informative and non-informative trials preceded by a no-go trial continued to be differentiated from other sequences (F(1,20)=12.04, p=.002), but this sequence effect was no longer moderated by trial type. A significant three-way interaction between current trial, trial type and electrode (F(2,40)=5.96, p=.010) indicated that there was a significant difference between switch and repeat trials parietally for informative trials only (F(1,20)=5.86, p=.025).

In summary, response-locked waveforms showed emergence of no-go/go sequence effects as early as 600ms pre-response for informative trials and 400ms pre-response for non-informative trials. Parietally, informative repeat trials preceded by a no-go trial showed a much smaller negativity than informative switch trials, an effect sustained at least until rLRP onset.

Discussion

Overall, behavioural findings were compatible with earlier studies showing a significant RT switch cost and a reduction in switch cost with increasing preparation time (e.g. Karayanidis et al., 2003; Meiran, 2000; Rogers & Monsell, 1995) as well as larger cost for non-informative as compared to informative trials (Swainson et al., 2006). We replicated the elimination of RT switch cost on trials following a no-go trial (e.g. Koch & Philipp, 2005; Schuch & Koch, 2003), an effect again driven by an increase in repeat trial RT. Importantly, sequence effects on RT switch cost were not specific to trials preceded by a no-go trial but also occurred with other non-repetitive trial sequences, with switch cost also eliminated in pNI/I sequences.

Cue-locked waveforms showed an early positivity for informative relative to non-informative waveforms (Swainson et al., 2006) and a later positivity for informative switch relative to informative repeat waveforms (e.g. Nicholson et al., 2005). Neither early nor late positivity was affected by sequence effects. Stimulus-locked waveforms showed only small differences between switch and repeat trials that were also not affected by trial sequence. No-go trials elicited a large frontal no-go P3, an ERP component

believed to index inhibition (Falkenstein et al., 2002; Smith et al., 2006). This result, together with the finding that sLRP onset was not differentially modulated by no-go/go sequence effects questions Koch and colleagues' assumption that, in the context of task-switching paradigms, no-go trials do not involve response selection and therefore no-go/go sequence effects do not arise from inhibition-related processes (Koch & Philipp, 2005; Schuch & Koch, 2003).

The most salient electrophysiological no-go/go sequence effect was observed in response-locked waveforms. Informative pNI/I sequences were differentiated from pI/I sequences as early as 600ms before response onset. Given a mean RT of just over 750ms, the timing of this differentiation seems to occur shortly after stimulus onset. The failure to find any evidence of sLRP or rLRP onset differences and the early emergence of the response-locked ERP difference suggest that RT sequence effects may be explained by reduced response readiness on informative trials preceded by a no-go trial (see General Discussion). This effect seems larger for, though not exclusive to, pNG/I repeat and is compatible with the finding that sequence effects reduce RT switch cost by increasing repeat trial RT.

Experiment 2

Before interpreting the above results, it is necessary to address some unforeseen findings in Experiment 1. Firstly, as we predicted, non-informative/informative (pNI/I) sequences showed a reduction in RT switch cost, similar to though smaller than that observed with no-go/informative (pNG/I) sequences, suggesting that other types of sequences can also affects RT switch cost. However, while pNG/I sequences differed from pI/I sequences in both stimulus-locked and response-locked ERPs, there was no ERP difference between pNI/I and pI/I sequences. Additionally, if the effect were merely due to a change in trial type (i.e., the shift from a non-informative to an informative trial becoming another type of switch trial), it should have also been observed when shifting from informative to non-informative conditions (pI/NI sequences) – but this was not the case.

Secondly, while behavioral data in the non-informative condition showed the expected increase in RT switch cost, electrophysiological data showed an unexpected pattern of outcomes. Non-informative

trials can be considered as having a cue-stimulus interval of zero, because the stimulus itself carries the information about which task is relevant. We therefore expected stimulus-locked ERPs to show an early positivity for non-informative switch as compared to non-informative repeat trials (Nicholson et al., 2005; Nicholson, Karayanidis, Davies, et al., 2006). However, stimulus-locked waveforms showed *reduced* positivity for non-informative relative to informative trials and no differentiation between non-informative switch and repeat trials.

The paradigm used in Experiment 1 differs from our previous paradigms in two important ways. Firstly, it includes a mixture of informative (prepared) and non-informative (unprepared) trials within the same block. Previous studies have shown that randomizing preparation intervals may result in the formation of different task strategies than blocked preparation intervals (eg., Rogers & Monsell, 1995; Strayer & Kramer, 1994). In blocked designs, participants may develop a different optimal strategy for each condition. In randomized designs, however, participants would need to adopt a strategy that is suitable for all conditions, even if this strategy is sub-optimal for each condition alone. Secondly, the paradigm included a large proportion of no-go trials. As these no-go trials produced a large behavioral effect on the subsequent trial, it is not unreasonable to assume that they may have had an effect on performance as a whole. Altmann (2004) showed that mere exposure to one condition (i.e., short CSI) may change participants' strategy and performance under another condition (i.e., anticipatory preparation in long CSI condition). In go/no-go tasks, the proportion of no-go trials has been shown to affect overall go trial RT, with low probability no-go trials resulting in faster go trial RT as compared to a block of go trials (Low & Miller, 1999). In Experiment 1, no-go trials constituted one third of all trials and this is likely to have affected performance on both informative and non-informative trials. Note also that whereas one half of informative cues were followed by a valid stimulus leading to a response, the other half were followed by a no-go stimulus. As informative and no-go trials were indistinguishable at cue onset, this produced an unintentional manipulation of cue validity, with informative cues leading to a valid stimulus on only 50% of trials. It is known that cue validity can affect task-switching performance (e.g. Dreisbach et al., 2002; Hubner, Kluwe, Luna-Rodriguez & Peters, 2004).

In order to examine whether pNI/I and pI/NI sequence effects persist in the absence of any confounding effect from no-go trials, we ran a second experiment that did not include no-go trials. Experiment 2 also included a second group of participants that experienced only blocked presentations of informative and non-informative trials in order to examine whether some of the unexpected findings in Experiment 1 could be due to randomized presentation of informative and non-informative trials.

Method

Experiment 2 was identical to Experiment 1, with only exceptions as mentioned below.

Participants

Thirty-two undergraduate students (mean age 25 +/- 10 years, 4 male) with no prior exposure to the current paradigm were randomly assigned to randomized (n=16) and blocked (n=16) presentation groups.

Procedure

The randomized group received identical training and testing to Experiment 1, except that there were no no-go trials. The blocked group completed two blocks of informative and two blocks of non-informative trials in counterbalanced order. Switch probability was 50%.

Data Analysis

Three subjects from the randomized group were excluded from all analyses due to high levels of movement artifact in the EEG. After artifact rejection an average of 75 trials per trial type per condition remained for each participant. Analyses comparing randomized and blocked groups included only pI/I and pNI/I trials from the former group, in order to avoid contamination from sequence effects. The same analysis was run using all trials averaged over previous trial type in the randomised group and produced largely identical outcomes. EEG recording parameters were identical to those in Experiment 1.

Results

Behavioral Results

RT and switch cost for each sequence are shown in Figure 6A. Sequence effects on RT and arc sine transformed proportion error rates were analyzed in the randomized group with a 2 current trial x 2 previous trial (informative, non-informative) x 2 trial type repeated-measures ANOVA. There was a significant main effect of trial type on RT ($F(1,12)=16.92$, $p=.001$). Previous trial had widespread effects on RT (main effect: ($F(1,12)=4.94$, $p=.046$; previous x current: $F(1,12)=6.97$, $p=.022$; previous x current and type: $F(1,12)=5.17$, $p=.042$). Switch trial RT was not significantly affected by previous trial type ($p>.10$). In contrast, repeat trial RT was slower when the preceding trial type (informative or non-informative) was different to the current one (current x previous: $F(1,12)=7.68$, $p=.017$), resulting in a reduction in RT switch cost from 122ms for pNI/NI to 87ms for pI/NI sequences and from 35ms for pI/I to 23ms for pNI/I sequences. As in Experiment 1, the interaction between trial type and other factors was examined further by analyzing switch cost at each level of the other factors. A significant switch cost was obtained for all sequences ($p<.008$) except pNI/I. Thus, significant sequence effects on RT switch cost were obtained for both informative and non-informative trials, both effects being driven by an increase in repeat trial RT. Error rate did not differ between conditions (all $p>.271$; Figure 6B).

ERP Results

Cue-Locked Waveforms

Figure 7 shows cue-locked waveforms superimposed for informative switch, informative repeat and non-informative trials for each sequence. Based on Experiment 1, we restricted cue-locked analyses to Pz where the effects of cue and trial type were maximal and defined two analyses windows based on difference waveform analyses. The early positivity (350-450ms) was larger for informative as compared to non-informative cues ($F(1,27)=34.13$, $p<.001$), but did not differ between informative switch and repeat cues ($F<1$). The later positivity (550-650ms) continued to be larger for informative as compared to non-informative cues ($F(1,27)=38.10$, $p<.001$), but was also larger for switch than for repeat cues ($F(1,27)=5.73$, $p=.034$). Both positivities were modulated by previous trial. Specifically, the early positivity was larger when the previous trial was informative ($F(1,12)=8.28$, $p=.014$), whereas the later positivity was larger when the previous trial was non-informative ($F(1,12)=7.33$, $p=.019$). However, again, these sequence effects did not interact with trial type (switch vs. repeat).

Stimulus-Locked Waveforms

As in Experiment 1, non-informative trials showed a broad parietally-maximal positivity emerging as early as 100ms and extending 700ms after stimulus onset (Figure 8A). However, the morphology of the informative waveforms differed substantially from those in Experiment 1. Here, informative trials showed a large centroparietally maximal negative deflection with a later onset LPC (around 300-350ms). In addition, informative switch trials showed a very large negative shift relative to repeat trials emerging as early as 150ms post-stimulus and extending in some instances beyond 600ms (stimulus-locked differential switch-negativity). These effects were examined over 300-400ms and 500-600ms windows using a 2 current trial x 2 previous trial (informative, non-informative) x 2 type repeated-measures ANOVA. We restricted stimulus-locked analyses to Pz where the effects of cue and trial type were maximal.

Both early and late windows showed significant previous trial x current trial interactions (300-400ms: $F(1,12)=11.56$, $p=.005$; 500-600ms: $F(1,12)=6.71$, $p=.024$) and marginal previous trial x trial type interactions (300-400ms: $F(1,12)=4.80$, $p=.049$; 500-600ms: $F(1,12)=4.01$, $p=.068$). Non-informative trials were not significantly affected by either trial type or previous trial. Informative trials, however, showed significant effects of trial type (300-400: $F(1,12)=6.32$, $p=.027$; 500-600: $F(1,12)=12.13$, $p=.005$), previous trial type (300-400: $F(1,12)=11.03$, $p=.006$; 500-600: $F(1,12)=9.20$, $p=.010$) and their interaction (300-400: $F(1,12)=5.69$, $p=.034$; 500-600: $F(1,12)=8.75$, $p=.012$) in both time windows. While pI/I sequences showed a large differentiation between switch and repeat trials, there was no difference evident for pNI/I sequences. This effect was driven by a significant negative shift in the repeat waveform in pNI/I sequences relative to pI/I sequences (300-400ms: $F(1,12)=9.20$, $p=.010$; 550-650ms: $F(1,12)=15.24$, $p=.002$; see Figure 8A). The amplitude of the switch waveform was not modulated by previous trial in either time window ($F<1.0$).

In summary, non- informative waveforms were again not affected by trial type or previous trial. However, the removal of no-go trials resulted in a substantial change in the morphology of the informative waveform. While informative switch waveforms were not affected by sequence effects,

informative repeat waveforms were indistinguishable from informative switch waveforms when the preceding trial was non-informative.

Response-locked waveforms

Neither sLRP (Figure 8B) nor rLRP (Figure 9A) onset were affected by any of the manipulations (all $F<1.0$). Response-locked waveforms showed a similar morphology to that seen in Experiment 1 (Figure 9B). Mean amplitude was measured in two windows: the early window (600-500ms pre-response) was at least 2 SD before rLRP onset, whereas the later window (350-250ms pre-response) targeted activity around rLRP onset (-275 +/- 4ms). These were analyzed with a 2 current trial x 2 previous trial x 2 trial type x 3 electrode ANOVA.

Sequence effects did not emerge until rLRP onset (-350 to -250ms; previous x electrode: $F(2,24)=4.96$, $p=.028$; previous x type x electrode: $F(2,24)=4.21$, $p=.039$). Switch trials and informative repeat trials were not significantly affected by sequence effects. However, non-informative repeat trials showed a smaller parietal positivity for pI/NI sequences as compared to pNI/NI sequences (previous x electrode interaction: ($F(2,24)=15.19$, $p=.001$). Thus, unlike Experiment 1, response-locked ERPs showed no sequence effects on informative trials, but a significant differentiation between non-informative repeat trials, depending on whether the preceding trial was informative or non-informative.

Blocked Versus Randomized Presentation of Informative and Non-informative Trials

RT and error rate are compared for blocked and randomized groups in Figure 10. The blocked group showed only a marginal reduction in RT switch cost on informative relative to non-informative trials ($F(1,15)=3.30$, $p=.089$), whereas the effect was much larger in the randomized group ($F(1,12)=18.22$, $p=.001$). Error rate mirrored this effect with a marginal cue type x group interaction ($F(1,27)=3.89$, $p=.059$). There was no significant difference between blocked and randomized groups on either cue-locked or stimulus-locked ERPs (Figure 11). Therefore randomization of cue types (informative vs. non-informative) cannot account for the unexpected pattern of stimulus-locked ERP waveforms seen in Experiment 1.

Discussion

The main aim of Experiment 2 was to examine whether some of the results in Experiment 1 could be attributed to spurious effects arising from the inclusion of no-go trials. Most behavioral and ERP results replicated the effects found in Experiment 1 and will be discussed in more detail in the General Discussion. However, the morphology of the stimulus-locked ERPs for informative cues differed substantially between experiments.

Recall that stimulus-locked waveforms of Experiment 1 showed an atypical pattern of results: informative trials showed a larger sustained positivity relative to non-informative waveforms with little differentiation between informative switch and repeat trials. In Experiment 2, informative trials produced a large central N2-like component that resulted in a much smaller parietal positivity and a large informative switch/repeat difference that emerged as early as 150ms and extended beyond 600ms in some conditions. This pattern is reminiscent of the stimulus-locked switch negativity reported previously (e.g., Karayanidis et al, 2003; Nicholson et al, 2005). This pattern was evident in both blocked and randomized groups, and in the latter group was affected by sequence (see General Discussion). It is notable that no other difference was observed between blocked and randomized condition groups.

There were three main differences between the experiments. Firstly, in Experiment 1, informative cues were followed on 50% of trials by a no-go stimulus, whereas in Experiment 2, they validly predicted a go stimulus requiring a response. It is possible that, in Experiment 1, participants delayed processing the cue until onset of the valid go stimulus, at least on a proportion of informative trials, resulting in post-stimulus reconfiguration for all trial types. This could explain the large stimulus-locked positivity for both informative and non-informative trials, but not the RT advantage for informative vs. non-informative trials in Expt 1. Secondly, in Experiment 1, switch probability was 33%, whereas in Experiment 2, it was 50%. However, our unpublished data (Karayanidis, Jamadar, Sanday & Provost, in preparation) show that stimulus-locked ERPs do not differ between 25% and 50% switch conditions, both showing a pattern similar to that seen in Experiment 2. Thirdly, Experiment 1 included no-go trials whereas Experiment 2 didn't. As we suggested earlier, the presence of no-go trials may have affected not only processing of the subsequent informative or non-informative go trial but also overall task strategy. Since the stimulus-locked ERPs for informative trials are consistent with the pattern reported in a number of previous studies

(e.g., Karayanidis et al., 2003; Kieffaber & Hetrick, 2005; Nicholson et al., 2005; Rushworth et al., 2005), it is likely that the novel pattern seen in Experiment 1 is related to strategy changes associated with the inclusion of no-go trials. Further analyses of behavioral data could help identify strategic differences, but these are outside the scope of this paper.

General Discussion

Components of task-set reconfiguration

In most cued trials paradigms, the cue provides two pieces of information: whether the next trial will involve repeating or switching task-sets, and which task will be completed. Therefore, within the CSI, participants can not only select the relevant goal (e.g. 'do the letter task') but also reorient attention to the relevant stimulus dimension ('attend to letters, ignore numbers') and remap these to responses ('left for vowel, right for consonant'). In both Experiments, two cue-locked differential positivities were observed: an early increase in positivity for informative relative to non-informative cues (Swainson et al., 2006), and a later increase in positivity for informative switch relative to informative repeat cues (Karayanidis et al., 2003; Kieffaber & Hetrick, 2005; Miniussi et al., 2005; Nicholson et al., 2005; Nicholson, Karayanidis, Bumak, et al., 2006; Nicholson, Karayanidis, Davies, et al., 2006; Poulsen et al., 2005; Rushworth et al., 2002; 2005). The early 'informative-positivity' is compatible with goal activation processes that can be activated by informative but not by non-informative cues. Similar early positive components have been found for partially informative cues that signal a change in task, without specifying the new task-set (Karayanidis et al., in revision; Nicholson, Karayanidis, Davies, Michie, 2006) and for mixed block cues relative to single task cues (Kieffaber & Hetrick, 2005). This early goal activation process may include goal setting (i.e., do the letter task) and orienting of selective attention (i.e., attend to letters, ignore numbers'; Rubinstein et al., 2001; Koch & Philipp, 2005; Schuch & Koch, 2003), as well as disengagement or inhibition of the irrelevant task-set (Karayanidis et al., in revision; Nicholson, Karayanidis, Davies, Michie, 2006).

Rubinstein et al. (2001) invoked a second reconfiguration process of category-response (C-R) rule activation – the process of (re)mapping the task-set categories to response (i.e., vowel=left; odd=right). In Rubinstein's model, C-R rule activation is triggered exogenously after stimulus

identification and before response selection (Rubinstein et al., 2001; p.771), and hence is not part of anticipatory reconfiguration (see also Koch & Philipp, 2005). We argue here that the later switch-related positivity is consistent with task-specific *C-R rule activation* (such as 'left is odd, right is even'), a process that can be activated before stimulus onset and that is necessary only on switch trials, which, by definition, require activation of a different set of task-specific C-R rules than the previous trial. In contrast, as repeat trials require the implementation of the same rules that were applied on the previous trial, they do not *require* C-R rule activation, although this may occur under some task conditions or on some proportion of trials. A second cue-locked switch-related positivity has also been found to cues that fully specify the upcoming task-set (Karayanidis et al., in revision; Nicholson, Karayanidis, Davies, Michie, 2006) and for switch vs. repeat cues in a mixed block (Kieffaber & Hetrick, 2005) and is compatible with this interpretation. The conceptualization of goal activation and C-R rule activation as constituents of anticipatory task-set reconfiguration is consistent with previous arguments by Schuch and Koch (2003), represents a slight modification to the original Rubinstein et al. (2001) framework and is compatible with Rogers and Monsell's (1995) task-set reconfiguration framework. We introduce the term *C-R rule implementation* to refer to the set of decision- and response-related processes that can only occur *after* stimulus identification and involve mapping the specific stimulus to a category, and selecting and activating the appropriate response ('A is a vowel, press left'). Even with fully completed anticipatory task-set reconfiguration, C-R rule implementation may require greater activation of conflict monitoring processes or greater attentional resources for switch than repeat trials, as the latter would invoke less interference from the irrelevant stimulus dimension and/or the alternative hand-response mappings that switch trials. We will use this framework to interpret the sequence effects findings.

No-go/go sequence effects

One of the main aims of this study was to use ERPs to examine the locus of no-go/go sequence effects on RT switch cost. In Experiment 1, RT was longer on both informative and non-informative trials that followed a no-go trial, as compared to those that followed a go (informative or non-informative) trial. This effect interacted with trial type, resulting in the elimination of RT switch cost in both no-go/informative (pNG/I) and no-go/non-informative (pNG/NI) sequences. This reduction, and in some

instances reversal, of RT switch cost was driven by a disproportionate increase in repeat trial RT, rather than a decrease in switch trial RT, an effect consistent with previous studies (Koch & Philipp, 2005; Schuch & Koch, 2003; but see Astle et al., 2006 for a reduction in switch trial RT). Koch and colleagues (Koch & Philipp, 2005; Schuch & Koch, 2003) argue that inhibitory processes cannot account for no-go/go sequence effects because although C-R rules may be activated, response selection does not take place and therefore there is no response to inhibit.

In Experiment 1, the cue was identical on informative and no-go trials, so goal activation and C-R rule activation could be completed within the cue-stimulus interval for both trial types. These two trial types were only differentiated upon stimulus onset, with informative trials including a valid stimulus mapped to a response and no-go trials including a non-alphanumeric stimulus for which no response was possible. No-go trials elicited a large frontal no-go P3 similar to that elicited in go/no-go paradigms (Falkenstein et al., 2002; Smith et al., 2006), suggesting that no-go trials did trigger an inhibition process. Since the stimulus was not mapped to any response, this no-go P3 is unlikely to represent response inhibition per se but rather inhibition of the recently activated C-R rule or the readiness to implement that rule.

This finding, along with the finding that sLRP onset did not differ between the sequences, questions Koch's argument that inhibition of activated C-R rules is an automatic process that accompanies response selection and therefore cannot contribute to sequence effects following no-go trials (e.g., Koch & Philipp, 2005; Schuch & Koch, 2003). It also supports our position that C-R rule activation can be temporally differentiated from C-R rule implementation. The former can be triggered by the cue, can occur independently of a validly mapped stimulus and can be inhibited by a no-go trial, whereas the latter cannot occur until stimulus identification.

The finding that our no-go trials did elicit a frontal no-go P3 suggests either the activated C-R rule or the resulting response preparedness is inhibited. In order to select between these two alternatives we have to consider how this inhibition affects processing of the subsequent trial. We examined sequence effects in cue-locked, stimulus-locked, and response-locked ERP waveforms in order to identify the timing and mechanism underlying this effect. If a no-go stimulus results in inhibition of the activated C-R

rule, we would expect this to affect the efficiency or time course of C-R rule activation on the subsequent trial, and this would be manifest in the late cue-locked switch-positivity. However, cue-locked ERP waveforms showed no modulation of either the early informative-positivity or the late switch-positivity as a function of previous trial type. This suggests that no-go trials did not inhibit the activated C-R rule and in turn indicates that the effects of task sequence on repeat trial RT were not mediated by anticipatory preparation processes. This result is compatible with Astle et al. (2006) who did not find a no-go sequence effect on the parietal switch-positivity[3] and partly compatible with Sinai et al. (2007) who found that sequence effects on cue-locked waveforms were task-dependent. It suggests that no-go sequence effects on anticipatory preparation processes are task-specific and cannot account for the increase in repeat trial RT and consequent elimination of RT switch cost in the current paradigm.

Informative trials showed no evidence of modulation of sLRP or rLRP onset latency as a function of previous trial type, indicating that no-go sequence effects did not affect response selection or activation processes. Both stimulus- and response-locked ERPs showed a positive shift for no-go/informative trials relative to informative trials preceded by another informative or a non-informative trial. In stimulus-locked ERPs, this emerged around 200ms after stimulus onset. However, this sequence effect did not differentially affect switch and repeat trials within the stimulus-locked waveform. In response-locked ERPs, the reduced pre-response negativity for no-go/informative sequences as compared to other informative trials emerged around 700ms before response onset. This sequence effect was significant over 500-600ms pre-response for both switch and repeat no-go/informative sequences, but was larger for repeat sequences centro-parietally. Considering these effects in the light of RT for repeat and switch no-go/informative sequences as compared to other informative trials, it appears that the differentiation between no-go/informative trials and other informative trials emerged around 150ms after stimulus onset.

We argue that the above results suggest that no-go trials inhibit the readiness to respond to the activated C-R rule. This results in reduced response readiness on the subsequent informative trial, which in turn differentially affects prepared repeat trial RT (i.e., informative repeats) as these are most likely to

[3] These authors did find a significant no-go/go vs. go/go sequence effect on a late frontal switch negativity, but our data showed no switch/repeat differentiation over 600-700ms (i.e., immediately pre-stimulus). Analyses over this mean amplitude window produced no interactions between previous and current condition ($F<1$).

benefit from high response readiness. Note that a similar, but considerably smaller, no-go sequence effect was evident for non-informative trials, but this emerged much later (though still well before rLRP onset) and was not significantly larger for these unprepared repeat trials. Thus, like Astle et al (2006), we suggest that, within the current paradigm, no-go trials inhibit the preparedness to initiate the response process. After a no-go trial, it is more difficult to activate *any* response because it is necessary to disinhibit the response system and re-initiate the C-R implementation process, resulting in an increase in RT. The outcome of this process is more evident on repeat trials because these require not only disinhibition of the response system but also implementation of the same C-R rules that had been prepared but not implemented on the previous trial – a type of *inhibition of return* (Posner & Cohen, 1984) to the C-R rules whose implementation was suppressed on the preceding no-go trial. These findings add to a growing body of literature providing converging evidence for a carry-over effect of response activation on RT switch cost (Gade & Koch, 2007; Philipp, Jolicouer, Falkenstein & Koch, 2007; Koch & Philipp, 2005; Schuch & Koch, 2003; Astle et al., 2006; Verbruggen, Liefooghe, Szmalec, & Vandierendonck, 2005; Verbruggen, Liefooghe & Vandierendonck, 2006).

Other sequence effects on RT switch cost[4]

Interestingly, sequence effects on RT switch cost did not occur exclusively following no-go trials. For all trial types, RT switch cost was smaller when the preceding trial type differed from the current trial type (i.e., pNI/I vs. pI/I and pI/NI vs. pNI/NI), and these effects were also due to an increase in repeat trial RT. Therefore, in the current context, sequence effects on RT switch cost can also arise when the preceding trial involves a different order of processing. Consistent with no-go sequence effects, sequence effects arising from a change in trial type did not affect cue-locked ERPs suggesting that they do not arise at the level of anticipatory preparation.

For informative trials, sequence effects arising from a previous non-informative trial were evident in stimulus-locked waveforms. The informative switch/repeat difference spread over 250-850ms when the preceding trial was also informative (i.e., prepared) and disappeared completely when the previous trial

[4] These effects will be discussed using Experiment 2 data which showed a more typical pattern of stimulus-locked findings.

was non-informative (i.e., unprepared). This effect was entirely driven by a negative shift in the repeat waveform for pNI/I sequences – that is, when the preceding trial was unprepared, the stimulus-locked waveform for the prepared repeat trial resembled a prepared switch trial, even though the same C-R rules were activated and implemented on both n-1 and n trials. The reduced positivity for pNI/I repeat trials was associated with an increase in RT. This pattern of findings is highly reminiscent of the repetition priming literature which shows reduced negativity and faster RT with stimulus repetition (e.g, Fabre, Lemaire & Grainger, 2007; Karayanidis, Andrews, Ward & McConaghy, 1991). In the current context, it would appear that pI/I repeat trials provide a processing benefit which is not available on pNI/I repeat trials. Thus, the increase in repeat trial RT and concomitant decrease in RT switch cost in pNI/I sequences appears to be due to the elimination of the repetition priming benefit when the previous trial did not afford the opportunity for anticipatory preparation.

Sequence effects were also obtained on non-informative trials. A similar pattern of reduced repeat trial positivity was evident for non-informative trials preceded by an informative trial (pI/NI), but this was not significant. However, for non-informative trials, RT switch cost was smaller and, in response-locked waveforms, the switch/repeat difference was less pronounced when the preceding trial was informative (pI/NI sequence) rather than non-informative (pNI/NI sequence). Again, this ERP effect was due to a shift in the repeat trial waveform closer to the switch trial waveform – that is, the difference between switch and repeat waveforms was reduced in pI/NI sequences. These effects emerged around 500ms before the response and well before rLRP onset, suggesting an effect on response activation processes.

Conclusions

The current study replicated no-go/go sequence effects on RT switch cost and extended this by showing that other types of sequences can also affect RT switch cost. While different types of sequence effects affected different levels of the processing continuum, the effect was consistently due to a modulation of repeat trial processing. No-go/go sequence effects emerged in response-locked ERPs well before any response selection was likely to have occurred. We argue that, in the current context, no-go trials involved inhibition of the preparedness to respond. No-go/go sequence effects appeared to arise because, having suppressed the readiness to respond on the preceding trial, it was necessary to reactivate

the response system and implement the prepared C-R rules. This process seems to be more difficult when these rules are the same as those that had been prepared but suppressed on the preceding no-go trial (i.e., repeat trials), and suggests a loss of repetition benefit for implementing the same C-R rules. Sequence effects arising from changes in condition (informative vs. non-informative) also appear to be due to the removal of the response repetition benefit, an effect seen earlier in the trial when the current trial is informative, and later when the current trial is non-informative. Our participants were highly practiced, young and motivated, so it seems that once performance reaches a certain criterion, switch trials are performed as efficiently as possible – it is the repeat trials that suffer as a result of sequence effects, due to the removal of stimulus-based priming that favors repetition.

The current study has also shed light on the nature of the stimulus-locked switch effect. Although we have previously labeled this effect as a 'switch-negativity' or 'D-Neg', we have acknowledged that this effect could well be a modulation of the LPC or P3b complex. In the current study, however, we have seen that this effect can be modulated by the amount of stimulus-based priming interference/facilitation. In Experiment 2, the switch-negativity was superimposed over a number of components, including the P2/N2 and the LPC. The magnitude of the switch/repeat difference was substantially reduced in pNI/I sequences, across these three components. Thus, the stimulus-locked switch effect is not simply a modulation of a single ERP component – i.e. P3b (Kieffaber & Hetrick, 2005). Furthermore, the fact that it was the *repeat* waveform, and not the switch waveform that was modulated by sequence effects is consistent with arguments that the stimulus-locked ERP effect may be more accurately described as reduced negativity for repeat trial rather than increased negativity for switch trials, and may therefore be an index of priming on repeat trials (Barcelo, Munoz-Cepedes, Pozo & Rubia, 2000; Swainson et al., 2006). This interpretation is consistent with the repetition priming literature that shows a reduction in N400 and increase in LPC amplitude for repeated words, the effect being larger with immediate as compared to delayed repetition (e.g. Fabre et al., 2007; Karayanidis et al., 1991). This repeat-positivity is therefore likely to index the amount of repeat trial facilitation due to stimulus-based priming (Swainson et al., 2006).

References

Allport, A., Styles, E. A., & Hsieh, S. (1994). Shifting intentional set: Exploring the dynamic control of tasks. In C. Umilta & M. Moscovitvh (Eds.), *Attention and Performance XV* (pp. 421-452). Cambridge, MA: MIT Press.

Allport, D. A., & Wylie, G. (2000). Task-switching, stimulus response bindings, and negative priming. In S. Monsell & J. Driver (Eds.), Attention and Performance XVIII (pp. 35-70). Cambridge, MA: MIT Press.

Altmann, E.M (2004). The preparation effect in task switching: Carryover of SOA. *Memory & Cognition,* 32, 153-163.

Arrington, C. M., Logan, G. D., & Schneider, D. W. (2007). Separating cue encoding from target processing in the explicit task-cuing procedure: Are there "true" task switch effects? *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 33, 484-502

Astle, D.E., Jackson, G.M., & Swainson, R. (2006). Dissociating neural indices of dynamic cognitive control in advance task-set preparation: An ERP study of task switching. *Brain Research*, 1125, 94-103.

Barcelo, F., Munoz-Cespedes, J.M., Pozo, M.A., & Rubia, F.J. (2000). Attentional set shifting modulates the target P3b response in the Wisconsin card sorting test. *Neuropsychologica, 38,* 1342-1355.

Burle B, Vidal F, & Bonnet M. (2004). Electroencephalographic no-go potentials in a no-movement context: the case of motor imagery in humans. *Neuroscience Letters*, 360, 77–80.

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

De Jong, R. (2000). An intention - activation account of residual switch costs. In S. Monsell & J. Driver (Eds.), Attention and Performance XVIII (pp. 357-376). Cambridge, MA: MIT Press.

Dreisbach, G., Haider, H., & Kluwe, R.H (2002). Preparatory processes in the task-switching paradigm: Evidence from the use of probability cues. *Journal of Experimental Psychology: Learning, Memory & Cognition*, 28, 468-483.

Fabre, L., Lemaire, P. & Grainger, J. (2007). Attentional modulation of masked repetition and categorical priming in young and older adults. *Cognition, 105,* 513-532.

Falkenstein, M., Hoorman, J., & Hohnsbein, J. (2002). Inhibition-related ERP components: Variation with modality, age, and time-on-task. *Journal of Psychophysiology*, 16, 167-175.

Fulham, W.R. (2006). EEG Display (Version 4.10) [Computer software]. Newcastle, Australia.

Gade, M. & Koch, I. (2007). The influence of overlapping response sets on task inhibition. *Memory & Cognition*, 35, 603-609.

Goffaux, P., Phillips, N.A., Sinai, M., & Pushkar, D. (2006). Behavioural and electrophysiological measures of task switching during single and mixed-task conditions. *Biological Psychology,* 72, 278-290.

Hubner, M., Kluwe, R., Luna-Rodriguez, A. & Peters, A. (2004). Task preparation and stimulus-evoked competition. *Acta Psychologica, 115,* 211-234.

Ivanoff, J. & Klein, R. (2001). The presence of a nonresponding effector increases inhibition of return. *Psychonomic Bulletin & Review*, *8*, 307-314.

Karayanidis, F., Andrews, S., Ward, P.B., & McConaghy, N. (1991). Effects of inter-item lag on word repetition: an event-related potential study. *Psychophysiology*, 28, 307-318.

Karayanidis, F., Coltheart, M., Michie, P. T., Murphy, K. (2003). Electrophysiological correlates of anticipatory and post-stimulus components of task-switching. *Psychophysiology*, 40, 329-348.

Kieffaber, P.D., & Hetrick, W.P. (2005). Event-related potential correlates of task switching and switch costs. *Psychophysiology*, 42, 56-71.

Koch, I., & Allport, A. (2006). Cue-based preparation and stimulus-based priming of tasks in task switching. *Memory & Cognition*, 34, 433-444.

Koch, I., & Philipp, A.M. (2005). Effects of response selection on the task repetition benefit in task switching. *Memory & Cognition*, 33, 624-634.

Lavric, A., Mizon, G., & Monsell, S. (in press). Neurophysiological signature of effective anticipatory task-set control: a task-switching investigation. *European Journal of Neuroscience.*

Logan, G.D., & Bundesen, C. (2003). Clever homunculus: Is there an endogenous act of control in the explicit task-cuing procedure? *Journal of Experimental Psychology: Human Perception and Performance*, 29, 575-599.

Low, K.A., & Miller, J. (1999). The usefulness of partial information: Effects of go probability in the choice/nogo task. *Psychophysiology, 36,* 288–297.

Mansfield, E., Galloway, K., Smith, J. Provost, A., Heathcote, A., Karayanidis, F., (submitted) Task-set inhibition as a component of anticipatory reconfiguration: Diffusion model parameters reconcile behavioural and ERP evidence. *Cognitive, Affective, and Behavioral Neuroscience*

Mayr, U., & Keele, S.W. (2000). Changing internal constraints on action: The role of backward inhibition. *Journal of Experimental Psychology: General*, 129, 4-26.

Meiran, N. (1996). Reconfiguration of processing mode prior to task performance. *Journal of Experimental Psychology: Learning, Memory and Cognition,* 22, 1423-1442.

Meiran, N. (2000a). Reconfiguration of stimulus task sets and response task sets during task switching. In S. Monsell & J. Driver (Eds.), Attention and Performance XVIII (pp. 377-399). Cambridge: MIT Press.

Miller, J., & Hackley, S.A. (1992). Electrophysiological evidence for temporal overlap among contingent mental processes. *Journal of Experimental Psychology: General,* 121, 195–209.

Miniussi, C., Marzi, C.A., & Nobre, A.C. (2005). Modulation of brain activity by selective task sets observed using event-related potentials. *Neuropsychologia*, 43, 1514-1528.

Monsell, S. (2003). Task switching. *Trends in Cognitive Sciences*, 7, 134-140.

Monsell, S., & Mizon, G.A. (2006). Can the task-cuing paradigm measure an endogenous task-set reconfiguration process? *Journal of Experimental Psychology: Human Perception and Performance*, 32, 493-516.

Mordkoff, J.T. & Gianros, P.J. (2000). Detecting the onset of the lateralized readiness potential: A comparison of available methods and procedures. *Psychophysiology*, 37, 347-360.

Nicholson, R., Karayanidis, F., Poboka, D., Heathcote, A., & Michie, P. (2005). Electrophysiological correlates of anticipatory task-switching processes. *Psychophysiology*, 42, 540-554.

Nicholson, R., Karayanidis, F., Bumak, E., Poboka, D., & Michie, P.T. (2006). ERPs dissociate the effects of switching task sets and task cues. *Brain Research*, 1095, 107-123.

Nicholson, R., Karayanidis, F., Davies, A., & Michie, P.T. (2006). Components of task-set reconfiguration: Differential effects of 'switch-to' and 'switch-away' cues. *Brain Research*, 1121, 160-176.

Otten, L.J. & Rugg, M.D. (2005). Interpreting event-related brain potentials. In T.C. Handy (Ed.), *Event-Related Potentials* (pp. 3-16). The MIT Press: Cambridge.

Philipp, A. M., Jolicoeur, P., Falkenstein, M. & Koch, I. (2007). Response selection and response execution in task switching: Evidence from a go-signal paradigm. Journal of Experimental Psychology: Learning, Memory & Cognition, 33, 1062-1075.

Posner, M., & Cohen, Y. (1984). Components of visual orienting. In H. Bouma & D. Bouwhuis (Eds.), *Attention and Performance X: Control of Language Processes* (pp. 531-556. Hillsdale, NJ: Erlbaum.

Poulsen, C., Luu, P., Davey, C., & Tucker, D. M. (2005). Dynamics of task sets: Evidence from dense-array event-related potentials. *Cognitive Brain Research*, 24, 133-154.

Rogers, R. D., & Monsell, S. (1995). Costs of a predictable switch between simple cognitive tasks. *Journal of Experimental Psychology*, 124, 207 - 231.

Rubia, K., Russell, T., Overmeyer, S., Brammer, M., Bullmore, E., Sharma, T., et al. (2001). Mapping motor inhibition: conjunctive brain activations across different versions of go/no-go and stop tasks. *NeuroImage, 13*, 250-261.

Rubinstein, J. S., Meyer, D. E., & Evans, E. (2001). Executive control of cognitive processes in task switching. Journal of Experimental Psychology: *Human Perception and Performance*, 27, 763-797.

Rugg, M. D. & Coles, M. G. H. (1995). *Electrophysiology of mind: Event-related brain potentials and cognition*. New York: Oxford University Press.

Rushworth, M. F. S., Passingham, R. E., & Nobre, A. C. (2002). Components of switching intentional set. *Journal of Cognitive Neuroscience*, 14, 1139-1150.

Rushworth, M. F. S., Passingham, R. E., & Nobre, A. C. (2005). Components of attentional set-switching. *Experimental Psychology*, 52, 83-98.

Schuch, S., & Koch, I. (2003). The role of response selection for inhibition of task sets in task shifting. *Journal of Experimental Psychology: Human Perception and Performance*, 29, 92-105.

Schwarzenau, P., Falkenstein, M., Hoorman, J., & Hohnsbein, J. (1998). A new method for the estimation of the onset of the lateralized readiness potential (LRP). *Behavior Research Methods, Instruments, & Computers*, 30, 110-117.

Semlitsch, H. V., Anderer, P., Schuster, P., & Presslich, O. (1986). A solution for reliable and valid reduction of ocular artefacts. *Psychophysiology*, 23, 695-703.

Sinai, M., Goffaux, P., & Phillips, N.A. (2007). Cue- versus response-locked processes in backward inhibition: Evidence from ERPs. *Psychophysiology*, 44, 596-609.

Smith, J.L., Johnstone, S.J., & Barry, R.J. (2006). Response priming in the go/nogo task: The N2 reflects neither inhibition nor conflict. *Clinical Neurophysiology*, 118, 343-355.

Strayer, D.L. & Kramer, A.F. (1994). Strategies and automaticity: I. Basic findings and conceptual framework. *Journal of Experimental Psychology: Learning, Memory and Cognition, 28,* 318-341.

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

Swainson, R., Jackson, S.R. & Jackson, G.M. (2006). Using advance information in dynamic cognitive control: An ERP study of task-switching. *Brain Research*, 1105, 61-72.

Ulrich, R., & Miller, J. (2001). Using the jack-knife-based scoring method for measuring LRP onset effects in factorial designs. *Psychophysiology*, 38, 816–827.

Vasey, M. W., & Thayer, J. F. (1987). The continuing problem of false positives in repeated measures ANOVA in psychophysiology: A multivariate solution. *Psychophysiology*, 24, 479-486.

Verbruggen, F., Liefooghe, B., Szmalec, A. & Vandierendonck, A. (2005). Inhibiting responses when switching: does it matter? *Experimental Psychology,* 52, 125-130.

Verbruggen, F., Liefooghe, B., & Vandierendonck, A. (2006). Selective stopping in task-switching: the role of response selection and response execution. *Experimental Psychology*, 53, 48-57.

Verbruggen, F., Liefooghe, B., Vandierendonck, A., & Demanet, J. (2007). Short cue presentations encourage advance task preparation: a recipe to diminish the residual switch cost. *Journal of Experimental Psychology: Learning Memory and Cognition*, 33, 342-356.

Waszak, F., Hommel, B., & Allport, A. (2003). Task-switching and long term priming: Role of episodic stimulus-task bindings in task-shift costs. *Cognitive Psychology*, 46, 361-413.

Wylie, G., & Allport, A. (2000). Task switching and the measurement of 'switch costs'. *Psychological Research*, 63, 212-233.

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Figure Legends

Figure 1: A. RT and RT switch cost and B. proportion error rate and error switch cost for informative and non-informative trials for each previous trial type. Note that, with the exception of pI/NI sequences, both RT and error switch cost reduced when the preceding trial was different to the current one, and this was largely due to increased RT and error scores for repeat trials. Standard error bars are shown. Asterisks signify a significant switch cost at $p<.05$. Abbreviations: pI, previous informative; pNI, previous non-informative; pNG, previous no-go.

Figure 2: Top: Cue-locked waveforms for informative switch, informative repeat and non-informative trials for each previous trial type at three midline sites. The first black bar represents the early informative positivity (informative – non-informative difference over 300-400ms) and the second represents the later switch positivity (informative switch – informative repeat difference 450-550ms). Note that the early informative positivity was larger for both informative switch and informative repeat cues, relative to non-informative cues, whereas the late switch positivity was larger for informative switch compared to repeat cues. These differential positivities were not affected by previous trial type. Stimulus onset is depicted by the broken line and negative is plotted up. Bottom: Scalp topographies of the informative positivity and the switch-positivity shows similar distribution for each type of previous trial.

Figure 3: A: Stimulus-locked ERPs for informative and non-informative trials at three midline sites. Repeat and switch trials for each type of previous trial are superimposed. Black bars represent the early (300-400ms) and late (550-650ms) analysis windows. Note the absence of the expected parietal switch-repeat negativity. NG/I sequences resulted in larger frontal early and late positivities, but there were no differential effects of sequence on switch vs. repeat waveforms. B: Stimulus-locked LRP onset was not modulated by any task parameters. For abbreviations, see Figure 1 legend.

Figure 4: Stimulus-locked waveforms for informative and no-go trials averaged over previous trial and trial type. Note the large frontal P3 after stimulus onset for no-go but not informative trials. The scalp

topography of the frontal P3 (no-go – informative difference 350-450ms) is compatible with that generally observed for no-go P3.

Figure 5: Response-locked LRPs (A) and ERPs (B) at three midline sites for informative and non-informative trials. rLRP onset is depicted by the broken line and regions of mean amplitude measures are shown as black bars. For abbreviations, see Figure 1 legend. No-go/go sequences showed reduced centroparietal negativity as early as 600ms pre-response for informative trials and 400ms pre-response for non-informative trials. This effect was more pronounced for informative repeat trials that showed the largest behavioral modulation by no-go/go sequence effects.

Figure 6: A. RT and RT switch cost, B. proportion error rate and error switch cost for informative and non-informative trials and for each previous trial type in the randomized group. Note that RT switch cost was smaller when the preceding trial was different to the current one (i.e., NI/I and I/NI as compared to I/I and NI/NI, respectively). Standard error bars are shown. Asterisks signify that the switch cost was significant at $p<.05$. For abbreviations, see Figure 1 legend.

Figure 7: Top: Cue-locked waveforms for informative switch, informative repeat and non-informative trials for each previous trial type at three midline sites. See Figure 2 legend for figure details. Note that the early informative positivity extends longer and overlaps the late switch positivity. The switch-repeat difference was again not affected by previous trial. Bottom: Scalp topographies for the informative-positivity and the switch-positivity.

Figure 8: A: Stimulus-locked ERPs for informative and non-informative trials at three midline sites and scalp topography of the switch negativity (informative switch-repeat difference 500-600ms). Note that the large differential switch negativity (informative switch - informative repeat) is only evident for I/I trials. See Figure 3 legend for figure details. B: Stimulus-locked LRPs.

Figure 9: Response-locked LRPs (A) and ERPs (B) at three midline sites are shown for informative and non-informative trials. Note sequence effects on non-informative repeat trials. Specifically, non-informative repeat trials were more similar to non-informative switch trials when the preceding trial was informative. See Figure 5 legend for figure details.

Figure 10: A. RT and RT switch cost, B. proportion error rate and error switch cost for blocked and randomised condition groups. Standard error bars are shown. Asterisks signify that the switch cost was significant at $p<.05$. Abbreviations: Inf – informative, Non-inf: non-informative.

Figure 11: Cue-locked (A) and stimulus-locked (B) waveforms at three midline sites for blocked and randomised groups. Regions of mean amplitude measures are depicted by black bars. There was no significant group main effect or interaction on any measure.

Figure1

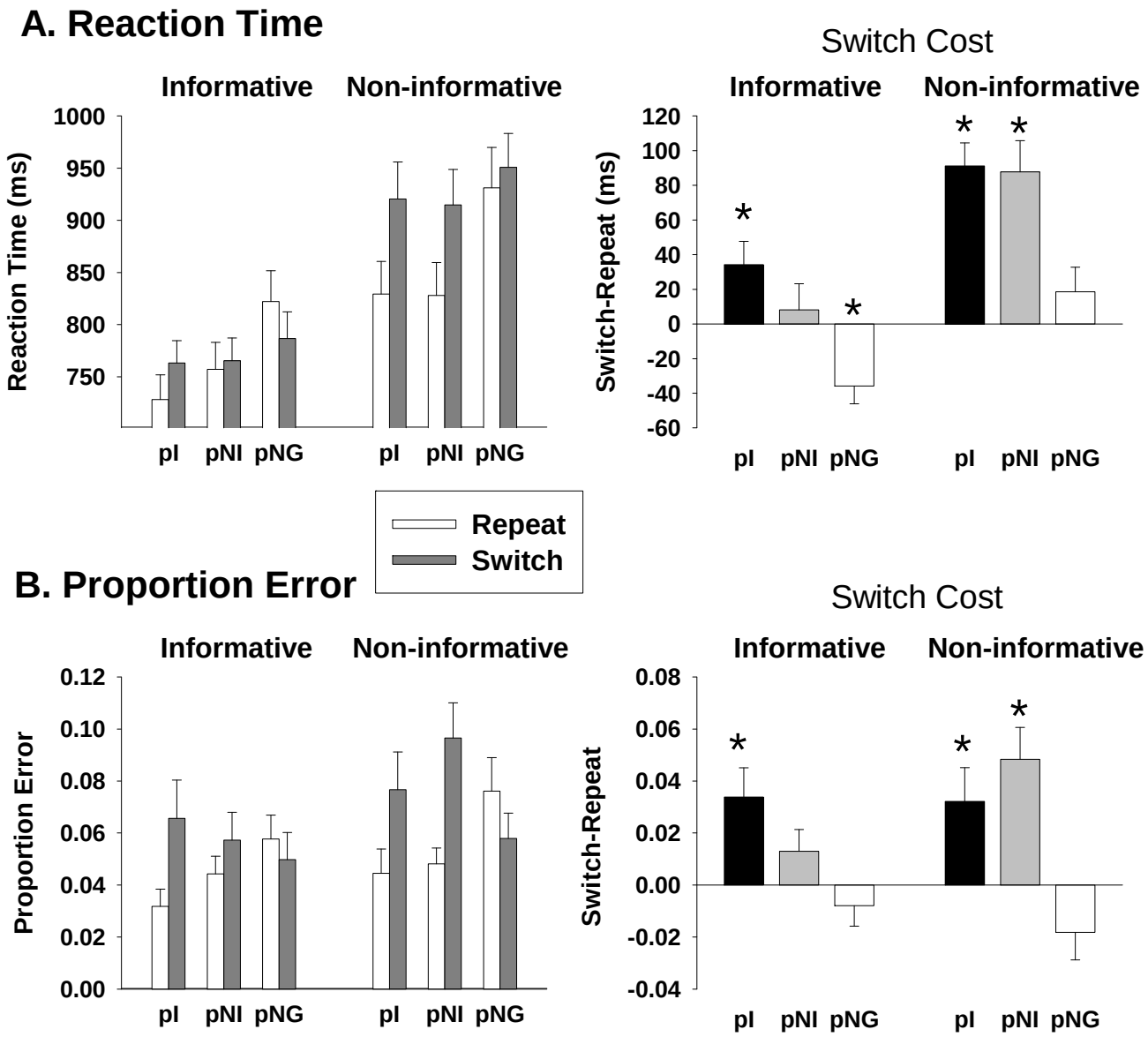
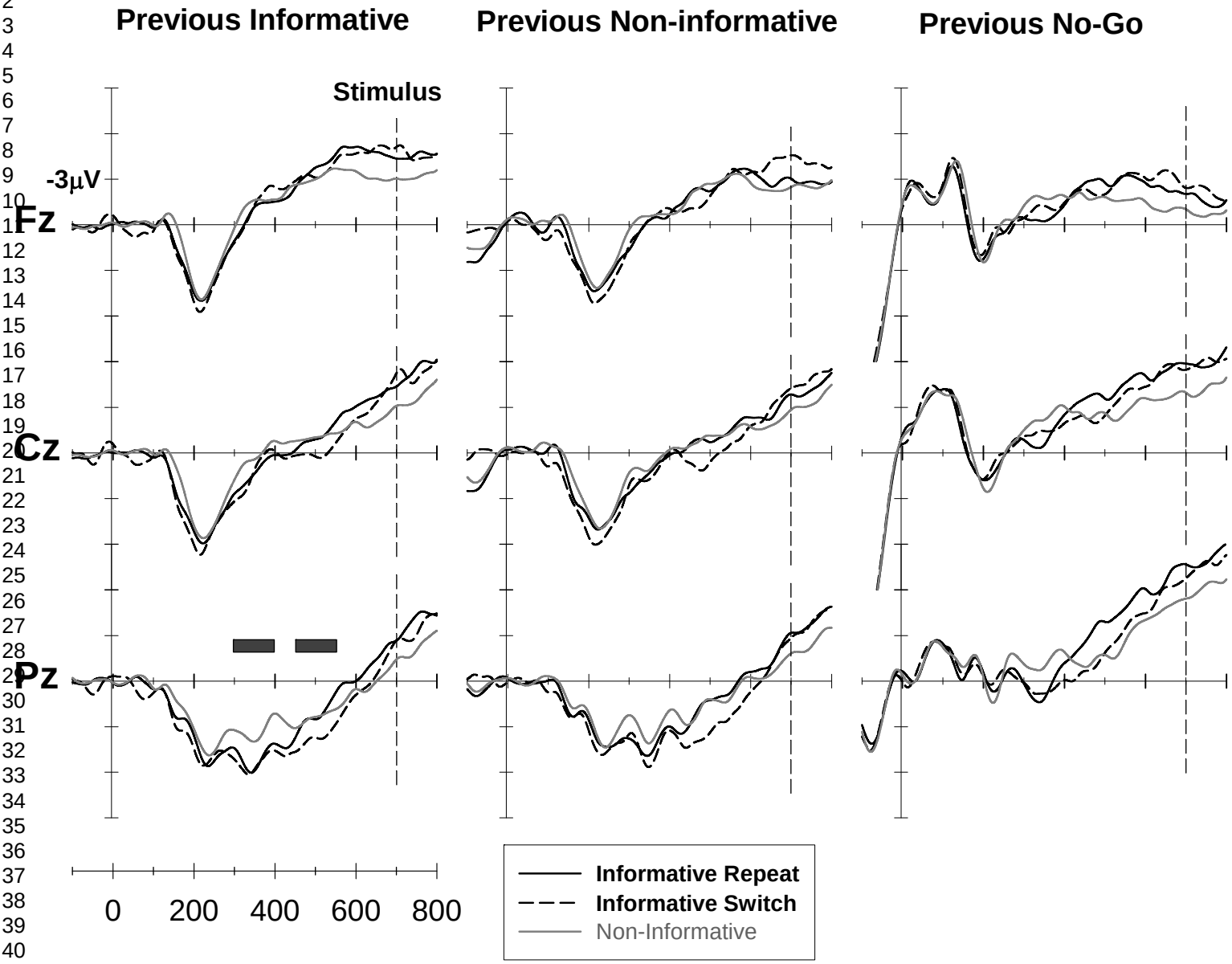

A. Reaction Time
Informative
Non-informative
Reaction Time (ms)
1000
950
900
850
800
750
pI
pNI
pNG
Switch Cost
Switch-Repeat (ms)
120
100
80
60
40
20
0
-20
-40
-60
*
Repeat
Switch
B. Proportion Error
Proportion Error
0.12
0.10
0.08
0.06
0.04
0.02
0.00
Switch-Repeat
0.08
0.06
0.04
0.02
0.00
-0.02
-0.04

Figure 2

Cue-Locked Waveforms



Scalp Topography

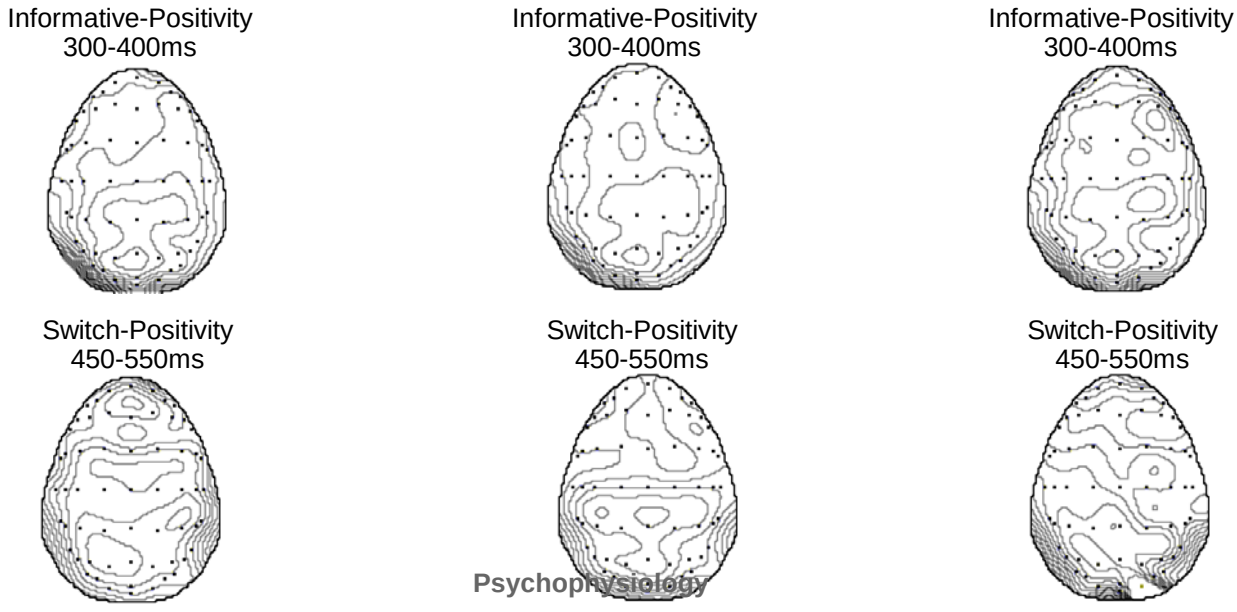


Figure 3

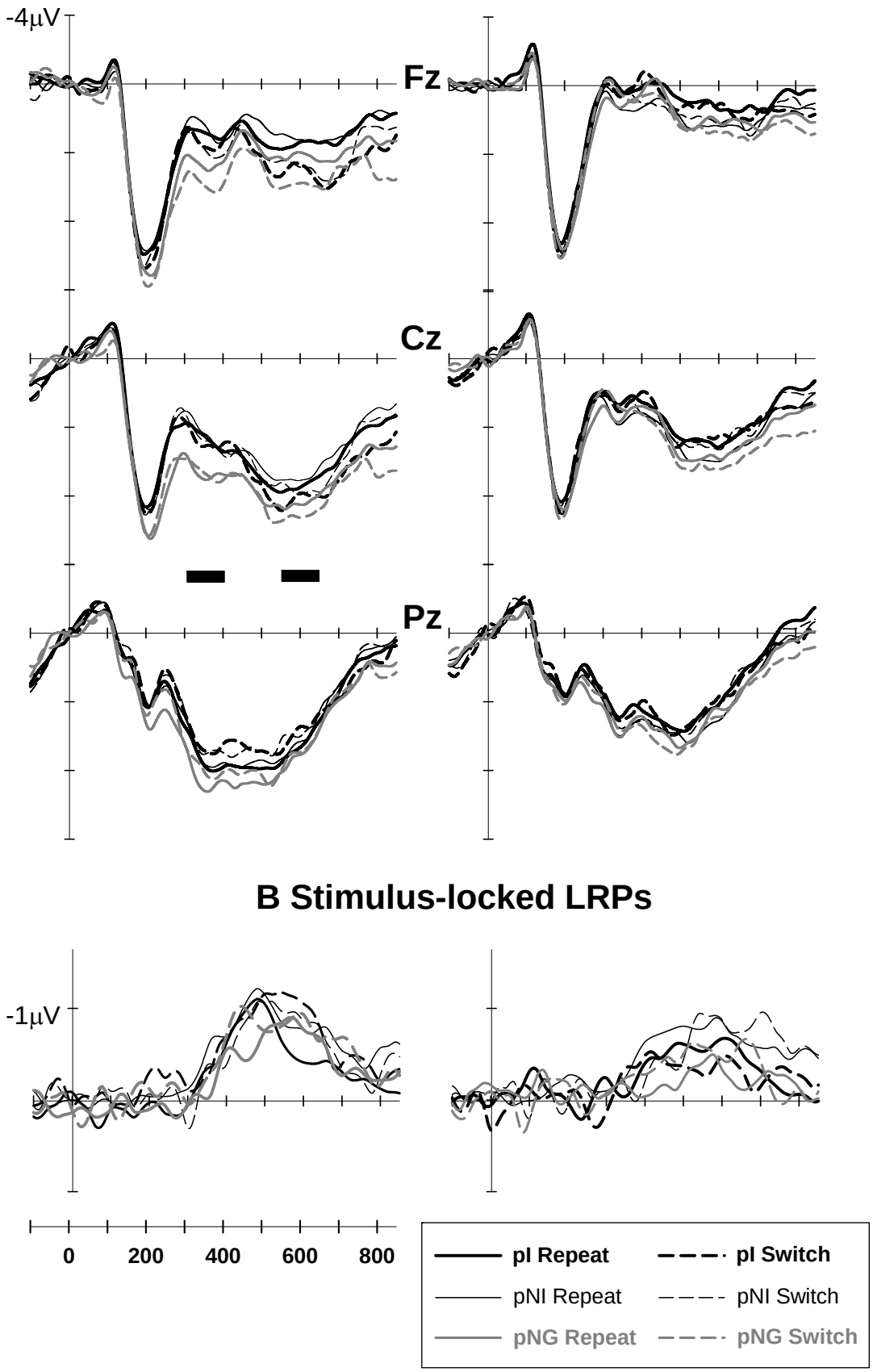

Informative
Non-Informative
A Stimulus-locked ERPs
-4μV
Fz
Cz
Pz
B Stimulus-locked LRPs
-1μV
0
200
400
600
800
pI Repeat
pI Switch
pNI Repeat
pNI Switch
pNG Repeat
pNG Switch

Figure 4

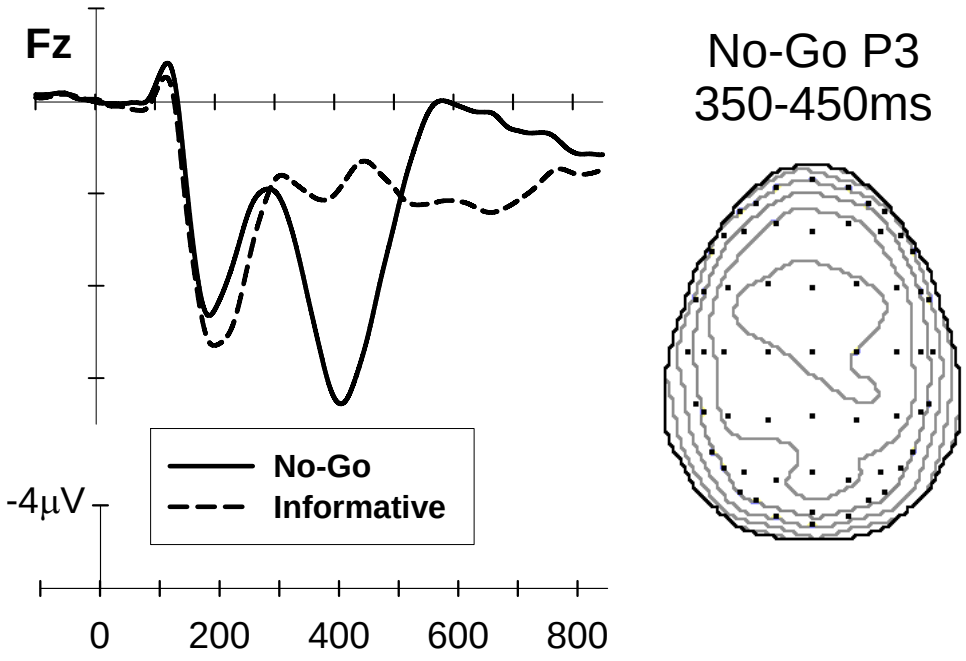

Frontal N2/P3 No-Go
Fz
-4μV
No-Go
Informative
0
200
400
600
800
No-Go P3
350-450ms

Figure 5

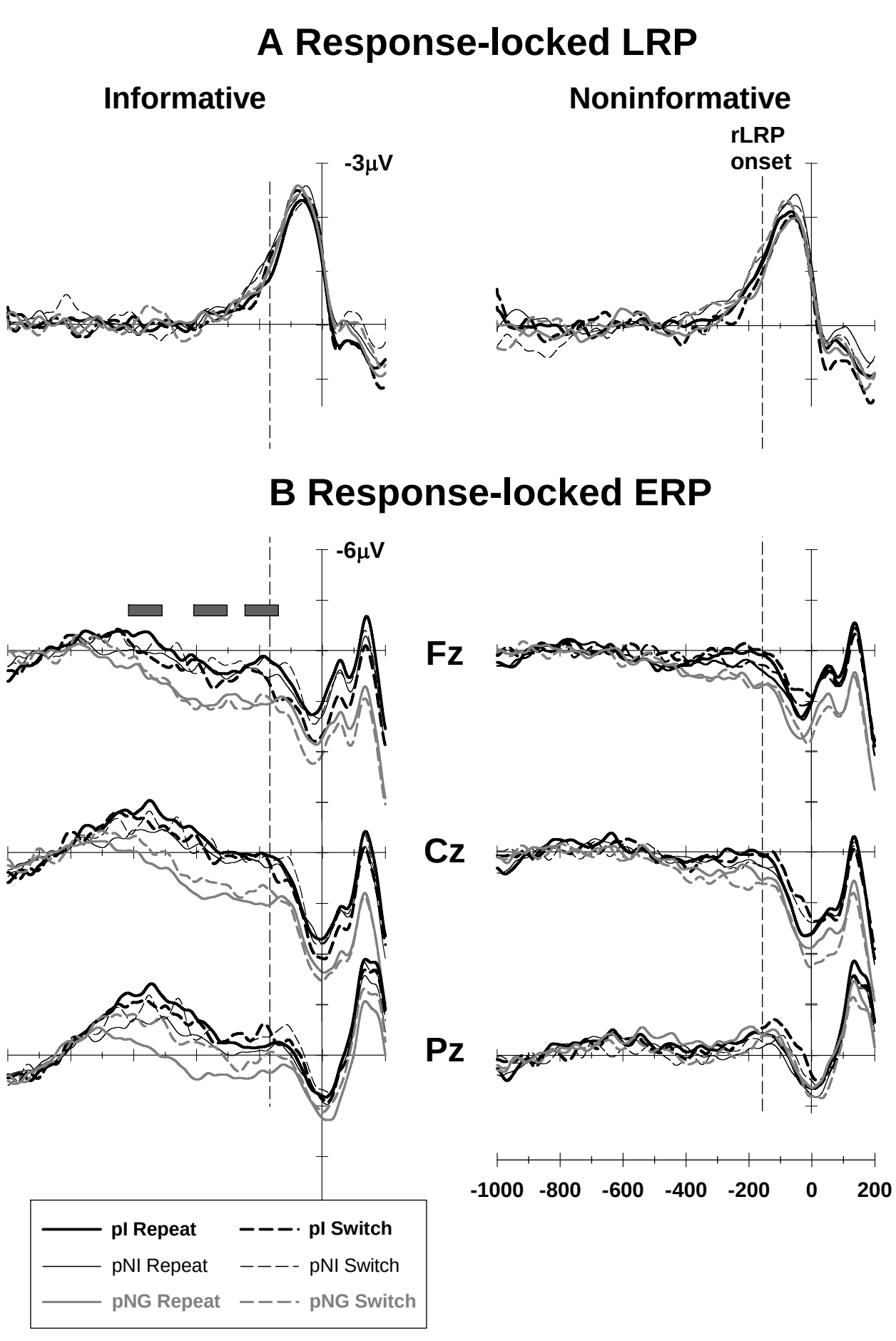

A Response-locked LRP
Informative
Noninformative
-3μV
rLRP
onset
B Response-locked ERP
-6μV
Fz
Cz
Pz
-1000
-800
-600
-400
-200
0
200
pI Repeat
pI Switch
pNI Repeat
pNI Switch
pNG Repeat
pNG Switch

Figure 6

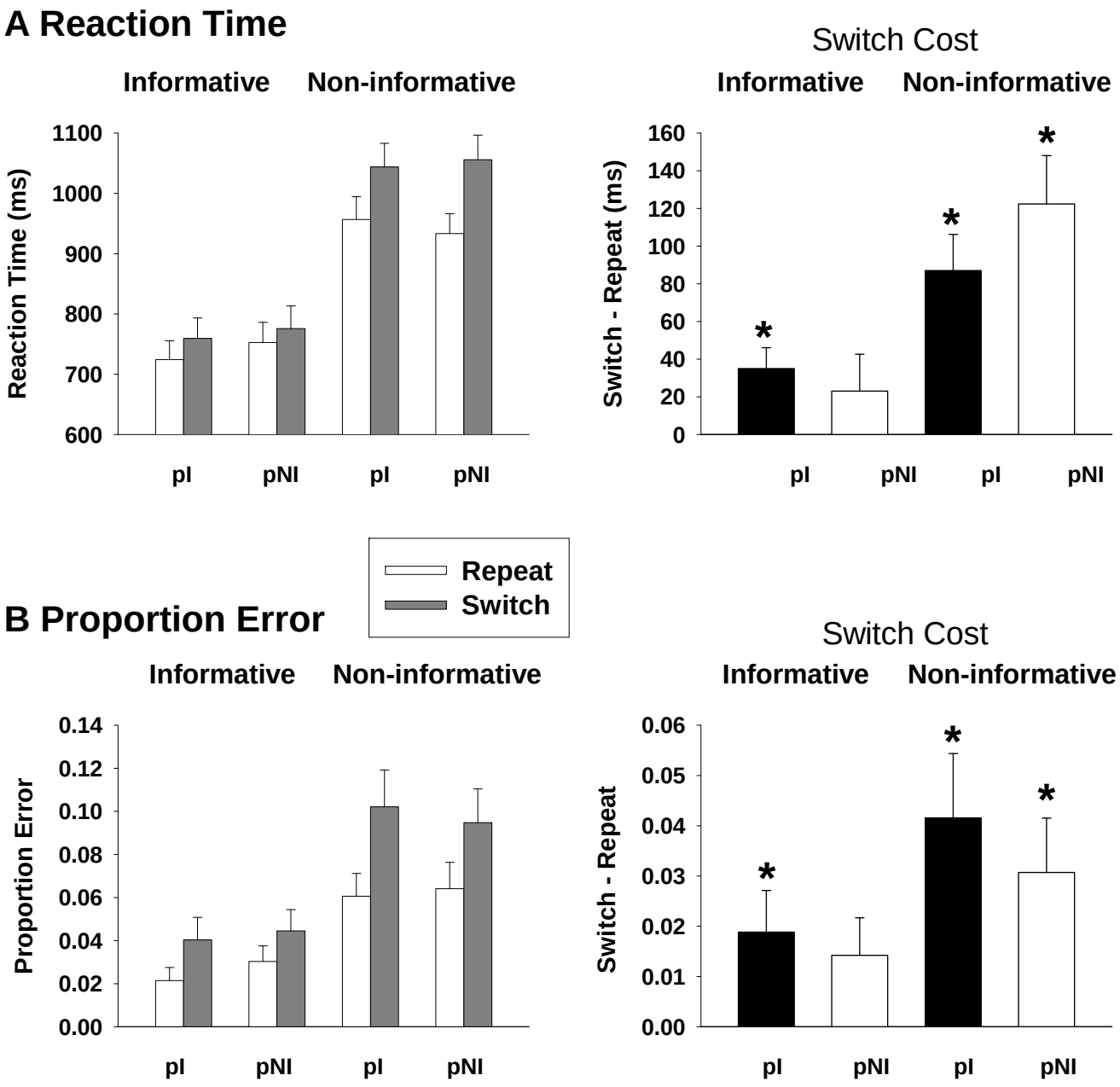
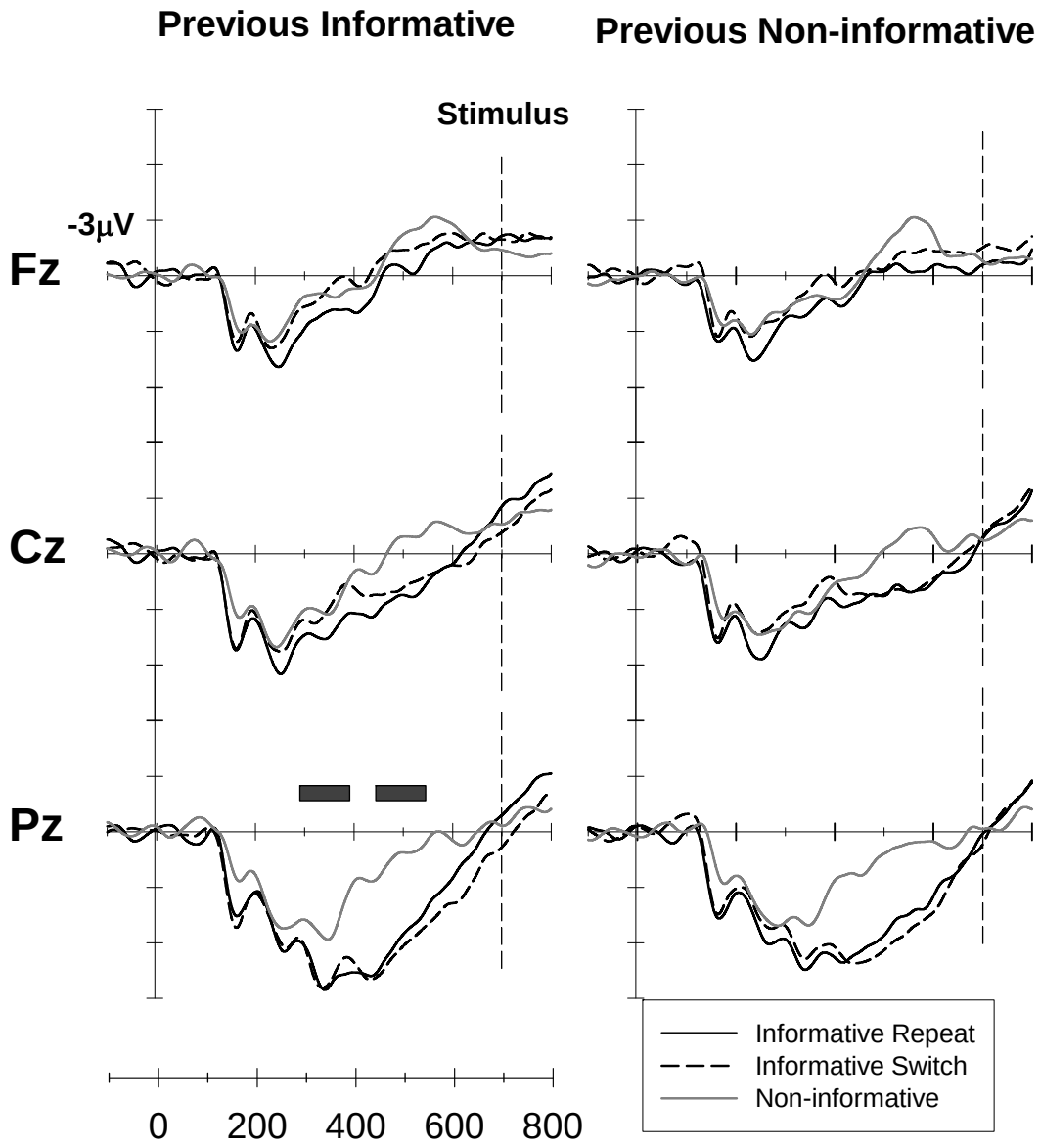

A Reaction Time
Informative
Non-informative
Reaction Time (ms)
1100
1000
900
800
700
600
pI
pNI
pI
pNI
Switch Cost
Informative
Non-informative
Switch - Repeat (ms)
160
140
120
100
80
60
40
20
0
*
*
*
pI
pNI
pI
pNI
Repeat
Switch
B Proportion Error
Informative
Non-informative
Proportion Error
0.14
0.12
0.10
0.08
0.06
0.04
0.02
0.00
pI
pNI
pI
pNI
Switch Cost
Informative
Non-informative
Switch - Repeat
0.06
0.05
0.04
0.03
0.02
0.01
0.00
*
*
*
pI
pNI
pI
pNI

Figure 7

Cue-Locked Waveforms



Scalp Topography

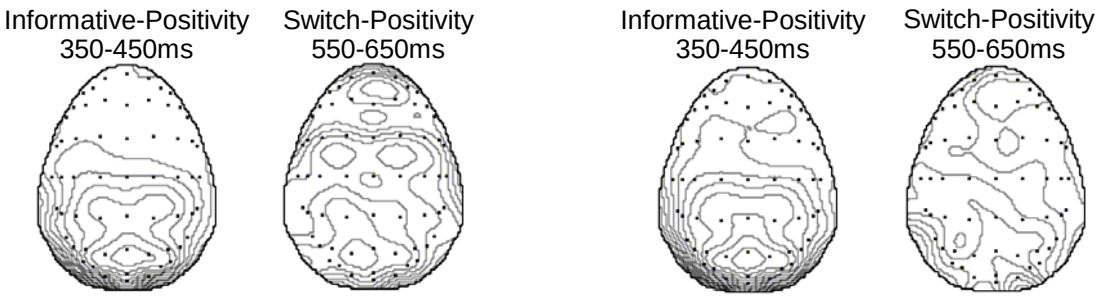


Figure 8

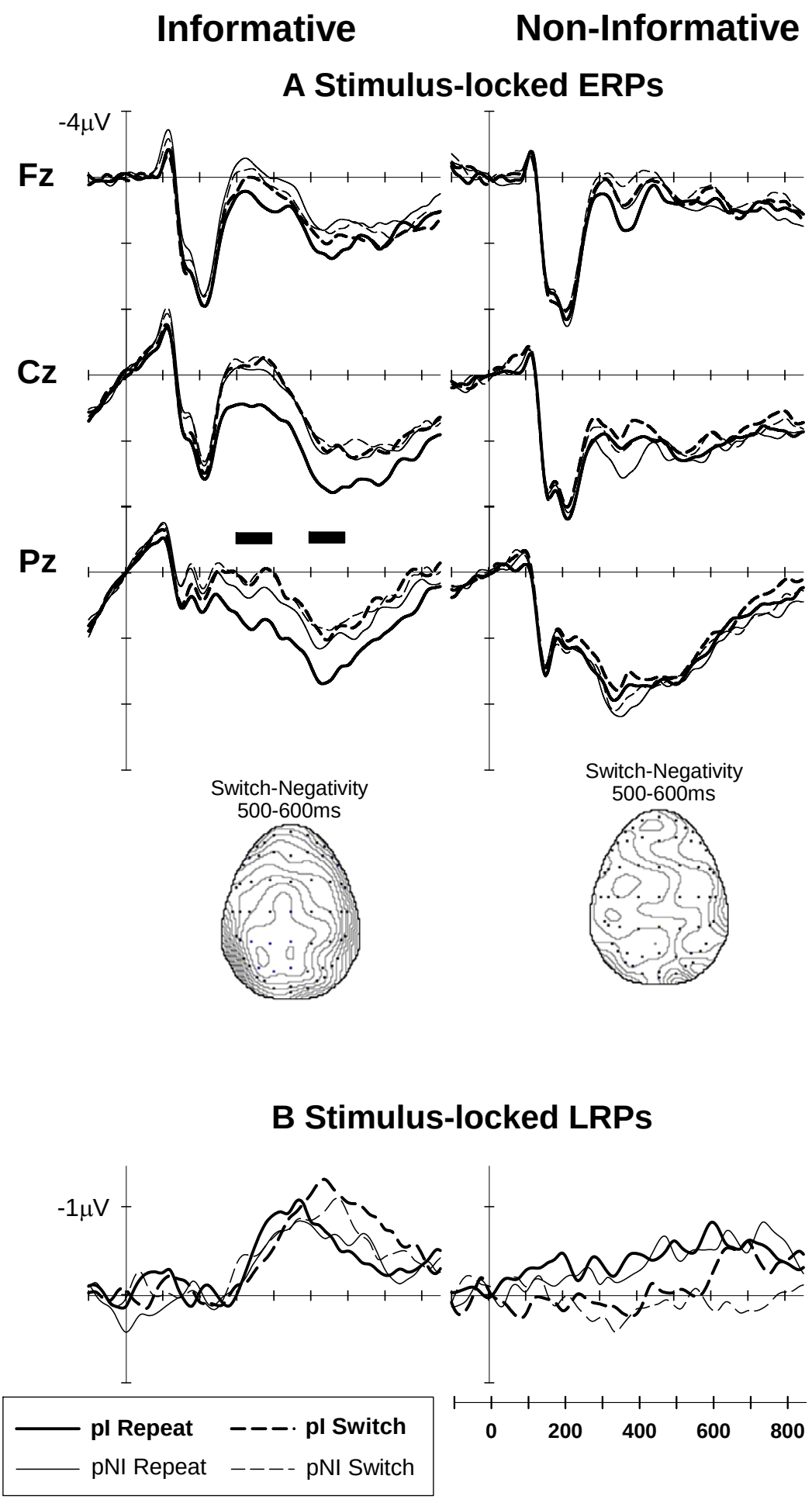

Informative
Non-Informative
A Stimulus-locked ERPs
-4μV
Fz
Cz
Pz
Switch-Negativity
500-600ms
Switch-Negativity
500-600ms
B Stimulus-locked LRPs
-1μV
pI Repeat
pI Switch
pNI Repeat
pNI Switch
0
200
400
600
800

Figure 9

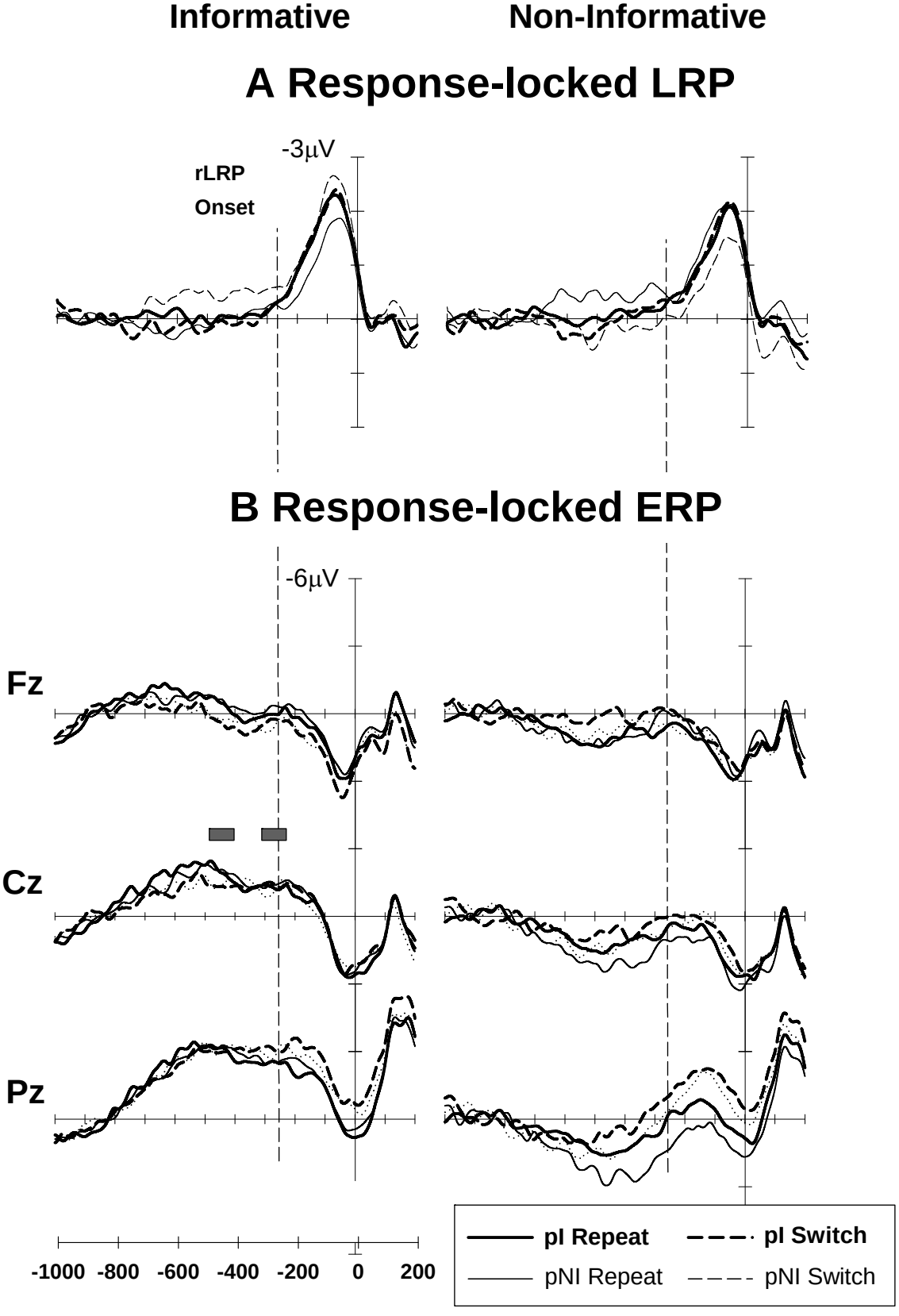

Informative
Non-Informative
A Response-locked LRP
-3μV
rLRP
Onset
B Response-locked ERP
-6μV
Fz
Cz
Pz
-1000 -800 -600 -400 -200 0 200
pI Repeat
pI Switch
pNI Repeat
pNI Switch

Figure 10

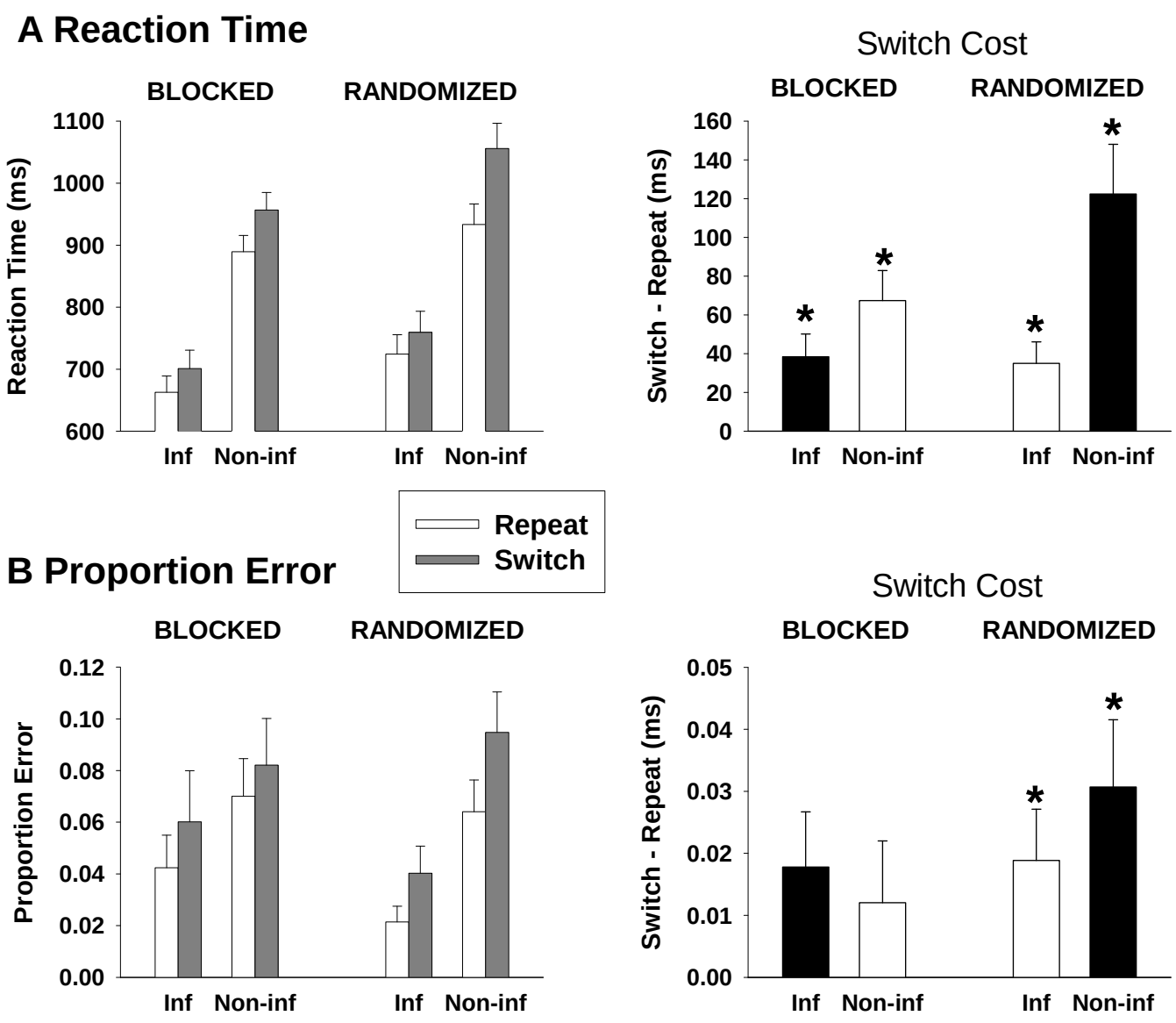
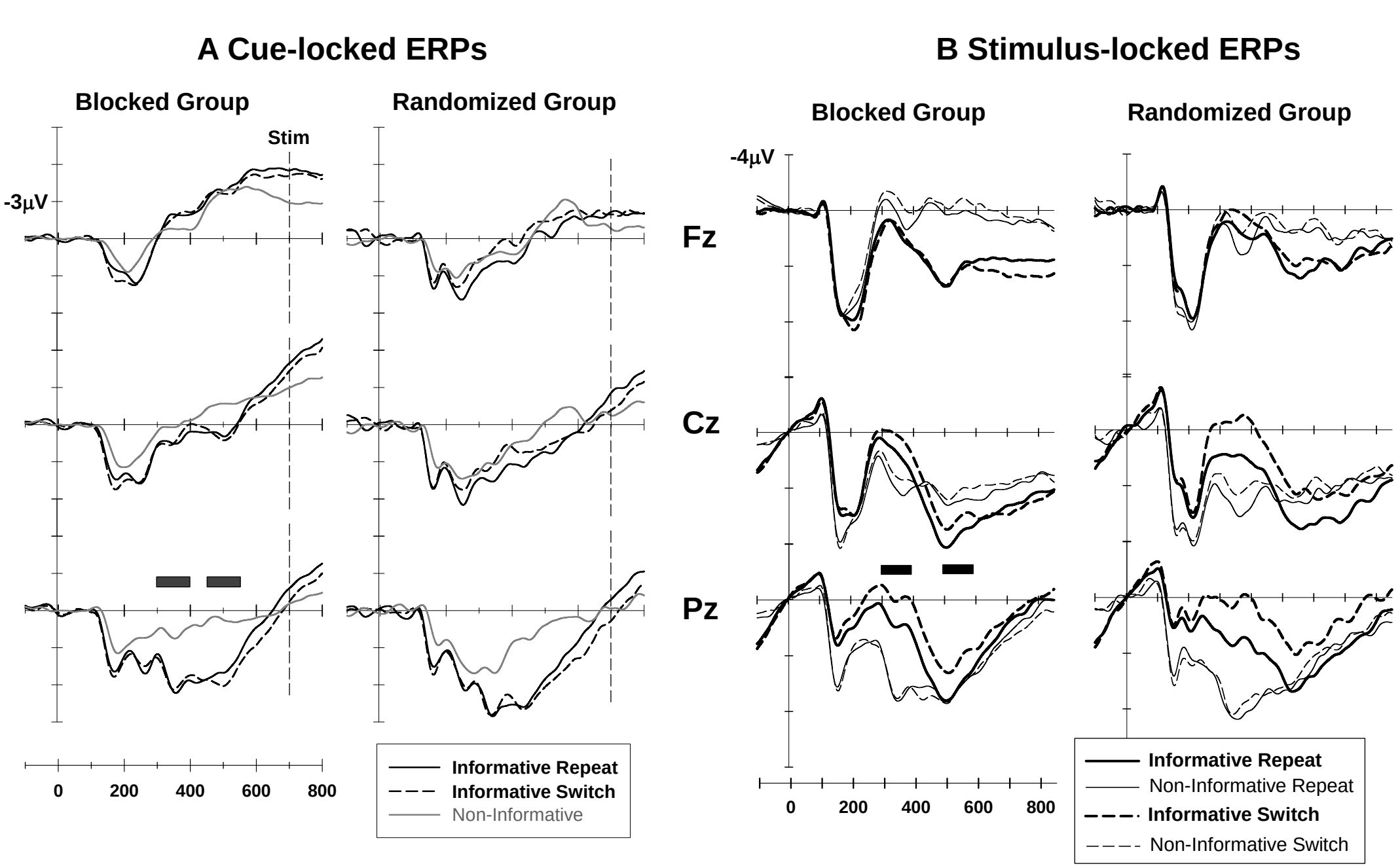

A Reaction Time
BLOCKED
RANDOMIZED
Reaction Time (ms)
1100
1000
900
800
700
600
Inf
Non-inf
Inf
Non-inf
Switch Cost
BLOCKED
RANDOMIZED
Switch - Repeat (ms)
160
140
120
100
80
60
40
20
0
Inf
Non-inf
Inf
Non-inf
Repeat
Switch
B Proportion Error
BLOCKED
RANDOMIZED
Proportion Error
0.12
0.10
0.08
0.06
0.04
0.02
0.00
Inf
Non-inf
Inf
Non-inf
Switch Cost
BLOCKED
RANDOMIZED
Switch - Repeat (ms)
0.05
0.04
0.03
0.02
0.01
0.00
Inf
Non-inf
Inf
Non-inf

Figure 11


A Cue-locked ERPs
Blocked Group
Randomized Group
Stim
-3μV
0
200
400
600
800
Informative Repeat
Informative Switch
Non-Informative
B Stimulus-locked ERPs
Blocked Group
Randomized Group
-4μV
Fz
Cz
Pz
0
200
400
600
800
Informative Repeat
Non-Informative Repeat
Informative Switch
Non-Informative Switch