

*Perceptual learning, motor learning and automaticity*

# Switching from automatic to controlled behavior: cortico-basal ganglia mechanisms

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**Most daily tasks are performed almost automatically, but occasionally it is necessary to alter a routine if something changes in the environment and the routine behavior becomes inappropriate. Such behavioral switching can occur either retroactively based on error feedback or proactively by detecting a contextual cue. Recent imaging and electrophysiological data in humans and monkeys support the view that the frontal cortical areas play executive roles in behavioral switching. The anterior cingulate cortex acts retroactively and the pre-supplementary motor area acts proactively to enable behavioral switching. The lateral prefrontal cortex reconfigures cognitive processes constituting the switched behavior. The subthalamic nucleus and the striatum in the basal ganglia mediate these cortical signals to achieve behavioral switching. We discuss how breaking a routine to allow more adaptive behavior requires a fine-tuned recruitment of the frontal cortical-basal ganglia neural network.**

## Breaking a routine: difficult but crucial

Driving to one's workplace is an easy task: a task that most of us do on a daily basis for several years. On our journey to work we see the same houses, the same trees and the same traffic lights. We might not be aware of our car accelerating or slowing down, despite being the driver. If there is unexpected congestion in the main road ahead then we can quickly decide to avoid the traffic jam by changing our route. But if the decision is late, even by only a second, the chance to turn and avoid the congestion could be missed. This example illustrates that most daily behaviors are composed of well-learned routines – occasionally, given the context, an important decision is made to switch from a routine behavior to an alternative and more appropriate behavior.

Behavioral switching has been an important question in experimental psychology [1], and there has been a recent surge of interest among neuroscientists in this area. Several recent studies using neuroimaging methods and transcranial magnetic stimulation with human subjects, including subjects with prefrontal lesions, indicate that several regions in the frontal cortex play different roles in behavioral switching [2–5].

However, how the brain actually executes behavioral switching is not fully understood from the human data alone. The switching-associated reconfiguration of cognitive processes indicated by the psychological studies is probably composed of serial and parallel neuronal activity changes which occur within a short period before the decision to switch. However, the spatiotemporal resolution of the imaging data might not be sufficient for elucidating such fast changes in neuronal activity. To this end, single unit recording experiments using trained animals provide important complementary data.

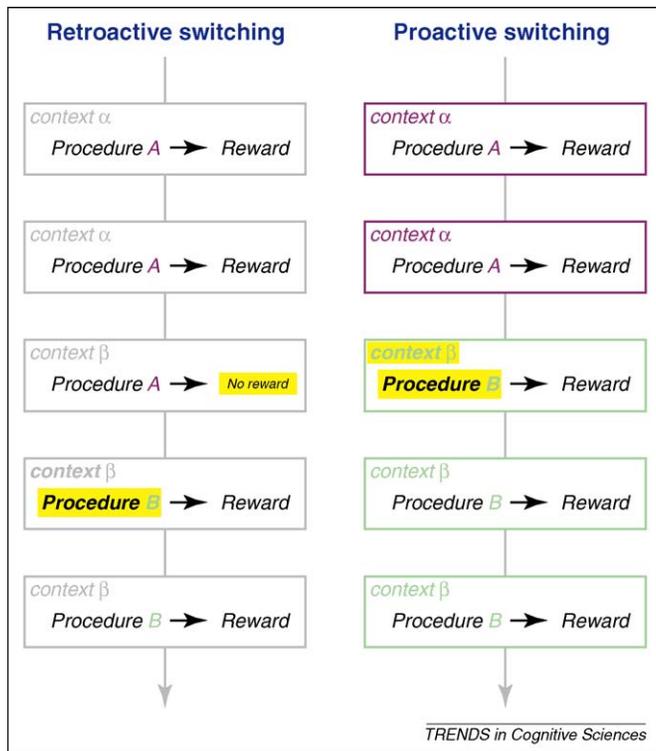
In this article, we synthesize the insights provided by human neuroimaging data and animal single neuron data and put forward a framework that specifies the neural circuits involved in the execution of behavioral switching.

## Two modes of behavioral switching

To understand the neural mechanisms of behavioral switching, it is important to determine what triggers such switching. Let us consider a situation in which procedure A is the appropriate behavior in order to obtain a reward in context  $\alpha$ , whereas procedure B is the appropriate behavior in context  $\beta$  (Fig. 1), and a motivated subject has already learned these associations. Suppose the context changes from  $\alpha$  to  $\beta$ . If the subject is unaware that the context has changed, s/he will perform procedure A and will therefore receive no or little reward (Fig. 1, left). This negative feedback signal triggers behavioral switching on the next trial. By contrast, if the subject is aware of the context change, s/he will perform procedure B instead of A and will obtain a reward (Fig. 1, right). We term these two modes of switching 'retroactive switching' and 'proactive switching' respectively.

Note that this classification is different from the proposal by Braver and colleagues on proactive and reactive control of cognitive function [6]. Proactive control in their framework refers to a sustained process before the onset of an imperative stimulus, whereas reactive control refers to a transient process after the onset of an imperative stimulus. There is no particular emphasis in their hypothesis on how behavior might switch when the context changes, whereas our main goal is to understand the switching process where, we believe, the retroactive–proactive distinction is useful.

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**Figure 1.** Retroactive and proactive switching. Retroactive switching (left) is triggered by a failure (decreased reward value or an error). In this case the context cue is either absent or unknown to the animal (indicated by gray rectangles). Proactive switching (right) is triggered by a cue signaling a context change so that the subject will not experience the failure. This is possible, however, only after the subject has learned the meaning of the cue (indicated by purple and green rectangles). Highlighted in yellow are triggers of behavioral switching and switched procedures.

In retroactive switching (Fig. 1, left), the subject's behavior is bound to fail on switch trials. This is costly in a dangerous world where one-time failure could be fatal. However, a change in context can be indicated in advance by a change in sensory inputs, which is often called a cue. Detection of the cue enables proactive switching in which the behavior can continue to be optimal even on switch trials. In a social context, the cue might be a change in facial expression or gaze direction of one's partner or manager [7]. It should be emphasized that the subject has to discover the cue from his/her experience. The discovery depends on learning, specifically learning of statistical relationships between the cues and the outcomes (e.g. rewarding or punishing).

A main proposal in this article is that retroactive switching and proactive switching are controlled by different regions in the medial frontal cortex, anterior cingulate cortex (ACC) and the pre-supplementary motor area (pre-SMA).

### The ACC and retroactive switching

The brain region that enables retroactive switching needs to be sensitive to negative feedback (e.g. reduced reward or punishment). It also needs to have access to the brain regions that implement alternative learned procedures. The ACC seems to fulfill both of these requirements.

First, many neurons in the monkey ACC are excited by negative feedback. In experiments using monkeys, the

monkeys are trained to perform a task in order to obtain a certain amount of reward. If the reward is absent (e.g. due to poor performance) or reduced in amount experimentally, some ACC neurons are excited [8–12]. Task-selectivity of ACC neurons is strongest after switching and declines thereafter, consistent with their role in retroactive switching [13]. Neuronal activity in the ACC after negative feedback can continue if and until the monkey switches procedures [9] (Box 1). Further, switching is impaired by inactivation of the ACC [9].

Second, the switching function of the ACC might be mediated by its connections to the lateral prefrontal cortex (LPFC) [14,15], which is thought to play an executive role in procedure implementation. An alternative pathway might be the connections to the striatum [16], which is equipped with mechanisms for behavioral selection [17]. The role of the ACC–striatum connection is perhaps supported by the finding that striatal neurons show rapid changes in activity after retroactive switching in associative learning [18].

Neuroimaging studies with human subjects support the above conclusion. Functional magnetic resonance imaging (fMRI) studies have indicated that the ACC is activated when the subject fails to perform a trial correctly (e.g. by failing to stop a button press) [19–24]. Human EEG studies have revealed error-related potentials immediately after the erroneous motor response or after the error feedback, which are thought to be generated in the ACC [2]. Similar error-related potentials are recorded using electrodes placed in the monkey and human ACC [25–27], which might be associated with the error-induced burst firing of ACC neurons described above.

The sensitivity of ACC neurons to negative feedback indicates that they could be related to motivational decision-making in general. In fact, some ACC neurons are excited by positive feedback (i.e. reward), but only when the reward is unexpected (i.e. immediately after the correct choice is discovered) [12,28]. These results support the view that the ACC enhances cognitive processes not only before switching (based on an unexpected error) but also after switching (based on an unexpected reward). Indeed, lesions of the ACC can cause general impairments in decision-making based on the history of actions and outcomes [29,30].

### The pre-SMA and proactive switching

A conflict in information processing characteristically occurs in proactive switching. The subject's performance on switch trials is much worse (high error rate and longer reaction time) than when the same context is repeated (non-switch trial), a phenomenon called 'switch cost' [1]. This is thought to occur because multiple cognitive operations are executed in response to the switch cue, which might include suppression of the old procedure and facilitation of the new procedure. The switch cost is particularly high if the old procedure has been repeated and therefore has become habitual or automatic.

Various lines of research support the view that the pre-SMA [31] is essential for proactive switching. Functional

MRI studies have shown that the pre-SMA is consistently activated when human subjects switch between two tasks proactively in response to a cue [32,33]. Repetitive transcranial magnetic stimulation over the pre-SMA disrupts subjects' performance in switch trials, but not in non-switch trials [33].

Such pre-SMA activation could be related to the cognitive operations described above. First, the pre-SMA seems to have a powerful mechanism to suppress body movements. For example, electrical stimulation of the pre-SMA suppresses ongoing or impending body movements in humans [34] and monkeys [35]. The pre-SMA is activated consistently when the human subject tries to stop an impending movement [19,36,37]. Such inhibitory control is impaired in subjects with lesions including the pre-SMA [38] and in normal subjects when transcranial magnetic stimulation is applied over the pre-SMA [39]. Second, the human pre-SMA is activated when two procedures compete with each other [19,22,37]. Thus, the conflict associated with proactive switching (i.e. conflict between the old

and new procedures) is likely to be processed in the pre-SMA [40,41].

The fact that transcranial magnetic stimulation over the pre-SMA disrupts performance only on switch trials [33] suggests that the pre-SMA generates switch-related signals transiently at the time of switching. This is in contrast to the ACC, where neural processing continues after an erroneous choice [42] and even after a correct choice [12,28]. The hypothesized difference is supported by a recent finding that the pre-SMA and the ACC show transient and sustained responses, respectively, to incentive cues [43].

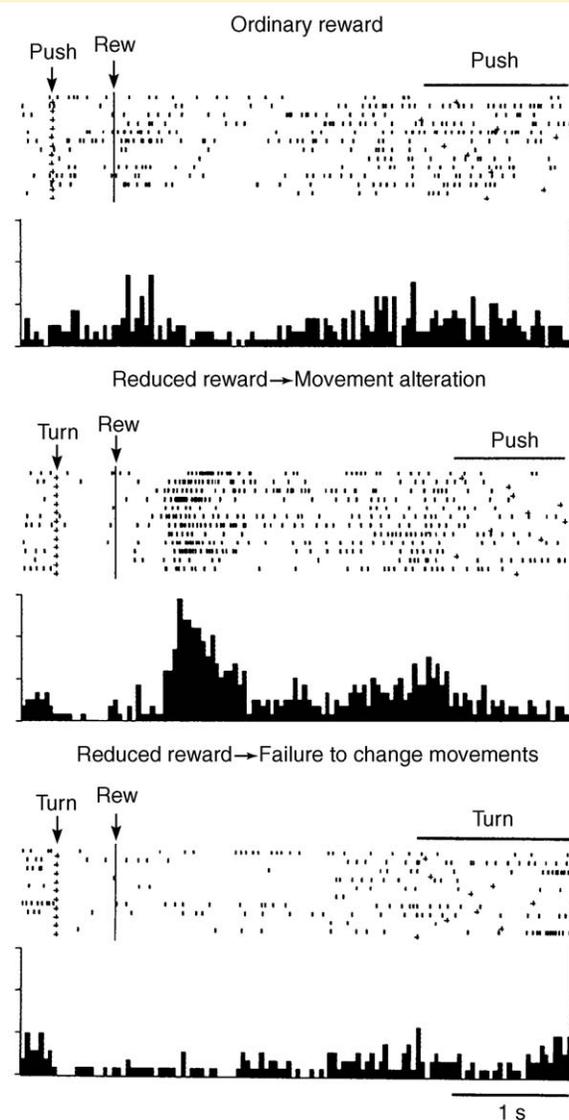
Another indication that the pre-SMA might be related to behavioral switching comes from studies with trained monkeys. Many pre-SMA neurons are activated before the monkey switches button presses from one target to the other in response to a sensory cue [44]. They are also active when the monkey switches from one learned sequential procedure to another learned procedure, but only on the first trial [45]. However, it is unclear from these

### Box 1. Retroactive switching by ACC neurons

There is empirical evidence that errors result in adjustments of behavior in several ways. First, subjects can correct their action slips resulting from premature responses immediately after they have committed an error [62]. Second, subjects slow down on subsequent trials after errors, a phenomenon known as post-error slowing [62]. As long as the correct action remains unchanged, such cautious responding is adaptive to attain the intended goal on the next trial. The ACC is implicated in both error detection [79,80] and post-error adjustments [81]. Third, once subjects realize on the basis of feedback (such as reduced reward) that the previously correct action becomes no longer valid, they switch behavior or learn a correct action (retroactive switching).

In a pioneering study designed to explore the role of the ACC in retroactive switching [9], monkeys were trained to perform one of two different arm movements, either pushing or turning a handle, in response to a movement trigger signal. Choosing a correct movement was rewarded and the correct movement remained unchanged in a block of trials, so that monkeys kept selecting the same movement. After a variable number of constant-reward trials, the amount of the reward decreased by 30 percent for each subsequent correct trial. At this stage monkeys were free to switch to the alternate movement. Once they did, the alternate movement was defined as the correct movement, and the reward reverted to the full amount. Thus, monkeys voluntarily selected one of the two movements based on the reduced amount of reward. An analysis of ACC neurons revealed that neuronal activity increased during the interval between the receipt of reduced reward and the switch to the alternate movement (Figure 1, middle). Notably, no such activation was observed when the monkey was given the full amount of reward in constant-reward trials (Figure 1, top) or when the reward was reduced but the monkey failed to switch to the alternate movement (Figure 1, bottom). Most importantly, chemical inactivation of the ACC impaired switching of movements based on the reduced amount of reward. These data indicate a crucial role for the ACC in retroactive behavioral switching. Similar activity properties were later found in the human ACC [82].

**Figure 1.** Retroactive switching by ACC neurons. Activity of a representative ACC neuron recorded while the monkey selected one of two movements (pushing or turning a handle) based on reduced reward. Top: The neuron was not very active after ordinary reward and the monkey continued to select the same movement. Middle: The same neuron increased discharges after the receipt of reduced reward and before the initiation of the alternate movement. Bottom: The neuron remained inactive when the monkey did not switch to the alternate movement despite a reduction of reward.

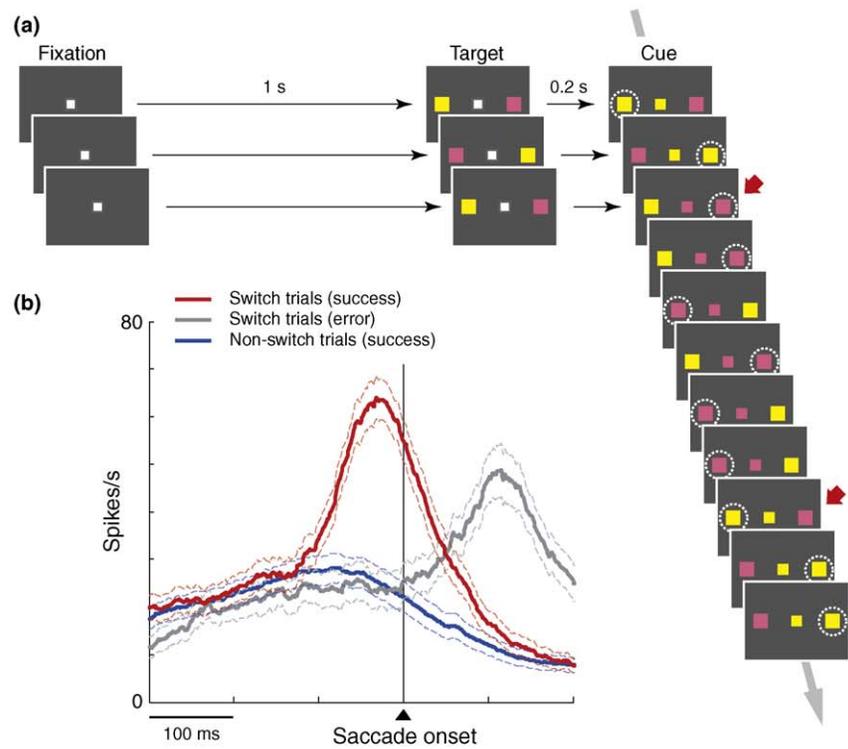


### Box 2. Electrophysiological evidence for the role of the pre-SMA in proactive switching

To study the neural mechanisms of proactive switching, Isoda and Hikosaka devised an oculomotor switching task (Figure 1a) [46]. The task can be viewed as a change-signal task in which, immediately before the subject is about to perform the prepotent response based on the previous cue, a different cue is presented [44]. Unlike most of the change-signal tasks, the prepotency is created internally by repeating the same response. This is the hallmark of automaticity or habit formation. Further, there is no special cue for switching.

In the oculomotor switching task the monkeys developed automaticity and showed a clear switch cost which was expressed as an increased rate of errors and increased reaction times [46]. On switch

trials they tended to make a prepotent but wrong saccade especially when the saccade occurred earlier than a latency which we called 'behavioral differentiation time'. Many pre-SMA neurons were activated on switch trials, but not on non-switch trials (Figure 1b). Importantly, the onset of the switch-selective activity preceded the behavioral differentiation time when switching occurred correctly. When the monkey failed to switch, the pre-SMA neurons did become active, but after the wrong saccade (Figure 1b). When the pre-SMA neuronal activity was boosted with electrical stimulation before the behavioral differentiation time, the success rate of switching increased.



**Figure 1.** Proactive switching by pre-SMA neurons. **(a)** Oculomotor switching task [46]. Each trial began with the onset of a white fixation point followed by the presentation of two stimuli on each side of the fixation point in two different colors. The positions of the pink and yellow stimuli were randomized out of two possible locations. After a short delay, the fixation point became either pink or yellow as a cue, instructing the monkey to make a saccade to the stimulus whose color was the same as the central cue. The central cue color remained unchanged in a block of 1–10 consecutive trials and then was switched in the next block. For simplicity, display panels demonstrating the onset of fixation point (Fixation) and two peripheral stimuli (Target) are illustrated only for the first three trials. White dotted circles, which were not shown to the monkeys during the actual experiments, indicate the correct saccade target. Red arrows indicate switch trials. **(b)** The population activity of switch-selective pre-SMA neurons for successful switch trials (red), erroneous switch trials (gray) and successful non-switch trials (blue).

experiments whether the pre-SMA can act rapidly enough to enable proactive switching under the time constraint described above. It is also unclear how the pre-SMA might enable switching.

In a recent study using an oculomotor switching task Isoda and Hikosaka presented evidence that the pre-SMA competes with automatic processes to enable behavioral switching (Box 2) [46]. Confirming the above prediction, switch-related pre-SMA neurons are activated transiently at the time of switching. It was also shown that pre-SMA neurons, as a population, perform the two operations hypothesized above: suppression of the old procedure and facilitation of the new procedure. Switching is successful if the activation of pre-SMA neurons precedes the initiation of the automatic process; switching fails if the

initiation of the automatic process precedes the activation of pre-SMA neurons.

#### The LPFC and rule implementation

Another cortical area that is thought to be essential for behavioral switching is the LPFC [47]. Subjects with prefrontal lesions show impairments in switching behaviors [48–50] or in inhibiting prepotent responses [51,52]. Similar to the pre-SMA, the LPFC is activated when response inhibition is required [36,53]. Other studies support the view that the LPFC is predominantly active when relevant rules are retrieved, maintained and implemented [13,54]. Strong activation of the LPFC occurs when the rules are complex and require changes in stimulus–response relationships in multiple dimensions, as typically

seen in the Wisconsin Card Sorting Task (WCST) [55,56]. Rule-selective activity is also found in single neurons in the monkey LPFC [57]. Switching between complex tasks requires reconfiguration of cognitive processes, and this might be done by changes in functional connectivity among frontal cortical areas [5,58,59].

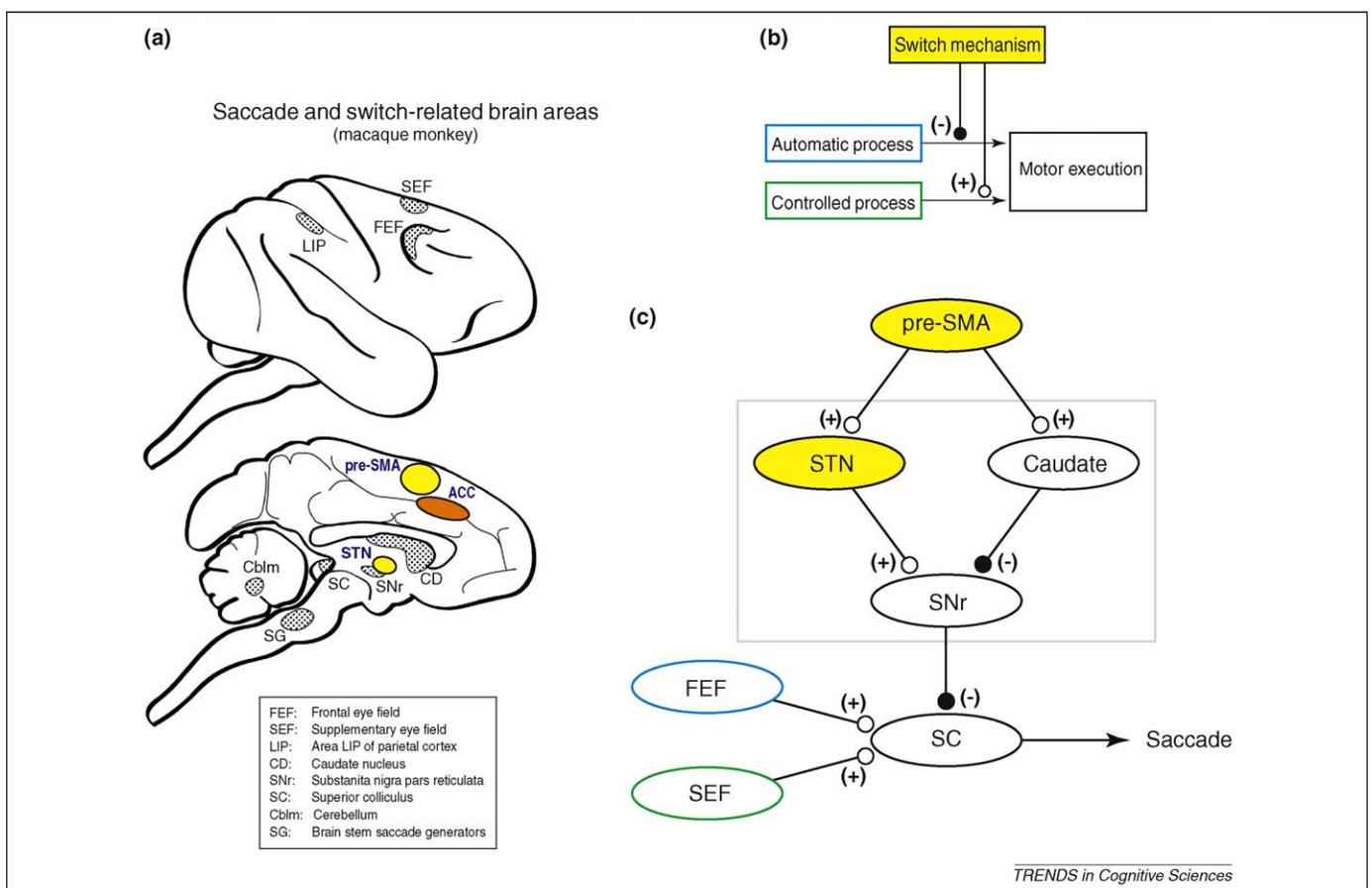
The task rules, which are presumably represented in different regions in the LPFC, need to be executed as motor outputs. Each sub-region in the LPFC can select a correct motor response by inhibiting an incorrect response because neurons specialized for a particular dimension (e.g. color), which are clustered in the LPFC, respond to WCST stimuli selectively when no-go responses are required [60]. Part of the LPFC is characterized as a negative motor area (i.e. stimulation of a cortical area which suppresses voluntary movements), along with the pre-SMA [34]. Thus, it is possible that the LPFC has a mechanism to inhibit motor behavior, but in a selective manner to choose the right behavior. The selection-related inhibition could constitute the LPFC activation during

response inhibition described above. Connections to the striatum might mediate such selective inhibitions as well as disinhibitions [61].

### Cortico-basal ganglia mechanisms and behavioral switching

The outcome of behavioral switching is a change in motor behavior. A crucial aspect of behavioral switching, as we have suggested above, is the suppression of prepotent body movements. This is particularly clear for proactive switching, but is also true for retroactive switching in which performance often becomes slower after an erroneous trial [62,63].

One possibility is that the switch-related cortical signals are mediated by an area that has a powerful capacity to inhibit motor areas. A candidate is the basal ganglia because their final outputs are exclusively inhibitory and are directed to a wide variety of motor structures including the cerebral cortex through the thalamus [64]. The basal ganglia contain parallel circuits which are capable of removing inhibition (direct pathway) or enhan-



**Figure 2.** Neural mechanism of proactive switching in oculomotor behavior. A neural mechanism of behavioral switching must be able to: (1) detect a change in the context; (2) suppress the prepotent, automatic process; and (3) facilitate the alternative, controlled process (b). The suppression must occur quickly because the automatic process emits a motor signal quickly; the facilitation can occur thereafter because the controlled process is slow. Recent studies have indicated that the pre-SMA, together with other frontal cortical areas, acts as a switch mechanism and the basal ganglia might mediate the switch-related signal from the cortical areas (a). In our study using saccadic eye movement, many neurons in the pre-SMA became active selectively and proactively on switch trials (Box 2). It was also shown, using a go-nogo task, that some pre-SMA neurons suppress the prepotent saccade, and the rest have both functions (c). The suppressive pre-SMA neurons tended to be active earlier than the facilitatory pre-SMA neurons, consistent with the conceptual scheme in (b). In the basal ganglia, the STN might serve to suppress the automatic saccade by enhancing the inhibitory output of the basal ganglia (SNr) on the SC or the thalamo-cortical network. The caudate nucleus might serve to facilitate the controlled saccade by disinhibiting the target of the basal ganglia. We speculate that the signals for the automatic and controlled saccades are carried mainly by the frontal eye field (FEF) and the supplementary eye field (SEF) respectively. In the possible neural network in (c), excitatory and inhibitory connections are indicated by (+) and (-) respectively.

cing inhibition (indirect and hyperdirect pathway) [65]. Most cortical areas, including the pre-SMA, ACC and LPFC, project to the striatum and the subthalamic nucleus (STN), both being input zones of the basal ganglia [66]. These anatomical features suggest that the basal ganglia are instrumental for selecting appropriate motor behaviors [17].

The function of the basal ganglia is heavily dependent on dopamine, as evidenced in Parkinson's disease. It has been shown that people with Parkinson's disease have difficulty in changing motor or cognitive behaviors [67] and that dopaminergic medication remediates impairments in switching between tasks [68]. The contribution of the basal ganglia in behavioral switching is also shown in human subjects without dopamine deficits. Subjects performing switching tasks show activations in the striatum [69–71] and the STN [36,72], or both [73]. There is a tendency for switching that is based on abstract rules to be associated with striatal activations, whereas switching relying on suppression of a prepotent response is associated with STN activations [73]. Using a stop-signal task, Aron and colleagues found that stopping a prepotent motor response activated the inferior frontal cortex (IFC), pre-SMA and STN [36], which were shown to be connected with each other using diffusion-weighted imaging tractography [72]. Recent studies by Li and colleagues suggest that the IFC is involved in orienting attention to a salient event (i.e. stop process), whereas the pre-SMA is more specialized for mediating response inhibition via the STN and caudate nucleus [74,75].

When monkeys perform the oculomotor switching task, a group of STN neurons show a switch-selective activity change (mostly an increase in activity) [76]. The activity is similar to that seen in pre-SMA neurons, but occurs slightly later, consistent with the hypothesis that STN neurons receive the switch-related signal from the pre-SMA. The neurons' actions, assessed with the go-nogo task, are usually suppressive, indicating that the STN works mainly to suppress the old no-longer-valid procedure. This conclusion is consistent with a study on people with Parkinson's disease. Electrical stimulation of the STN in these subjects improved their motor symptoms, but the stimulation interfered with the normal ability to slow down when faced with decision conflict [77].

Because the STN has excitatory connections to the final output neurons in the basal ganglia located in the substantia nigra pars reticulata (SNr) or the globus pallidus internal segment (GPi) (Fig. 2) [65], their phasic activation will lead to a phasic inhibition of motor-related neurons in the basal ganglia-recipient thalamus and subcortical motor-related neurons including those in the superior colliculus (SC). Because signal transmission through the hyperdirect pathway is fast [65], the activity of pre-SMA neurons will be translated into an actual stopping action rapidly. These features fulfill one of the two mechanisms requisite to proactive switching: suppression of the old procedure.

The striatum (caudate or putamen) might also be involved in the execution of behavioral switching. Its output via the direct pathway could be used for the facilitation (disinhibition) of the new procedure (Fig. 2). This

could serve as the other mechanism for proactive switching, facilitation of the new procedure, such as the saccade to a different colored target [46] or the antisaccade [71,78]. On the contrary, the output of the striatum via the indirect pathway might be used for the suppression of the old procedure or the task rule-related inhibition of motor outputs.

### Concluding remarks

When the circumstances necessitate it, we make the important decision to change our behavior by breaking a routine. Recent studies with human and non-human primate subjects have begun to elucidate the neural mechanisms underlying such behavioral switching. These studies support the view that different areas in the medial and lateral frontal cortices play executive roles in behavioral switching and do so using different algorithms.

What triggers behavioral switching represents one aspect of the switching algorithm. Switching might occur retroactively based on error feedback indicating that the current behavior is no longer appropriate. A critical structure for this retroactive switching is the ACC. In many cases, however, there is a sensory cue that predicts a change in the context. The subject can use the cue to switch behaviors proactively so that failure can be avoided. Such proactive switching is mainly governed by the pre-SMA. Importantly, the subject might not be aware of the presence of the cue initially, but can learn the meaning of the cue with experience.

Another aspect of the switching algorithm arises if the task rule changes before and after switching. In this case, cognitive processes need to be reconfigured to accommodate the rule change. Such cognitive reconfiguration seems to be performed by changes in functional connectivity among frontal cortical areas, including the LPFC. Even if the ACC or pre-SMA sends signals for switching, the switching would not be accomplished if the new rule has not been implemented (e.g. due to malfunction of the LPFC).

However, it is debatable whether each of the ACC, pre-SMA, and LPFC performs an exclusive function as described above and is thus requisite for a certain type of behavioral switching. In fact, a lesion in each area might not lead to impairment in switching. Instead, these prefrontal cortical regions could constitute a large network in which different switching algorithms are computed differentially but in an overlapping manner.

These switching algorithms need to be executed by selecting an appropriate motor behavior. The basal ganglia are considered to be a major mediator of the switch execution signals. In particular, the STN receives the switch-related signal from the pre-SMA and suppresses the ongoing but no-longer-valid behavior so that the new behavior can be executed. The striatum might also contribute to switching based on its input from the frontal cortical areas. Parallel neural circuits in the basal ganglia (direct, indirect and hyperdirect pathways) might underlie these neural operations through which a valid behavior can be selected and invalid behaviors suppressed.

However, behavioral switching is only part of what animals would do to adapt to changing worlds. Changing

**Box 3. Outstanding questions****Is the ACC necessary for retroactive switching?**

We have proposed that the ACC is essential for retroactive switching. However, unlike a reversible inactivation study [9], recent lesion studies indicate that retroactive switching *per se* is impaired neither by ACC lesions [29] nor by lesions in different parts of the LPFC or the orbitofrontal cortex [30]. This raises the possibility that, although the ACC is necessary for retroactive switching in the intact animal, other brain areas take over after ACC lesion and enable switching.

**What are the roles of neuromodulators in behavioral switching?**

The brain areas related to behavioral switching, especially the ACC and pre-SMA, receive substantial dopaminergic inputs from the ventral tegmental area and the substantia nigra [83]. Because some dopamine neurons carry reward-related value signals [84,85], it is plausible that dopamine in the medial frontal cortex is essential for behavioral switching [2]. Experimental evidence in support of this hypothesis is currently lacking, however. These medial frontal cortical areas are also mutually connected with the locus coeruleus, which is a major source of noradrenergic signals. Because the locus coeruleus is thought to regulate the balance between exploration and exploitation [86], it might also be related to behavioral switching.

**How is behavioral switching related to reward-based learning?**

A dominant theory proposes that reward-based learning is based on plasticity in corticostriatal synapses which are conditioned by dopaminergic inputs [87]. However, because the animal experiences two alternating task conditions repeatedly, reward-based changes in behavior tend to become faster [88]. It is thus likely that all reward-based change in behavior involves both striatum-based plasticity and medial frontal cortex-based switching. The transition of the dopamine neuron's response from the reward outcome to a predictive cue [84] might be related to the hypothetical transition from retroactive switching to proactive switching.

**How important is behavioral switching in social contexts?**

Behavioral switching might be particularly important in social contexts: an animal (or human) is surrounded by many animals (or humans) that have different behavioral traits. It is then crucial to switch behaviors in anticipation of (rather than in response to) the other individual's behavior. Facial expressions, gestures, vocalization and gaze direction can provide many cues for switching, which the animal might need to learn to enable proactive switching [7].

behavior gradually, based on reward outcome, is another important type of behavioral adaptation. It is still unclear, however, whether rapid adaptation (i.e. switching) and slow adaptation (i.e. reward-based changes) are controlled by the same or different brain networks (see also Box 3).

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