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RESEARCH ARTICLE

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Switching, plasticity, and prediction in a saccadic task-switch paradigm

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Abstract Several cognitive processes are involved in task-switching. Using a prosaccade/antisaccade paradigm, we manipulated both the interval available for preparation between the cue and the target and the predictability of trial sequences, to isolate the contributions of foreknowledge, an active switching (reconfiguration) process, and passive inhibitory effects persisting from the prior trial. We tested 15 subjects with both a random and a regularly alternating trial sequence. Half of the trials had a short cue–target interval of 200 ms, and half a longer cue–target interval of 2,000 ms. When there was only a short preparatory interval, switching increased the latencies for both prosaccades and antisaccades. With a long preparatory interval, switching was associated with a smaller latency increase for prosaccades and, importantly, a paradoxical reduction in latency for antisaccades. Foreknowledge of a predictable sequence did not allow subjects to reduce switch costs in the manner that a long preparatory cue–target interval did. In the trials with short preparatory intervals, the effects on latency attributable to active reconfiguration processes were similar for prosaccades and antisaccades. We propose a model in which the passive inhibitory effects that persist from the prior saccadic trial are due not to task-set inertia, in which one task-set inhibits the opposite task-set, but to inhibition of the saccadic response-system by the antisaccade task, to account for the paradoxical set-switch benefit for antisaccades at long cue–target intervals. Our findings regarding foreknowledge show that previous studies used to support task-set inertia may have conflated the effects of both active reconfiguration and passive inhibitory processes on latency. While our model of response-system plasticity can explain a number of effects of dominance asymmetry in switching, other models fail to account for the paradoxical set-switch benefit for antisaccades.

Keywords Task switch · Prediction · Foreknowledge · Reconfiguration · Antisaccade

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Introduction

In studies of cognitive function, the response on a trial is influenced by events in the previous trial (Fecteau and Munoz 2003). One important type of interaction between current and prior trials is the task-switch, when set of tasks performed differs between the two trials. The ability to switch between task-sets is an important manifestation of control over cognitive operations. Implementing such a switch incurs costs to the system, usually measured as increases in errors and reaction time. The study of task-switch costs is of interest because these costs reflect the various factors at play during the operation of control processes.

There have been several hypotheses about the origins of task-switch costs. One model proposes that an active reconfiguration process switches the system from one

task to another. At least part of this reconfiguration can be done in advance of a target, if an instructional cue precedes the target by an interval long enough to allow preparation. This active preparatory switching has been labeled "advance reconfiguration" (Rogers and Monsell 1995; Monsell et al. 2000). A second model, "task-set inertia," proposes that activation of one task-set inhibits the competing task-set, and that this inhibition persists into following trials. If the next trial requires the subject to switch to the competing task-set, their response will need to overcome this persistent inhibition, leading to an increase in latency (Allport et al. 1994). These two models are not mutually exclusive. For example, it is possible to separate active reconfiguration effects from passive inhibitory effects of the prior task by independently manipulating two intervals. First, varying the interval between the instructional cue and the target affects preparation time, and can reveal latency variations due to advance reconfiguration. Second, varying the interval between the prior response and the current trial's target reveals the gradual decline in the inhibitory effects of the prior task. A study that manipulated both intervals independently produced evidence of both active reconfiguration and passive inhibitory effects persisting from the prior trial (Meiran 2000).

Evidence for task-set inertia derives mainly from studies of switching between task-sets with a dominance asymmetry. Dominance asymmetry exists when one task-set is easier to perform than the other, through learning, habit, or compatibility. These studies found that switching from the dominant to the non-dominant task-set has less latency cost than switching in the reverse direction (Allport et al. 1994; Wylie and Allport 2000). The proffered explanation is that a non-dominant task-set requires significant inhibition of the dominant task-set, and that this inhibition persists into the following trials (Fig. 1a). Because there is less need to suppress the non-dominant task-set during a dominant response, there is less inhibition to persist into the next trial. The result is less switch cost when switching from dominant to non-dominant responses.

Asymmetric switch costs are not always found, though, and their existence may depend on the degree of dominance asymmetry (Monsell et al. 2000; Yeung and Monsell 2003). We previously studied task-switching with a paradigm that randomly alternated prosaccades and antisaccades (Cherkasova et al. 2002; Manoach et al. 2002). A prosaccade trial requires the subject to simply look towards a suddenly appearing target, a natural response made almost every waking minute by humans. An antisaccade trial requires the subject to look in the direction opposite to the target (Hallett 1978), a response seldom, if ever, made in normal life. Thus, this pairing of prosaccades and antisaccades has one of the most extreme dominance asymmetries possible. Our studies produced a novel finding. Switching to the non-dominant antisaccade task was associated not with reduced switch cost, but with a paradoxical task-switch benefit. That is, antisaccades following a prosaccade had

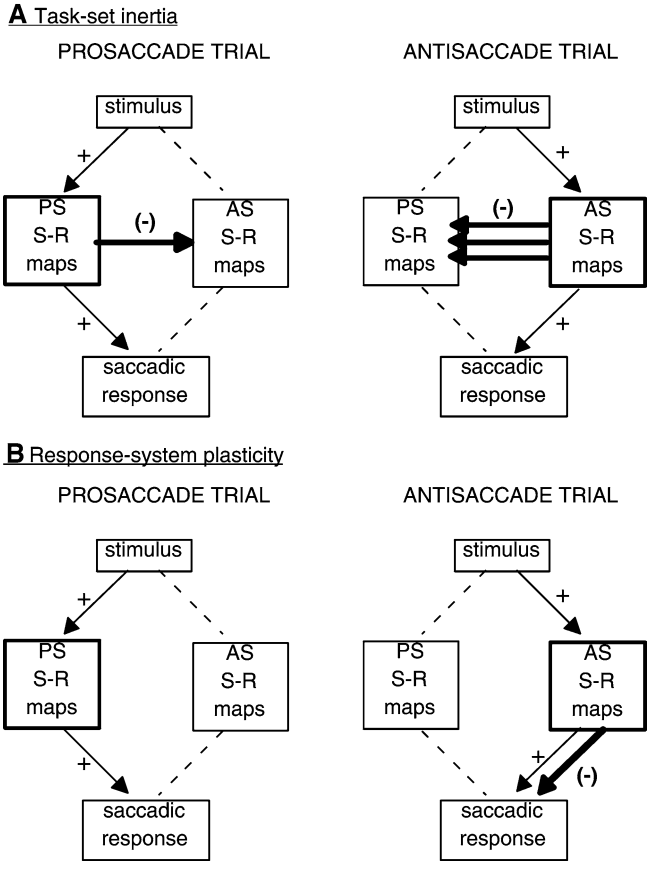

Fig. 1 Different models of inhibition during saccadic switching. The diagrams show proposed flows of activation and inhibition during execution of a prosaccade (*left*) or an antisaccade (*right*). Processing of a stimulus leads to one of two possible stimulus–response (*S–R*) mappings, one generating prosaccades (*PS*) and one generating antisaccades (*AS*), the outputs of which are passed to the saccadic response-system to generate an action. Arrows with plus signs indicate activation, bold arrows with negative signs indicate inhibition, and dashed lines indicate inactive pathways. **a** Task-set inertia hypothesis . In the left graph, a prosaccade needs only slight inhibition of the weak AS S–R map. In the right graph, an antisaccade requires much stronger inhibition of the dominant PS S–R maps. Persistence of these inhibitory effects leads to switch costs in the next trial, more when going from an AS to a PS than in the other direction. However, this cannot account for the fact that antisaccades have reduced latencies when preceded by a prosaccade. **b** Response-system plasticity. This proposes that activating an AS S–R-map leads to inhibition of the saccadic response-system in general. Persistence of this type of inhibition will delay any saccade in the next trial, whether prosaccade or antisaccade. This leads to a residual switch cost for prosaccades but a switch benefit for antisaccades, since antisaccades preceded by prosaccades face less persistent inhibition from the prior trial than antisaccades preceded by antisaccades

shorter latencies than antisaccades following an antisaccade, a result replicated recently by other investigators (Fecteau et al. 2004). We label this switch benefit as "paradoxical" because all models of task-switching, including active reconfiguration and task-set inertia, predict that repeating a set should be advantageous and switching should always incur a cost.

One possible explanation of this task-switch benefit is that the antisaccade task-set inhibits not the prosaccade

task-set, as task-set inertia supposes, but the saccadic response-system itself (Fig. 1b). Persistence of antisaccadic inhibition of the response-system would increase the latency of any future response, whether prosaccade or antisaccade. Physiologic data support the possibility of this type of inhibition. In monkeys, antisaccades are associated with a generalized depression of preparatory (pre-target) activity in saccade-related motor structures such as the frontal eye field (Everling and Munoz 2000) and the superior colliculus (Everling et al. 1999). (Such depression has been invoked to explain other saccadic data in cognitive psychology, such as the reduced effect of fixation offset for antisaccades (Forbes and Klein 1996).) As the characteristics of the prior trial can influence the preparatory activity in the next trial (Dorris et al. 2000), it is plausible that antisaccadic depression of pre-target activity in the saccadic response-system could persist from one trial to the next. Since depression of pre-target activity is directly related to saccadic latency (Dorris and Munoz 1998), a persistent antisaccadic depression could generate the pattern of latency effects we found. Following Dorris et al. (2000), we suggest that this effect reflects ''response-system plasticity.''

Our results raise the question of why other studies of task-switching with dominance asymmetry failed to find the paradoxical task-switch benefit for antisaccades that we found. One possibility is variations in methodology. In particular, the saccadic and the Stroop studies used different strategies to allow subjects to prepare for an upcoming switch. The goal of these studies was to study what is sometimes referred to as ''residual switch cost,'' the remaining switch-related differences in latency after active reconfiguration has been completed. An effective technique to remove active reconfiguration effects is to provide a long preparatory interval between the instructional cue and the target (Meiran 2000). This allows subjects to prepare the switch prior to the target's appearance. The remaining effects on latency can then be attributed to persistent inhibitory effects from the prior trial. Our studies followed this strategy, using random trial sequences with long intervals between the cue and the target in each trial. The Stroop studies, on the other hand, provided either no cue (Allport et al. 1994) or used target location as the cue (Wylie and Allport 2000), essentially providing no interval between the cue and the target. Instead, they relied on a predictable sequence of task-sets to eliminate advance reconfiguration from their data. Their assumption was that, possessing foreknowledge of the coming task-set because of its predictability, subjects would be able to actively reconfigure the switch prior to the target, in the same way that they do with a long cue-triggered preparatory interval.

However, in the few studies that have assessed the effects of predictable sequencing of task-sets (but using paradigms without dominance asymmetries), prediction has increased switch costs, not reduced them (Sohn and Anderson 2001; Tornay and Milan 2001). These findings challenge the assumption that prediction is as effective as a long cue-triggered preparatory interval in promoting reconfiguration in advance of the target. These methodological differences may have led to different cognitive operations being reflected in the switch effects reported in our studies and the Stroop studies. Our saccadic studies (using a long cue-to-target interval, CTI) may have isolated the persistent inhibitory effects from the prior trial, without active reconfiguration. The latency data of the Stroop studies (using prediction and no cue–target interval) may have inadvertently included the effects of both active reconfiguration and persistent inhibition from the prior trial.

To investigate this possibility, we studied two experimental effects in a single group of subjects. First we examined the effect of CTI on our switch costs. A very short CTI does not allow time for reconfiguration before the target appears, whereas a long CTI does. The result is that latency switch costs for short-CTI trials are always greater than those of long-CTI trials, since the short-CTI data include the costs of active reconfiguration whereas those of the long-CTI trials do not. This difference in switch costs between long- and short-CTI trials indexes the time costs of active reconfiguration (Meiran 1996; Meiran et al. 2000). We anticipated that, with long-CTI trials that reflect only the passive inhibitory effects of the prior trial, we would replicate our finding of a task switch cost for prosaccades but a paradoxical task-switch benefit in latency for antisaccades. However, in short-CTI trials, where both active reconfiguration and passive inhibitory influences are reflected in response times, we predicted that adding the extra cost from active reconfiguration would create, first, an even larger switch cost for prosaccades and, second, reverse the switch benefit for antisaccades to a small switch cost (Fig. 2). If so, this would replicate the pattern of latency effects of the Stroop studies, of apparently easier switching to a non-dominant response, the key finding used to support the theory of task-set inertia (Allport et al. 1994; Wylie and Allport 2000; Yeung and Monsell 2003).

Second, we compared the effects of predictable and random sequences on switching behavior. This directly tests the assumption that prediction or foreknowledge of the upcoming trial allows a subject to reconfigure in advance of the instructional cue. Since perfect predictability renders the cue redundant, the duration of the CTI should no longer matter. In other words, the switch costs with predictable sequences should not vary with CTI. Also, the switch costs with predictable sequences should be equivalent to those from random sequences with a long-CTI, where the preparatory interval is long enough to allow reconfiguration before the target appears. This is the intuitive view expressed by others that either a long preparatory interval or a predictable schedule should allow subjects to initiate reconfiguration (Nieuwenhuis and Monsell 2002).

However, if other studies of foreknowledge (Sohn and Anderson 2001; Tornay and Milan 2001) are correct in concluding that prediction is an ineffectual trigger of

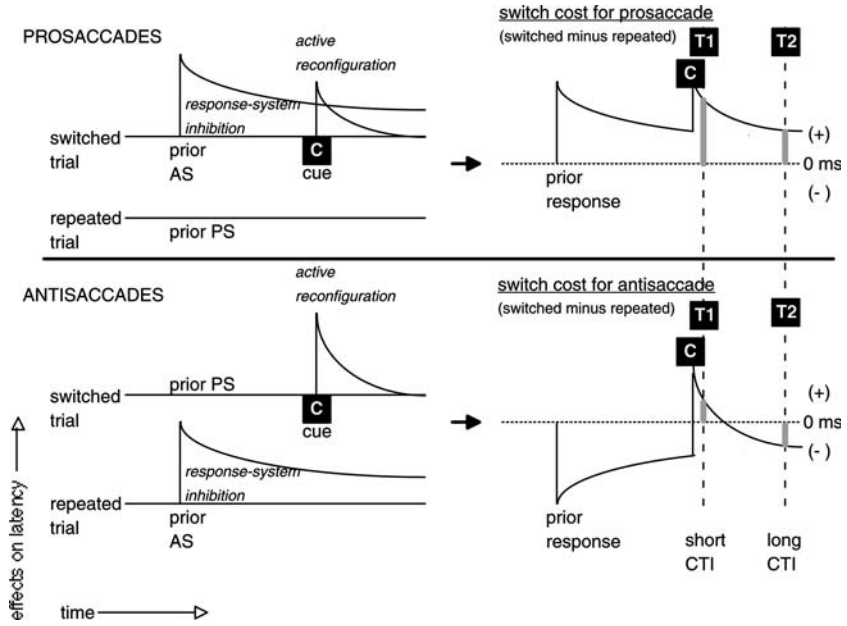

Fig. 2 Model of the temporal interactions of response-system plasticity and active reconfiguration. On the left are the temporal profile of the two latency costs being incurred by repeated and switched saccades. First, a prior antisaccade (*AS*) inhibits the response-system, which would be evident as an increase the latency of the next trial (a switched PS or a repeated AS). This "passive" effect decays slowly to an asymptote. In contrast, a prior PS does not inhibit the system. Second, if the cue (*C*) indicates the need to switch, this triggers an "active" reconfiguration process, which will be evident as an increase in latency for switched trials. This decays more rapidly than the response-system inhibition. The switch costs we measure are a subtraction of repeated from switched trials, and this is represented on the right, showing how active and passive effects summate in a dynamic time-dependent fashion to generate switch costs. Note how response-system inhibition generates a negative switch cost for antisaccades related to the prior response. This diagram illustrates the importance of the cue–target interval (CTI). If the target (*T1*) occurs shortly after the cue, there will be large effects of active reconfiguration on switch costs. Summation of this with the effects of response-system inhibition, generates a positive switch cost for both prosaccades and antisaccades, but larger for prosaccades than antisaccades (thick grey bars at "short-CTI"). This mimics the results of the Stroop studies (Allport et al. 1994; Wylie and Allport 2000). If there is a long-CTI, as with target T2, active configuration is completed or minimal, leaving mainly the more long-lasting effects of passive response-system inhibition. The result is a small positive switch cost for prosaccades and a negative switch cost for antisaccades, i.e., the paradoxical switch benefit in our prior studies (thick grey bars at long-CTI) (Cherkasova et al. 2002; Manoach et al. 2002)

advance reconfiguration, then we should not obtain these two results. Instead, we would find that the latency effects of manipulating the CTI were the same for both random and predictable sequences. A key finding would be that the predictable sequences with short-CTI were more similar to random sequences with short-CTI, than to sequences with long-CTI, since subjects would not be able to use prediction to overcome the handicap of having only a short-CTI in which to prepare.

To summarize, we expected to first reproduce our paradoxical task switch benefit for antisaccades at long-CTI. That is, there would be a positive switch cost for prosaccades and a negative switch cost for antisaccades, which would be attributable to antisaccade-induced response-system plasticity. Second, at short-CTI we would find larger switch costs because of the additional effect of active reconfiguration. The addition of active reconfiguration costs to the effects of response-system plasticity would produce a pattern of asymmetric costs, larger for prosaccades than antisaccades (Fig. 2), mimicking the findings used to support task-set inertia. Third, we would find that prediction did not alter this pattern of results, because it would fail to trigger advance reconfiguration before the appearance of the cue or target. If all three of these results were found, we would postulate that the combined but temporally independent effects of active reconfiguration and passive persistent effects of response-system plasticity could provide a unifying explanation of the switch costs in our saccadic studies and the Stroop studies.

Methods

Participants

We studied 19 healthy subjects, none of whom had been subjects in our prior study (Cherkasova et al. 2002). Subjects were screened to exclude personal histories of mental illness, neurological disorders, or severe head injury. Four did not return for a second test session, leaving 15 subjects with complete datasets. The analysis reported is restricted to these 15 subjects, eight men and seven women, with mean age of 39.5 years (range 22–56).

Apparatus

We recorded eye movements with a magnetic search coil technique, using three-foot field coils (Crist Instruments,

Bethesda, MD, USA). A scleral coil was placed in the subject's left eye, though the subject was permitted to view the stimulus binocularly. Images generated by a Power Macintosh 9600/233 using the VisionShell programming platform were back-projected by an Eiki LC-7000U projector onto a screen 81 cm from the subject. Participants' heads were secured in a chin-rest. Eye position was digitized at 500 samples/s and velocity was derived from eye position by a five-point central difference algorithm. The system was calibrated for each subject by having the subject successively fixate on nine targets in a grid spanning 50°.

Eye movement protocol

Subjects were tested in two sessions on different days. In one, the random test session, the blocks contained a random order of prosaccades and antisaccades. In the other, the predictable test session, the prosaccades and antisaccades were arranged in a predictable "AABB" sequence, where two prosaccade trials were followed by two antisaccade trials, followed by two prosaccade trials, and so on.

Each session consisted of eight blocks, each containing 48 trials. Four of these blocks had a short CTI of 200 ms, and four had a long-CTI of 2,000 ms (Fig. 3). We balanced the order of testing. Approximately half the subjects began with the session using random sequences and half with the predictable sequences. Of all testing sessions (two per subject), half began with the short-CTI and half with the long-CTI.

The initial display had a dark background with a white fixation ring at center, of 0.4° diameter and luminance of 20 cd/M^2. The fixation ring was flanked by two dots of 0.2° diameter and the same luminance at 20° eccentricity right and left, which remained visible throughout the test. Each trial began when a subject's eye fell within 3° of the fixation point. After a brief period the fixation point was replaced by one of two symbols—a yellow "O" with a surrounding ring of 0.8° diameter, or a blue "X" spanning 0.8°. The yellow O was the cue for visual saccades, while the blue X was the cue for antisaccades. The cues lasted 200 ms. In the trials with a short 200 ms CTI, the cue was immediately followed by the appearance of the target, which was the appearance of the white ring around one of the two peripheral dots. In those with a long 2,000 ms CTI, the cues were replaced by the return of the white fixation ring at screen center for another 1,800 ms, after which it shifted to become the target around one of the peripheral dots. The side of the target was randomly ordered. The white ring remained in the peripheral target location until either the subject's eye had fallen within 3° of the desired end position or 3 s had elapsed, then it reappeared at the central fixation point for the next trial.

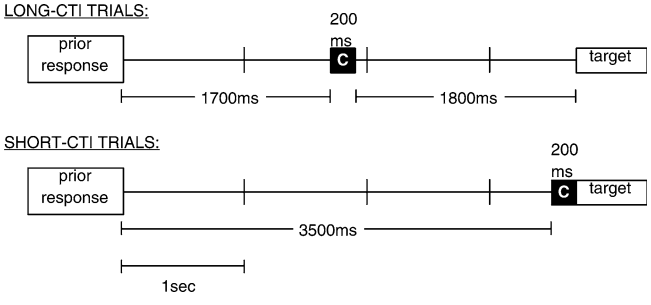

Fig. 3 Trial intervals. In long-CTI trials the 200 ms cue (C) appears 2,000 ms before the target. In short-CTI trials it appears 200 ms before the target. The interval between the prior response and the cue is varied to keep the interval between the prior response and the target constant at 3,700 ms, so that any passive carry-over effects from the prior response are the same in both types of trials

The interval between the end of the last trial and the appearance of the target in the next trial was kept constant at 3,700 ms (Fig. 3). Thus, in the short 200 ms CTI trials, there was an interval of 3,500 ms between the prior-trial and the cue; whereas in the long 2,000 ms CTI trials, this interval was 1,700 ms. By keeping the prior response to target interval constant, passive decay effects from the prior task-set would not differ between our short- and long-CTI conditions. Hence, differences in switch costs between the short- and long-CTI conditions would be solely attributable to active reconfiguration processes triggered by the cue (Meiran 1996).

Before each session, all subjects did a practice session of 20 trials. For the predictable session, we told the subjects that the sequence was predictable and that they should use this knowledge to enhance their performance. Monetary rewards of $0.025 per correct saccade were given to enhance motivation.

Data analysis

We identified saccades as eye movements with velocities exceeding 47°/s. The onset of a saccade was taken as a point at which the velocity of the eye first exceeded 31°/s. The first saccade after target onset was considered the saccadic response. The first saccade of each block was eliminated from analysis. We excluded trials whose saccadic responses had latencies less than 130 ms, as these would be anticipatory responses in advance of rather than in response to the appearance of the target (Kalesnykas and Hallett 1987). We also excluded trials with latencies greater than 800 ms as being too prolonged to accurately reflect the processes we wished to isolate.

Saccadic responses were classified as directionally correct or erroneous, according to target location and whether the trial required a prosaccade or an antisaccade. Analysis of latency was restricted to trials that not only had correct responses but had also been preceded by correct trials. While this eliminates a number of trials, the remaining data are more likely to reflect the true influences of prior responses.

Each trial was classified by its conditions: trial sequence (predictable versus random), CTI interval (200 vs. 2,000 ms), saccade type (prosaccade vs. antisaccade), and task transition (repeated vs. switched). We used ANOVA with these four conditions as repeated measures. Pair-wise comparisons were evaluated with linear contrasts.

Results

General effects of antisaccades and switching

Latency

We found significant main effects for saccade type and task transition. Antisaccades had longer latencies that prosaccades ($F(1,14) = 268$, $p < .0001$), and switched saccades longer latencies than repeated ones ($F(1,14) = 59.9$, $p < .0001$). As in our prior report, there was a significant interaction between saccade type and task transition ($F(1,14) = 15.47$, $p < .0001$), with greater switch cost for prosaccades than antisaccades. At the long-CTI, this study replicated our prior finding for antisaccades of a significant "paradoxical switch benefit": that is, shorter latencies for switched than repeated antisaccade trials (Figs. 4 and 5).

Accuracy

There were significant main effects of saccade type ($F(1,14) = 49.7$, $p < .0001$) and task transition ($F(1,14) = 40.7$, $p < .0001$). Antisaccadic error frequency was 6% more than that of prosaccades, and switched trials had 5% greater errors than repeated trials. As in our prior report, there was no significant interaction between saccadic type and task transition for accuracy, in contrast to the findings for latency. Hence, the effects of switching are similar on both antisaccadic and prosaccadic error rates (Fig. 6).

The effects of the CTI preparatory interval (short vs. long-CTI)

Latency

There was a significant main effect for CTI. Increasing preparation time reduced saccadic latencies by 20 ms on average ($F(1,14) = 145$, $p < .0001$) (Fig. 4a).

There was a significant interaction between CTI and task transition ($F(1,13) = 41$, $p < .0001$, Fig. 4b). Repeated and switched trials did not differ at the long-CTI when all saccades were grouped, mainly because the switch costs of prosaccades were negated by the switch benefits for antisaccades. However, at the short-CTI the latencies of switched trials were on average 24 ms longer than those of repeated trials ($t = 9.80$, $p < .0001$). One important effect of the increased switch cost when there was little preparation time was that the paradoxical switch benefit for antisaccades at long-CTI reversed to a switch cost at short-CTI (Fig. 5). However, the switch costs at short-CTI were still about 8 ms less for anti-

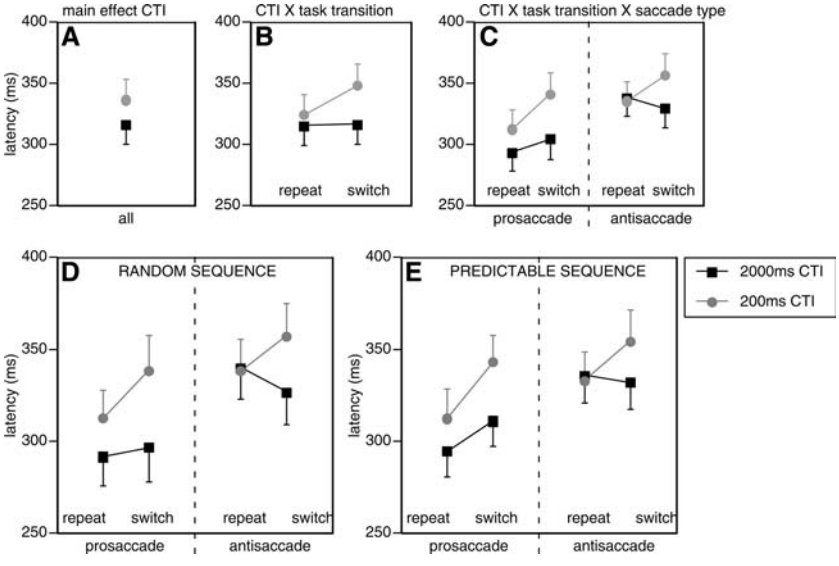

Fig. 4 Response latencies and the effects of CTI. Graphs **a**–**c** show the main effect and significant interactions of CTI (cue–target interval). Graph **d** shows the random sequence data, graph **e** those from the predictable sequence. Error bars indicate standard error. Latencies of the short-CTI trials are longer than those of the long-CTI trials (**a**), and this effect is greater for switched trials (**b**). At the long (2,000 ms) CTI, there is a switch cost for prosaccades but a paradoxical switch benefit for antisaccades, switched antisaccades having shorter latencies than repeated antisaccades (**c**). At the short (200 ms) CTI, the paradoxical switch benefit reverses to a switch cost (C). Note that a predictable sequence does not affect the pattern of switch costs at either CTI (graph **d** vs. **e**)

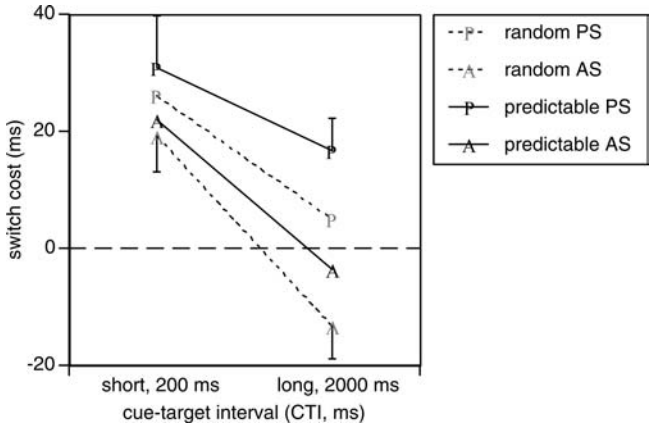

Fig. 5 Latency switch costs. The effect of CTI on switch costs (the difference between repeated and switched responses) is shown for prosaccades and antisaccades in the two different sequences. Error bars are one standard error. A short-CTI increases switch costs of all responses, more so for antisaccades. This is the change attributed to the advance reconfiguration that can be prepared before the target in the long-CTI trials but not the short-CTI trials. Prediction does not reduce switch costs but elevates them non-specifically. Note the change from a paradoxical switch benefit for antisaccades at long-CTI, to an asymmetric switch cost at short-CTI, with smaller costs for the non-dominant antisaccade response

saccades than prosaccades. Hence, this replicates the key finding of the Stroop studies (Allport et al. 1994; Wylie and Allport 2000), that switching from a dominant to a non-dominant response is more rapidly accomplished than switching in the reverse direction.

This change in switch costs with CTI was reflected in a significant three-way interaction between CTI, task transition and saccade type ($F(1,14) = 4.13$, $p < .046$, Fig. 4c). While shortening the CTI increased the switch costs for both antisaccades and prosaccades, the net effect of this on switch costs differed. Antisaccades changed from a task-switch benefit at long-CTI to a task-switch cost at short-CTI, while prosaccades merely changed from a small task-switch cost to a larger one.

The subtraction between switch-costs at long-CTI and switch costs at short-CTI can serve as an index of active reconfiguration (Meiran 1996). This was 29 ms for antisaccades and 18 ms for prosaccades. Thus, while the switch cost at short-CTI is larger for prosaccades, the relative difference between long-CTI and short-CTI switch costs might be slightly greater for antisaccades, not for prosaccades (Fig. 5).

Accuracy

There was a significant main effect of CTI ($F(1,14) = 26.9$, $p < .0001$). Subjects made more errors on the short-CTI trials than on the long-CTI trials (11% vs. 6%, Fig. 6a).

There was a significant interaction between CTI and saccade type ($F(1,14) = 12.69$, $p < .0005$, Fig. 6b). Increased preparation time did not improve prosaccadic accuracy (error rate 6% for short-CTI vs. 5% for long-CTI), but did reduce antisaccadic errors (error rate 15% for short-CTI vs. 8% for long-CTI). Thus, as might be expected, a longer preparatory interval improves accuracy, particularly for the more difficult antisaccade task.

The effects of set predictability (random vs. predictable sequences)

Latency

The effects of set predictability were minimal compared to the effects of CTI. There was no significant overall difference between the random and predictable sequences (Fig. 7a). Thus, set predictability did not allow subjects to reduce reaction times in general.

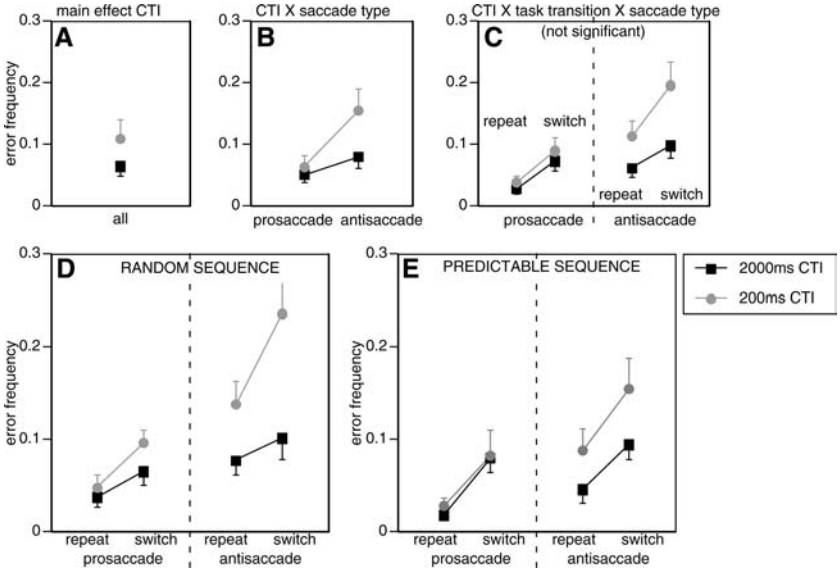

Fig. 6 Error rates and the effects of CTI. Graphs **a** shows the insignificant main effect of CTI. Graph **b** shows the interaction of CTI and saccade type, with antisaccade more delayed than prosaccades by the short-CTI. Graph **c** shows the interaction of CTI, saccade type, and switch context, for comparison with Fig. 4. Graph **d** shows the random sequence data, graph **e** those from the predictable sequence. Error bars indicate standard error. Both sequence prediction and a long preparatory interval (2,000 ms CTI) improve accuracy

What about more specific effects of prediction? If prediction could be used to prepare task-sets in advance, then it should mitigate against the effects of shortening the CTI. Because the cue is made redundant by a predictable sequence, long- and short-CTI trials should give the same result when a predictable sequence is used. Furthermore, the latencies with predictable sequences should be equivalent to the latencies generated by random sequences with a long-CTI, where preparation was also possible through advance warning. The net result should be that, at short-CTI, where the cue provides little preparation time, there would be a significantly shorter latencies for the predictable sequence compared to the random sequence, whereas at long-CTI, the predictable and random sequences should be equivalent.

Two possible statistical effects might follow from this view of prediction: first, prediction might enhance task preparation in general. If so, the statistical result would be a significant interaction between CTI and trial sequence, and linear contrasts should show that this interaction was due to a significant effect of trial sequence for short-CTI but not long-CTI trials. While we did find an interaction between CTI and trial sequence ($F(1,14) = 7.13$, $p < .008$), the origin of this interaction were not what was predicted (Fig. 7b). Linear contrasts showed that the predictable sequence had slightly longer latencies than the random sequence with the long-CTI (4 ms difference, $t = 2.81$, $p < .005$) but not with the short-CTI. Reaction times were clearly longer for the short-CTI, not just for the random sequence but also for the predictable sequence.

Second, prediction might more specifically enhance preparation of a task switch. (This essentially is the assumption made by others that prediction allows active reconfiguration in advance of the cue (Nieuwenhuis and Monsell 2002).) If so, the increase in switch cost caused by reducing the CTI from 2000 to 200 ms should occur only with the random sequence, not with the predictable sequence. A three-way interaction between CTI, trial sequence, and task transition would result. This was not found. Rather, there was a trend to the interaction between trial sequence and task transition ($F(1,14) = 3.60$, $p < .058$, Fig. 7c). This stemmed from switched trials having slightly longer (rather than shorter!) latencies of about 5 ms in the predictable sequence compared with the random sequence ($t = 2.21$, $p < .028$), whereas repeated trials did not differ between the two sequences. The result was consistently higher switch costs for the predictable sequence, for both prosaccades and antisaccades and at both CTIs (Fig. 5).

In general, these small differences notwithstanding the pattern of response latencies in the predictable sequence were remarkably similar to those in the random sequence (Fig. 4d, e). Set prediction did not allow either the increased latencies in general or the switch costs in specific at the short-CTI to be reduced to the smaller values found at long-CTI. Thus set prediction does not allow the active reconfiguration component of task-switching in saccadic responses to be implemented in advance of the cue.

Accuracy

In contrast to the findings with latency, there was a significant main effect of trial sequence on accuracy ($F(1,14) = 9.21$, $p < .003$). The predictable sequence reduced overall error rate to 7%, down from the 10% in the random sequence (Fig. 7d).

There were two trends in the interaction effects. First, was the interaction between trial sequence and CTI ($F(1,14) = 3.61$, $p = .059$, Fig. 7e). While there was no difference between random and predictable sequences at long-CTI, the error rate at short-CTI improved from 13% in the random trials to 9% in the predictable trials ($t = 3.49$, $p < .0006$). Second was the interaction between trial sequence and saccade type ($F(1,14) = 3.83$, $p < .052$, Fig. 7f). Prosaccades in both predictable and

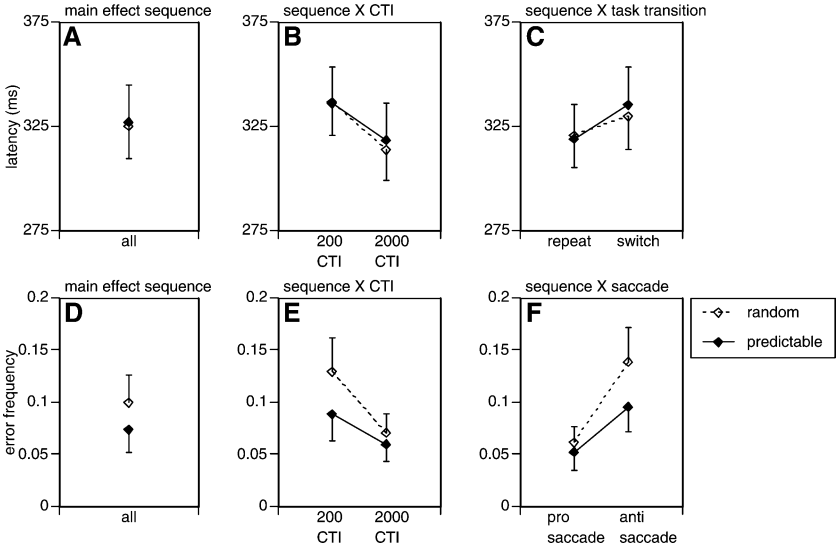

Fig. 7 Effects of sequence prediction. Main effects and interactions of the type of sequence (random vs. predictable) on latency (**a**–**c**) and error rates (**d**–**f**). Prediction has little effect on reaction times, showing only a small increase in latency for the long 2000 ms CTI trials and switched trials. In contrast, prediction significantly improved accuracy, particularly for certain difficult conditions, namely the antisaccades and the short 200 ms CTI trials

random sequences had a similar overall error rate of 5–6%. Antisaccades, however, had an error rate of 14% in the random sequence and 10% in the predictable sequence ($t = 3.53$, $p < .0005$).

Thus, set prediction allowed subjects to reduce error rate in general, and more particularly for the difficult short-CTI condition and the difficult antisaccade task.

Discussion

By varying sequence predictability and the length of the preparatory interval after an instructional cue, we have directly tested the assumption that foreknowledge allows subjects to prepare a task-switch in advance of the target, in the same manner that having a long preparatory (CTI) interval does. The results clearly show that this is not true. Foreknowledge did not render the cue redundant, nor did it allow subjects to overcome the disadvantage of a short-CTI in preparing a task switch. The result suggests that advance reconfiguration is a process that is triggered only by an instructional cue, not by internal prediction of the upcoming task-set. The implications of this result for prior data on task-switching are important. It suggests that the switch costs in some previous studies with Stroop stimuli (Allport et al. 1994; Wylie and Allport 2000) reflect not only inhibitory effects from the prior trial but also reconfiguration costs, which were thought to have been eliminated by use of a predictable sequence. Our data suggest that a re-appraisal of the origins of the asymmetric switch costs between dominant and non-dominant trials may be in order.

The effects of the cue–target preparatory interval and advance reconfiguration

Allowing subjects only a brief period to prepare after the cue significantly degraded accuracy, particularly for the non-dominant antisaccade response, and increased latencies in general. Of greater interest is the specific effect of CTI on switch costs (the difference between switched and repeated trials). As others have suggested (Meiran 1996), the difference in switch costs between long and short-CTI trials reflects an active switching process that can be executed before the target appears (advance reconfiguration). In our study, shortening the CTI did not affect the switch cost in errors but it did increase the switch cost in latency. This increase, the "advance reconfiguration cost," was found for both tasks, prosaccade and antisaccade, and both trial sequences, random and predictable.

The data also suggested that advance reconfiguration might require slightly more time for antisaccades (29 ms) than prosaccades (18 ms). This is the opposite of the findings for residual task-switch costs, where non-dominant responses like antisaccades have either lower switch costs or, in our studies, even negative switch costs (i.e., benefits). Our result shows that the "advance" reconfiguration process of task-switching may take the same time or slightly longer to accomplish for the non-dominant task, a more intuitive conclusion.

Task-set prediction

The main effect of sequence predictability was to improve accuracy, most notably at the short CTI and for antisaccades. Thus, subjects were able to use foreknowledge to reduce the error rate for the more difficult tasks. Similar general effects of set prediction on accuracy have been reported by others (Tornay and Milan 2001). In contrast to the findings on error rate, set prediction had minimal effects upon latency, and the few small effects were due to increases rather than reductions in latency. Thus, set prediction increased the latency of long-CTI trials and switched trials by about 5 ms. The resulting increase in switch costs replicates the report by others of increased switch latency cost induced by set prediction (Sohn and Anderson 2001; Tornay and Milan 2001).

The most important finding concerning set prediction was that it did not trigger advance reconfiguration. With foreknowledge subjects should have been able to prepare for a set-switch without waiting for the cue. If so, the switch costs with the predictable sequence would not have varied with CTI, and all switch costs with the predictable sequence would have resembled those of the long-CTI random trials. That is, either set prediction or a long preparatory period after the cue should have allowed preparation of the switch, as assumed by others (Nieuwenhuis and Monsell 2002). Instead, we found that only a long-CTI was effective in promoting advance reconfiguration, and not set prediction, despite the fact that set prediction improved accuracy. Hence, in our saccadic paradigm, the cue was a mandatory trigger for reconfiguration in advance of the target.

Models of component processes in switching

One important result of this study was the replication of a paradoxical task-switch benefit for antisaccades with random sequences at long-CTIs. This finding has now been replicated in four different samples, the normal subjects in this report, the normal subjects in our prior study (Cherkasova et al. 2002), a schizophrenia group (Manoach et al. 2002) and a series of patients with developmental social processing disorders (Manoach et al. 2003). Other saccade studies (Hunt and Klein 2002) have also confirmed that task switch costs present at short-CTIs can change to task-switch benefits at long-CTIs (though this was found for both prosaccades and antisaccades). Also, our pattern of task-switch costs for prosaccades and task-switch benefits for antisaccades has recently been confirmed independently by another

antisaccade study that used a CTI of 1.2 s (Fecteau et al. 2004).

This finding challenges existing models of the processes involved in task-switching. Neither an active reconfiguration process nor a passive persistence of inhibitory influences on opposing task-sets predict that switching sets should ever result in shorter latencies than repeating task-sets. Other models of switching also fail to explain this finding. For example, the "failure to engage" hypothesis proposes a single preparation process, "intention activation." Depending on conditions, this process can range from fully prepared to fully unprepared, and it is this variable readiness that generates variations in latency (De Jong 2000; Nieuwenhuis and Monsell 2002). This hypothesis proposes that repeated tasks with adequate warning (i.e., long-CTI) approximate a fully prepared state, and hence generate responses with very short latencies. On the other hand, switched tasks with little warning (i.e., short-CTI) represent a fully unprepared state and have responses with very long latencies. Switched tasks at long-CTI would have an intermediate preparedness, and therefore their latencies should be some intermediate value between the fully prepared and fully unprepared latencies. This is clearly not the case for antisaccades, where the switched trials at long-CTI consistently have the shortest latencies of all antisaccades.

Similarly, task-switch benefit is not explained by the contrast between algorithmic computation and episode retrieval, which Hunt and Klein (2002) used to explain the elimination of task-switch costs in a prosaccade/antisaccade paradigm when long-CTIs were used. This relied on the proposal that responses to a stimulus could stem from either the output of an algorithm or retrieval of the memory of a similar stimulus–response pairing that had been recently executed, whichever is completed first. The authors suggested that "hypercompatible" responses like prosaccades have very efficient algorithms, which would be completed before episode retrieval, and that residual switch costs might be associated with the latter alone. Whereas this proposal may successfully explain the elimination of switch costs in certain paradigms, it does not account for switch benefits for antisaccades.

Rather, we believe that the results indicate that the key factor prolonging latencies for both prosaccades and antisaccades is not a preceding switch but a preceding antisaccade, as also suggested by others (Fecteau et al. 2004). We have provided further evidence of this inhibitory antisaccade effect by showing that antisaccades from two trials back also increased both prosaccadic and antisaccadic response latency in schizophrenia (Barton et al. 2005).

This antisaccade effect can be understood in terms of the physiology of the frontal eye field and superior colliculus. Antisaccades suppress the preparatory pre-target activity of all saccade-related neurons in the frontal eye field and superior colliculus, without regard to directional specificity. This suppression may be generated by fixation neurons in the frontal eye field and/or superior colliculus, possibly under control of the supplementary eye field or dorsolateral prefrontal cortex (Munoz and Everling 2004). The degree of this suppression correlates with longer response latencies and decreased error rates (Everling et al. 1999; Everling and Munoz 2000). While it is not yet known whether this suppressive effect persists into the preparatory activity of the next trial, such persistence has been shown for the variations in pre-target activity related to directional congruency (Dorris et al. 2000). Thus, it is plausible that the inhibition of pre-target activity by antisaccades can persist into future trials as a type of "immediate neural plasticity" in the saccadic system, prolonging the latency of any upcoming response, whether prosaccade or antisaccade. This is a distinctly different effect from task-set inertia, where antisaccade-induced inhibition should be limited to the prosaccade task-set. In our proposal, inhibition from the non-dominant task-set occurs on the response-system, not the opposing task-set, and thus this inhibition affects both upcoming prosaccades and antisaccades (Fig. 1). We call this "response-system plasticity" to emphasize that the locus of change is in the motor response, not in sets of stimulus–response associations.

The second important result of this study was that we could consistently produce a pattern of small switch costs for antisaccades and large switch costs for prosaccades by using short-CTI. We propose in accordance with Meiran (1996) that at short-CTI the latency costs reflect both the effects of advance reconfiguration as well as persistent inhibitory influences from the prior trial. At long-CTI, costs reflect only the inhibitory influences, which we propose to be the effects of response-system plasticity. Adding the latency costs of advance reconfiguration to those of response-system plasticity increased the switch cost of prosaccades and reversed the paradoxical switch benefit of antisaccades to a small switch cost (Fig. 2).

This pattern of smaller switch costs for the non-dominant response is the key finding upon which the theory of task-set inertia rests. However, the studies of Allport used predictable sequences with no cue or CTIs that were effectively zero (Allport et al. 1994; Wylie and Allport 2000). Since our study shows that prediction does not allow advance reconfiguration in the manner that a long-CTI does, the switch costs measured by Allport and colleagues likely included the costs of a cue-triggered reconfiguration process. We believe that their results can be explained by a combination of reconfiguration costs and antisaccade-like inhibition of response-systems, just as we found, rather than task-set inertia. In contrast, task-set inertia cannot explain the paradoxical switch benefit for antisaccades at long-CTI.

In this model, because switch costs combine the effects from active reconfiguration and those from response-system plasticity, the pattern of switch costs in any given paradigm will reflect a ratio of the two processes. This ratio will depend upon the relative magnitude of the two processes to each other, as well as the

time (CTI) at which switch costs are measured. The dynamic nature of this mixture in the model can explain a number of unusual findings in the literature.

First, consider the fact that at long-CTI (~1500 ms) some paradigms have no switch cost (Meiran 1996; Tornay and Milan 2001). These tend to be studies that use tasks without a dominance asymmetry. Task-set inertia would argue that these responses nevertheless require some inhibition of the competing task-set, and therefore there should be some persistent inhibition and a switch cost at long-CTI. In our model of response-system plasticity, when there is no dominance asymmetry, there is no difference in the effects of the two task-sets on the response-system and hence no difference in persistent inhibition. The result is no switch cost.

Another important finding is a recent demonstration that the reduced switch costs for non-dominant task-sets only occur when the dominant and non-dominant tasks require the same types of responses, and not when the responses are entirely different (Yeung and Monsell 2003). Task-set inertia has difficulty explaining this, but our model of response-system plasticity predicts this result. If a non-dominant task-set persistently suppresses its effector response-system, this would not affect the dominant task-set if that used a different set of responses. Furthermore, the active reconfiguration process in this scenario may still possess the asymmetry we found, with more active cost for switching to the non-dominant response. Hence, with short-CTI there should be more rather than less switch cost for the non-dominant task-set when the type of response differs. This, indeed, is exactly what was found by Yeung and Monsell (2003).

A model that incorporates response-system plasticity and active reconfiguration can also explain a number of interesting findings by Wylie and Allport (2000). In their experiment 2, the latencies of a dominant task declined after the non-dominant (Color naming) task had been replaced by a neutral task in switching. However, switch trials still had latencies greater than the most recent repeat trials, even though there was no further repetition of the non-dominant trial. Without such repetition, task-set inertia predicts that latencies should decline monotonically regardless of task transition. To explain this anomaly they hypothesized a stimulus-triggered component to inhibition. However, our model suggests that what was triggered by their stimuli (which also served as their cue) was active reconfiguration, a process conclusively proven by Meiran (1996). The importance of our study is that it shows that their use of a predictable sequence did not eliminate active reconfiguration from their latency effects, since reconfiguration only occurs after a cue or stimulus.

Their experiment 2 also showed that when alternation between dominant and non-dominant tasks resumed, the switch costs of the dominant response gradually increased while the switch costs of the non-dominant response decreased. Why should this be? One possibility is that the non-dominant inhibition in response-system plasticity is not just additive but facilitatory with repeated occurrences. That is, recent repetitions of non-dominant tasks enhance the plastic inhibitory changes of a non-dominant task in upcoming trials. If so, there would be a gradual increase of passive switch costs for the dominant response and of passive switch benefits for the non-dominant response. When added to the active switch costs of reconfiguration, the pattern of gradually increasing switch costs for the dominant task and decreasing switch costs for the non-dominant task is the result.

Thus, we believe that a model of response-system plasticity, in which non-dominant task-sets inhibit the effector arm of the process rather than the opposing task-set, can account for a variety of intriguing switch effects in the literature. This includes the paradoxical residual task-switch benefit of antisaccades we reported (Cherkasova et al. 2002; Manoach et al. 2002) and replicated in this study. The interactions of this passive plasticity effect with active reconfiguration, and the fact that prediction is an ineffectual trigger of the latter, allow us to explain some of the findings used to support task-set inertia as the origin of dominance effects in switching. This does not mean that task-set inertia is non-existent. Studies that show that the switch costs for tasks without dominance asymmetry decline with time since the last response (Meiran 2000) would be hard to explain otherwise. However, the lack of switch costs at CTI of 1500 ms (Meiran 1996) suggests that the effects of task-set inertia decay more quickly than others believe (Wylie and Allport 2000). Our results suggest that the more durable effect in trials with dominance asymmetry is response-system plasticity, an effect with parallels in animal physiology, and not task-set inertia. Furthermore, our analysis of the costs attributable to advance reconfiguration show that, at least as far as this active process goes, it is at least as difficult—or even slightly harder—to switch to the non-dominant response after all.

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