Challenging A Decade of Brain Research on Task Switching: Brain Activation in the Task-Switching Paradigm Reflects Adaptation Rather Than Reconfiguration of Task Sets

Wouter De Baene,^{1,2*} Simone Kühn,^{1,2} and Marcel Brass^{1,2}

¹Department of Experimental Psychology, Ghent University, Ghent, Belgium

²Ghent Institute for Functional and Metabolic Imaging, Ghent University, Ghent, Belgium

Abstract: In daily life, we permanently need to adapt our behavior to new task situations, requiring cognitive control. Such adaptive processes are commonly investigated with the task-switching paradigm. Many fMRI studies have interpreted stronger activation for switch than repeat trials in fronto-parietal brain areas as reflecting an active reconfiguration process in switch trials, tuning the cognitive system for proper task execution. From the single cell literature, however, one could deduce the alternative interpretation that switch-specific activity reflects reduced brain activity in repeat trials due to adaptation. These alternative explanations cannot be distinguished by simply comparing brain activity in switch and repeat trials. Therefore, we used a parametric approach to examine which interpretation is more powerful to account for the data. In all areas of the fronto-parietal network, adaptation explained the data better than reconfiguration. Therefore, our results call the classical reconfiguration interpretation into question and provide first evidence for adaptation of abstract task representations. *Hum Brain Mapp* 33:639–651, 2012. © 2011 Wiley Periodicals, Inc.

Key words: task switching; adaptation; reconfiguration; abstract representations; cognitive control

INTRODUCTION

In daily life, people show a remarkable flexibility in switching between multiple tasks. Despite the subjective impression that alternating between different tasks does

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*Correspondence to: Wouter De Baene, Department of Experimental Psychology, Ghent University, Henri Dunantlaan 2, Ghent, B-9000, Belgium. E-mail: Wouter.DeBaene@ugent.be

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not require much effort, several behavioral studies showed that cognitive control processes are involved in task switching [Allport et al., 1994; Rogers and Monsell, 1995]. This is reflected in the so-called switch cost: people are generally slower and less accurate at switching than at repeating tasks [Jersild, 1927]. Furthermore, it has been demonstrated that when participants are able to prepare the next task, reaction times decrease and the switch costs are reduced [Meiran, 1996; Meiran et al., 2000]. Many researchers have interpreted switch costs as reflecting a reconfiguration process. The reduction of switch costs with additional preparation time has been interpreted as reflecting advanced reconfiguration of the task set [Meiran, 1996; Monsell and Mizon, 2006; Rogers and Monsell, 1995]. According to the task set reconfiguration account, the cognitive system has to be tuned for a proper execution of the task when switching tasks. Such reconfiguration processes will normally not be needed when the same task is repeated, since the previous task set is still active.

Although there are great inconsistencies between studies (see Discussion), several fMRI-studies seem to provide support for this account by showing increased BOLD-activation during switching compared to repetition in cognitive control-related brain regions [Braver et al., 2003; Dove et al., 2000; Kimberg et al., 2000; Rushworth et al., 2001, 2002; Smith et al., 2004; Sohn et al., 2000].

Moreover, a number of fMRI task-switching studies tried to isolate the neural mechanisms involved in advanced preparation of task sets. Preparation-related activation was found in lateral prefrontal cortex (PFC), intraparietal sulcus (IPS), pre-supplementary motor areas (pre-SMA), and the anterior cingulate cortex (ACC) [Dosenbach et al., 2006; Dreher et al., 2002; Luks et al., 2002; MacDonald et al., 2000; Rushworth et al., 2001; Sohn et al., 2000]. According to the reconfiguration view, this larger preparation-related activation in switch compared to repeat trials reflects additional processes, necessary to configure the cognitive system for the new task at hand.

From neurophysiological research, however, one could generate an alternative interpretation of switch-specific activation differences. This interpretation would suggest that differences between switch and repeat trials are not due to stronger activation in switch trials caused by task-set reconfiguration but rather to a reduction of activation in repeat trials caused by adaptation of task-related neural populations. In line with its tentative role in representing task-relevant information to guide cognitive processing [Miller and Cohen, 2001], neurons in prefrontal cortex have been shown to encode arbitrary task rules, both concrete and abstract in nature. Under identical sensory and attentional conditions, prefrontal neurons respond to a given cue when a monkey uses a specific rule, but show weak or no activity when the monkey uses another rule [Asaad et al., 2000; Johnston et al., 2007; Sakagami and Niki, 1994; Wallis and Miller, 2003; Wallis et al., 2001; White and Wise, 1999]. Additional support for this cardinal function of prefrontal cortex in representing task rules came from human brain-imaging studies. Sakai and Passingham [2003, 2006] showed sustained rule-specific neural activity in prefrontal cortex during the cue-target interval, which was interpreted as the neural correlate of a task set [for a review, see Sakai, 2008]. Bunge et al. [2003] reported similar findings using match and non-match rules. Also in parietal cortex, rule sensitive neurons have been found [Freedman and Assad, 2006; Gail and Andersen, 2006; Gottlieb, 2007; Oristaglio et al., 2006; Stoet and Snyder, 2004, 2007; Toth and Assad, 2002].

However, if prefrontal and parietal brain areas carry task representations, it would be only logical to assume that such representations show neural adaptation. Adaptation [Krekelberg et al., 2006; Ringo, 1996] or repetition suppression [Desimone, 1996; Grill-Spector et al., 2006] refers to the decrease in neuronal activity when a stimulus is repeated. Adaptation has been observed in many cortical areas, across many different experimental conditions and at multiple spatial scales, from the level of individual cortical

neurons in monkeys [De Baene and Vogels, 2010; Miller and Desimone, 1994; Sawamura et al., 2006] to the level of haemodynamic changes in humans [Grill-Spector and Malach, 2001; Kourtzi and Kanwisher, 2000]. Importantly, adaptation has also been demonstrated in cognitive control related brain areas. Both single cell and fMRI studies have reported adaptation in prefrontal [Buckner and Koutstaal, 1998; Miller et al., 1996; Rainer and Miller, 2000; Wagner et al., 1997] and parietal areas [James et al., 2002; Lehky and Sereno, 2007; Piazza et al., 2004; Vuilleumier et al., 2002].

In the present study, we examined whether the activation difference in preparing switch compared to repeat trials is better explained by adaptation in repeat trials than by enhanced activation in switch trials. Importantly, it is impossible to disentangle the adaptation view from the reconfiguration view on the basis of the common switch versus repetition contrast since both accounts predict higher activation in switch compared to repeat trials. To disentangle these two accounts, we therefore examined longer sequences of trials and modeled brain activation continuously by using a parametric model. While a classical reconfiguration approach would not predict a differential change of activation level in repeat trials over longer sequences of trials, the adaptation account clearly predicts a decrease of activation with successive repeat trials. We derived a model of the adaptation rate from the literature [Grill-Spector and Malach, 2001; Sawamura et al., 2006] and compared it to the reconfiguration model, which is equivalent to the classical switch versus repeat contrast.

To summarize, the aim of the present study was to test whether switch-specific differences in brain activation when preparing an upcoming task are related to additional brain activation in switch trials due to task set reconfiguration, as has been suggested previously, or to decreased brain activation in repeat trials caused by adaptation. These two theoretical perspectives on switch costs could be dissociated by investigating how brain activation developed over a sequence of trials.

MATERIALS AND METHODS

Participants

Twenty-one, neurologically normal, right-handed volunteers were recruited from Ghent University and were paid for participation. We obtained written consent from all 21 participants prior to the scanning session. All subjects (four males, mean age $\pm$ SD = 23.1 $\pm$ 3.0) had normal or corrected-to-normal vision. The study was approved by the local ethical committee of the Medical Department of Ghent University.

Stimuli

All stimuli were stored as 180 $\times$ 180-pixel image sequences and were presented for 250 ms as a continuous movie of frame sequences at a frame rate of 60 Hz on a

Figure 1.

Design of the experiment. A trial started with the presentation of a cue for 500 ms which instructed the participants which of the two tasks (motion or color task) to perform. After a variable cue-target interval (ranging between 200 and 6,150 ms following a pseudo-logarithmic distribution), the stimulus was presented for 250ms. The participants were instructed to respond as fast as possible, without sacrificing accuracy. After a variable response-cue interval (ranging between 200 and 6,150 ms following a pseudo-logarithmic distribution), the next trial was initiated.

black background on a screen positioned 120 cm from the subject. The stimuli (size = 4.6 visual degrees) were filled with a random texture pattern (50% colored and 50% black pixels) moving at a standard speed of 1.5°/s. The colored pixels were either red or blue and were matched for luminance. The pixels moved up and down (125 ms each) or left and right (125 ms each).

Experimental Procedures

On each trial (see Fig. 1), subjects were presented with one out of four cues (a square, circle, triangle or diamond; size: 1.0°) for 500 ms. Each task (see below) was associated with two cues. These cue-to task assignments were counterbalanced across subjects. After a jittered cue-target interval (CTI; 200–6,150 ms, in steps of 350 ms, distribution with pseudo-logarithmic density), the stimulus was presented for 250 ms after which the subjects had to respond as fast as possible, without sacrificing accuracy. The jittering enabled separating the activity of cue and target within a trial. In the motion task, subjects judged the direction of motion (up and down vs. left and right) of the stimulus. In the color task, subjects judged the color (red vs. blue) of the colored pixels of the stimulus. Subjects used the index finger of their right and left hand to answer. The stimulus-response assignments for each task were counterbalanced

across subjects. After a jittered response-cue interval (RCI; 200–6,150 ms, in steps of 350 ms, distribution with pseudo-logarithmic density), the next trial started.

Prior to scanning, all subjects were instructed about the two tasks and worked through one practice block for each task separately (each 32 trials). The order of these practice blocks was counterbalanced across subjects. Afterwards, subjects worked through a practice block (48 trials) in which these two tasks were randomly intermixed. In the scanner, subjects went through four blocks of 92 trials, each which were equally distributed across the two tasks and randomly intermixed. Switch and repeat trials occurred with identical frequency. The maximum number of successive repeat trials was set to 5. The same limitation was used for the number of successive switch trials. Each block started with an instruction screen reminding the subjects of the cues and the stimulus-response assignments associated with each task.

Scanning Procedure

Subjects were positioned head first and supine in the magnetic bore. Images were collected with a 3T Magnetom Trio MRI scanner system (Siemens Medical Systems, Erlangen, Germany), using a standard eight-channel radio-frequency head coil. Participants were instructed not to move their heads to avoid motion artifacts.

First, 176 high-resolution anatomical images were acquired using a T1-weighted 3D MPRAGE sequence [TR = 1,550 ms, TE = 2.39 ms, TI = 900 ms, acquisition matrix = 256 × 256 × 176, sagittal FOV = 220 mm, flip angle = 9°, voxel size = 0.9 × 0.86 × 0.86 mm³ (resized to 1 × 1 × 1 mm³)]. Whole brain functional images were collected using a T2*-weighted EPI sequence, sensitive to BOLD contrast (TR = 2,000 ms, TE = 35 ms, image matrix = 64 × 64, FOV = 224 mm, flip angle = 80°, slice thickness = 3 mm, distance factor = 17%, voxel size 3.5 × 3.5 × 3.5 mm³, 30 axial slices). A varying number of images were acquired per run.

fMRI Data Pre-Processing and Main Analysis

Data processing and analyses were performed using Matlab and the SPM8 software (Wellcome Department of Cognitive Neurology, London, UK). The first four scans of all EPI series were excluded from the analyses to minimize T1 relaxation artifacts. Data processing started with slice time correction and realignment of the EPI datasets. A mean image for all EPI volumes was created, to which individual volumes were spatially realigned by rigid body transformation. The high-resolution structural image was co-registered with the mean image of the EPI series. The structural image was normalized to the Montreal Neurological Institute (MNI) template. The normalization parameters were then applied to the EPI images to ensure an anatomically informed normalization. Motion parameters were estimated for each session separately.

A commonly applied filter of 8 mm FWHM (full-width at half maximum) was used. The time series data at each voxel were processed using a high-pass filter with a cut-off of 128 s to remove low-frequency drifts.

Basic Analyses

The subject-level statistical analyses were performed in the context of the general linear model (GLM) in SPM8. A parametric regression analysis was applied [Buchel et al., 1998] to determine the relationship between a parametric regressor (see below) and the BOLD signal. The main events of interest were the periods after the onset of the cues. A single vector containing the event onsets for all experimental trials was modulated by convolution with a parametric regressor that allowed testing for areas showing an increase or decrease of the BOLD signal as a function of (1) reconfiguration or (2) adaptation/repetition suppression. The cue onset vector was also convolved with the temporal derivative. The design matrix furthermore contained the target onset vector and the six translational and rotational motion correction parameters. All regressors were convolved with the canonical haemodynamic response function (HRF). The statistical parameter estimates were computed separately for each voxel for all columns in the design matrix.

Since the reconfiguration and the adaptation view predict an identical activation pattern when only looking at the activation level as a function of trial $n - 1$, we had to model brain activation continuously by using a parametric approach. A hypothesis on how this parameter should evolve across several subsequent trials according to the adaptation view could be derived from a single cell study [Sawamura et al., 2006] and an fMRI study [Grill-Spector and Malach, 2001]. Both studies showed that adaptation gradually increased as the number of repetitions increased: activation after a second repetition was lower compared to a first repetition; activation after three repetitions was again lower than after two repetitions, and so on. Importantly, the size of activation reduction by each stimulus recurrence was monotonically decreasing, but not at a constant rate: the first repetition produced a more substantial reduction in the signal strength compared to the second repetition, which caused by itself a larger reduction than the third repetition, and so on. No evidence could be found suggesting that the reconfiguration view would predict an effect of successive repetitions on brain activity. Since the reaction time results showed this same kind of exponential decrease with successive repetitions, we defined the adaptation coding parametric regressor on the basis of the mean reaction times. For this regressor, all switch trials were assigned a weight of 100. The weights assigned to the repeat trials depended on the number of preceding repeat trials. Repeat trials following 0 (i.e., after a switch trial) to 4 repeat trials were assigned a respective weight of 76.7, 63.7, 56.4, 52.3, and 50.0, following an expo-

ponential decay function fitted to the reaction time data. Note that different definitions of this adaptation coding parametric regressor (which all followed an exponential decrease with successive repetitions) showed very similar results. Since an effect of successive repetitions was not predicted by the reconfiguration view, we assigned a weight of 100 to all switch trials for the reconfiguration coding parametric regressor. A weight of 50 was assigned to all repeat trials. This is similar to using a non-parametric model and searching for areas displaying higher activation for switch compared to repeat trials as is commonly used in fMRI task-switching studies.

To control for the possibility that a difference in fit between these two models was caused by the fact that both models allowed a different level of flexibility (i.e., the reconfiguration regressor only had two possible values whereas the adaptation regressor had six possible values), we also included an adjusted version of the reconfiguration model in which successive switch trials lead to decreasing activation. This adjusted version thus assumed adaptation for the switch process itself and allowed the same level of flexibility as the adaptation model. For this regressor, we assigned a weight of 50 to all repeat trials. The weights assigned to the switch trials, however, depended on the number of preceding switch trials. Switch trials following 0 (i.e., after a repeat trial) to four switch trials were assigned a respective weight of 100, 85.4, 77.2, 72.6, and 70, following an exponential decay function.

Group analyses were performed according to the random effects procedure using the single-subject contrast images as input. Group SPMs were generated using a one-sample t test with a voxel threshold of $P < 0.005$. Based on Monte Carlo simulations (AFNI AlphaSim; 10,000 iterations) with our brain volume, it could be determined that a cluster size of 147 mm³ (42 contiguous voxels) ensured an overall image-wise false-positive rate of 5% (corrected $P < 0.046$).

Bayesian Model Comparison

To select the most optimal model for each voxel, we used the Bayesian model selection (BMS) procedure. This procedure is a powerful tool to determine the most likely model among a set of competing models on the mechanisms generating the observed data [Stephan et al., 2009] by providing information on the probability that one model is more likely than another model, given the group data. From a Bayesian perspective, a model is selected on the basis of the model evidence, i.e., the probability $P(y|m)$ of the data y given a particular model m [Raftery, 1995]. The ratio of two model evidences is the log-evidence ratio, commonly known the Bayes factor [Kass and Raftery, 1995]. In this study, we followed the analysis procedure described by Rosa et al. [2010]. We first estimated each model, described above, using the first-level Bayesian estimation procedure [Penny et al., 2005] for every subject. This resulted in a voxel-wise whole-brain log-evidence

Figure 2.

Reaction time results. RTs for repeat trials (with SEs) are plotted in black as a function of the position in the sequence of successive repeat trials. RTs for switch trials (with SEs) are plotted in grey as a function of the position in the sequence of successive switch trials.

map for every subject and for every model we have estimated. We then applied the random effects approach [Stephan et al., 2009] to the group model log-evidence data in a voxel-wise manner, providing a posterior probability map [PPM; Friston and Penny, 2003] and an exceedance probability map (EPM) for each model [Rosa et al., 2010] at group-level. The posterior probability is a measure of likelihood that a specific model generated the data of a randomly chosen subject. A posterior probability map (PPM) shows regions where the posterior probability of a specific model is greater than a certain pre-defined threshold. The exceedance probability describes the probability that one model is more likely than another model, given the group data. An exceedance probability map (EPM) then shows the regions where the exceedance probability of a specific model exceeds a pre-defined threshold. An exceedance probability threshold of 0.80 already suggests strong evidence in favor of a model [Rosa et al., 2010]. In this article, we will show the results using a very conservative threshold of 0.95. Results using other thresholds are reported in Table II.

RESULTS

Event-related fMRI scans were acquired from adults while they carried out a task-switching paradigm with a color and a motion task. We separated trials into those in which the task switched relative to the previous trial (switch trials) from those in which the task repeated (repeat trials).

In accordance with previous findings, the behavioral data revealed substantial switch costs: participants were significantly slower in switch than in repeat trials (852 ms vs. 777 ms; GLM Repeated Measures ANOVA with trial condition as within-subject variable: $F(1,20) = 29.07$, $P <$

0.001). Reaction times decreased as the number of successive repeat trials increased (GLM Repeated Measures ANOVA with number of previous successive repeat trials as within-subject variable: $F(3,60) = 3.33$, $P < 0.05$). No such effect of number of successive switch trials on reaction times was found (GLM Repeated Measures ANOVA with number of previous successive switch trials as within-subject variable: $F(3,60) = 1.10$, $P = 0.36$; Fig. 2).

To determine brain areas that show stronger activation for switch versus repeat trials, we carried out a parametric analysis in which all trials were coded according to the reconfiguration model (see grey solid line in Fig. 3). Results are presented in Figure 4 and show that activation within the pre-SMA, left inferior frontal gyrus (IFG) and left intra-parietal sulcus (IPS) followed the reconfiguration model, thus displaying higher activation in switch trials compared to repeat trials (Table I).

Next to the reconfiguration model, we also defined an adaptation model (see black dotted line in Fig. 3). Using a Bayesian model selection (BMS) procedure, we examined for each voxel of these three areas which of the two models, the reconfiguration or the adaptation model, better fitted the data (see Materials and Methods). When comparing the adaptation model to the reconfiguration model, 87% of the voxels displaying switch-specific effects showed an exceedance probability of at least 0.95 for the adaptation model. This indicates very strong evidence for the adaptation model (Fig. 4). Thus for all these voxels, the probability of the adaptation model being a more likely

Figure 3.

Illustration of parameter values assigned to a sequence of switch and repeat trials according to the reconfiguration model (grey solid line) and the adaptation model (black dotted line). The two models differ in their assumption on the effect of successive repeat trials. The adaptation model predicts a decrease of activation with successive repeat trials, whereas the reconfiguration approach does not predict such a differential change of activation level in repeat trials over longer sequences of trials.

Figure 4.

Areas displaying stronger activation for switch trials compared to repeat trials are displayed in blue. Activation map averaged across 21 subjects ($P < 0.005$, uncorrected, cluster size = 42) mapped onto a standard Colin brain template. These areas were used as masks in the Bayesian model selection procedure

comparing the adaptation model to the reconfiguration model. Voxels within these areas showing an exceedance probability greater than 0.95 for the adaptation model (which indicates very strong evidence for this model) are colored red.

model than the reconfiguration model was at least 95%. Only 1% of the voxels exceeded this threshold in favor of the reconfiguration model. When lowering the threshold to 0.80, 94% of the voxels exceeded this value, indicating strong evidence for the adaptation model [cf. Rosa et al., 2010], whereas still only 2% showed evidence in favor of the reconfiguration model. Thus, in general, the exceedance probability map for the adaptation model almost completely overlaps with the switch-specific activity (Fig. 4).

To control for the possibility that the better fit for the adaptation model was caused by its higher level of flexibility (i.e., the reconfiguration regressor only had two possible values whereas the adaptation regressor had six possible values), we also tested a modified version of the reconfiguration model in which successive switch trials lead to

decreasing activation, thus assuming adaptation to the switch process itself (see Materials and Methods). Although not backed-up by the behavioral results, a Bayesian model comparison showed that the adjusted reconfiguration model fitted the data in the three areas better than the basic reconfiguration model: 70% of the voxels showed a higher exceedance probability compared to the 0.95 threshold for the adjusted reconfiguration model (Table II), whereas only 6% of the voxels showed evidence in favor of the basic reconfiguration model. However, the exceedance probabilities for the adaptation model when comparing this adaptation model to the adjusted reconfiguration model showed that activation in these areas was still strongly in favor of the adaptation model, despite the increased flexibility of the adjusted reconfiguration model (compared

TABLE I. Anatomical location and MNI coordinates

Area	Peak coordinates	z-score	extent
A) Reconfiguration model			
(pre-)SMA	4, 4, 60	4.22	98
Left inferior frontal gyrus	-49, 38, 18	3.78	43
Left inferior parietal sulcus	-35, -46, 49	3.68	44
B) Adaptation model			
(pre-)SMA/ACC	7, 4, 60	4.64	213
Left inferior frontal gyrus	-49, 42, 14	3.79	65
Left inferior parietal sulcus	-35, -49, 49	3.71	203
Left inferior frontal junction	-46, 4, 28	3.44	146
Right inferior frontal gyrus	49, 42, 24	3.40	50

$P = 0.005$, uncorrected.

to the basic reconfiguration model): very strong evidence (exceedance probabilities > 0.95) for this adaptation model was found in 61% of the voxels, whereas only 4% showed these high exceedance probabilities for the adjusted reconfiguration model. When lowering the threshold to 0.80, 78% of the voxels showed strong evidence for the adaptation model whereas 7% of the voxels showed evidence in favor of the adjusted reconfiguration model. Activity in these regions was thus best explained by adaptation to successive repeat trials, as estimated by the adaptation model, suggesting that these areas show adaptation to abstract representations of the task set. This is nicely illustrated in Figure 5, in which the results of a region of interest (ROI) analysis for both the adaptation model and the adjusted reconfiguration model are presented. Data for the different conditions (switch and first to fourth repeat for the adaptation model; repeat and first to fourth switch for the adjusted reconfiguration model) for each ROI were extracted from the peak voxel identified for each of the three areas. The results, averaged across these three areas, clearly show that activity in these regions indeed is better explained by adaptation to successive repeat trials (upper part) compared to adaptation to successive switch trials (lower part).

Since the adaptation model was very successful in explaining brain activation that was extracted from the classical switch versus repeat contrast, we were wondering

whether additional brain areas follow the adaptation model. We therefore performed a parametric analysis in which all trials were coded according to the adaptation model. Results are presented in Figure 6 and show extended activation in the same three areas as in the previous analysis (pre-SMA, left IFG and left IPS) and two additional areas: the left inferior frontal junction (IFJ), extending to the primary motor cortex (PMC), and right IFG. Contrary to the analyses based on the basic reconfiguration model, the activation in pre-SMA now extended to the ACC, and much more voxels in the IPS reached significance (Table I).

The results above suggest that the pre-SMA, left IPS, left IFJ and bilateral IFG show adaptation to the abstract representation of the task set. However, since in half of the repeat trials also the cue repeated, these results might reflect mere adaptation to the low-level visual representation of the cue. To disentangle cue adaptation from task set adaptation, we performed a region of interest (ROI) analysis for each of these five areas to compare the signal change in switch trials, repeat trials in which the cue switched (i.e., task repeat trials) and repeat trials in which the cue repeated (i.e., cue repeat trials) separately. Data for these three conditions for each ROI were extracted from the peak voxel identified for each of the five areas. Figure 7 shows the percent signal change in each of these areas for the cue repeat, task repeat and switch trials. For the pre-SMA and IFJ, no difference was found between the cue repeat and task repeat trials (one-sided t -test for dependent samples, $P = 0.21$ and $P = 0.32$, respectively), whereas both repeat conditions differed from the switch condition ($P < 0.05$ for all comparisons). These areas thus showed adaptation to abstract task set representations but no adaptation to the cue. The IPS showed both cue adaptation (cue repeat vs. task repeat: $P < 0.05$) and task set adaptation (task repeat vs. task switch: $P < 0.05$). The results of the left and right IFG were less straightforward: left IFG clearly showed task set adaptation (task repeat vs. task switch: $P < 0.05$) and a tendency towards significant cue adaptation (cue repeat vs. task repeat: $P = 0.065$). Right IFG showed no cue adaptation (cue repeat vs. task repeat: $P = 0.36$) and a tendency towards significant task set adaptation (task repeat vs. task switch: $P = 0.064$).

TABLE II. Proportion of voxels best explained by the different models

Model comparison	Exceedance probability threshold				
	0.50	0.70	0.80	0.90	0.95
A) Adaptation vs. basic reconfiguration model					
Adaptation model	97%	96%	94%	91%	87%
Basic reconfiguration model	3%	3%	2%	1%	1%
B) Adjusted vs. basic reconfiguration model					
Adjusted reconfiguration model	88%	83%	81%	77%	70%
Basic reconfiguration model	12%	9%	9%	7%	6%
C) Adaptation vs. adjusted reconfiguration model					
Adaptation model	87%	83%	78%	66%	61%
Adjusted reconfiguration model	13%	8%	7%	6%	4%

Figure 5.

Region of interest analyses. Percent signal changes averaged across the peak voxels of areas pre-SMA, IFG (left) and IPS (left) are shown for the different trial conditions (grey circles), as defined by the adaptation model (upper part) and the adjusted reconfiguration model (lower part). The predicted percent signal changes for the respective models (based on the parameter values for that model) are presented by the black line.

DISCUSSION

The present results show that the activation pattern in areas displaying higher preparation-related activation in switch trials compared to repeat trials is better explained by task set adaptation than by task reconfiguration. This finding questions the common view that activation differences between switch and repeat trials are primarily due to a reconfiguration in switch trials. Furthermore, our results demonstrate that using an adaptation model leads to reliable activation in cognitive control related brain areas that are not activated when simply contrasting switch and

repeat trials. Finally, our results demonstrate that adaptation effects are not restricted to simple perceptual representations but also occur for abstract task representations.

A Representational Perspective on Cognitive Control

About a decade ago, the task switching paradigm was first applied to functional MRI [Dove et al., 2000]. Ever since, a growing number of studies have used this paradigm to investigate the brain basis of cognitive control. Most of these studies have implicitly or explicitly assumed that the comparison of switch versus repeat trials reflects additional processes in switch trials. This logic is derived from the idea that in switch trials we have to configure the new task set which is not required in repeat trials [Meiran, 1996; Monsell and Mizon, 2006; Rogers and Monsell, 1995]. However, recent behavioral research has questioned this assumption and suggested that the performance difference between switch and repeat trials is better interpreted in terms of a facilitated performance on repeat trials than in terms of a worse performance on switch trials [Dreisbach et al., 2002; Ruthruff et al., 2001; Sohn and Carlson, 2000]. These authors suggested referring to the difference between switch and repeat trials as a repetition benefit rather than a switch cost. Translated to the neural level, this would suggest that switch-specific brain differences are not due to additional activation in switch trials but rather to a reduction of activation in repeat trials. By simply comparing switch and repeat trials, it is not possible to dissociate these two accounts. Both predict very similar patterns of brain activation. To dissociate an adaptation view from a reconfiguration view, it is necessary to consider longer sequences of trials. The present study is to our knowledge the first fMRI study that follows this logic by using a parametric approach. Our data clearly show that switch-related brain activation is better explained by repetition benefits rather than reconfiguration costs.

The adaptation account, however, implies a completely different way of thinking about the role of the prefrontal and parietal cortex in cognitive control. Rather than assuming that activation in these brain regions reflects specific processes such as reconfiguration, this approach rather promotes a representational view of cognitive control. From this perspective, prefrontal and parietal brain regions, like perceptual brain regions [for a review, see Grill-Spector, 2003], house specific representations. Representations can be defined as memories localized in neural networks that encode information and, when activated, enable access to this stored information [Wood and Grafman, 2003]. The prefrontal and parietal representations only differ from the representations in visual brain areas with respect to their content and how enduring they are.

In this sense, our findings strongly support the perspective put forward by Wood and Grafman [2003]. After

Figure 6.

Areas displaying larger activation for switch trials compared to repeat trials and increasing adaptation with increasing number of successive repeats, as coded by the adaptation model. Activation map averaged across 21 subjects ($P < 0.005$, uncorrected, cluster size = 42) mapped onto a standard Colin brain template.

reviewing the primary theories of prefrontal cortex function, Wood and Grafman [2003] argued that only theories promoting a representational approach, such as the structured event complex framework [Grafman, 2002] or the guided activation theory [Miller and Cohen, 2001], provide a useful framework to understand prefrontal cortex function, since this perspective is highly compatible with electrophysiological, neuroimaging and monkey neurophysiological results. Miller and Cohen [2001], for instance, proposed that cognitive control stems from the active maintenance of activity patterns in prefrontal cortex that represent goals and means to achieve them. Indeed, single cell studies showed that the neuronal properties in prefrontal cortex are consistent with representing a task set [Wallis, 2007]. Not only are many of the prefrontal responses dependent on the precise task that the subject is

performing [Asaad et al., 2000; Hoshi et al., 1998, 2000; White and Wise, 1999], some neurons also showed an increased firing rate whenever the monkey was performing one task relative to another [Asaad et al., 2000]. Prefrontal neurons do not only encode concrete task rules, they also code abstract rules [Wallis and Miller, 2003; Wallis et al., 2001]. Furthermore, the neuronal encoding of a task [Stoet and Snyder, 2009] is not restricted to the prefrontal cortex. Stoet and Snyder [2004, 2007] showed that also parietal neurons responded selectively to cues for different task rules. Their results indicated that these parietal neurons reflect abstract rules, rather than merely representing a particular motor command. This suggests that the representational approach could also be valid for the understanding of the parietal cortex function. Both prefrontal and parietal neurons thus seem to contain abstract

Figure 7.

Region of interest analyses. Percent signal changes for the peak voxel of areas pre-SMA, IFG (bilateral), IFJ (left) and IPS (left) are shown in the cue repeat (dark grey bars), task repeat (light grey bars) and task switch (white bars) condition.

task set representations, similar to the way visual brain areas house representations of concrete objects.

Adaptation to the Cue or to the Task?

In studies of human perception, many cortical areas thought to house concrete object representations showed repetition suppression with repeated presentation of these visually presented objects [for a review, see Grill-Spector et al., 2006; Krekelberg et al., 2006]. In all these studies, as well as in recent adaptation studies on language [Chee et al., 2003; Klein et al., 2006] and memory [Turk-Browne et al., 2006], however, the stimuli that were repeated were concrete in nature. In the present study, we observed adaptation to the repeated presentation of a concrete, visual object (i.e., the cue), in left IPS (a tendency towards cue adaptation was found in left IFG). However, adaptation to this cue fell short explaining all our results. Even when the cue switched, the activation was reduced in left IFG, left IPS, pre-SMA, and left IFJ when the same, but not a different task had to be prepared in two subsequent trials (a tendency toward task set adaptation was found in right IFG). This finding argues against the claim of Logan and Bundesen [2003] that switch costs are primarily due to switching of the cue instead of switching of the task. A fairly common assumption in cognitive psychology is that in order to prepare the execution of a particular task, one need to activate a task set. Since neurophysiological data indicate that parietal and prefrontal neurons house task set representations and since only the representation of this task set is the same in two subsequent trials when repeating the task and different in two subsequent trials when switching tasks, we can conclude that the activation reduction in repeat trials reflects the adaptation to the abstract task set representation. To our knowledge, this study is the

first to show adaptation to such abstract representations. The different areas we have observed possibly adapt to different components of the task set (e.g., stimulus dimension, stimulus-response mapping, response category, response modality, etc.). However, with the present design, we cannot disentangle these different parts.

It is important to consider that the repetition suppression we observed in the present study is different from the repetition-related activity reductions caused by experience-dependent learning [Dobbs et al., 2004; Horner and Henson, 2008; Race et al., 2009]. These studies showed that stimulus-decision or stimulus-response learning decreased the demands on prefrontal cortex. However, the sharpening mechanism, thought to underlie such kind of repetition priming or long-lag repetition suppression [Li et al., 1993] is different from the mechanism that underlies the short-lag adaptation observed here [De Baene and Vogels, 2010].

In a recent study using a similar parametric approach in a combined location-cueing/oddball paradigm, Vossel et al. [2011] suggested a different interpretation of the observed decreasing neural activity with increasing numbers of repetitions of valid standard trials in right inferior and middle frontal gyrus. Instead of interpreting this decreasing activity with increasing repetitions as a reflection of adaptation, Vossel et al. suggested that this activation pattern reflects the neural representation of event regularities. In the present study, however, the result pattern for successive switch trials is quite different from the result pattern for successive repeat trials although the regularity of switch and repeat trials is very similar, which does not fit this event regularity perspective put forward by Vossel et al. [2011].

The Adaptation Approach and Brain Activation Related to Cognitive Control

One puzzling finding of brain imaging research on cognitive control using the task switching paradigm is the heterogeneity of results. Very often, switch specific brain activation was restricted to only one brain area. Kimberg et al. [2000], for instance, found an activation difference only in the superior parietal lobule, whereas no frontal areas showed up. Brass and von Cramon [2004] did not find any activation difference between switch and repeat trials. Cole and Schneider [2007] showed, however, that the cortical regions, involved in many forms of cognitive control, form a functionally connected cognitive control network (CCN). Regions within the CCN include the ACC/pre-SMA, dorsolateral PFC, IFJ, anterior insular cortex (AIC), dorsal pre-motor cortex (dPMC), and posterior parietal cortex (PPC). In the present study, only a small part of this CCN was revealed when comparing switch and repeat trials using the reconfiguration model (i.e., pre-SMA, left IFG, and IPS). A much larger part of the CCN, however, appeared using the adaptation model (i.e., ACC/pre-SMA, bilateral IFG, left IFJ, PMC, and IPS). This suggests that applying an adaptation approach to model fMRI data in a task switching paradigm leads to

more reliable activation in the cognitive control network than the classical switch versus repeat analysis.

Interestingly, this finding provides a new perspective on the failure of some task switching studies to find reliable switch specific brain activation. According to Brass and von Cramon [2004], some cognitive control brain regions do not show a consistent switching effect in task switching studies with a high proportion of switch trials, since, under these circumstances, participants tend to prepare the task equally in each trial. This, indeed, could explain why Kimberg et al. [2000] did not find frontal switch-related activation using the alternating runs procedure and why Brass and von Cramon [2004] did not find any switch-related activation at all, since, in these studies, the probability of a switch was quite high. Dove et al. [2000], however, used a design with a considerably smaller switch likelihood and did find activation differences between switch and repeat trials in almost all areas of the CCN. Possibly, however, the lack of significant activation differences in switch vs. repeat trials is not due to the particular characteristics of the design (i.e., the switch likelihood) that motivates the participants to equally prepare the task in both sorts of trials. Based on our results, it is more plausible that in circumstances with a high switch probability, the amount of successive repeat trials is low, which results in a low level of adaptation to repeat trials, making the activation difference between switch and repeat trials quite small. As a consequence, fewer or no areas show a significant activation difference between switch and repeat trials, as is the case in the Kimberg et al. and the Brass and von Cramon study. With a smaller switch likelihood, it is more likely that the number of successive repeat trials is larger, resulting in more adaptation. As a consequence, the overall activation difference between switch and repeat trials is larger, resulting in more areas showing a significant switch vs. repeat activation difference, as is the case in the Dove et al. study. With the design of the current study, however, it is not possible to dissociate this hypothesis from the hypothesis proposed by Brass and von Cramon [2004].

Adaptation or Decay of Interference

Finally, one needs to discuss the possibility that adaptation effects are related to task interference. The basic assumption of the adaptation model is that with successive repetitions, activation for a specific task representation adapts leading to a decrease of activity. However, a task interference account would lead to a very similar prediction. According to the interference view, the performance difference between switch and repeat trials represents an automatic carryover effect of the wrong task set from previous trials as a result of task-set inertia or proactive interference [i.e., the passive dissipation of the preceding task set; Allport et al., 1994]. In repeat trials, the carryover effect is less harmful because it supports performance in the current trial. In switch trials, however, the carryover effect is distracting because the wrong task set is pre-acti-

vated. The interference account assumes a decay of activation of the previous task set. One could assume, however, that, as long as the previous irrelevant task is still somewhat activated, interference will also occur in repeat trials. As a consequence, increasing the number of successive repeat trials should lead to a decrease in interference from the other, irrelevant task. Although we cannot totally exclude interference as an alternative account to explain the activation decrease with an increasing number of repeat trials, an additional analysis (cf. Supporting Information) suggests the interference account is very unlikely to hold true in this present study. This analysis started from the assumption that decay of task set activation is time-dependent [Altmann and Gray, 2008]. As a consequence, the activation difference between two successive trials should be larger if the time between these two trials is longer. For sequences in which the task was repeated at least three times, we computed the activation difference between the first repeat trial and the third repeat trial in the two areas that clearly showed task set adaptation and no cue adaptation (i.e., pre-SMA and left IFJ). We then subdivided these activation differences based on the time between the first and third repeat trial in four parts and compared the activation difference for the fastest part (i.e., the first quartile) with that for the slowest part (i.e., the fourth quartile). According to the interference assumption mentioned above, a fast succession of trials should lead to smaller activation differences compared to a slow succession of trials. However, this effect was not found. Moreover, if any, the (non-significant) effect went in the opposite direction: faster succession of trials led to larger activation differences (Supporting Information Fig. 1), which severely diminishes the probability that the interference account is valid in the present study.

Note, however, that the interference and adaptation accounts do not need to be mutually exclusive. Both views argue against an additional control process in switch trials, as claimed by the reconfiguration view. Both the interference and the adaptation account, based on the present findings, also assert that the difference between switch and repeat trials might be better referred to as a repetition benefit, since it seems to reflect an activation decrease when the task is repeated (cf. Above).

Taken together, the findings reported above provide strong evidence that neurons in different parts of the cognitive control network show adaptation to abstract task set representations. We have argued that it is this adaptation to abstract task set representations in repeat trials that causes the activation difference in preparing switch compared to repeat trials in task switching studies, which was up till present generally accredited to additional reconfiguration processes in switch trials.

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