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A computational model of fractionated conflict-control mechanisms in task-switching

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Abstract

A feature of human cognition is the ability to monitor and adjust one's own behavior under changing circumstances. A dynamic balance between controlled and rapid responding is needed to adapt to a fluctuating environment. We suggest that cognitive control may include, among other things, two distinct processes. Incongruent stimuli may drive top-down facilitation of task-relevant responses to bias performance toward exploitation vs. exploration. Task or response switches may generally slow responses to bias toward accuracy vs. speed and exploration vs. exploitation. Behavioral results from a task switching study demonstrate these two distinct processes as revealed by higher-order sequential effects. A computational model implements the two conflict-control mechanisms, which allow it to capture many complex and novel sequential effects. Lesion studies with the model demonstrate that the model is unable to capture these effects without the conflict-control loops and show how each monitoring component modulates cognitive control. The results suggest numerous testable predictions regarding the neural substrates of cognitive control.

Keywords: Cognitive control; Task switching; Sequential effects; Performance monitoring; Neural network; Computational modeling

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1. Introduction

Environments often change unpredictably over time. In some cases, adaptation to such changing environments requires the learning of new responses. However, in other cases, the same response must be generated with slightly changed parameters. This necessitates an ongoing adjustment of behavioral control. A classic example is the tradeoff between speed and accuracy (Osman et al., 2000; Plamondon & Alimi, 1997; Strayer & Kramer, 1994). If the cost or likelihood of errors is low and speed essential, then one will do well to execute a given action as quickly as possible with less regard for accuracy. Conversely, if the cost of errors is high and speed less important, then one will do well to increase behavioral control, slow down, and be more careful. Subjects may adopt a strategy ranging between rapid (emphasis on speed) and controlled (emphasis on accuracy) responding. In this case, shifting the bias toward accuracy rather than speed might be considered an example of a simple form of cognitive control. More complex forms of control might involve adjusting attentional allocation between focused exploitation of known aspects of an environment versus exploration of unknown components (Ishii, Yoshida, & Yoshimoto, 2002; Kaelbling, Littman, & Moore, 1996; Sutton & Barto, 1998; Usher, Cohen, Servan-Schreiber, & Rajkowski, 1999) or between fast, pre-potent versus controlled, non-prepotent responding (Pardo, Pardo, Janer, & Raichle, 1990; Weissman, Gopalakrishnan, Hazlett, & Woldorff, 2005). Notably, the cognitive control strategy may change independently of changes in the tasks being performed. The domain of cognitive or executive control may thus include many different kinds of control effects and underlying mechanisms (Norman & Shallice, 1986). Recent efforts have been made to dissect various components on empirical and meta-analytic grounds into such categories as shifting, monitoring or updating, inhibition, and selective attention (Miyake et al., 2000; Wager & Smith, 2003). Our goal in this paper is to develop a fractionation of cognitive control that derives from an integration of theoretical, computational, and behavioral analyses. To do this, we focus on task switching as a well-studied representative paradigm (Allport, Styles, & Hsieh, 1994; De Jong, Berendsen, & Cools, 1999; Dreisbach, Haider, & Kluwe, 2002; Hodgson et al., 2002; Jersild, 1927; Meiran, 1996, 2000a, 2000b; Meiran, Chorev, & Sapir, 2000; Meiran & Gotler, 2001; Meiran & Marciano, 2002; Nieuwenhuis & Monsell, 2002; Rogers & Monsell, 1995; Sohn & Anderson, 2001; Wylie & Allport, 2000b). Studies of switching between two cognitive tasks afford significant insight into control of both cognitive and motor processes by dissociating changes in task set (cognitive) from changes in the required response (motor).

1.1. Cognitive control in task switching

In this paper, we have focused the scope of the analysis and simulations toward the specific aims of elucidating cognitive control mechanisms. There has been significant recent controversy regarding the extent to which executive control mechanisms are necessary to drive a task switch (Altmann, 2003; Logan & Bundesen, 2003; Monsell, 2003; Rogers & Monsell, 1995). We do not take an absolute position on this issue. Nonetheless, the controversies suggest that further specification is needed of different types of control functions and how they might influence performance during task-switching. Rather than investigating whether control functions are necessary to drive a task switch, we focus instead on delineating mechanisms of performance monitoring and control that modulate response

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parameters *within* a particular task set (Altmann & Gray, 2002). In so doing, we develop a computational model of cognitive control in standard task switching experimental paradigms. Implementing theoretical hypotheses as computational models provides a critical means to clarify the relationship between complex effects and relatively simpler underlying mechanisms (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Braver, Barch, & Cohen, 1999; Cho et al., 2002; Gilbert & Shallice, 2002; Jones, Cho, Nystrom, Cohen, & Braver, 2002; Melara & Algom, 2003; Miller & Cohen, 2001; Phaf, Van der Heiden, & Hudson, 1990). However, our purpose in developing a computational framework is not to provide a comprehensive model of task switching but rather to use it as a means to an end. We use task switching as a representative domain in which multiple control mechanisms may interact within a single experimental paradigm. Our aim in this endeavor is twofold. First, we aim to explore competing hypotheses that behavioral effects in task-switching can be accounted for solely by bottom up mechanisms, versus the hypothesis that top-down effects such as control loops provide better accounts of a range of data, even if such control loops are not required for task-switching *per se*. Second, we aim to concretely delineate specific mechanisms of cognitive control that may generalize beyond task switching to a range of behavioral paradigms.

A central issue in any task-switching study is the importance of sequential relationships between trials. At a basic level, a task switch involves information from two consecutive trials, in that the task changes from trial $n-1$ to trial n. Studies of task-switching have, with some exceptions (Altmann & Gray, 2002; Mayr, 2002) generally focused on the current and immediately preceding trial, in that a task switch involves task A in the preceding trial and task B in the current trial. Perhaps the most prominent finding is that of switch costs, which refer to the slower, less accurate performance when the task switches relative to the preceding trial as compared to when it does not switch (Jersild, 1927). The switch cost effect persists residually even with long preparation times (Allport et al., 1994; de Jong, 2000; Meiran et al., 2000; Nieuwenhuis & Monsell, 2002; Rogers & Monsell, 1995).

The origin of the switch cost is controversial. Some investigators (Rogers & Monsell, 1995) interpret the residual switch cost as the time needed to reconfigure the system, which cannot be completed until the target stimuli appear. This interpretation implies a putative *top-down* executive control mechanism, possibly distinct from task-specific stimulus-response pathways, that implements the task switch and produces the residual switch cost. On the other hand, Allport and colleagues argue for a *bottom-up* explanation of switch costs (Allport et al., 1994; Wylie & Allport, 2000b). According to their associative task-set-interference (TSI) hypothesis, the switch cost originates from associative strengthening between task-related stimuli and an internal representation of the task-set with which they are paired on a previous trial. In the current trial, stimuli will tend to re-evoke the previous task-set, even if the previous task-set is different from the one relevant for the current trial (i.e., the task switched). This pairing may lead to interference that lengthens RTs (Waszak, Hommel, & Allport, 2003). In the same vein, recent modeling work (Gilbert & Shallice, 2002) has shown that switch costs can be accounted for in part by a combination of residual activity in task set representations and associative TSI. In addition to target-related priming effects, some portion of the apparent switch cost may be due to a loss of cue repetition benefits (Arrington & Logan, 2004a, 2004b; Logan & Bundesen, 2003; Mayr & Kliegl, 2003) related to stimulus-specific cue priming effects. Also in favor of a bottom-up explanation of switch costs, several authors have argued that switch costs represent a failure to *proactively* reconfigure task set on a subset of trials and maintain this

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configuration across a delay (De Jong, 2000; De Jong et al., 1999; Meiran, 1996; Nieuwenhuis & Monsell, 2002). This is referred to as the "failure-to-engage" (FTE) hypothesis. Thus, the switch cost would result from the time required to *reactively* instate the correct task set representation at the time of target stimulus presentation, while also overriding interference from the prepotent but inappropriate task-set. Computational modelling work has shown that the FTE hypothesis can, in fact, provide a quantitative account of the relevant behavioral data (Reynolds, Braver, Brown, & Stigchel, 2006). Others have suggested that switching away from a given task involves a possibly top-down inhibitory process, namely backward inhibition, that suppresses the previous task set representation, making it more difficult to return to it again (Mayr, 2002; Mayr & Keele, 2000). Finally, on the basis of previous work (Cho et al., 2002; Jones et al., 2002), we will suggest below that top-down mechanisms might impose a protracted slowing effect that persists into subsequent trials. While all of these factors may contribute to the switch cost to some degree, further specification is needed regarding the relative contributions of each.

Aside from the switch cost, incongruency effects are also prominent in task switching studies. It is well-established that stimuli associated with incongruent responses lead to interference and thus poorer performance in a range of cognitive tasks (Botvinick et al., 2001; Botvinick, Nystrom, Fissel, Carter, & Cohen, 1999; Egner & Hirsch, 2005; Eriksen & Eriksen, 1974; MacLeod, 1991). Moreover, incongruency effects have been associated with a conflict adaptation effect (Mayr, Awh, & Laurey, 2003), i.e. reduced incongruency effects on subsequent trials, possibly due to a corresponding increase in cognitive control (Kerns et al., 2004; MacDonald, Cohen, Stenger, & Carter, 2000). Within task-switching paradigms, incongruency effects occur when a feature of the target stimulus is associated with an incompatible response according to the currently irrelevant task. In the case of incongruent stimuli, cognitive control mechanisms may be needed to increase activity of the (possibly already active) task set representation or pathway, and directly or indirectly suppress irrelevant task set representations or pathways (Egner & Hirsch, 2005). Given incongruent stimuli, a performance monitor may serve to increase persistent task-set-related activity and subsequent attentional focus to the current task; once increased, the effect may persist into subsequent trials. Several predictions follow from this hypothesis. First, despite the cost of incongruency on both response time and error rate in the current trial, subsequent trials of the same task would be expected to show an *improvement* in performance. In support of this account, recent work (Goschke, 2000) suggests that incongruency leads to greater switch costs in the *subsequent* trial. Specifically, prior incongruency increased response time on switch trials and reduced response time on no-switch trials. This may reflect facilitation of repeating the same task (though not necessarily the same response), as well as increased inhibition of the previously irrelevant task, both of which are expected if incongruency does lead to an enhancement of representations of the current task set (at the expense of representations of the currently irrelevant task set), which persists into subsequent trials. Furthermore, if the subsequent trial is incongruent as well as a switch, then this incongruency should interact with the persistent previous task set representation to increase switch costs further. Although some of the effect of incongruency in the previous trial may be due to stimulus repeat effects (Mayr et al., 2003), in the case of task switching, the effect persists even when episodic stimulus repeats are eliminated in the sequence of interest (Goschke, 2000). In the experiment below, we begin by attempting to replicate these findings.

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1.2. Higher-order sequential effects

As the above suggests, cognitive control mechanisms may exert protracted and persistent effects on performance, and therefore they may be evident most clearly in the effects of longer sequences of trial conditions. Higher order sequential effects refer to conditions involving sequences of more than two trials. In general, a full descriptive model of performance monitoring will need to make reference to higher order effects (Laming, 1968). To our knowledge, there have been no previous studies that have systematically examined the presence of higher-order sequential effects in task-switching. However, there are strong *a priori* reasons to suggest that these effects will be present and significant. Specifically, within simpler task paradigms, such as two-alternative forced choice (2AFC), there has been a great deal of examination of the role of higher-order sequential effects in modulating behavioral performance. For example, there is a long tradition of research establishing that error commission (Laming, 1968; Rabbitt, 1966) or a change in the required response (Bertelson, 1961) produces a relatively persistent slowing in response time on subsequent task trials. Furthermore, there is evidence that such sequential effects may reflect specifically top-down control mechanisms, such as expectation of particular sequences (Soetens, Boer, & Hueting, 1985), in addition to bottom-up mechanisms. Evidence for the co-existence of top-down and bottom-up mechanisms of sequential effects has also been found previously with the aid of computational modeling of 2AFC tasks (Cho et al., 2002).

In principle, different forms of conflict might usefully modulate distinct components of cognitive control. One form of conflict may be evoked by switching tasks from one trial to the next. Specifically, if the required task or response changes frequently, then it is difficult to predict where to focus attention for optimal performance. Thus, conflict between the expected and actual required responses due to changes in the required task or response may be effectively addressed by generally slowing responses in subsequent trials, to prevent an anticipated response from being prematurely (and erroneously) generated before external stimuli can be adequately processed. On the other hand, another form of conflict may be evoked by incongruent stimuli. If the task requirements change little but strong task-irrelevant, conflicting stimuli appear, then performance may be best served if conflict due to task-irrelevant stimuli drives increased attentional focus to the relevant stimuli. Conversely, if there were only a single form of conflict-control mechanism used to adjust performance, responses could not be effectively adapted to the constraints of specific task situations. Thus, a non-specific response slowing mechanism would not produce an appropriate shift in attentional focus towards task-relevant stimuli and away from task-irrelevant ones. Likewise, an attentional focusing mechanism would be ineffective in responding to unexpected changes in task-requirements. Thus, we postulate that there are multiple conflict-control loop mechanisms in the brain that are each associated with regulating adjustment in specific forms of cognitive control.

It is well-established that incongruency effects across a range of task paradigms are associated with a form of conflict, and that this conflict engages the anterior cingulate cortex (ACC) and related brain areas that appear to perform performance monitoring and control functions (Botvinick et al., 1999; Carter et al., 1998; Kerns et al., 2004). Likewise, the results of 2AFC and related paradigms (e.g., go-nogo) indicate that response switching also appears to engage similar conflict and performance monitoring processes in the ACC and elsewhere (Jones et al., 2002; Stuphorn, Taylor, & Schall, 2000). In a task-switching environment, switches in both response and task also occur frequently from one trial to

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the next, and as such may engage a conflict-detection mechanism that non-specifically slows responses on subsequent trials. There is some evidence that task-switching engages conflict-detection mechanisms in the ACC (Dove, Pollmann, Schubert, Wiggins, & von Cramon, 2000; Luks, Simpson, Feiwell, & Miller, 2002).

Below, we present a systematic analysis of sequential effects from an experimental study of task-switching that demonstrates the presence of potentially complex and confusing higher-order sequential relationships in behavioral performance. We then show that a computational model with two performance monitoring and control mechanisms can account for the observed complex behavioral effects, while the model without the two conflict-control loops cannot account for the observed effects. The model thus supports the hypothesis of multiple generalized cognitive control mechanisms and produces quantitative predictions that can be tested with functional imaging and lesion studies.

2. Behavioral study: Sequential effects in task-switching

Our aim was to test for the existence of effects consistent with cognitive control mechanisms as discussed above. To this end, we conducted a task switching study and looked at factors of: (1) task switch (S) vs. no-switch (N); (2) response alternation (A) vs. repetition (R), and (3) stimulus incongruency (I) vs. congruency (C). Trials were coded this way in accordance with previous task-switching studies that have observed significant effects or interactions of these factors on behavioral performance (Allport et al., 1994; Rogers & Monsell, 1995). To avoid confusion, we use the terms *repetition* and *alternation* to refer to changes in the required response from trial to trial, and *switch* or *no-switch* to denote changes in the required task set. In order to analyze the interaction of these factors in higher-order sequential effects, we crossed each of the eight possible current-trial conditions with the eight previous trial conditions, for a total of 64 task conditions (6 factors). Following earlier conventions (Botvinick et al., 1999; Kerns et al., 2004), we use uppercase letters to denote current-trial conditions and lowercase letters to denote previous trial conditions. Importantly, this coding method implicitly includes information from three consecutive trials (though not all information from the earliest trial was analyzed—in particular, the incongruency factor was omitted). For example, one possible trial condition is nrcSAI, i.e., the *previous* trial conditions were no-task-switch, response repetition, congruent stimulus followed by *current* trial conditions of task switch, response alternation, and incongruent stimulus.

The complexity of this analysis method was justified in that it allowed us to investigate several issues. First, we attempted to replicate previous results (Goschke, 2000), to show increased switch cost as a function of prior trial incongruency. Second, we attempted to demonstrate effects consistent with a non-specific, task-switch-induced slowing that persisted to subsequent trials, regardless of subsequent trial type. This would be found as a slowing in response times due to task switching in the *previous* trial, regardless of the current trial type. Furthermore, if such slowing due to task switches in the *previous* trial was found even when no switch occurred in the *current* trial, then we would conclude that the slowing differed from backward inhibition effects, since backward inhibition entails a switch in the current trial to a previously abandoned task set. A positive result would suggest that the cognitive control mechanisms of the Jones et al. (Jones et al., 2002) model, and associated with the ACC, might also govern the tradeoff between control and prepotency in cognitive as well as motor mechanisms.

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3. Methods

3.1. Participants

Sixteen participants, age 19–22 (9 female) underwent behavioral testing. These participants came from the Washington University area and were compensated for their participation by being paid $10/hour.

3.2. Tasks and stimuli

Subjects performed a variant of the Rogers and Monsell (1995) letter-digit paradigm which involved two different tasks performed on visually presented stimuli: consonant/vowel classification of letters and odd/even classification of digits (see Fig. 1). On each trial, a single uppercase letter and digit were centrally presented side-by-side in 24-point Times New Roman font, white on a black background. The location of each stimulus type (letter or digit) was random and varied across trials. Letters and digits were selected randomly and with uniform probability from the following two sets: letters: (A, E, I, U, X, P, L, Z); digits: (2–9, inclusive). Classification judgments were indicated via a manual button press with the index finger of each hand. Only two buttons were provided for classification, which produced response overlap across tasks. The mapping of response (odd/even, consonant/vowel) to hand was counterbalanced across participants, but was fixed for a participant across all trials. On any given trial, only a single task was to be performed, and this was indicated by a task-cue presented prior to the onset of the target stimuli. The task cue was the word "LETTER" or "NUMBER" presented visually at central fixation in 24-point Times New Roman font. Each task cue occurred randomly with 50% probability, leading to an equal proportion of trials in which the current task switched or repeated from the one performed just previously.

The timing of each trial was as follows: (1) task-cue presented for 300 ms; (2) constant preparatory interval of 1500 ms starting at cue offset, during which the display went blank; (3) presentation of target stimuli (e.g., "A 2" or "9 P") immediately following the prepa-

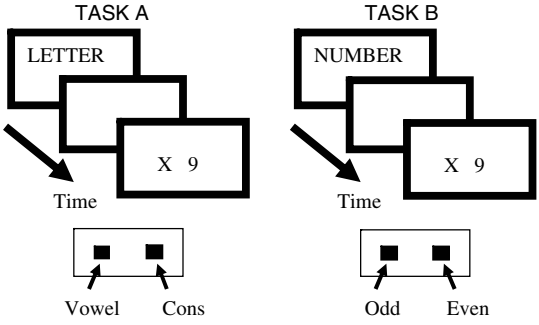

Fig. 1. The task-switching task, adapted from (Rogers & Monsell, 1995). Each trial begins with the cue word "LETTER" or "NUMBER" appearing briefly in the middle of the screen. After a delay during which the screen is blank, a letter and a number appear side-by-side. If the cue word was "LETTER," the subject must respond differentially for vowels and consonants. If "NUMBER," the subject must respond differentially for odd and even numbers.

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ratory interval, with stimulus duration lasting until a response was made or 5000 ms elapsed; (4) constant response-cue-interval (RCI) of 200 ms occurring prior to onset of the next trial.

3.3. Procedure

Testing was performed on a Macintosh G3 computer running PsyScope software. Button press responses were made on the PsyScope button box. After receiving task instructions, participants performed an initial 50 practice trials. Following the practice phase, participants performed an additional 6 blocks of 200 trials each. A rest break was provided following each block. The total experimental session lasted approximately 1 h.

3.4. Data analysis

Response times and error rates were recorded for each trial. Trials in which a response was not made in the allotted period were excluded from further analysis. Because of the sequential nature of the analysis, the two trials immediately subsequent were also excluded. The following trials were also excluded: (1) the first two trials of each block; and (2) trials with response times faster than 250 ms or slower than 3 standard deviations from the subjects' mean response time (Ratcliff & Tuerlinckx, 2002). These data-trimming procedures resulted in the exclusion of 4.01% of total task trials. Response times reported below reflect only correct trials. Response times and error rates were analyzed with repeated measures ANOVAs using factors of current and previous task switch, response alternation, and incongruency.

4. Results and discussion

4.1. Current trial effects

The results showed a significant spread of response times (mean correct 885 ms) and error rates (mean 9.8%) for the various conditions (Tables 1, 2, and Fig. 2). The response time results (Fig. 2A) replicated previous findings of switch cost (73 ms, $F(1,15) = 36.87$, MSe = 36480, $p < .0001$), alternation cost (32 ms, $F(1,15) = 5.40$, $MSe = 26079$, $p < .04$), and incongruency cost (31 ms, $F(1,15) = 5.59$, $MSe = 50517$, $p < .04$) (Allport et al., 1994; Rogers & Monsell, 1995). Due to the long preparatory interval, the switch costs measured were most likely the "residual" switch costs (Allport et al., 1994; Meiran et al., 2000; Rogers & Monsell, 1995). Switching and alternation interacted ($F(1,15) = 54.93$, $MSe = 13401$, $p < 10^{-5}$), such that switch costs were 103 ms larger ($t(15) = 6.93$, $p < 10^{-5}$) for response repetition trials (122 ms, $t(15) = 6.74$, $p < 10^{-5}$) than response alternation trials (19 ms, $t(15) = 2.08$, $p < .03$), consistent with previous results (Meiran, 2000a; Rogers & Monsell, 1995). In terms of errors (Fig. 2B), there was a switch cost (2.2%, $F(1,15) = 11.55$, $MSe = 0.009734$, $p < .004$) and an incongruency cost (1.7%, $F(1,15) = 26.03$, $MSe = 0.0265$, $p < .0002$), but no significant alternation cost (1.2%, $F(1,15) = 1.50$, $MSe = .02650$, $p < .24$). Alternation interacted with task-switching ($F(1,15) = 4.63$, $MSe = 0.0059$, $p < .05$) such that error switch costs appeared to be greater for response alternation trials (2.9%, $t(15) = 8.13$, $p < 10^{-6}$) than for response repetition trials (1.1%, $t(15) = 0.96$, $p < .83$) (Fig. 2B).

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Table 1
Mean human (bold) and model RT as function of current and previous trial types (correct trials only)

	Current trial							
	NRC	NRI	NAC	NAI	SRC	SRI	SAC	SAI
nrc	**701**	**793**	**861**	**912**	**850**	**976**	**865**	**849**
	745	787	824	831	950	975	903	989
nri	**742**	**769**	**816**	**839**	**925**	**953**	**867**	**892**
	735	739	792	860	963	1011	906	983
nac	**800**	**827**	**895**	**883**	**939**	**929**	**906**	**925**
	765	810	842	862	937	970	951	1017
nai	**803**	**816**	**881**	**873**	**945**	**967**	**894**	**955**
	792	793	833	850	950	1055	945	987
src	**796**	**859**	**912**	**910**	**926**	**972**	**897**	**910**
	822	856	883	956	931	998	924	981
sri	**836**	**827**	**906**	**885**	**866**	**879**	**918**	**952**
	806	835	883	937	931	999	935	975
sac	**848**	**885**	**887**	**943**	**952**	**1012**	**934**	**959**
	838	850	930	995	913	1011	901	932
sai	**840**	**878**	**919**	**945**	**921**	**1037**	**886**	**953**
	800	830	910	956	954	1053	880	925

Table 2
Mean human (bold) and model error rate (%) as function of current and previous trial types

	Current trial							
	NRC (%)	NRI (%)	NAC (%)	NAI (%)	SRC (%)	SRI (%)	SAC (%)	SAI (%)
nrc	**5.1**	**12.9**	**4.7**	**6.4**	**6.6**	**10.9**	**5.8**	**15.1**
	5.1	9.1	7.7	18.0	6.6	11.8	6.6	15.1
nri	**8.5**	**11.5**	**5.8**	**8.4**	**6.0**	**11.7**	**9.1**	**17.1**
	5.1	6.3	11.4	12.7	5.2	15.8	3.5	12.6
nac	**3.0**	**12.6**	**10.4**	**10.1**	**8.4**	**12.9**	**10.3**	**11.1**
	4.2	11.4	4.9	12.3	3.9	13.3	5.5	16.9
nai	**5.4**	**6.5**	**8.5**	**12.2**	**8.2**	**15.3**	**10.2**	**16.3**
	4.4	9.1	7.6	11.9	4.2	17.7	5.2	18.9
src	**7.0**	**12.2**	**5.2**	**9.8**	**6.8**	**12.6**	**9.5**	**12.6**
	5.0	7.0	7.3	13.5	4.9	12.2	4.2	14.4
sri	**5.4**	**13.5**	**7.5**	**11.0**	**7.7**	**11.5**	**4.4**	**16.1**
	3.2	8.9	6.7	10.4	5.1	11.0	3.5	12.5
sac	**3.8**	**9.5**	**10.0**	**12.6**	**3.2**	**14.2**	**11.6**	**17.6**
	4.4	6.8	4.4	13.7	4.3	11.2	5.6	8.6
sai	**8.1**	**13.0**	**6.0**	**13.9**	**6.3**	**10.7**	**10.2**	**15.1**
	5.4	5.8	4.8	15.7	3.2	18.9	4.1	9.8

4.2. Effects of preceding trials

The preceding trial type could affect response time on the current trial, collapsed across all current trial conditions (Fig. 3). Previous switch trials led to longer response times on the current trial than did previous no-switch trials (39 ms, $F(1,15) = 13.69$, $MSe = 25438$, $p < .003$). Likewise, previous alternate trials led to longer response times compared with previous repeat trials (47 ms, $F(1,15) = 15.49$, $MSe = 22427$, $p < .002$). There was no interaction between these two factors ($F(1,15) = .09$, $p > .77$). Thus, we verified the

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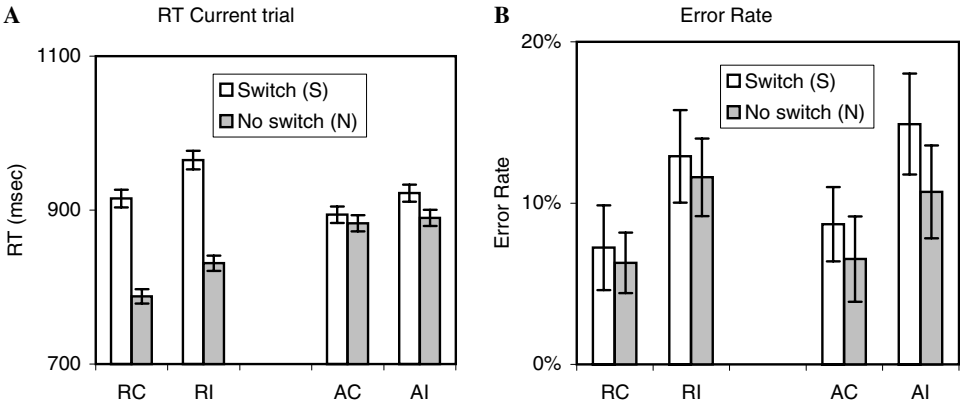

Fig. 2. Current trial effects. (A) Response time. Switch costs are reflected in the response time difference between switch ("S") and no-switch ("N") trials, and they are greater for response repetition ("R") than response alternation ("A"). Incongruent trials ("I") showed a trend toward longer response times than did congruent trials ("C"). (B) Error rate. Switch costs are seen in the increased error rate with switch vs. no-switch trials. Error bars here and in subsequent figures are ± standard error unless otherwise noted.

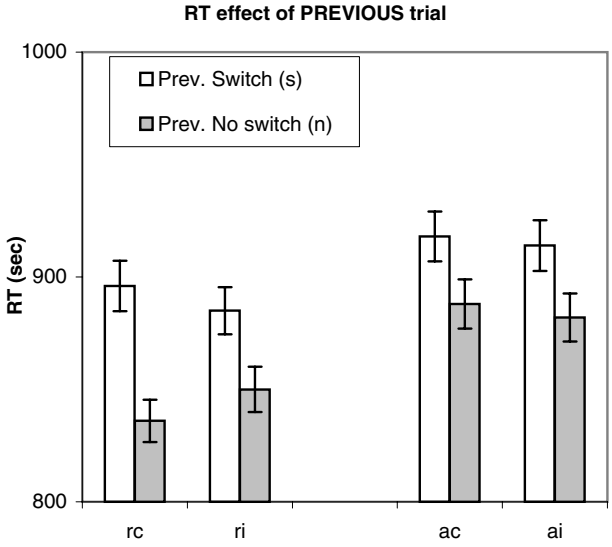

Fig. 3. Effect of preceding trial type on current trial RT. Factors are previous trial congruency, previous trial switch, and previous trial response alternation. All lowercase conditions refer to the type of the previous trial, not the current trial.

hypotheses that parallel effects lead to a persistent response slowing due to task switches and response alternations.

The RT effect of a previous switch trial occurred even in the absence of a current-trial switch. That is, a switch in the previous trial followed by no-task switch in the current trial (sN sequence) led to a 56 ms increase in RT, as compared with two consecutive no-switch trials nN sequences ($t(15) = 3.88$, $p < .002$). Because in both nN and sN trials the same task is repeated from the previous trial, the effects cannot be due to returning back to a

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previously abandoned task-set; therefore, backward inhibition was unlikely to have played a role in this particular effect. However, this effect is consistent with a mechanism of persistent task-switch-induced slowing, as proposed above. Of course, this result by no means excludes a role for backward inhibition in other circumstances when the current trial is a switch trial. There was no corresponding error rate effect for sN (9.3%) vs. nN (8.7%) when the previous trial was correct ($t(15) = 0.77$, $p < .5$).

Analogously, the effect of a response alternation vs. repetition in the previous trial was an increase in RT, even when the current trial was a response repetition. The RT cost under these circumstances was 56 ms ($t(15) = 4.47$, $p < .001$). This agrees with previous findings suggesting a non-specific, persistent slowing effect due to response alternations in 2AFC tasks (Jones et al., 2002).

There was no main effect on response times for previous congruent vs. incongruent trials ($p > .60$). The effects of preceding trial on error rate were generally less pronounced and none of the effects reached significance (all p's $> .10$).

4.3. Interactions between current and previous trials

The effect of previous trial type interacted with the current trial type. There was a three-way interaction among current switch, preceding congruency, and *preceding switch* ($F(1,15) = 10.24$, $MSe = 5357$, $p < .006$), which suggested that response time switch costs are increased if the previous trial was a no-switch/incongruent trial (Fig. 4A). A three-way interaction tendency (Fig. 4B) was found among current switch, preceding congruency, and *current congruency*, but this failed to reach significance ($p < .09$). However, subsequent t-tests showed that the increased switch cost due to prior incongruency vs. prior congruency with current incongruent trials (iI vs. cI effect on switch cost) was significant ($t(15) = 2.22$, $p < .05$). The effect of the preceding switch condition further amplified this effect such that the switch cost difference was greater if the preceding trial was a non-switch. Specifically, response time switch costs (Fig. 4C) were larger ($t(15) = 2.37$, $p < .02$) for previous no-switch, current incongruent trials (niI vs. ncI) when the previous trial was incongruent (mean 124 ms) than when the previous trial was congruent (mean 53 ms). Notably, previous trial incongruency vs. congruency speeded response time by 28 ms ($t(15) = 2.41$, $p < .03$) for current no-switch, incongruent (iNI vs. cNI) trials. There was no discernible effect of preceding incongruency on switch cost when the current trial was congruent (iC vs. cC). These results involving previous-trial incongruency are consistent with earlier results (Goschke, 2000) and further demonstrate that the effect of prior incongruency is amplified by a previous no-switch trial.

Were the effects of previous trial incongruency on current trial reaction time due to the incongruency itself or rather to the increased response time of the previous trial? To examine this issue, we partialled out the previous trial RT from current correct trial RT for each subject. We found that despite controlling for preceding response times, preceding incongruent trials still speeded response time by 31 ms ($t(15) = 2.58$, $p < .03$) for current no-switch, incongruent (iNI vs. cNI) trials. Likewise, trials with a preceding incongruency still showed a strong trend toward increased switch costs (62 ms on average) for previous no-switch, current incongruent trials (niI vs. ncI), as in Fig. 4C ($t(15) = 2.11$, $p = .052$). These results suggest that the effects of preceding incongruency on the current trial were due to the preceding incongruency itself and not simply an artifact of the lengthened response times of the previous trial.

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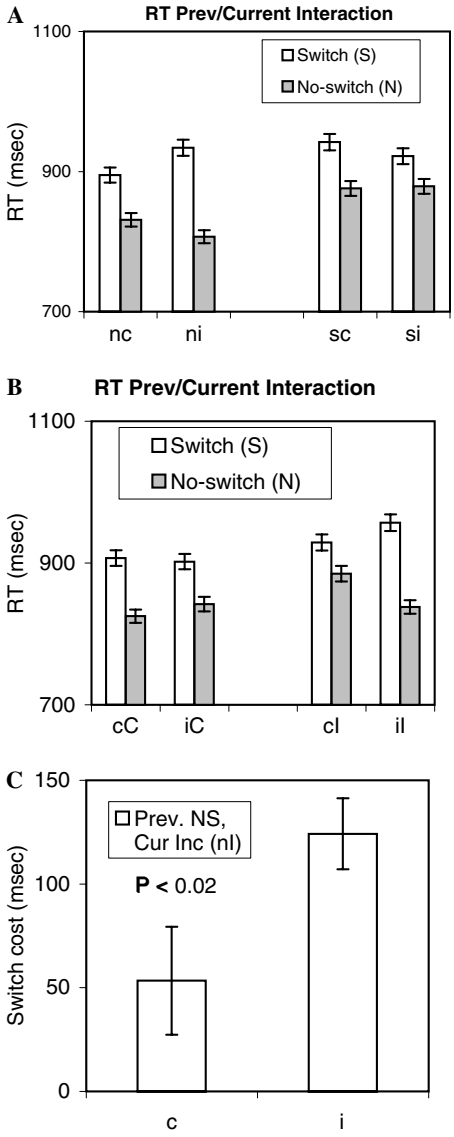

Fig. 4. Interactions of current and previous trial types on RT. Previous trial factors denoted by lowercase, e.g., "n" for previous no-switch trial. (A) Significant interactions of current switch, previous switch, and previous congruency factors. (B) Interactions among current switch, current congruency, and previous congruency factors. (C) Increased response time switch cost for previous incongruent trials, restricted to previous no-switch, current incongruent trials.

The full 64 conditions of current- and previous-trial type combinations yielded a range of effects and mean RTs ranging from 701 to 1047 ms. With 1200 trials per subject and 16 subjects, each of the 64 conditions was estimated on the basis of an average of 18.75 trials per condition per subject. The minimum average number of trials per condition across subjects was 13.06, and the minimum number of trials sampled in a given condition in

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a given subject was 3. Thus, none of the 64 RT conditions was missing data from any subjects. The standard error across subjects in each condition averaged 68 ms. For effects of less than 6 factors, more data points were available, and the analysis was proportionally more powerful.

4.4. Error rate

With respect to error rates, a four-way interaction was found among previous alternation and all three current trial factors (Fig. 5A) ($F(1,15) = 5.24$, $MSe = .0019$, $p < .04$). This indicated that switching interacts with alternation (i.e., greater error switch costs for response alternate than response repeat trials) only when the current trial is incongruent and the previous trial was a response repeat (rRI vs. rAI effect on error switch cost) ($F(1,15) = 31.625$, $MSe = 13320$, $p < .0001$). The finding suggests that the response priming effect of two response repetition trials in a row counteracts the increased error tendency associated with switch incongruent trials. However, when the current response also alternates, the response priming effect works in the opposite direction to increase error tendencies.

Another four-way interaction (Fig. 5B) was found among current and previous alternation and current and previous switching ($F(1,15) = 7.51$, $MSe = .0065$, $p < .02$). The interaction indicated that the general trend for error switch costs to be greater on current response alternate trials compared to response repeats reversed when the previous trial was a no-switch response-alternate (naR vs. naA effect in error switch cost). This might occur if the previous alternation sets up an expectancy for a current alternation that provides relative facilitation on task-switch trials if the expectancy is confirmed, but increased interference if the expectancy is violated (with a current trial response repeat). The effect seemed to be cancelled out though if the previous trial was also a switch trial. Such a pattern supports the hypothesis that switching and alternation exert effects on cognitive control via a common pathway.

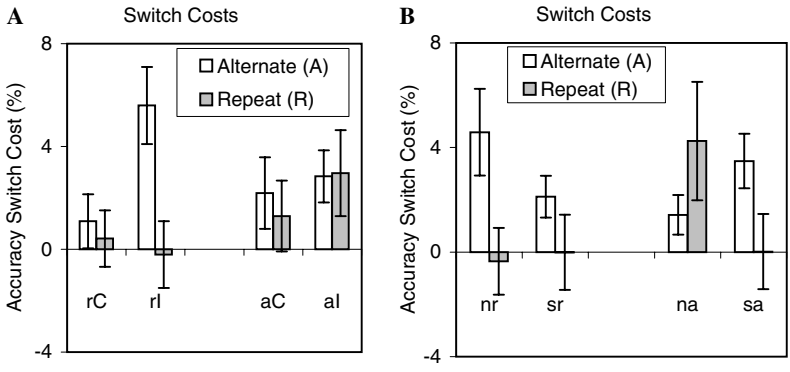

Fig. 5. Interactions of current and previous trial types on error rate. Same conventions as Fig. 4. (A) Accuracy switch cost is largest for alternate, incongruent, previous repeat trials. (B) Accuracy switch cost is greater for repeat trials following alternate/no-switch (naR) trials.

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4.5. Speed–accuracy effects

We examined whether subjects showed evidence of speed–accuracy effects. For this analysis, we first controlled for condition effects as follows. We regressed out the condition means so that the mean RT for each of the eight current-trial conditions (combined across correct and error trials) in each subject was the same as the grand mean over all conditions and subjects. We refer to these adjusted RTs as condition-controlled RTs. Trials for each subject were then binned by response time into 100 ms wide bins, centered on integer multiples of 100 ms. Error rates for each bin were calculated. Overall, accuracy showed an inverted U-shaped function (Fig. 6), such that across all subjects, accuracy was highest for middle RT bins and lower for extremely fast RTs. To assess whether a speed/accuracy tradeoff existed, we first looked at all trials with condition-controlled RTs in bins from 100 to 2000 ms and found no effect ($r = .31$, $t(18) = 1.37$, $p = .19$). However, when we looked at trials in the fastest bins with midpoints from 100 to 1000 ms, inclusive, we found a positive correlation between RT and accuracy ($r = .86$, $t(8) = 4.67$, $p < .002$), consistent with a speed/accuracy tradeoff. Further analysis showed that subjects showing a strong speed/accuracy tradeoff generally had higher error rates. Thus, there may have been a floor effect in that subjects with already low error rates at fast RTs would not show significant further error rate reductions with increasing RTs.

Similarly, we found an opposite effect for slower RT trials. For trials with condition-controlled RT in bins from 1000 to 2000 ms, accuracy decreased with increasing RT ($r = .75$, $t(9) = -3.40$, $p < .01$). This may reflect an occasional failure to engage the task (de Jong, 2000; Nieuwenhuis & Monsell, 2002), in which subjects performed some trials both more slowly and less accurately. These results are consistent with a speed–accuracy tradeoff for faster, "engaged" trials superimposed on a periodic failure to engage.

We further tested explicitly to see whether task switches as compared with no task switch would lead to response slowing and a corresponding increase in RT and reduction in error rate in the subsequent trial. In the fastest 200 trials per subject (absolute RTs), task switches led to increased response time in subsequent trials ($F(1,15) = 9.06$, $p < .01$), but there was no apparent effect on error rate ($p = .57$) despite the observed overall speed accuracy tradeoff in fast trials. An error rate effect was not found even when the analysis was restricted to the 100 fastest trials per subject ($p = .38$). We then tested another (but not mutually exclusive) hypothesis, which is that switch-induced slowing also modulates a tradeoff in favor of exploration (i.e., more broadly focused attention) at the expense

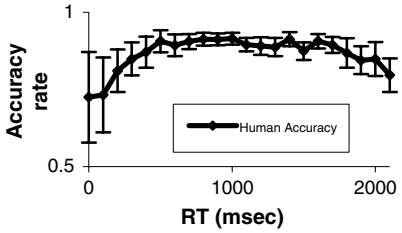

Fig. 6. Accuracy vs. response time. Effects of each of the eight current trial conditions on the means in each subject have been regressed out. The inverted U-shaped curve may reflect two separate effects. For the fastest trials, a speed–accuracy tradeoff effect appears. For the slowest trials, increased response time is associated with higher error rates, which may reflect a general failure to engage the task in the slower trials.

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of exploitation. If so, then a previous-trial switch vs. no-switch trial should increase the error-rate effect of incongruent distractors in the current trial, leading to an interaction between previous trial switch and current incongruency on error rate. We found a trend in this direction ($F(1,15) = 3.55$, $p < .08$).

5. Experiment discussion

As a whole, the results reveal a complex interplay of higher-order sequential effects. Nonetheless, several principles stand out. First, while incongruency in the current trial generally slows response time, we replicated the paradoxical finding that an incongruent trial can actually speed subsequent responses (Goschke, 2000). Preceding incongruency led to faster responses when the task repeated on the subsequent trial (i.e., current no-switch), but slower responses when the task did not repeat again (i.e., current switch). Furthermore, we found that this effect is amplified by a preceding no-switch trial. This may be due greater activity of the established task set in the previous trial, which affords incongruency more opportunity to enhance the then-current task set and inhibit the irrelevant task set. We interpret this finding to mean that a prior incongruency helps increase focus on the current task set, thereby facilitating subsequent performance of the same task but increasing the cost of a task switch. This result is consistent with the hypothesis that cognitive control mechanisms such as ACC may be part of a conflict-control circuit that drives increased attentional focus to task relevant stimuli (Posner & DiGirolamo, 1998).

Second, in addition to the switch cost and response alternation cost, we confirmed the presence of longer-lasting effects that persist across multiple trials, consistent with a cognitive control mechanism. These effects include both response alternation-induced slowing and task switch-induced slowing. The persistence of these effects across trials and their presence even when there is no task switch in the current trial suggest that the effect is distinct from backward inhibition (Mayr & Kliegl, 2000). It might be argued that the slowing effect of a previous switch trial even with a current no-switch trial (e.g., sN vs. nN) reflects simply a lack of cumulative priming in the previous switch condition. In that case, arguments from Occam's razor might counter a putative control loop as the supposed basis of the slowing effect. However, if priming were a significant factor, then the task sequence sS (e.g., BAB) should be faster than nS (e.g., AAB), since the repeated task B should facilitate the response in the repeated switch condition. However, studies of backward inhibition show that the opposite is true (Mayr & Kliegl, 2000). The results are, however, consistent with a mechanism that causes a persistent slowing of responses on the basis of detected task switches, a possibility we explore further below. The absence of a significant interaction between the two persistent slowing effects due to task switches and response alternations leaves open the question of whether they are due to a shared cognitive control mechanism. The computational model developed in the following section examines the degree to which these mechanisms can account for the observed higher-order sequential effects of task switching.

6. Modelling study: Simulating task-switching sequential effects

The observed sequential effects of task switching in humans presumably reflect a complex interplay of various mechanisms. At this level of complexity, including 3- and 4-way interactions, the behavioral effects are not easily or intuitively mapped to underlying

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mechanisms. However, this same complexity that makes direct conclusions from the data difficult can be leveraged as an advantage for computational modeling, because the abundance of effects provide stronger constraints on candidate computational models. Given the above behavioral results, we hypothesized that the effects could be accounted for in large part by several relatively simple mechanisms. We first present these principles conceptually, and then we describe specific implementation details and processing in the model. Following this, we present the results of simulations demonstrating the success of the model in capturing relevant behavioral phenomena as well as predicted effects of specific model lesions.

7. Model mechanisms

1. Active maintenance of task set representations bias responding according to the currently relevant task. This concept has been widely discussed previously (Jacobsen, 1935; Miller & Cohen, 2001) and has formed an essential part of previous models of controlled responding in cognitive tasks (Braver & Cohen, 2000; Brown, Bullock, & Grossberg, 2004; Gilbert & Shallice, 2002; Reynolds et al., 2006). In our model (Fig. 7A), a task cue activates a persistent representation of task set. Recurrent excitation in this task set layer allows for stable recirculation and active maintenance of the current task set. The pattern of activity represents the task set, and this activity in turn biases the transformation of target signals into movement commands according to the current task set. The model defines movement (such as a button press) as a behavioral action initiated when a corresponding model output layer cell's activity reaches a fixed threshold (Hanes & Schall, 1996). We define a hidden or ''plan'' layer between the target stimuli input and the output layer cells, where the movement is specified prior to its execution by the output layer cells. Specifically, each plan layer cell responds to a unique combination of signals from task set representations and target-stimuli-representing input layer cells, and in turn drives the response. Thus, the task-set layer activity pattern ensures execution of the task-appropriate response to the target.
2. An Incongruency detector (INCd) monitors control of task set, detecting conflict between incongruent response processes and enhancing activity of the current task set representation. Under this hypothesis, conflict between incompatible stimulus-response processes drives INCd (Fig. 7B), as in previous studies of conflict (Carter et al., 1998). In this case, conflict consists of the coactivation of incompatible plan layer representations, driven by incongruent target stimuli. The conjunction of simultaneously active incompatible plans drives transient activity in specific cells in the INCd layer, whose activity signals the presence of response conflict. Following the observed dissociation between areas of the brain that detect conflict and areas of the brain that implement cognitive control (Botvinick et al., 1999, 2001), the conflict-related activity in INCd does not directly implement cognitive control. Instead, it drives a longer-lasting increase in the activity of downstream cells, which in turn augment the activity of task set representations implementing cognitive control, thus amplifying the processing of task-relevant stimulus-response processes (Posner & DiGirolamo, 1998). The conflict-control loop pathway is completed as amplified processing of the task-relevant pathway overrides conflict from the inappropriate response process. Thus, following an incongruent trial with this model mechanism, switch costs are expected to increase (as found

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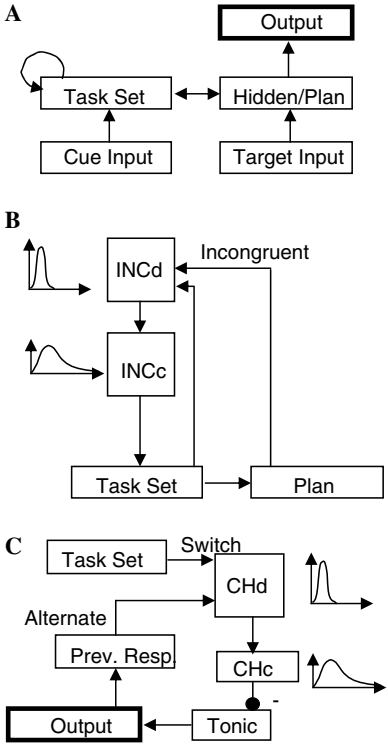

Fig. 7. Model components. (A) Cues activate persistent representations of task set, and these bias the Hidden Plan layer to respond appropriately to the target (B) Incompatible response processes are detected by INCd, which activates a slowly decaying INCd output signal, which in turn excites the corresponding task set representation. This facilitates responses for the same task set but opposes responses according to the other task set if a switch occurs. (C) When the task set switches or the response alternates, CHd detects the change and responds phasically. The CHd in turn activates a slowly decaying control signal (CHc), which suppresses tonic excitation of the Response layer. Thus, the net effect of task set or response changes is a response slowing that persists across trials.

behaviorally above). This is due to the fact that amplification of the present task set may speed RTs when the same task is repeated, as has been found previously (Goschke, 2000) but slow RTs when the task switches, because amplification of the previous task set persists even though it may no longer be relevant. A previous model of task switching (Gilbert & Shallice, 2002) has suggested that response processes feed back to amplify task set representations, so that longer response times (such as those associated with incongruency) might integrate over time to amplify current task set or "task demand" (Gilbert & Shallice, 2002) representation activity (and likewise persist into subsequent trials). In this case, it is unclear why a separate incongruency detector loop would be needed here. However, as we have shown above in the human data, the effects of previous-trial incongruency on current trial response times and switch costs persist even when controlling for previous-trial response time. These results suggest the need for a separate incongruency detector. Notably, the current task set representation contextualizes the model INCd layer, so that INCd layer units respond to unique combinations of task set and incongruent stimulus representations. These in turn augment the activity

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of the corresponding task set representations. Finally, the model makes a crucial distinction here between executive control as driving a switch in task set (Rogers & Monsell, 1995) and executive control as increasing the strength of an already established task set. For this task, the model posits that the INCd performs primarily the latter (task set*strengthening*) rather than the former (task set *switching*).

3. A change detector (CHd) monitors control of responding by detecting task set or response changes across trials and subsequently slowing responses. In contrast with INCd, which monitors for simultaneous incompatible response cues *within* a trial, CHd monitors *across* trials. The model CHd (Fig. 7C) detects brief periods of conflict as coactivation when the new task set representation becomes active and the previous one has not yet been deactivated by lateral inhibition. Similarly, conflict-related activity in an oculomotor countermanding task has been found in the monkey (Stuphorn et al., 2000), where conflict is engendered during a brief period when a new plan becomes active before the previous plan has been deactivated. By extension, in the present model, the conflict is between conditions of consecutive trials rather than the same trial. Likewise, a persistently maintained representation of the previous response (response buffer) allows a similar coactivation to be detected briefly as an alternated response representation replaces the previous response representation that was activated in the preceding trial. Following the distinction between performance monitoring and control mechanisms as with the INCd above (Botvinick et al., 1999), the net result of CHd activation is to remove (''brake'') a tonic arousal signal to the response pathway, as in previous models (Jones et al., 2002). Critically, the effect of this brake is a persistent slowing of responses not only in the current trial but also in subsequent trials, as suggested by the behavioral result that an RT cost of previous switches persists even though the current trial is a no-switch trial (see Section 5). Thus, this second conflict-control loop mechanism contributes in part to the switch cost, but its effects are not limited to the switch cost and persist to slow responses on subsequent trials. That the slowing effect of this mechanism leads to increased accuracy can be demonstrated by an increased error rate with lesions of CHd (see Section 10).
4. The contribution of CHd to the task switch cost is distinct from that induced by associative task set interference (TSI). Associative TSI has been argued to contribute to the switch cost (Allport et al., 1994; Waszak et al., 2003; Wylie & Allport, 2000a). According to this theory, target stimuli that have been paired with one task will lead to a switch cost if presented in the context of the other task. Previous computational modeling studies (Gilbert & Shallice, 2002) have implemented this mechanism as representations of target stimuli learn to activate the task set representation with which they have been paired. The present model implements a similar mechanism by assuming that plan representations have a reciprocal, adaptive excitatory projection to task set representations. This allows target stimuli, via the movement plan representations they activate, to become associated with and thereby more strongly evoke a particular task set. The net result is effectively an implementation of the TSI model, which allows learned stimulus-to-task pairing. Nevertheless, CHd makes an additional contribution to the switch cost over and above associative TSI effects. This can be demonstrated by showing that lesions of the CHd reduce this switch cost without completely eliminating it (see Section 10). Thus, CHd drives response slowing effects in both the current and subsequent trials.

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5. Priming from the plan to the output layer allows plan representations to more efficiently drive responses that are repeated in the same task context. The model was designed to capture the crossover interaction between task switch and response alternation in the current trial, as shown behaviorally above. This result replicated previous studies that have found the same pattern (Fagot, 1994; Meiran, 1996; Rogers & Monsell, 1995). Specifically, while response repetition leads to facilitation when the task does not switch, a response repetition leads to paradoxical slowing when the task does switch. To account for this finding, the model posits a mechanism similar to previous models (Meiran, 2000a). Specifically, making a particular response (e.g. left) in a given task leads to a strengthening of that response within in that specific task context, by strengthening synapses from the plan layer to the output layer. This strengthening may correspond to biological processes of long-term potentiation (LTP). This strengthening occurs at the expense of synaptic strength from the plan to the output layer cells driving the leftward response in the other task, due to normalization of synaptic strengths for a given response across tasks (Grossberg, 1982). Although such normalization mechanisms have typically been promoted on purely computational grounds (i.e., that they avoid "synaptic explosion" effects), initial empirical evidence for normalization of synaptic strength by postsynaptic cells has recently been found (Koester & Johnston, 2005). At the same time, the plan-to-output layer projections for both responses in the other task remain constant (and equal). Thus, a trial in which the task switches and the response repeats entails activation via the competitively weakened pathway for the same response in the other task. We have implemented a similar mechanism in another model of task switching (Reynolds et al., 2006).

8. Model methods

8.1. Architecture

The model (Fig. 8) was simulated in a new computational modeling framework, RNS++ (Brown, 2003), that we developed to facilitate integrated computational neural

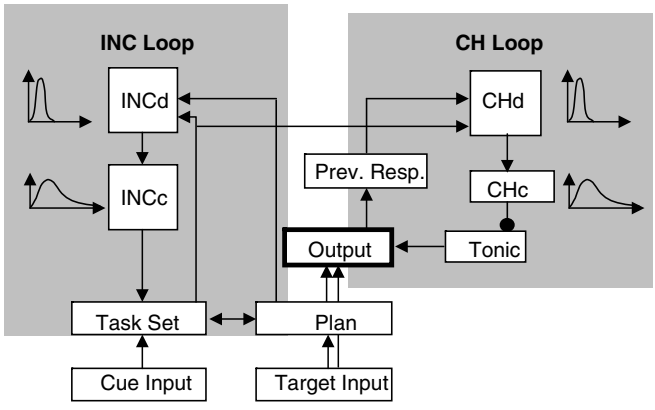

Fig. 8. The complete model, showing both cognitive control loops.

modeling of behavior, electrophysiology, and functional imaging. The network can be conceptualized in terms of two major divisions: (1) a network that can accomplish task-switching; and (2) a supervisory control system that interacts with this task-switching network (and is discussed in detail below). Within the task-switching network, the architecture consisted of five layers of units: a target input layer, a cue input layer, a plan (or hidden) layer, a task-set layer and a response output layer. There were two task cue inputs that represented the two different cues (LETTER/DIGIT) in a localist fashion. For simplicity, all stimuli that mapped to a particular response in a given task set were collapsed and represented by a single input node to the model. Thus, the target input layer was composed of four units, two for each of the task dimensions (VOWEL/CONSONANT for the letter task and ODD/EVEN for the number task). The target inputs connected in a one-to-one fashion with the four units of the plan layer while the cue inputs connected in a one-to-one fashion with the two units of the task-set layer. The task-set units had recurrent self-excitatory connections that enabled activity to be recirculated, and thus actively maintained over preparatory intervals. The task-set units also had a feedback connection to the plan units such that preparatory task-set activity could bias activation in the plan layer. In particular, the task-set unit associated with each cue excited both of the features of the relevant task dimension (e.g., the LETTER task-set unit excited both the CONSONANT & VOWEL plan units, but not the other two units). Finally, there was strong lateral inhibition among units in the task-set layer such that the presentation each new cue caused an updating of the relevant task-set activity. The connection between the task-set units and plan units was bi-directional, such that target-induced activity in the plan layer could also enhance task-set activation.

The plan units connected to the output units such that the appropriate response was made for each task. The network was judged to have made a behavioral response when the activity of one output unit reached a pre-specified activation value. An additional layer, the response buffer, had two units that were activated by the response given on a particular trial and maintained this activity pattern until a response occurred in the subsequent trial.

8.2. Processing

Activity in the model was simulated as a dynamical system with continuous-time rate-coding of individual cells, which allows for a neurobiologically realistic representation of the time course of neural activity (Brown et al., 2004). Below, we provide a conceptual description of the simulation methods. Specific details of the equations governing processing and model parameter selection are described in the Appendix A. Units in the model compute their output activation based on integration of both excitatory and inhibitory signals (whether these are coming from other units or represent passive forces—e.g., leak currents). Updating of activation was simulated in very fine time steps to approximate the real-time dynamics of neural activity during a given trial and to closely simulate the fine timing of environmental events as presented to human subjects.

Each simulated trial consisted of the same series of events and timing experienced by the human subjects performing the behavioral study above. At 200 ms after the beginning of each trial, a cue appeared. This was represented by clamping one of the two corresponding Cue input layer cells (Fig. 9) to 1.0 for 300 ms, at which point the Cue input cell was clamped back to its default activity value of 0. The cue units

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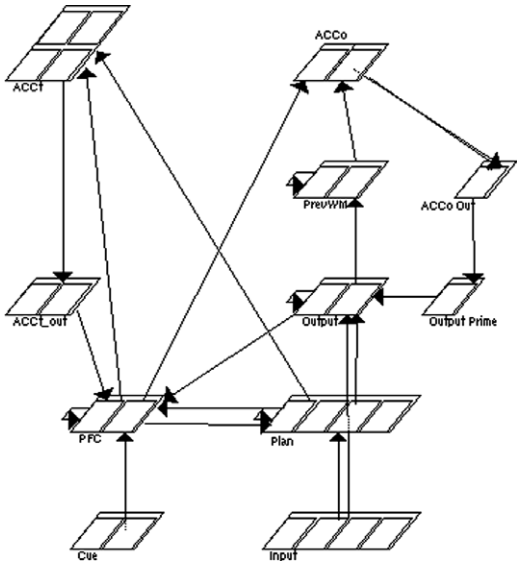

Fig. 9. A screenshot of the model as simulated, showing individual neuronal units in each layer.

then directly activated the corresponding task set representations, causing the appropriate task-set unit to increase activity to an asymptotic level. The self-recurrent connections caused this activation to be sustained at a stable fixed level. Lateral inhibition ensured that only one task-set unit remained active during the preparatory period. Additionally, a tonic inhibitory current ensured that baseline activation of these units remained low. The task-set units also received a persistent excitatory input coming from the supervisory control module that served to modulate processing dynamics, which is described further below.

After a delay of 1500 ms, the target input appeared and remained present until either a response was made or the trial ended. The target input was represented by the activation of two out of the four input units, corresponding to the presence of one letter feature (VOWEL or CONSONANT) and one digit feature (ODD or EVEN). Active units were clamped to 1 and inactive units clamped to 0. The target input was then propagated to the hidden/plan layer where it was integrated with excitatory input coming from the task-set layer.

The propagation of plan unit activity to the response layer enabled the simulation of one of two behavioral outputs to match the leftward or rightward manual response used in the behavioral study. The response units also received a small and non-specific input from the target units, in that each target unit excited both response units. This additional non-specific input was intended to simulate a degree of response ambiguity, such that some level of activity was always present in the incorrect response channel. This provided a means for the network to produce errors, when combined with the effects stimulus priming and noise. The response units also received a non-specific, tonic excitatory input that served to generally enable quick responding. The actual response made by the network on a given trial was coded by the first unit to reach a prespecified activation threshold. Response times were encoded by the latency required to reach this threshold.

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8.3. Priming mechanisms

Input to units was governed by the strength or weight of synaptic connections. These synaptic weights were not fixed for many of the pathways, but were instead dynamically changing in an experience-dependent manner that corresponded to priming effects. Two sets of connections were specifically affected by experience: the connection from the hidden to task-set layer and the connection from hidden to output layer. In both sets of connections the weight changes corresponded to a basic associative or Hebbian (Hebb, 1949) learning rule, such that co-activation of a sending and receiving unit led to a strengthening of the connection between them, in proportion to the magnitude of co-activation. The effects of changing the hidden layer to task-set connection implemented a form of associative TSI, since targets that have previously been associated with a specific task-set in a previous trial will more strongly activate this task-set when the targets are activated in a current trial (even if the previous task-set is no longer appropriate). The connections from the hidden to output layer implement a form of response priming, such that a response activated on a previous trial will have a strengthened connection to the hidden units that correspond to the stimulus features that activated the response. For both sets of adaptive connections the priming effect was time-dependent such that there was a passive exponential decay of any strengthening back to baseline values with a relatively short time constant (i.e., with a half-life of several seconds).

8.4. Performance monitoring modules

The basic task-switching network interacted with two additional modules that subserved performance monitoring functions. Each module had two components: a conflict detector and a control signal output activated by the conflict detector (Botvinick et al., 1999). The conflict detector responded phasically to transient states of conflict, and drove the control signal to respond with a rapid onset of slowly decaying activity. Thus, the control signal persisted beyond the time at which conflict is detected and into subsequent trials.

The two different performance monitoring modules were distinguished by the type of conflict detected in each as well as the control effects they drive. The INC module was responsible for the detection and control of *incongruency conflict*: the simultaneous presentation of stimuli associated with incompatible responses.[1] The detection units in this module received inputs from both the target layer and the task-set layer and responded to particular conjunctions of task and stimulus features that were associated with incongruency in each task. There were four INC detection units, with one unit responding to each of the two incongruent stimulus patterns present for each of the two tasks.

When an incongruent stimulus pattern was detected, a specific unit in the detection layer (INCd) was phasically activated and in turn excited one of two units in the control output layer that corresponded to the currently active task. This control unit then responded with a persistent increase in activity which was transmitted back to the task-set layer, and served to modulate activity there. The effect of the control input was to

[1] The astute reader will note that we have chosen to model INCd conflict detection on the basis of incongruent stimuli in the hidden layer rather than on the basis of simultaneous responses in the output layer. Both approaches may work well, although we did not explore the latter possibility.

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enhance the difference in activation among task-set units, such that the currently active unit was further excited while the inactive unit was more strongly suppressed. Because of the persistent nature of the control output signal, the signal remained across trials, and therefore facilitated responding under conditions where task-set remained constant across trials (no-switch conditions) but impaired responding when the required task-set changed across trials (switch conditions).

A second performance-monitoring module, CH, was responsible for the detection and control of *change-related conflict*: cross-trial changes in the required task-set or response. The detection units (CHd) in this module received inputs from two different sources, the task-set layer and the response buffer layer. There were two CHd units, which each responded to a brief period of coactivation in the layer (task-set or response buffer) that was being monitored, as a new representation became active before the previous one had shut off. This brief coactivation period occurred only under conditions in which the response or task-set changed across trials. The transient activity of CHd units excited more persistent activity in a corresponding change-driven control output unit. This activity in turn caused inhibition of the non-specific tonic excitatory input to the response layer. The control unit activity can thus be conceptualized as a ''brake'' system that reduces in a nonspecific manner the baseline activity level in the response layer. Such a mechanism will produce non-specific slowing of responding.

9. Model analysis methods

Several analyses of the model were carried out, as follows. First, the model was fit to corresponding human behavioral data (see Appendix A). Second, overall goodness-of-fit was evaluated between model and human data, taking into account reductions in effective degrees of freedom due to free parameters used in the data fitting process. Third, a number of response time and error-rate effects predicted from the human data were examined in the model, looking at effects of current and previous trial types, as well as interactions between them. Fourth, sequences of more than three trials in length were specifically examined with regard to effects of task-switching, even though the model was not explicitly fit to these longer sequence data. Finally, the model performance monitoring mechanisms were individually lesioned, and the model was refit in order to further specify the contributions of each model performance monitor to the model's ability to fit the human data.

The model was tested under the same experimental conditions as the human subjects above. For simplicity, all stimuli that mapped to a particular response in a given task set were collapsed and represented by a single input node to the model. Thus, the model had four stimulus input nodes (odd, even, vowel, and consonant) in addition to two separate task cue representations (odd/even, and vowel/consonant). To evaluate statistical significance of the model effects, the model was run for 19,200 trials. These were grouped into sets of 1200 trials, with each trial group corresponding to a virtual subject. This allowed a direct comparison with the 16 human subjects, each of whom performed 1200 trials. The model output was then analyzed statistically using the same analysis routines as for the human data above. One potential issue with this approach is that the lack of individual differences among the 16 virtual model subjects may lead to alpha inflation. However, as we discuss below, we found that the statistically significant model effects were also numerically significant and similar in size to those found in the human data. Furthermore, as we show later, this supports the conclusion that the absence of effects in the

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lesioned model demonstrates the essential role of various model mechanisms. Thus, the individual variability not simulated in the current model does not appear to negatively impact the interpretation of positive vs. null statistical effects in the simulation results.

10. Model results

The model provided a comprehensive fit to the data, capturing a number of sequential and higher-order effects. The model fit all 64 response time data points in addition to the 8 current trial error rate data points, for a total of 72 data points. There were 21 free parameters in the model, which left 51 degrees of freedom (DOF). The model fit the 64 RT data points with a Pearson correlation of $r = .83$, which was highly significant even with fewer degrees of freedom due to the model's free parameters ($t(41) = 9.5$, $p < 10^{-9}$), as shown in Fig. 10. The model captured the specific effects on human RT. Specifically, the model showed current trial RT effects of switch cost (112 ms, $F(1,15) = 550$, $MSe = 5783$, $p < 10^{-12}$), alternation (24 ms, $F(1,15) = 62$, $MSe = 2607$, $p < 10^{-5}$), and incongruency (43 ms, $F(1,15) = 110.24$, $MSe = 4281$, $p < 10^{-7}$), as seen in Fig. 11A. The model also captured the RT effects of previous trial conditions (Fig. 11B) of switch (27 ms, $F(1,15) = 38.74$, $MSe = 4827$, $p < 10^{-4}$) and response alternation (8 ms, $F(1,15) = 6.17$, $MSe = 3463$, $p < .03$). There was no main RT effect of previous trial incongruency in humans, and the model did not predict one ($F(1,15) = 0.35$, $MSe = 5004$, $p > .57$). Fig. 11 reveals an apparent discrepancy between the model and human RTs, in that the model responds too slowly when the preceding trial was a no-switch, repeat (nr) trial. At least part of the discrepancy may be attributable to inherent noise in the fitting process (see Appendix A). Nonetheless, the greater slowing effect of previous trial task switches (s) and response alternations (a) on current-trial RT in the human data as compared with the model suggests that if anything, the simulations may have underestimated the contribution of the change-induced subsequent-trial slowing effect driven by the CHd loop. Similarly, we analyzed the 64 RT data points in Fig. 10 to see which ones showed a significant difference ($p < .05$, Bonferroni corrected) between model and data. Only 1 point (nrcSAI) was significant, followed closely by the corresponding current no-switch condition (nrcNAI), which failed to reach significance. We found that the model switches were especially slow and no-switches were especially fast (compared to the human data) for current alter-

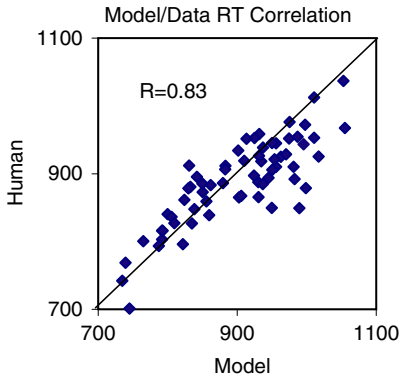

Fig. 10. The response time correlation between the model and the data. $R = .83$, $t(41) = 9.51$ ($p < 10^{-9}$).

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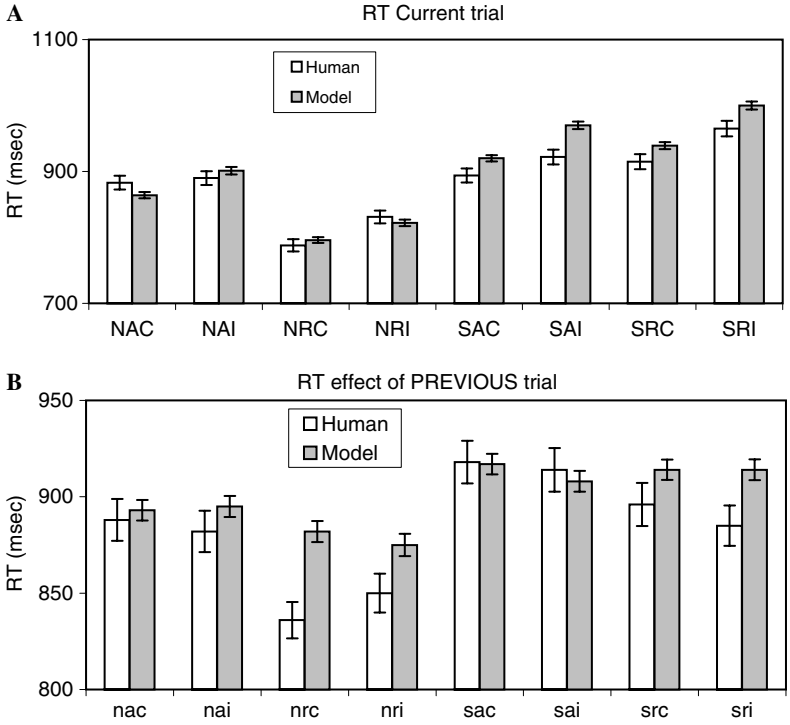

Fig. 11. Response time effects (A) RT as a function of current trial condition, showing fit between model and data. (B) RT as a function of *previous* trial conditions. The model captures many of the effects seen in the human data.

nate, incongruent trials when the previous trial was a no-switch, repeat, congruent trial (nrcAI). In both of these cases, the previous trial conditions elicit the smallest control signals possible. This again suggests that the model may have overestimated the contribution of current-trial effects to the switch cost. It may be possible to improve the model fit in this regard by simulating a higher baseline level of control signal in the CHd control loop, but this was not tested.

The model simulated the effects of interactions between current trial task switch and current trial response alternation ($F(1,15) = 154$, $MSe = 4096$, $p < 10^{-8}$), as well as between current and previous task switch ($F(1,15) = 168$, $MSe = 2684$, $p < .01$). As in the human data, previous-trial incongruency increased the current-trial switch cost, when the previous trial was a no-switch trial and the current trial was incongruent ($F(1,15) = 7.79$, MSe $= 1028$, $p < .02$). Conversely, previous trial incongruency, as compared with previous congruency, led to a facilitation trend (16 ms, $t(15) = 1.96$, $p < .07$) in current no-switch, incongruent trials, in agreement with effects in the human data. Notably, the model also captured a 4-way interaction among current and previous trial task switch and response alternation factors ($F(1,15) = 12.29$, $MSe = 4515$, $p < .004$).

The model also captured several effects of error rate as well (Fig. 12). Specifically, the model captured the main effect of switch cost ($F(1,15) = 7.24$, $MSe = 0.0005$, $p < .02$), incongruency ($F(1,15) = 268$, $MSe = 0.0006$, $p < .0001$), and the interaction between current trial switch and response alternation ($F(1,15) = 13.7$, $MSe = 0.0009$, $p < .003$). As in

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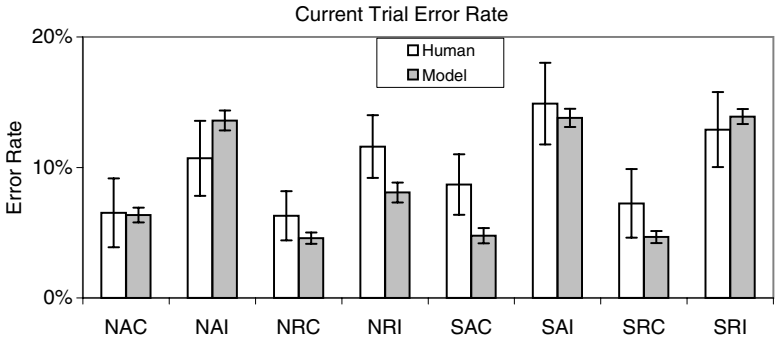

Fig. 12. Error rate effects of the current trial, showing model fit.

the human data, there were no main effects of previous trial task switch, response alternation, or incongruency on error rates ($p > .10$). Furthermore, error rates did not differ significantly for sequences culminating in a no-switch trial sN (8.2%) and nN (9.2%) ($t(15) = 1.38$, $p < .2$), although RTs did differ (sN: 870 ms, nN: 796 ms, $t(15) = 14.6$, $p < 10^{-9}$), in agreement with the human data.

The model also showed speed–accuracy effects (Fig. 13A). With the same analysis used above for the human data (Fig. 6), the model correlation between condition-controlled RT and accuracy in bins from 100 to 1000 ms was positive ($r = 0.85$, $t(6) = 3.88$, $p < .01$). This is consistent with the speed–accuracy tradeoff effects observed in humans above. The model did not show a clear effect of decreasing accuracy with longer RTs, most likely because there was no mechanism that allowed task set representations to fail to engage (de Jong, 2000; Nieuwenhuis & Monsell, 2002). However, we have addressed this issue directly in other models (Reynolds et al., 2006). Also, we directly compared condition-controlled RT distributions for correct and error trials from the model and human data. For the model data (Fig. 13C), RTs were slightly faster for error than correct trials (868 ms error vs. 896 ms correct, $t(15) = 4.25$, $p < .002$). For the human data overall, the error RT data trended in the opposite direction (944 ms error vs. 882 ms correct, $t(15) = 1.93$, $p = .07$). However, this was again due to slow human errors that were not simulated in the model. Human trials with condition-controlled RTs faster than 800 ms showed a trend toward faster errors than correct responses (585 ms error vs. 601 ms correct, $t(15) = 2.06$, $p < .06$), consistent with the model and a speed/accuracy tradeoff in the faster trials (Fig. 13B). Overall, the human and model distributions were similar in both mean and variance, although the model did not reproduce the skewness of the human RT distribution. Ideally, the model should account for skewness. However, the overall difference in skewness between the model and human data did not hinder the model's ability to account for the sequential effects that are the focus of this paper. An account of skewness in the RT distributions is beyond the scope of the model.

We then tested the model explicitly to see whether task switches as compared with no task switch in a previous trial would lead to response slowing and a corresponding reduction in error rate. In the fastest 200 trials per model subject (absolute RTs), task switches led to increased response time in subsequent trials ($F(1,15) = 6.24$, $p < .03$), but there was no apparent effect on error rate ($p = .12$) despite the observed overall speed accuracy tradeoff in fast trials. These results again match the counter-intuitive findings of the human data. However, analysis restricted to the fastest 100 trials per model subject revealed that

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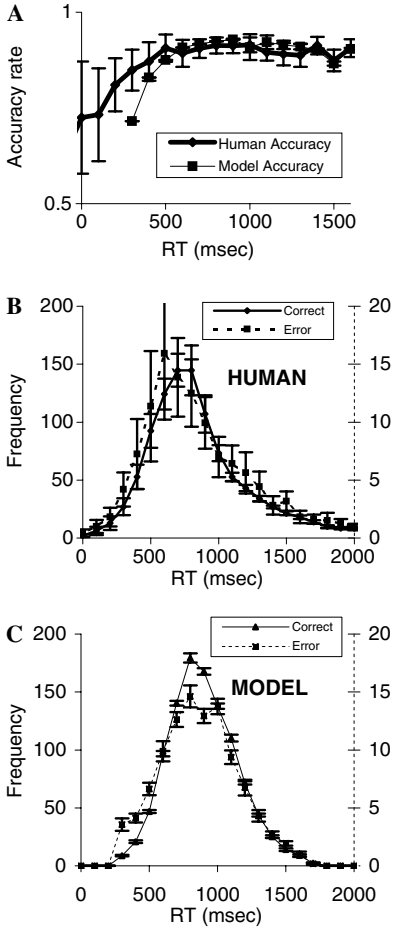

Fig. 13. Comparison of human and model (A) accuracy function. Same analysis as in Fig. 6. (B) Human RT distributions for correct and error trials, with condition means regressed out as in panel A. (C) Model RT distributions for correct and error trials, with condition means regressed out as in panel B.

previous switch trials led to reduced error rates relative to previous no-switch trials ($F(1,15) = 6.10$, $p < .03$), despite no interaction between previous trial switch vs. no-switch and current trial congruency on error rate effects ($F < 1$). Overall, these results are consistent with the possibility that despite the presence of a speed–accuracy tradeoff, increased accuracy driven by switch-induced slowing is partially masked by increased exploration relative to exploitation as the model spends more time processing input stimuli. Furthermore, the model mechanisms are able to account for these paradoxical speed–accuracy effects.

10.1. Higher-order sequences

We also investigated the ability of the model to capture effects of longer sequences of five trials. Previous studies of two-alternative forced choice tasks showed effects of longer

sequences of five trials (Soetens et al., 1985). Recently, other models have shown that performance-monitoring mechanisms similar to those modeled here can account for a significant component of the variance in response time in two-alternative forced choice tasks (Jones et al., 2002). Therefore, we coded trials according to whether the current and preceding three trials were switch (S) or no-switch (N) trials. To do so, for the analysis shown in Fig. 14 only, we used a slightly different convention (Soetens et al., 1985) to code sequences longer than two trials, such that the sequence read left to right describes consecutive trials. For example, the condition "NSNS" refers to a no-switch trial, followed by a switch trial, then followed by a no-switch trial, then followed by a current trial condition of switch. This approach implicitly includes task information from five consecutive trials and yields a total of $2^4 = 16$ conditions. Although the model was not fit to behavioral data on higher-order sequences, the model nonetheless provided a good though not perfect fit to the human data ($r = .90$), as shown in Fig. 14.

10.2. Performance monitor activity signatures

The intact model predicts a specific, quantitative pattern of activity related to the two distinct conflict-control loop mechanisms. Activity in the CHd shows strong current-trial effects (Fig. 15A) of task switching ($F(1,15) = 682$, $MSe = 0.71$, $p < 10^{-10}$) and response alternation ($F(1,15) = 141$, $MSe = 0.23$, $p < 10^{-7}$), and strong counter-intuitive effects of previous-trial task switching ($F(1,15) = 523$, $MSe = 0.62$, $p < 10^{-10}$), previous-trial response alternation ($F(1,15) = 25$, $MSe = 0.31$, $p < 10^{-6}$), and previous-trial incongruency ($F(1,15) = 5.31$, $MSe = 0.40$, $p < .04$), as shown in Fig. 15B. Essentially, task switches and response alternations led mainly to greater activity in the CHd module, as expected, and this effect appeared to be slightly amplified by previous-trial incongruency. Incongruency in the previous trial may have led to amplification of the previous task set, which would lead to greater coactivation of the task sets (and therefore greater CHd activity) when the task switches in the current trial.

In contrast, activity in the INCd showed a strong current-trial effect of incongruency ($F(1,15) = 74848$, $MSe = 0.1$, $p < 10^{-15}$) as expected (Fig. 15C). It also showed smaller

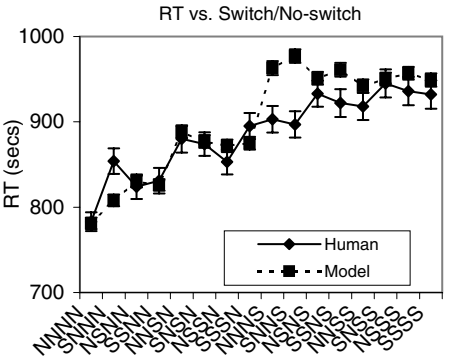

Fig. 14. Model prediction of higher-order sequential effects. The last character in each sequence denotes the most recent (current) trial. For example, "NNNS" denotes three no-switch trials followed by a switch trial. $R = .90$. The model was not fit to these higher order effects, so the fit can be considered predictive.

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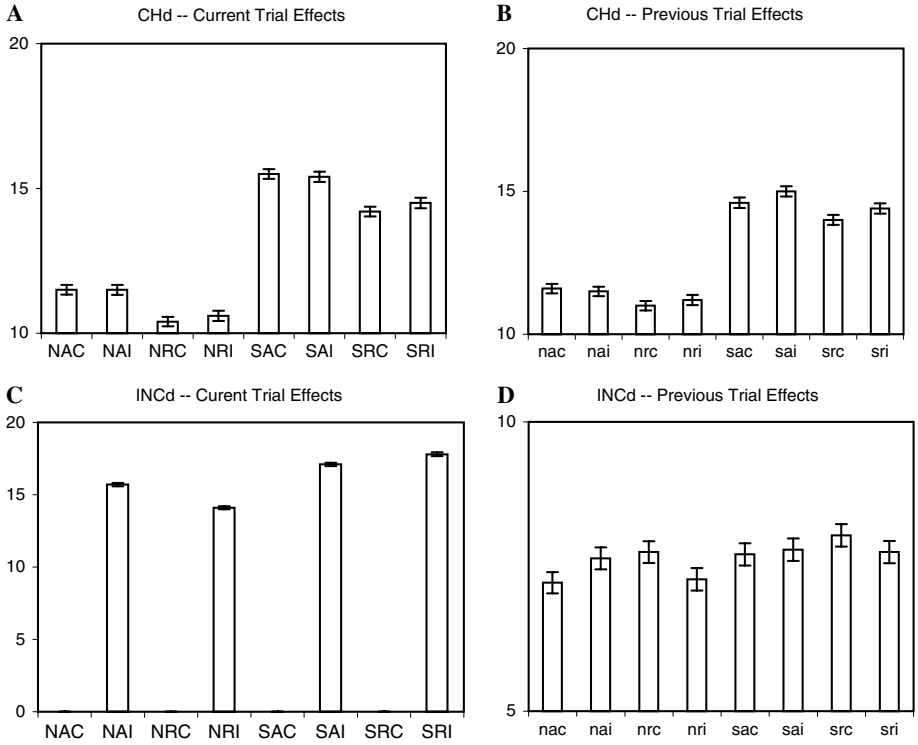

Fig. 15. Activity patterns in the model cognitive control mechanisms. (A) The model CHd shows strong effects of switching and response alternation. (B) Task switches or response alternations in the previous trial yield CHd activity effects in the current trial. (C) The INCd shows a strong current trial effect of incongruency and a smaller but still significant effect of current-trial task switch. (D) Smaller but still significant effects on INCd were due to task switches or response alternations occurring in the previous trial.

but still significant unexpected effects of current-trial task switching ($F(1,15) = 294$, $MSe = 0.17$, $p < 10^{-10}$) and response alternation ($F(1, 15) = 18$, $MSe = 0.11$, $p < .002$). Specifically, task switches seemed to enhance incongruency-driven activity relative to trials with no task switch. This may have occurred as a newly changed task set was less effective in reducing conflict than an established task set from the previous trial. Activity in the INCd also showed smaller but still significant effects of previous-trial switch ($F(1,15) = 6.43$, $MSe = 0.59$, $p < .03$), but not previous-trial response alternation or congruency (Fig. 15D). The INCd response to task-switching was an emergent and counter-intuitive property of the model, as we initially expected the INCd to show effects only of current-trial incongruent stimuli. Overall, these results provide quantitative and testable predictions regarding cognitive control mechanisms in the brain.

10.3. Nested model analysis

Having shown that the model captures a number of significant behavioral effects, we can now establish the significance of the conflict-control loops. To do this, we examined a version of the model without the conflict-control loops, which is effectively nested within

the full model with conflict-control loops. Specifically, we lesioned the model CHd and INCd individually by clamping the activity levels of each one in turn to zero, and then we tested the model with both INCd and CHd lesioned. Whenever CHd was lesioned, we also clamped B in Eq. (15) to zero, because doing so minimized the impact of unchecked tonic bias excitation on the output that made the CHd lesion effects appear more dramatic. Thus, the effects of the CHd lesion reported here are conservative. Also, after lesioning both CHd and INCd, we refit the remaining model parameters to the same data, to avoid the possibility that the effects of CHd and INCd lesions reflected merely an artifact of overall parameter choice in the model. The results (Table 3) show that the control loops improve the overall correlation between model and data most strongly in capturing the effect of previous trial type. We used a generalized likelihood ratio test (GLRT) for nested models (Bickel & Doksum, 1977; Mood, Graybill, & Boes, 1974) to examine whether the improved fit was significant despite the increased number of free parameters used in the control loops. This test is more sensitive than multiple regression analysis (Cohen & Cohen, 1983), because it accounts not only for the change in free parameters but also for the variance of individual data points, which is discarded by multiple regression analysis. Briefly, when both the full and reduced models are best fit to the data by adjusting the free parameters, the GLRT(Λ) can be computed according to $\Lambda = -2\ln(P(\text{full model})/P(\text{reduced model})) \sim \chi^2(N)$, where N is the difference in the number of parameters between the full and reduced models. After subtracting off between subject RT variance from the global mean, we tested the 64 RT conditions. The full model provided a significantly better fit than the reduced model (χ^2 (10) = 30.87, $p < .001$), in agreement with the full model's ability to capture effects that the lesioned and refit model could not. Similarly, the full model provided a significantly better fit than the lesioned model according to the Akaike information criterion (Akaike, 1987), with AIC(full model) = 290.66 and AIC(reduced model) = 301.53. Of note, these results suggest that the sample size of the human data was sufficient to allow model discrimination even in the case of the 64 crossed current and previous trial conditions. With regard to incremental model fits, adding each control loop individually improved the model fit relative to the lesioned model with no control loops (CH: χ^2 (7) = 41.96, $p < .001$; INC: χ^2 (3) = 39.72, $p < .001$). The incremental fits between the full model and lesions of either control loop individually

Table 3
Effects of model lesion on RT effects (P-values) All significant human effects up to sixth order shown

Effect	Human	Intact model	INCd lesion	CHd lesion (no tonic)	INCd, CHd lesioned	INCd, CHd lesioned (fit)
Switch	0.00002	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$
Alternate	0.035	$<10^{-5}$	0.012	0.0002	0.0003	0.0002
Congruent	0.032	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$
Prev. Switch	0.002	0.00002	$<10^{-5}$	**0.3**	**0.65**	**0.74**
Prev. Alternate	0.0013	0.025	0.002	**0.96**	**0.9**	**0.07**
Switch X Alternate	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$
Switch X Prev. Switch	0.035	$<10^{-5}$	$<10^{-5}$	$<10^{-5}$	0.001	$<10^{-5}$
Switch X Prev. Switch X Prev Cong	0.006	**0.8**	**0.85**	0.033	**0.83**	**0.38**
Switch X Prev Cong (nI)	0.017	0.013	**0.88**	0.001	**0.55**	**0.23**
Switch X Alternate X Prev. Switch X Prev. Alt	0.027	0.003	0.0007	0.008	0.00002	0.00006

Bold indicates model failure to capture significant human effects.

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were not significant, although each control loop was necessary for the model to account for specific behavioral effects, as discussed next (Table 3).

10.4. Lesions of cognitive control mechanisms

We used the reduced model with lesioned control loops to examine what the model predicts regarding the role of putative cognitive control mechanisms. Lesions generally rendered the model unable to capture specific effects found in the human data. By virtually lesioning the model, we can elucidate the significance of model performance-monitoring mechanisms' contributions to behavior. Individuals with schizophrenia show reduced conflict-related activity in the ACC and a corresponding reduction in persistent slowing effects following conflict trials (Kerns, Cohen, MacDonald, Johnson, & Stenger, 2005). Also in humans, lesions of the putative performance monitoring areas such as the ACC lead to specific cognitive impairments, (Gaymard, Ploner, Rivaud, Vermersch, & Pierrot-Deseilligny, 1998; Swick & Turken, 2002; Turken & Swick, 1999), although there is some disagreement in the literature as to the effects of performance-monitoring areas on behavioral performance (Fellows & Farah, 2005). The effects of lesions on the model predictions are summarized in Tables 3 and 4.

The model predicts that a previous-trial incongruency results in increased attention to the then-current task set, meaning that prior incongruency increases switch costs. Indeed, this result was found in both the human data and the intact model (Fig. 16A). However, when the INCd was lesioned in the model, this effect was abolished (Fig. 16A, Table 3). Lesions of CHd abolished the previous-trial switch effect and the previous-trial response alternation effect (Fig. 16B). All of these lesion effects persisted even when the CHd- and INCd-lesioned model was refit to the data. In the human and intact model, the reduction of switch costs due to a previous trial switch was mainly due to an increased no-switch response time (Fig. 14). Furthermore, Fig. 14 shows that with more immediately preceding switch trials over longer sequences of trials, the switch cost is further reduced. This suggests that as the likelihood of switch trials increases, the switch cost may vanish. A similar effect attributed to top-down expectancy has already been found with two-alternative forced-choice tasks (Soetens et al., 1985).

Overall, lesions of the model performance-monitoring components affected the model's ability to capture the effects of previous trials more severely than current trials (Table 4). The correlation between human and model RTs was only slightly affected by performance-monitor lesions for the full 64 RT data points, but model performance-monitor lesions virtually abolished the model's ability to capture previous-trial effects. With regard to error rates, lesions of CHd (but with intact tonic excitation B to the response layer) led to higher error rates and shorter RTs. However, if the

Table 4
Lesion fit effects

Condition	Intact model	INC/CH lesion
Model and human RT Pearson correlation		
Human vs. model (64 pt)	0.829	0.753
Human vs. model current trial (8 pt)	0.963	0.928
Human vs. model prev trial (8 pt)	0.686	0.142

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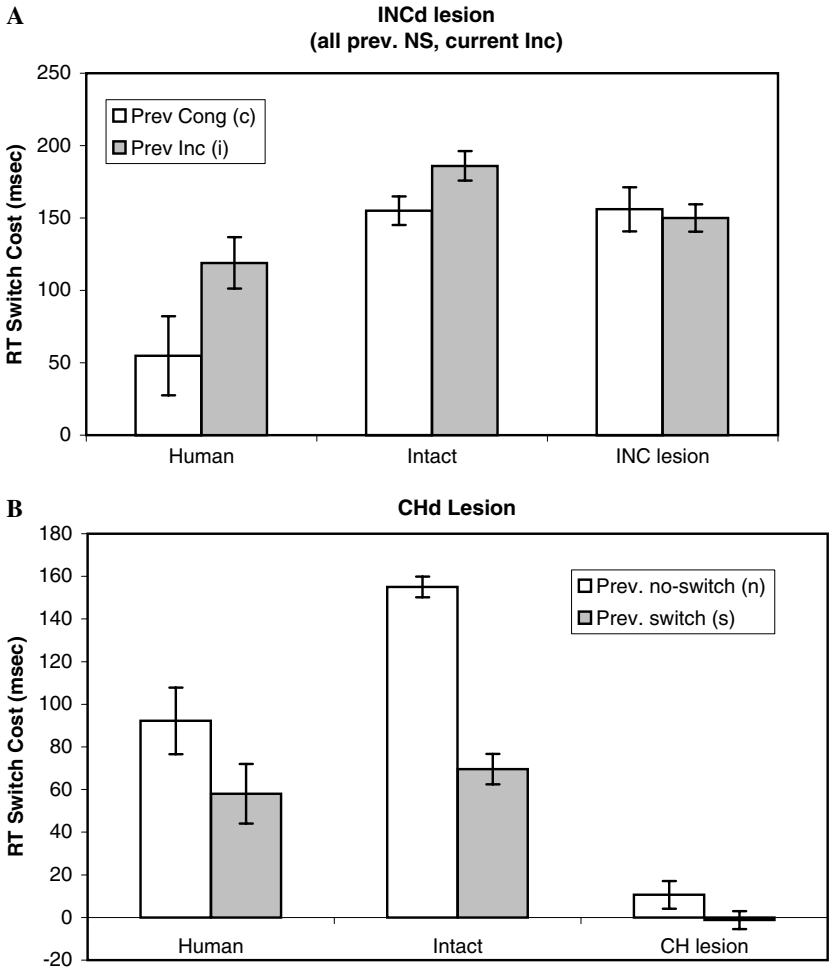

Fig. 16. Effects of model cognitive control mechanism lesions on RT. (A) Lesions of INCd abolish the effect of prior incongruency on switch costs, which are otherwise seen in both the intact model and the human data. Thus, INCd serves to increase the switch cost for previous incongruent trials. (B) Lesions of CHd abolish the effect of previous switches on switch cost, which is significant in both the human data ($p < .05$, paired) and the intact model. The persistent switch-induced slowing is due to the model CHd, and the braking activity saturates such that repeated switches have less additional effect.

CHd lesion also included the tonic excitation to the response layer, then the network instead displayed lower error rates and longer RTs. Beyond this, although we looked for additional error rate effects, the conflict-control loops seemed to influence mainly response time rather than error rates. We cautiously speculate that although the task we used provided strong constraints on the model, it may not have been optimal for revealing error rate effects. No additional stimulus information is presented subsequent to the target, and therefore waiting longer to respond provides no clear benefit. This stands in contrast with the stop signal task, for example, in which useful new information may be received to aid accuracy as responses are delayed (Logan, 1985).

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10.5. Switch costs

It is notable that although lesions of the model performance monitor components reduced the switch cost, some significant switch costs remained. The RT switch cost was 112 ms in the intact model, 98 ms in the INCd-lesioned model, 74 ms in the CHd-lesioned model, and 68 ms when both CHd and INCd were lesioned. Subsequent investigation of the model revealed that the remaining 68 ms switch cost was abolished when learning in the Hidden-to-task-set layer [Eq. (7) in the Appendix A] was shut off (i.e., the learning rate set to 0) along with lesions of both INCd and CHd components. According to these results, some of the model RT switch cost is due to the role of conflict-control loops, especially CHd. However, another portion of the switch cost is due to priming from target stimuli to the task set layer, via the hidden layer, which essentially implements a kind of associative TSI (Allport et al., 1994; Wylie & Allport, 2000a). This result agrees with similar mechanisms found in previous models (Gilbert & Shallice, 2002) accounting for switch costs. Thus, the model is consistent with multiple factors contributing to the switch cost (Meiran et al., 2000).

11. Model discussion

The quantitative simulation demonstrates that the five postulates as embodied by the model are sufficient to account for a range of complex sequential effects. Although some noise is inevitably present in both the human data and the model fit (see Appendix A), the ability of the model to capture effects present in the human data validates the model fitting approach based on the 64 RT conditions and 8 current-trial error rate conditions. The success of the model fit further validates the sufficiency of the human sample size even for the 64 conditions. More striking are the observations that the model captured several behavioral effects that were not part of the data set used to fit the model. For example, the model captured effects of five-trial sequences, although it was fit to data involving sequences of no more than three trials in length (Fig. 14). The model also makes quantitative and non-trivial predictions regarding cognitive control mechanisms. In particular, the addition of conflict-control loops allows the model to capture a significantly larger portion of the variance, including rich and subtle higher-order effects. The results thus suggest that certain aspects of behavior are functionally dependent on these mechanisms. In that case, similar effects would be generally predicted in paradigms involving task switching and/or incongruency. The model predicts specific activity pattern signatures for the various conflict-control loop components. These predictions can in principle be tested with electrophysiological or functional neuroimaging methods. Virtual lesions of the CHd and INCd confirmed the expected effects of each of these cognitive control mechanisms, which were apparent in the effects of the previous trial history on performance. In particular, the INCd was necessary for the increased switch cost due to previous trial incongruency, because INCd drove the increased activity of a task set representation that facilitated performance if the task did not switch but hindered performance if the task did switch. Similarly, the CHd was necessary for the switch-induced slowing that persisted into subsequent trials, because its activity led to inhibition of response layer activity even in subsequent trials in which no task switch occurred. Lesioning the CHd and/or INCd abolished these effects in the model. The model also predicts that in asymmetric tasks such as the Stroop task (Stroop, 1935), activation of the non-dominant task should lead to greater

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conflict from the irrelevant stimulus, and therefore greater INCd activation. The result would be an increased activation of the current, less pre-potent task, which opposes a switch to the more pre-potent task. For example, naming colors in the Stroop task allows conflict from the more pre-potent word-reading pathways, which leads to increased conflict-related activity and therefore increased attention to color-naming. The increased activity of color naming task representations in turn makes switching away from color-naming to word-reading more difficult. Thus, although we did not test it directly, the model should account for the kind of asymmetry in switch costs that has been observed with these tasks (Allport et al., 1994; Monsell, Yeung, & Rayna, 2000).

12. General discussion

Much of the recent debate regarding switch costs has focused on whether or not they reflect the work of an executive controller (Altmann, 2003; Monsell, 2003; Rogers & Monsell, 1995), which has thus far proven somewhat elusive (Logan, 2003; Logan & Bundesen, 2003). The question has been framed in terms of whether or not an executive is responsible for implementing the task switch as well as the switch cost. However, executive control encompasses many faculties (Norman & Shallice, 1986), of which task set switching may (or may not) be only one potential component. Indeed, in our model, the switch cost persisted even when the performance monitoring components were lesioned. We suggest that the search for executive control as the mechanism that implements task switches in explicitly cued paradigms may be ill-posed in that it defines the role of an executive too narrowly. Instead, as the behavioral and modeling results above suggest, the contribution of an executive controller to task switching paradigm performance may not be that of directly implementing task-set shifts, but rather to monitor and control responses within an active task set (Logan, 2003). Recasting the issue in these terms need not emasculate the central executive, because certain uncued task-switching paradigms still require endogenously driven task switches on the basis of error feedback, and there is evidence that these require executive processes localized to the medial wall (Bush et al., 2002; Shima & Tanji, 1998). However, by embracing a broader definition of executive control, the above results suggest that such mechanisms are sufficient to account for higher-order sequential effects of task switching that have not been previously accounted for.

12.1. Switch cost

This paper complements several previously proposed theories of the switch cost. In general, the model addresses the residual switch cost (Rogers & Monsell, 1995), given the length of the cue-target interval. First, the TSI (Allport et al., 1994) and associative TSI theories (Wylie & Allport, 2000b) essentially suggest that previously active task sets carry over and interfere with the current task set. This carry-over may be passive and related to persistent neural activity (Sohn & Anderson, 2001), or in the case of the associative TSI, may be reactivated by a stimulus that was recently associated with the task set. These mechanisms were incorporated into a recent model of task switching (Gilbert & Shallice, 2002), which showed the mechanisms sufficient to account for aspects of the switch cost. However, the Gilbert and Shallice model does not address effects of longer sequences, since the path weights are reset after each trial. Nonetheless, the associative TSI mechanism, by which stimuli can reactivate task sets that they have recently been paired with,

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may account for part of the switch cost. In the present model, the hidden-to-task-set weights (Eq. (7) in the Appendix A) are adaptive, which implements this kind of associative learning to contribute to the switch cost. Our model is consistent with a role for associative TSI, although our interest in sequential effects led us to focus on persistent task-switch induced slowing mechanisms underlying switch costs as part of a more general class of sequential effects.

In one of our other models (Reynolds et al., 2006), this associative learning mechanism serves not only to account for the residual switch cost but also to implement a kind of reactive cognitive control. Several investigators (De Jong, 2000; De Jong et al., 1999; Nieuwenhuis & Monsell, 2002) have suggested the *failure-to-engage* (FTE) hypothesis, which proposes that on a fraction of trials, the task set representations fail to persist and must be reactivated or retrieved by the appearance of the target. In these cases, associative learning allows target stimuli to reactivate the recently activated task set representation, so that the task can be performed correctly despite failures of task set activity to persist. Thus, although we did not investigate the FTE issue explicitly, the model is consistent with a role of failures to engage in accounting for the switch cost as well as the observed positive correlation between response time and error rate for the slowest trials.

Recent work has further probed the time scale on which such associative learning effects may act. Apparently, individual instances of pairing can have long lasting effects (Volkow et al., 1998), such that a stimulus previously paired with one task can evoke an increased switch cost in the other task even many trials later (Waszak et al., 2003). In other words, once the pairing had occurred, it was relatively stable and less sensitive to the recent trial history. Thus, this kind of persistent associative learning may indeed be present in our data, but it would not significantly contribute to the sequential effects that varied with recent trial history (with timecourses on the order of a few trials) as observed here. However, the model is consistent with associative learning that acts and decays on a shorter time scale of several trials.

Backward inhibition, as revealed in the set alternation cost (Mayr, 2002; Mayr & Keele, 2000), and cue repetition priming (Arrington & Logan, 2004a, 2004b; Logan & Bundesen, 2003; Mayr & Kliegl, 2003) are two additional phenomena that have been proposed to account for part of the switch cost. Our paradigm used two task sets rather than three, and a single type of task so testing for distinct set alternation costs and cue repetition benefits was beyond the scope of the simulations. However, our model could be augmented with either or both of two mechanisms that have been proposed to explain the set alternation cost, namely persistent self-inhibition and lateral inhibition of task set representations (Mayr & Keele, 2000). Essentially, these two mechanisms might allow a task that is switched away from to be persistently inhibited so as to render a reactivation of those task set representations more difficult. In any case, the model suggests that cognitive control processes are evident not in the current trial task switch (or cue switch) condition, but rather in the effects of previous trial conditions. The finding that switches in the previous trial lead to a response time cost in a current no-switch trial (i.e. for RTs, $sN > nN$) is beyond the scope of an explanation in terms of backward inhibition, because backward inhibition effects would not apply to this sequence. Nonetheless, the finding is consistent with the CH conflict-control loop mechanism simulated here. Furthermore, if the cue-repetition benefit such as proposed by (Logan & Bundesen, 2003) persisted beyond the first trial, then in cases with one cue per task, the response time for sequences of two switch trials should be faster than for a no-switch followed by a switch trial (i.e. $sS > nS$), due

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to repetition in the current trial of the task (and cue) presented two trials back (e.g., BAB). On the contrary, because of apparent backward inhibition effects, exactly the opposite is true (Mayr & Keele, 2000). More recent work suggests that backward inhibition may mask effects of cue repetition priming which are nonetheless present, at least in some cases with relatively few cues (Mayr & Kliegl, 2003). By itself, this observation might suggest cue repetition priming benefits as a potential alternative explanation to the model's CH control loop account of slowing effects in the case of a switch followed by a no switch trial (sN), as compared with a no-switch followed by a no-switch trial (nN). The main limitation of this argument is that in the case of voluntary task switches, no task cue is presented, yet the data show that RTs remain increased in the trial subsequent to the switch and decrease thereafter (Arrington & Logan, 2004a, 2004b). This is consistent with the CH control loop simulated here but cannot be accounted for by cue repetition priming, since no task cues are presented.

12.2. Cognitive control modulates performance tradeoffs

Our results are consistent with the general notion that multiple cognitive control mechanisms dynamically modulate behavioral parameters in a continual manner during task performance, in order to optimize behavior when environments are non-stationary (Mozer, Colagrosso, & Huber, 2002). With regard to the effect of incongruency on subsequent trial performance, increased focus on the current task comes at the expense of higher switch costs. The finding of increased switch cost due to previous trial incongruency is consistent with previous findings (Goschke, 2000). Furthermore, to the extent that the task set representations in humans fail to engage on a subset of trials (De Jong, 2000; De Jong et al., 1999; Nieuwenhuis & Monsell, 2002), we suggest that the likelihood of engagement may be modulated by the recent frequency of incongruent trials, such that task engagement increases and response time for incongruent trials decreases (as long as the task does not switch) when incongruent trials increase in frequency. This agrees with prior studies on the relative frequency of incongruent trials in the Stroop task (Logan & Zbrodoff, 1979). Thus, the model is consistent with a fundamental tradeoff between exploration and exploitation (Ishii et al., 2002; Kaelbling et al., 1996; Sutton & Barto, 1998; Usher et al., 1999), such that incongruency drives increased control to focus effort and attention on exploiting known stimuli at the expense of exploring/attending to other potentially useful cues.

Likewise, the observed change-induced slowing effects are consistent with cognitive control mechanisms that modulate the observed speed–accuracy tradeoff in favor of accuracy for faster, engaged trials in both model and human data (Fig. 13). Consistent with this observation, fast switch trials vs. no-switch trials lead to longer response times in subsequent trials in both human and model data. However, while the model further shows a reduction in error rate following fast switch vs. no-switch trials, the human data do not show a corresponding reduction in error rate. Thus, the human data show a discrepancy between the general speed–accuracy tradeoff in fast human trials and the lack of such tradeoff effects in trials subsequent to a switch. One possible reason for this discrepancy is that switch trials might also drive change-detecting cognitive control mechanisms to bias a performance tradeoff in favor of exploration vs. exploitation, i.e. oppose the incongruency conflict-control loop effect. This was not simulated in the model, but the human trend towards greater incongruency effects on error rate subsequent to switch trials in humans suggests this possibility. An increased exploration bias might increase the rate of errors driven by distractors in such a way as to

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mask an opposing reduction in errors due to increased response time under a speed–accuracy tradeoff. Another un-tested possibility is that task switches increase the likelihood of failure to engage the task in subsequent trials, which might in turn increase error rates and mask the effect of error rate reduction due to RT slowing.

12.3. Cognitive neuroscience

Studies of the neural bases of performance monitoring/conflict detection and cognitive control are consistent with the present model. A general consensus has been emerging in the literature that the ACC and related areas of the frontal medial wall are critically involved in monitoring performance (Blakemore, Rees, & Frith, 1998; Botvinick et al., 1999; Braver, Barch, Gray, Molfese, & Snyder, 2001; Carter et al., 1998; Carter, MacDonald, Ross, & Stenger, 2001; Gehring & Knight, 2000; Liddle, Friston, Frith, & Frackowiak, 1992; MacDonald et al., 2000; Menon, Adleman, White, Glover, & Reiss, 2001; Nordahl et al., 2001; Scheffers & Coles, 2000; Ullsperger & von Cramon, 2001; Veen, Cohen, Botvinick, Stenger, & Carter, 2001). Evidence suggests that the ACC detects conflict between incompatible response processes and in turn signals other areas of frontal cortex, such as dorsolateral prefrontal cortex (PFC), to increase the level of cognitive control (Botvinick et al., 1999; Carter et al., 1998; Carter et al., 2000; Kerns et al., 2004; MacDonald et al., 2000), particularly when the frequency of these events in recent trials is low, although some controversy exists on this point regarding specific behavioral paradigms (Carter, Kerns, Sohn, & Cohen, 2003; Mayr et al., 2003). Furthermore, we have recently shown that ACC may learn to signal increased error likelihood, which can lead to apparent response conflict detection effects (Brown & Braver, 2005). In the above results, task switches and stimulus incongruency are both associated with significantly increased error rates relative to no-switch and congruent conditions respectively. Therefore, apparent response conflict detection effects in and around ACC may be expected in incongruent vs. congruent as well as switch vs. no-switch conditions in the present task.

The ACC may signal response conflict due to incongruent stimuli and subsequently drive increased attentional focus to the current task set (Egner & Hirsch, 2005; Posner & DiGirolamo, 1998), which constitutes an increase of cognitive control consistent with the human data and model simulations above (Botvinick et al., 1999; Carter et al., 2000; Cohen, Botvinick, & Carter, 2000). Thus, some part of the ACC may perform a function similar to that of the model INCd.

In the case of task switching and response alternation, conflict may arise between the previously performed response or task set, and the new response or task set. This kind of conflict bears resemblance to that engendered in supplementary eye fields of macaque monkeys when a planned saccade is subsequently countermanded (Stuphorn et al., 2000), in that the previous response process conflicts with a newly introduced response process. Functional imaging studies of the medial prefrontal cortex (including ACC and pre-SMA) also show activity in response to task switches, when the stimulus-response mapping changes (Dove et al., 2000; Kimberg, Aguirre, & D'Esposito, 2000; Luks et al., 2002; Rushworth, Hadland, Paus, & Sipila, 2002; Shima & Tanji, 1998). While the cingulate motor area of the ACC may be involved in driving changes in task set under certain conditions lacking explicit task cues (Shima & Tanji, 1998), neighboring areas might also detect changes in task set evidenced as conflict between the previous and new task sets. In support of this hypothesis, Jones et al. (2002) found specific areas of

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ACC that activate when responses alternate. In macaque monkeys, the supplementary eye fields (SEF, which are adjacent and dorsal to ACC) show activity consistent with a role in performance monitoring and control. Some SEF cells are more active prior to antisaccades (Schlag-Rey, Amador, Sanchez, & Schlag, 1997), which require greater control. However, in a countermanding task, SEF cells do not modulate in time to play a causal role in driving eye movements (Stuphorn et al., 2000). Together, these results suggest that SEF cells may play a role in monitoring and increasing cognitive control, even without directly driving movements. Consistent with this interpretation, microstimulation of SEF can generally slow saccadic eye movement initiation without preventing movements (Stuphorn, Brown, & Schall, 2001). Thus, SEF in monkeys may implement a kind of switch-induced slowing as simulated by the model CHd above.

Overall, the tentative localization of the two cognitive control mechanisms in Fig. 7 to the ACC or pre-SMA again raises the intriguing possibility of multiple functional subregions of these areas, which has been a topic of recent discussion (Barch et al., 2001; Garavan, Ross, Murphy, Roche, & Stein, 2002; Peterson et al., 1999; Ullsperger & von Cramon, 2001). If this localization can be verified, the results as a whole would suggest that the analysis of sequential effects on behavior may increase the sensitivity of clinical evaluations of lesion patients, especially those with lesions involving neurobiological correlates of the model cognitive control mechanisms.

13. Conclusion

The present work has explored the effect of sequences of trials in task switching studies and demonstrated several effects. First, we have shown that recurring task-switch or response alternations tend to have a general slowing effect on response time that persists across multiple trials. Second, we demonstrated that both the response time and error cost of task-switching is significantly modulated by effects occurring in preceding trials, and that these often interact in complex ways. On the basis of our results, we suggest two conflict-control loop mechanisms that are distinct from the mechanisms that reconfigure task set. These putative mechanisms are two-fold. First, *change-detection* mechanisms may bias performance toward accuracy rather than speed and exploration rather than exploitation, with the general effect of slowing responses when the task or response changes from one trial to the next. Second, *incongruency-detection* mechanisms may increase the attentional focus on the currently active task set, effectively biasing performance toward exploitation rather than exploration. Furthermore, we believe that analysis of sequential effects and the proposed mechanisms of cognitive control may generalize to variety of task paradigms. Within task switching studies, there are many factors that we have not explored here in terms of their modulation by higher-order sequential effects, including manipulations of response-cue interval (RCI), cue-stimulus interval (CSI), and use of other tasks with asymmetrical switch costs, such as the Stroop task (Stroop, 1935) and the antisaccade task (Cherkasova, Manoach, Intriligator, & Barton, 2002), among others. In many cases, existing data may provide useful new insights provided that the trial order information is preserved.

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Appendix A

Below are the equations which govern activation dynamics, priming mechanisms, conflict detection and control effects in the model. The model was simulated in the RNS++ software program (Brown, 2003) which simulates rate-coded neural activity as a dynamical system. In RNS++, the activity of an individual cell is updated according to a finite-difference equation using a first-order Euler approximation. RNS++ updates the neural activity at time intervals much smaller than the trial duration, which simulates the real-time dynamics of the cell activity during a given trial. Thus, for all equations, the units of activation cycles corresponding directly to real-time units of milliseconds, which affords a direct quantitative comparison with empirically derived values. Tables 5 and 6 give a summary of model conventions, parameters, and the values used for these parameters in the simulation results described in the text. The optimization approach used to derive parameter values is described below.

Activation dynamics

Network cell dynamics are governed by a basic shunting equation formalism (Grossberg, 1982) which provides a rate-coded value of activation. Although RNS++ supports other governing equation models of neural activity, including spiking neurons, only variants of Eq. (1) below were used to model neural activity in the present model:

Table 5
Conventions

Symbol	Description
P	Task set
C	Cue input
T	Target input
H	Hidden/plan layer
R	Response output
L	Working memory for last response
B	Output bias tonic excitation
$A^{(\mathrm{INCd})}$	INCd
$A^{(\mathrm{INCc})}$	INCc
$A^{(\mathrm{CHd})}$	CHd
$A^{(\mathrm{CHc})}$	CHc
Function	Description
$\mathrm{Squelch}(x, v)=$	$\begin{cases} x & \text{if } x > v \\ 0 & \text{otherwise} \end{cases}$
$\mathrm{Rect}(x, v) = [x - v]^{+}=$	$\begin{cases} x - v & \text{if } x > v \\ 0 & \text{otherwise} \end{cases}$
$\mathrm{Sat}(x, v)=$	$\begin{cases} v & \text{if } x > v \\ x & \text{otherwise} \end{cases}$
$\mathrm{Step}(x, v)=$	$\begin{cases} 1 & \text{if } x > v \\ 0 & \text{otherwise} \end{cases}$
$N(x, v)=$	Gaussian random variable with mean x and variance v

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Table 6
Parameters

Parameter name	Value	Description
k_1	0.289	INCc → Task Set wt gain
k_2	0.078	INCc → Task Set inhib wt gain
k_3	2.157	RtUnitSpec_0 gov time const
k_4	2.940	Input → Plan wt gain
k_5	1.692	Task Set → Plan wt gain
k_6	0.446	Output gov time const
k_7	1	Bias → Output wt gain
k_8	16.018	Plan → Output wt gain
k_9	0.486	Plan → Output signal_func thresh
k_{10}	0.096	Output gov hyperpol
k_{11}	0	Output recur inhib wt gain
k_{12}	0.134	Output noise var
k_{13}	29.730	Plan → Output lrate
k_{14}	0.007	Plan → Output pass_wt_decay rate
k_{15}	19.500	INCd → INCc wt gain
k_{16}	8.299	CHd prev_aff wt_gain
k_{17}	9.338	CHd afferent wt_gain
k_{18}	1.714	CHc gov time_const
k_{19}	38.040	CHd(task) → CHc (brake) wt_gain
k_{20}	50.547	CHd(resp) → CHc (brake) wt_gain
k_{21}	2.867	CHc → bias inhib_spec wt gain

$$x_{t+1} = x_t + \frac{1}{\tau}[(1 - x_t)I_e - (x_t + H)I_i]\mathrm{d}t. \tag{1}$$

where x_t, is the rate-coded neural activity at time t; I_e, is net excitatory input to a cell, including random noise; I_i is net inhibition of the cell; τ is the time constant of the cell, i.e., how much time is needed for the activity level to change in response to input; H is the hyperpolarization potential of a neuron; dt is 0.001 s.

Individual layers of units were governed by activation equations that reflect their specific sources of input. The task-set representation is governed by:

$$P_i(t+1) = P_i(t) + 3\mathrm{d}t\left[(1 - P_i)\left(0.1 + 5C_i + 5\sum_{j\in S} W_j^{(HP)} sat\,(H_j, 0.7) + 0.5k_1 A_i^{(\mathrm{INCc})} + 2\frac{P_i^8}{P_i^8 + 0.3^8}\right) - (P_i + 1)\left(1 + 1.5\sum_{j\neq i} P_j + 0.5k_2 \sum_{k\neq i} A_k^{(\mathrm{INCc})}\right)\right] \tag{2}$$

There are two P_i nodes, which represent the Letter and the Number task. Eq. (2) says that each task set representation P_i is directly activated by a tonic bias excitation 0.1, Cue input C_i from the corresponding task cue representation, and the two hidden nodes H_j representing the left and right responses for the given task set. The hidden layer inputs are multiplied by synaptic weights $W^{(HP)}$. Also, the INC controller ($A_i^{(\mathrm{INCc})}$) provides persistent, slowly decaying excitation to amplify the activity of the corresponding task set and suppress other task sets. Finally, sustained activity in the task set layer is maintained by a sigmoidal recurrent excitation. The P_i units inhibit each other to implement lateral inhibition, and a tonic inhibitory signal also ensures activity decay in the absence of excitatory inputs.

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The hidden (or plan) layer (H) drives responses given a combination of contextual input from the task set layer (P) specifying the current task to perform and target information from the target input layer (T):

$$H_j(t+1) = H_j(t) + k_3 * \mathrm{d}t * [(1 - H_j)(0.5k_4 T_i + 0.5k_5 P_m) - (H_j + 0.5)] \quad (3)$$

where P_m is the task set representation that drives the hidden nodes implementing the given task set, and T_i is the target stimulus representation.

The response output layer (R) is driven by the hidden layer (H), the Target input layer (T), and the output bias (B):

$$R_i(t+1) = R_i(t) + k_6 \mathrm{d}t \left[(1 - R_i) \left(0.5k_7 B + 0.44 \sum_j T_j + k_8 \sum_{j \in D} W_{ij}^{HR} [H_j - k_9]^+ \right) - (R_i + k_{10}) \left(1.1 + k_{11} \sum_{m \neq i} R_m \right) + \sqrt{\mathrm{d}t} N(0, k_{12}) \right] \quad (4)$$

Eq. (4) indicates that the responses are driven in part by the mere presence of a stimulus, as inputs T_j drive the response nodes directly without bias toward either a left or right response. The tonic excitatory signal B is attenuated by the Change controller (CHc) so that CHc activity effectively slows response times. Gaussian noise is added in order to fit error rate data and to yield a distribution of response times. The actual response made is determined largely by signals H_j from the Hidden layer. The set D represents those hidden layer cells that drive the particular responses represented by the node R_i. There are two hidden layer nodes that drive the left response layer node (one pertaining to each task), and likewise two that drive the right response layer node.

Units in an additional layer (L) served as a buffer of previous response-related activity. When the current response differed from the last response before it (e.g., previous left response and current right response), then a brief period of coactivation between the current and previous response representations (L) occurred in the same way that a task switch led to transient coactivation of task set representations (P). The CHd uses these detections of task switches and response alternations to non-specifically slow responses.

The response buffer representations (L) are given by:

$$L_i(t+1) = L_i(t) + 3\mathrm{d}t \left((1 - L_i) \left(50 squelch(R_i, 0.5) + 3 \frac{x^8}{x^8 + 0.3^8} + 0.1 \right) - (L_i + 1)(1 + 1.5 \sum_{i \neq j} L_j) \right) \quad (5)$$

In Eq. (5), maintenance of previous responses is driven by the response R_i and sustained by recurrent excitation. Lateral inhibition prevents multiple response representations from being simultaneously active.

Priming

All synaptic weights were bounded between 0 and 1. Individual weights were initialized to 0.5 and were scaled by a weight gain parameter in many of the equations below. Additionally, certain synaptic connections in the model were adaptive and hence changed dynamically via an activity-dependent priming mechanism. The priming effects occurred

via a modified Hebbian (Hebb, 1949) learning law, which can be expressed in finite difference form as:

$$w_{t+1} = w_t + \mathrm{d}t[\gamma xy - \alpha(w_t - 0.5)] \tag{6}$$

where w_t is the synaptic efficacy or priming strength from presynaptic cell with activity x to postsynaptic cell with activity y at time t; γ is the rate of increase of the weight in response to paired activity of pre- and post-synaptic cells; α is the rate of passive weight decay toward the baseline, in this case 0.5, which represents the decay of priming effects; the hidden-to-task-set weights ($W_{ij}^{(HP)}$) are given by:

$$W_{ij}^{(HP)}(t+1) = W_{ij}^{(HP)}(t) + \mathrm{d}t\,(1000(sat\,(H_j, 0.7) * squelch(P_i, 0.5)) - 0.5(W_{ij}^{(HP)} - 0.5)) \tag{7}$$

and bounded such that $0 < W_{ij}^{(HP)} < 1$. Eq. (7) says that these weights increase quickly when the pre-synaptic hidden layer is active and the post-synaptic task-set cell activity exceeds 0.5. Decay towards the baseline weight of 0.5 occurs gradually in the absence of paired pre- and post-synaptic activity.

The weights W_{ij}^{HR} from the hidden layer to the response layer were also adaptive:

$$W_{ij}^{HR}(t+1) = W_{ij}^{HR}(t) + \mathrm{d}t[k_{13}squelch(R_i, 0.4)[H_j - 0.5]^{+} - k_{13}k_{14}(W_{ij}^{HR} - 0.5)] \tag{8}$$

Eq. (8) follows the form of Eq. (6) and says that the path weight from a hidden layer node to a response layer node increases when the response layer node is active above 0.4, and when the hidden layer node is simultaneously active above 0.5. In the absence of such coactivity, the weight decays passively to a baseline of 0.5. The passive decay rate is such that 22% of the difference between the weight and its baseline decays away after 1 second, so that approximately 78% of the difference remains. This decay is exponential. For example, after 2 seconds, only approximately 61% of the original weight difference remains. Thus, as a pathway is used, the path efficacy increases temporarily, which simulates a kind of priming effect, seen in the RT interaction between current task switch and current response alternation.

Conflict Detection

The INC conflict detector $A^{(\mathrm{INCd})}$ is governed by:

$$A_{ij}^{(\mathrm{INCd})}(t+1) = A_{ij}^{(\mathrm{INCd})}(t) + 50\mathrm{d}t\left[0.5\left(1 - A_{ij}^{(\mathrm{INCd})}\right)\left(\sum_{k\in C_j}\mathrm{step}(H_k, 0.4) + \mathrm{step}(P_i, 0.4)\right) - (A_{ij}^{(\mathrm{INCd})} + 1)\right] \tag{9}$$

where variable i indexes the task set, and variable j indexes the set of possible combinations of conflicting response plans. Each INCd conflict detector cell responds to a unique combination of conflicting stimuli and a task-set representation. There are two possible combinations of conflicting stimuli and two task set representations; hence, there are four INCd conflict detector cells in all. In Eq. (9), the *step*() function provides an upper bound on the individual contributions of any one input from the hidden layer as well as inputs from the task-set layer, so that only the conjunction of inputs is sufficient to elicit a response representing conflict.

The CH conflict detector ($A_i^{(\mathrm{CHd})}$) is defined by:

$$A_i^{(\mathrm{CHd})}(t+1) = A_i^{(\mathrm{CHd})}(t) + 50\mathrm{d}t\,((1 - A_i^{(\mathrm{CHd})})(A_i^{(\mathrm{CHdexc})}) - (A_i^{(\mathrm{CHd})} + 1)), \tag{10}$$

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which is driven by transient coactivations due to transitions of task set or the response buffer. For input from the response,

$$A_i^{(\mathrm{CHdexc})} = 0.1k_{16}\sum_j \mathrm{step}(L_j, 0.3). \tag{11}$$

For input from the task set representation,

$$A_i^{(\mathrm{CHdexc})} = 0.1k_{17}\sum_j \mathrm{step}(P_j, 0.3). \tag{12}$$

Control adjustment

Changes in INC conflict detection units drive a control output signal $A^{(\mathrm{INCc})}$ defined by:

$$A_i^{(\mathrm{INCc})}(t+1) = A_i^{(\mathrm{INCc})}(t) + \mathrm{d}t\left(k_{15}\left(1 - A_i^{(\mathrm{INCc})}\right)\sum_j A_{ij}^{(\mathrm{INCd})} - 0.05\left(A_i^{(\mathrm{INCc})} + 0.5\right)\right) \tag{13}$$

Eq. (13) says that phasic INC detector layer activity $A_{ij}^{(\mathrm{INCd})}$ rapidly activates a slowly decaying control signal $A_i^{(\mathrm{INCc})}$. This signal in turn selectively excites activity in the task-set layer cf. Eq. (2), such that the task-set unit currently active (which represents the current task-set) becomes enhanced while the inactive task-set unit is more strongly suppressed.

Likewise, activity in the CH conflict detector ($A^{(\mathrm{CHd})}$) drives a parallel slowly decaying control signal ($A^{(\mathrm{CHc})}$) which ultimately slows responses:

$$A^{(\mathrm{CHc})}(t+1) = A^{(\mathrm{CHc})}(t) + k_{18}\mathrm{d}t\left((1 - A^{(\mathrm{CHc})})\sum_i W_i^{(C)} A_i^{(\mathrm{CHd})} - 0.03(A^{(\mathrm{CHc})} + 0.5)\right) \tag{14}$$

For signals $A^{(\mathrm{CHc})}$ driven indirectly by task set representations, $W^{(C)} = 0.5k_{19}$. For signals $A^{(\mathrm{CHc})}$ driven ultimately by working memory representations of the last response, $W^{(C)} = 0.5k_{20}$.

The CHc($A^{(\mathrm{CHc})}$) suppresses a signal (B) that provides tonic excitation to the response layer (R):

$$B(t+1) = B(t) + 1.76\mathrm{d}t\left((1 - B) - 0.5k_{21}(B + 0.5)A^{(\mathrm{CHc})}\right) \tag{15}$$

Thus, activity in the CH control loop suppresses tonic excitation of the response layer (cf. Eq. (4)), which generally slows responses.

A.1. Parameter optimization

Model parameters were adjusted in order to optimize the network's ability to capture the empirical effects. The optimization procedure was performed using the RNS simulator's built-in subplex (Rowan, 1990) optimization algorithm, which essentially performs gradient descent on a monte carlo objective function. This entails an inherently noisy process, and therefore some error in the final parameter set therefore

cannot be ruled out. Nonetheless, we attempted to minimize error in the parameters by increasing the sample size, at the expense of increased computational requirements. In addition, the subplex algorithm is particularly suited to optimization of noisy objective functions (Bogacz & Cohen, 2002). A total of 21 parameters were adjusted to yield the best fit (Table 6). Of note, these parameters are distinct from the learned connection weights between nodes in the model. The model parameters govern the properties of the model and are fixed for a given model simulation, while the learning laws allow connection weights to adapt and change during the simulation as a function of simulated neural activity in the model.

The optimization procedure involved minimizing a cost-function using a least squares fit of model performance to the empirical data. The data fit were the 64 response time data points obtained by crossing factors of task switch/no-switch, response alternate/repeat, and stimulus congruent/incongruent, associated with both current and preceding trials. The fitting procedure also used the 8 error-rate data points obtained from these conditions in just the current trial. Additionally, the model was constrained to optimize the fit to 4 effect-size (d') measures specifying contrasts between task conditions. Specifically, to reveal an interaction of previous congruency and current switch, the RT effect size of switch vs. no-switch trials for previous incongruent trials (where previous trials were no-switch and current trials were incongruent) was constrained to 1.0. Conversely, for previous congruent trials (where again previous trials were no-switch and current trials were incongruent), the effect size was constrained to 0.2. The effect size on RT of previous switch vs. previous no-switch trials was constrained to 0.5, and the effect size on RT of previous response alternation vs. repetition was likewise constrained to 0.5. These constraints on effect size guarded against the type II error scenario that the subtle but significant sequential effects of the human data would be lost in the model parameter fit (a noisy process) even though the structure of the model in principle allowed them to be captured.

Appendix B. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.cogpsych.2006.09.005.

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