Beta Oscillations in Working Memory, Executive Control of Movement and Thought, and Sensorimotor Function

R Schmidt[1], M Herrojo Ruiz[2,3], BE Kilavik[4], M Lundqvist[5], P Starr[6], AR Aron[7]

1. Department of Psychology, University of Sheffield
2. Department of Psychology, Goldsmiths University of London
3. Center for Cognition and Decision Making, Institute for Cognitive Neuroscience, National Research University Higher School of Economics, Moscow, Russian Federation
4. Institut de Neurosciences de la Timone (INT), UMR 7289, CNRS, Aix-Marseille Université, Marseille, France
5. The Picower Institute for Learning and Memory, Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology
6. Department of Neurosurgery, University of California San Francisco
7. Department of Psychology, University of California San Diego

Correspondence:
Robert Schmidt
Department of Psychology
University of Sheffield
robert.schmidt@sheffield.ac.uk
or
Adam R Aron
Department of Psychology
University of California San Diego
adamaron@ucsd.edu

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Abstract

Beta oscillations (~13 to 30Hz) have been observed during many perceptual, cognitive and motor processes in a plethora of brain recording studies. While the function of beta oscillations (hereafter 'beta' for short) is unlikely to be explained by any single monolithic description, we here discuss several convergent findings. In prefrontal cortex, increased beta appears at the end of a trial when working memory information needs to be erased. A similar clear-out function might apply during the stopping of action and the stopping of long-term memory retrieval (stopping thoughts), where increased prefrontal beta is also observed. A different apparent role for beta in prefrontal cortex occurs during the delay period of working memory tasks: it might serve to maintain the current contents and/or to prevent interference from distraction. We confront the challenge of relating these observations to the large literature on beta recorded from sensorimotor cortex. Potentially, the clear-out of working memory in prefrontal cortex has its counterpart in the post-movement clear-out of the motor plan in sensorimotor cortex. However, recent studies support alternative interpretations. In addition, we flag emerging research on different frequencies of beta and the relationship between beta and single neuron spiking. We also discuss where beta might be generated: basal ganglia, cortex, or both. We end by considering the clinical implications for adaptive deep brain stimulation.

Introduction

Since the first descriptions of sensorimotor rhythms (Berger, 1929) many researchers have pondered the functional role of beta (~13-30Hz). These oscillations are often prevalent during stable postures and rare during movement, and some researchers have proposed that they indicate a brain state of 'neuronal activity equilibrium', or alternatively, at a more functional level, a state of 'status quo' or akinesis (e.g. Jasper and Penfield, 1949; Engel and Fries, 2010; Khanna and Carmena, 2017). These neural and functional descriptions fit well with the exaggeration of beta in Parkinson's disease, with its symptoms of rigidity and slow movement (Hammond et al., 2007). However, several experimental findings do not seem readily compatible with these ideas. This has led to proposals that sensorimotor beta also has a functional role in sensorimotor integration, temporal anticipation, and confidence in expectations (Kilavik et al., 2013; Torrecillos et al., 2015; Tan et al., 2016). Furthermore, beta is also observed *outside* of the sensorimotor system. For example, beta occurs in prefrontal cortex (PFC) during executive control of action (Swann et al., 2009; Ruiz et al., 2011; Wessel et al., 2013), working memory (Lundqvist et al., 2016; Miller et al., 2018) and preventing distraction (Hanslmayr et al., 2014; Zavala et al., 2017); and they increase in the basal ganglia in relation to sensory cues (Leventhal et al., 2012) and the encoding of sequence boundaries (Herrojo Ruiz et al., 2014). In this article, we address similarities across studies, aiming towards the larger goal of integrating these observations under a common rubric for beta.

Beta is observed using scalp electroencephalography, magnetoencephalograpy, intracranial electrocorticography, and local field potentials (LFPs). While most studies have averaged beta power across trials (producing so-called event-related beta synchronizations, or desynchronizations, compared to a baseline period), recent studies have focused on beta 'bursts' in single trials (Leventhal et al., 2012; Feingold et al., 2015; Lundqvist et al., 2016; Shin et al., 2017; Tinkhauser et al., 2017). The analysis of bursts reveals a rich dynamics of timing, duration, and other features. Below, we will discuss results from averaged power and also from single trial analysis, including bursts.

We start this review by considering the role of beta in PFC, in both retaining and clearing working memory. We then draw a connection to the suppression of movement and thought. Next, we discuss how these prefrontal and basal ganglia roles of beta relate to the well-described sensorimotor beta. We then consider how beta may be generated in the cortex and basal ganglia. We end by considering the clinical implications, especially for real-time adaptive brain stimulation.

Prefrontal beta for controlling contents of working memory

While beta has been widely studied for movement, recent findings also suggest a role in cognitive functions such as working memory (Lundqvist et al., 2016; Lundqvist et al., 2018). For example, recent studies recorded PFC activity in monkeys performing a delayed match-to-sample task, in which several objects had to be encoded, maintained, and tested sequentially, over several seconds (Lundqvist et al., 2016). During encoding, brief gamma bursts were associated with spiking activity

while beta bursts were reduced. Then, in the following delay period, beta was increased, except at the very end, when information was needed again. At that point, beta was reduced and gamma increased. Since working memory tasks typically involve a motor component (a saccade) to make the choice, this beta and gamma modulation before the test could in principle be related to movement and not cognitive aspects. However, in a follow-up study, the tests and responses were dissociated (Lundqvist et al., 2018). The observed suppression patterns of beta, and the selective upregulation of spike information about the object needed for a particular test, were consistent with a role in the flexible control of working memory rather than anticipation of movement. The pattern of beta changes is shown in **Figure 1A**. Overall, beta was reduced during encoding and test epochs, intermediate during delays, and strongly elevated after the response.

We speculate that the intermediate and strong beta increases have different functional roles. The intermediate elevation of beta during the delay period relative to the low levels seen at encoding and read-out may serve to protect the current working memory contents from interference. Indeed, human studies have shown increases of prefrontal beta when subjects must filter out distractors (Zavala et al., 2017) or prevent encoding (Hanslmayr et al., 2014). In contrast, the strong level of beta at the end of the trial might reflect a 'clear out' of the working memory content. It's noteworthy that this 'beta rebound' clear-out in PFC was specific to recording sites that carried working memory information during the trial (Lundqvist et al., 2018); i.e. it was not merely motor-related. This opens up the intriguing possibility that, in sensorimotor cortex, the so-called post-movement beta rebound could serve a similar function for motor plans (discussed below). Overall, these studies suggest that beta bursting, originating in deep layers of PFC (Bastos et al., 2018), might explain how information is regulated during encoding, retention, read-out, and working memory reallocation (Lundqvist et al., 2018; Miller et al., 2018).

Non-invasive human studies have also provided evidence for an inhibitory role of alpha/beta oscillations in working memory (Jokisch and Jensen, 2007; Tuladhar et al., 2007). These signals were observed primarily in sensory cortex, in a lower frequency range (8-16 Hz) and are thought to reflect the inhibition of task-irrelevant areas. This led to speculation that these large scale (electroencephalography-level findings) of alpha/beta-inhibition are analogous to the fine-scale beta inhibition discussed above (Miller et al., 2018). In summary, these findings suggest that beta acts as an inhibitory filter throughout cortex, predicting when and where the contents of working memory are expressed. They also suggest possible functional similarities between cognitive and motor beta.

Prefrontal–basal-ganglia beta for stopping action and thought

As described above, beta occurred at an intermediate level in PFC during the delay period of working memory tasks, possibly to protect against interference, whereas beta occurred at high levels at the end of the trial possibly related to 'clear out' of the working memory content. While those data were from

monkeys during various tasks requiring control over working memory, striking parallels in prefrontal beta are seen in human tasks requiring executive control over movement and thought.

Executive control over movement can be studied with the stop-signal task (Verbruggen et al., 2019). On each trial, the subject initiates a motor response; in a minority of trials, the subject has to try to stop the movement when a subsequent stop signal occurs. A critical prefrontal region for stopping is the right inferior frontal gyrus (reviewed by Aron et al., 2014). Intracranial electroencephalography showed that, after the stop signal, and within a few hundred milliseconds, there was an increase in right inferior frontal beta on successful stop trials (Swann et al., 2009; Wessel et al., 2013), **Figure 1B**. A similar pattern of increased beta has been shown in several scalp electroencephalography studies (Wagner et al., 2018; Castiglione et al., 2019). The wider network for rapidly stopping action is thought to include a hyperdirect pathway from the PFC to the subthalamic nucleus (STN) of the basal ganglia (reviewed in Wessel and Aron, 2017). Consistent with this, some studies of STN LFPs during stop-signal response inhibition have revealed a relative increase of beta-band power on successful stop trials, within approximately the same time frame as for the right inferior frontal gyrus (reviewed in Zavala et al., 2015; Aron et al., 2016) **Figure 1C**. Further, deep brain stimulation of the STN in patients with Parkinson's disease led to a relative increase in right frontal beta when stopping action (Swann et al., 2011). Taken together, these results suggest that increased frontal and subthalamic beta reflect a network signature of the stopping process, although how communication occurs is unclear. Further, because the beta increase after the stop signal is strongly above baseline, we suppose prefrontal beta during stopping is more akin to the 'clear out' mode rather than protecting against interference, although this remains to be established.

Stopping might extend from movement to thought, which can be studied with the Think/No-Think paradigm (Anderson and Green, 2001). In the first phase, participants learn cue-target word pairs such as 'oil'-'pump'. In the second phase, Think/No-Think, they are sometimes asked to stop the retrieval process. They perform trials in which they receive the reminder word from one of the studied pairs (e.g. 'oil'), presented either in green (cuing them to think of the associated word) or in red (cuing them to stop retrieval); and they are probed, at the end of each trial, regarding whether they experienced an intrusion of the associated memory into awareness (Levy and Anderson, 2012). A recent scalp electroencephalography study showed that, just as for movement-stopping mentioned above, there was an increase in right frontal beta during No-Think trials (Castiglione et al., 2019). Strikingly, this early right frontal beta effect (beginning ~300 ms after the No-Think cue) was more pronounced during No-Think trials in which retrieval was successfully stopped (i.e., there was no intrusion). These results indicate that the beta increases for successful movement-stopping and NoThink trials have a common function.

How could this putative prefrontal stopping system affect the retrieval of long-term memories? Above we saw that prefrontal beta is implicated in the control of working memory contents, including clear-out. Applying this view to the processes engaged on No-Think trials, we

suppose that pattern completion begins for the target word via the medial temporal lobe, but this has to then trigger reinstatement in neocortex to achieve recollection, perhaps via basal ganglia (Scimeca and Badre, 2012; Chatham and Badre, 2015). The stopping process on NoThink trials, reflected in increased right frontal beta, may interfere with this latter reinstatement aspect of retrieval (also see Michelmann et al., 2016), perhaps also via basal ganglia.

A different form of stopping might be involved in the interruption of *ongoing* thought (rather than preventing long-term memory retrieval), for example when an unexpected event occurs. Because unexpected events increase beta in right frontal areas (Wagner et al., 2018) and the STN (Wessel et al., 2016), it has been proposed that unexpected events recruit a frontal-STN stopping system to interrupt working memory (Wessel and Aron, 2017).

In summary, a right frontal beta increase is associated with engagement of the stopping system for movement and also for long-term memory retrieval. It also occurs with unexpected events, which can interrupt working memory. The functional role of beta in these scenarios is perhaps most compatible with clear out. We next consider how these putative beta functions of protecting against interference and clear-out compare to beta in sensorimotor cortex.

Sensorimotor beta: amplitude, frequency and beta bursts

Beta in sensorimotor cortex has been characterized in more detail in terms of frequency and amplitude changes than in PFC and basal ganglia. Decades of research show that sensorimotor beta increases at rest and for stable postures, is reduced during movement, and re-emerges prominently following movement or even completion of imaginary movements (reviewed by Kilavik et al., 2013) and also even after a passive movement (Cassim et al., 2001). For example, one study showed increased beta in both pre-cue and pre-go epochs of movement tasks, with a temporary drop in beta amplitude following the cue (Kilavik et al., 2012). This post-cue amplitude drop mainly occurs for cues containing information relevant for movement planning, and parallels the decreased beta burst probability in PFC during stimulus encoding in working memory tasks (Figure 1D). However, it remains unclear whether the increased beta amplitude in pre-cue and pre-go epochs are in some way functionally analogous to the prefrontal beta described above in reflecting, for example, protection of the posture or motor plan.

The beta rebound following movement has been linked to inhibitory GABAergic activity (reviewed by Kilavik et al., 2013) and has been interpreted as an implementation of resetting mechanisms that prepare the cortical networks for the execution of upcoming movements (Pfurtscheller et al., 2005). This could align well with the putative clear-out function of beta in working memory. On the other hand, recent studies reveal a multifaceted picture. We start by considering the relationship between beta and single-unit spiking, then we show how sensorimotor beta may have different bands with different functions, and we end with new findings on how single trial burst parameters relate to different aspects of movement.

In order to compare beta modulations across different studies, it is important to first understand the underlying relationship between the LFP and neuronal spiking activity. Many studies have shown that sensorimotor LFP beta at least partly reflects local activity, with the spikes of inhibitory interneurons and pyramidal tract neurons locking to the phase of beta (Murthy and Fetz, 1996; Donoghue JP, 1998; Baker et al., 1999 ; Jackson et al., 2002; Denker et al., 2011; Canolty et al., 2012; Confais et al., 2019). However, whether there is also an intrinsic relationship between the amplitude of beta oscillations and neuronal spike rates has been controversial (Canolty et al., 2012; Rule et al., 2017). A recent study resolved this issue (Confais et al., 2019), by showing that spike rates and beta amplitude have no intrinsic correlation, but are both modulated by external factors, such as a behavioral task.

A different issue is that the term 'beta' is broad and actually involves several types of oscillations in distinct frequency bands (Kopell et al., 2011). First, in parkinsonian rats, slow and fast beta seem to take different routes through cortical and basal ganglia circuits (West et al., 2018) and see for human evidence: (Lopez-Azcarate et al., 2010). Second, in the human, some evidence suggests beta frequency is effector specific, with frequencies >20 Hz associated with lower limbs and frequencies <20 Hz with upper limbs (Pfurtscheller et al., 2000; Neuper and Pfurtscheller, 2001). Third, in the macaque monkey, two beta bands, at ~20 and ~30 Hz, are present in motor cortical LFPs (Kilavik et al., 2012), and phase-locking analysis of neuronal spiking activity suggest both bands have at least partly a local origin within motor cortex (Confais et al., 2019). Whereas those particular studies found similar modulations of both bands with behavioral context and movement direction, other work found that pre-stimulus beta frequencies <20Hz were positively correlated with reaction times, while higher beta frequencies (>20 Hz) were negatively correlated (Zhang et al., 2008; Chandrasekaran et al., 2019). One interpretation is that lower beta (<20 Hz) is ‘anti-kinetic’ (Engel and Fries, 2010), while higher beta band (>20 Hz) reflects attention and anticipation (Saleh et al., 2010; Fujioka et al., 2012; Kilavik et al., 2012; Kilavik et al., 2014).

Finally, in addition to amplitude and frequency changes in beta, the duration, distribution and onset of beta bursts influences different properties of the movement. It was suggested that changes in beta bursts before movement was related to ‘specifying the movement goal’ while fewer bursts and later bursts after an error were related to ‘error evaluation and monitoring’ (Little et al., 2018). However, those results are perhaps also compatible with a ‘protection of the current state’ function before movement (also see Shin et al., 2017) and, after movement error, a reduced and delayed ‘clear-out’ to ‘buy time’ to learn. We note, however, that Torrecillos et al (2015) showed reduced post-movement beta power also for errors that do not induce motor adaptation, suggesting the reduced beta power instead reflects the saliency of the error, irrespective of whether the motor plan should be preserved or updated. Other recent findings are from reward-dependent motor learning (Sporn et al., 2018). That study showed that a phasic, post-reward, increase in the rate of long beta bursts (duration > 500 ms) attenuated the update in predictions about the rewarded movement goal (also see Tan et al.,

2016). While further work is needed to integrate these new proposals for post-movement and post-feedback sensorimotor beta, these studies highlight the usefulness of analyzing features such as duration, rate, and timing of beta bursts to better understand sensorimotor function.

In summary, while some aspects of sensorimotor beta might be compatible with protection of motor contents or posture and with clear-out, the picture is complicated. Recent insights into sensorimotor beta suggest that 1) an intrinsic relationship between beta in the LFP and spikes is only present for phase-locking, not amplitude correlations, 2) there are multiple beta bands at different frequencies: these might relate to different limbs, beta frequency changes within trials, and possibly beta has different functional roles (akinetic, attention, sensorimotor integration, and updating motor predictions), and finally, 3) the parameters of beta bursts, such as the duration, distribution, and timing onset, relate to motor performance and learning in quite complex ways that we are just beginning to probe.

Mechanisms of generating beta: basal ganglia and cortex

Executive control, as exemplified in the context of stopping movement described above, employs beta in the cortex as well as in the basal ganglia. We start this section by considering how beta might be generated in basal ganglia.

The basal ganglia are composed of the striatum, the globus pallidus interna (GPi) and externa (GPe), the STN and the substantia nigra. Beta is present in all subregions of the basal ganglia and is modulated during the processing of sensory cues and motor signals (Leventhal et al., 2012; Herrojo Ruiz et al., 2014). As in the sensorimotor cortex and PFC, basal ganglia beta occurs in healthy animals in brief bursts, and changes in beta power typically reflect changes in the probability of beta bursts.

Much evidence has implicated the STN-GPe network in the generation of beta (Hammond et al., 2007; Mallet et al., 2008). Computational modelling has demonstrated that beta can be generated in the STN-GPe network when the inhibitory input to GPe, or the excitatory input to STN, is increased (Kumar et al., 2011). Changes in the inhibitory input to GPe (from striatal medium spiny neurons) also occur in awake behaving animals during movement (Cui et al., 2013). Furthermore, excitatory inputs to STN include cortical and subcortical areas, providing motor and sensory inputs (Parent and Hazrati, 1995). Therefore, the sensory and motor signals that are processed in the striatum, GPe, and STN might be related to the generation of beta in the STN-GPe networks.

A recent study (Mirzaei et al., 2017) tested whether this computational model for beta generation applies in awake behaving animals. This was done by first generating artificial activity patterns, mimicking single-unit activity recorded in the striatum, GPe and STN of rats performing a cued choice task (Schmidt et al., 2013; Mallet et al., 2016). Second, these activity patterns were used as inputs to a spiking model of the STN-GPe network (Kumar et al., 2011). Intriguingly, the computational model generated transient beta, modulated by sensory and motor events in a way

strikingly similar to that in rats performing the task. It even accounted for the positive correlation of beta with reaction times (Leventhal et al., 2012), providing a potential neural mechanism for the akinetic aspect of beta. More generally, the model demonstrated how brief changes in firing rate of the inputs to GPe and STN could lead to beta bursts. An open question is whether beta in the GPe-STN network is coordinated with cortical beta. For example, basal ganglia beta could potentially propagate to the cortex, or an independent generation of cortical beta could enable a "communication through coherence" between cortex and basal ganglia (Fries, 2015).

We now consider how cortical beta could be generated. Several lines of evidence point to cortical deep layers as a source of beta (Bollimunta et al., 2008; Buffalo et al., 2011), and possibly implicate a local circuit involving pyramidal cells and fast-spiking interneurons (via the so-called "PING" mechanism) (Miller et al., 2018). Alternatively, interactions between excitatory and inhibitory neurons in deep and superficial layers might create beta oscillations (Sherman et al., 2016; Spitzer and Haegens, 2017), (also see Kopell et al., 2011). Strong excitation, e.g. from the mediodorsal thalamus (Ketz et al., 2015) to the deep layers, could lead to the generation of beta there, also in the absence of sensory inputs as required for working memory (Miller et al., 2018). Interestingly, the cortical deep layers are connected to the basal ganglia via projections to STN (Rouzaire-Dubois and Scarnati, 1985) and via thalamocortical loops (McFarland and Haber, 2002); this might be a circuit for coordinating or propagating beta between cortex and the basal ganglia (**Figure 2**).

The coordination of cortical and basal ganglia beta might orchestrate cognition and movement. One option is 'top-down' communication, in which beta is generated in the cortex and then propagates to the basal ganglia. This might reflect a situation in which cortical circuits use beta to maintain stimulus information in working memory (see above), and exert control on subcortical structures to protect them against interference. In contrast, 'bottom-up' communication could potentially generate beta in the STN-GPe network due to (non-oscillating) sensory and motor inputs, including ramping activity in STN (Mirzaei et al., 2017). This beta could affect reaction times and propagate through cortex via the mediodorsal thalamus. Finally, beta could be generated in the cortex and in the basal ganglia separately, perhaps relying on a shared input signal, e.g. increased excitation from the mediodorsal thalamus, to both areas (**Figure 2**). This might open a privileged communication channel between cortex and basal ganglia (Fries, 2015), so that spiking activity related to working memory or stopping can be processed across these circuits. Even though this remains speculative at this point, some evidence for bidirectional communication involving beta in cortical and basal ganglia circuits has been found in humans with Parkinson's disease (Lalo et al., 2008).

In summary, the interaction between STN and GPe can generate transient beta bursts observed in the BG of healthy, awake behaving rats. We do not yet know how these transient beta bursts in the BG are coordinated with cortical beta in executive function.

Using the beta signature in clinical medicine

Improving our understanding of the mechanisms and function of beta has direct clinical implications, especially for Parkinson's disease in which there is abnormally increased beta synchronization throughout the motor network. Indeed, the aim of clinical interventions is to reduce or prevent pathological beta. Thus, understanding non-pathological beta is essential to make clinical interventions more precise and reduce potential side effects due to the removal healthy beta.

Manifestations of increased beta synchronization in Parkinson's disease include elevated resting-state beta in LFP recordings from basal ganglia nuclei (STN and GP) (Oswal et al., 2013), alteration of beta burst dynamics in the basal ganglia (Tinkhauser et al., 2017), increased beta coherence between structures of the motor network (Wang et al., 2018), and changes in the relationship between the phase of beta-frequency oscillations and the amplitude of higher-frequency oscillations in basal ganglia (Lopez-Azcarate et al., 2010) and cortex (de Hemptinne et al., 2015; Swann et al., 2015). An important mechanism of deep brain stimulation may be reduction of coherent oscillations between basal ganglia output (Meidahl et al., 2017) and cortex (Wang et al., 2018). Since basal ganglia beta amplitude can index the effectiveness of (levodopa) therapy (Kuhn et al., 2006) or deep brain stimulation (Kuhn et al., 2008), beta amplitude recorded from basal ganglia stimulation electrodes is a promising control signal for adaptive (feedback controlled) deep brain stimulation. However, caution is warranted in using STN beta for adaptive deep brain stimulation, because this signal is affected by normal movement, as well as changes in parkinsonian motor signs (Kuhn et al., 2004), and the site of the maximal beta band activity within STN has connections not only within the motor system, but also with prefrontal areas that may mediate stimulation-induced adverse effects (Accolla et al., 2016). In the cortex, one effect of the parkinsonian state may be to increase beta waveform "sharpness", reflecting abnormally synchronized thalamocortical inputs (Cole et al., 2017). This raises the possibility of using waveform shape, assessed in the time domain, to index the severity of Parkinson's motor signs.

Much of the work on oscillatory phenomena in Parkinson's disease has been done using acute intraoperative recording in patients undergoing deep brain stimulation surgery in the awake state, or from temporarily externalized deep brain stimulation electrodes in the hospital. Yet these recordings happen in an unnatural environment, there is a "microlesion" effect of lead insertion, there are restrictions on subject movement, and there is a limited time window for research. Helpfully, since 2013, investigators have had access to an investigational bidirectional neural interface (Activa PC+S, Medtronic) that both delivers therapeutic stimulation, senses LFPs, and wirelessly streams data to an external computer (Quinn et al., 2015; Swann et al., 2018). A second-generation sensing interface, the Summit RC+S device (Medtronic), was introduced in 2018 and is the first implantable neural interface capable of continuously streaming electrophysiologic data for many hours, at home.

Wireless transmission of data at a distance allows full freedom of movement. Current research is using these devices to record chronic STN LFPs and primary motor cortex electrocorticography potentials in patients with Parkinson's disease, during daily motor fluctuations, and during normal activities such as hiking, driving, and sleeping (Stanslaski et al., 2018).

Chronic recordings have been used to prototype several adaptive deep brain stimulation algorithms using primary motor cortex electrocorticography signals, to deliver different levels of stimulation depending on movement state. One paradigm used motor cortex beta to increase stimulation when patients initiated movement (Herron et al., 2017). Adaptive stimulation may allow delivery of fully therapeutic deep brain stimulation without adverse effects associated with chronic "open loop" (unvarying) stimulation.

Apart from such research on sensorimotor motor beta in Parkinson's disease, future work may also focus on the cognitive aspects of beta. For example, one might predict that the difficulty of switching tasks in such patients with off-medication, and the improvement of switching with on-medication (Cools et al., 2001), relates to changes in clear-out.

Conclusions

Recent recording studies from monkeys motivated the theory that prefrontal beta has two modes: protection and clear-out. In humans, the protective mode is perhaps compatible with studies showing increased prefrontal beta when filtering out distractors or preventing of encoding, while the clear-out mode may occur in relation to the stopping of movement and thoughts (canceling an incipient motor response or long-term-memory retrieval). It remains challenging to connect these possible functional roles of prefrontal beta (protection and clear-out) with the complex beta modulation observed in sensorimotor cortex during a variety of tasks. One specific avenue is to investigate possible similarities between the putative clear-out mechanism for working memory content and the strong post-movement beta rebound in sensorimotor areas, and to relate this to the findings on how feedback and reward are integrated to update movements. Further research on the neural mechanisms that generate beta will also help to address these open questions about the cognitive and motor functions of beta and also, clinically, will help us better distinguish pathological from non-pathological beta.

Figure 1: Schematic illustration of how beta is recruited for different tasks in different brain regions. A. In lateral prefrontal cortex (PFC) of monkeys, a working memory task required encoding two objects, then a test (Lundqvist et al., 2018). Beta decreased during the first encoding, then increased during the delay, decreased during the second encoding, then increased during the delay; finally, beta increased strongly at the end of the trial. Functionally, it was proposed that the strong beta increase at the end of the trial corresponds to 'clear out', while the moderate increase during the delay period mediates 'protection' from interference. B. In the stop signal task, human subjects initiate a button press to a leftward pointing arrow, and then, when it changes color, they have to try to stop. Beta power in right inferior frontal gyrus (rIFG) increases strongly above baseline during the stop (Swann et al., 2009), possibly corresponding to 'clear out'. C. A similar pattern is seen when recording from the subthalamic nucleus of the BG during the stop-signal task (Ray et al., 2009): there is a desynchronization (reduction of power) relative to baseline as the subject initiates movement, but a strong increase with stopping [note however differences in the rodent (Leventhal et al., 2012)]. D. In sensorimotor cortex (SM), in a pre-cued motor task in monkeys, beta amplitude is high prior to the cue and drops temporarily following it, before increasing again towards the Go signal. Beta amplitude is minimal during the movement and then increases at the end of the trial (Kilavik et al., 2012; Kilavik et al., 2013). It is currently unclear how much the pre-frontal 'protection and 'clear out' notions apply to sensorimotor beta.

Figure 2: Schematic illustration of potential mechanisms of beta generation and interaction in thalamocortical–BG circuits. In the cortex (1), beta oscillations can be generated in deep cortical layers, by interactions between pyramidal neurons (triangles) and interneurons (circles), and potentially with neurons in superficial layers (not shown). Transient beta oscillations could be triggered by excitation from the mediodorsal thalamus (MD, black arrows). In the BG, beta oscillations can be generated in the subthalamo-pallidal loop (2) as a result of increased striatal inhibition of GPe (e.g. due to increased input from MD) or increased excitation of STN (relevant pathways marked with grey arrows). Despite local generation in cortex and BG, the resulting beta oscillation could open a communication channel between cortical and BG circuits (3).

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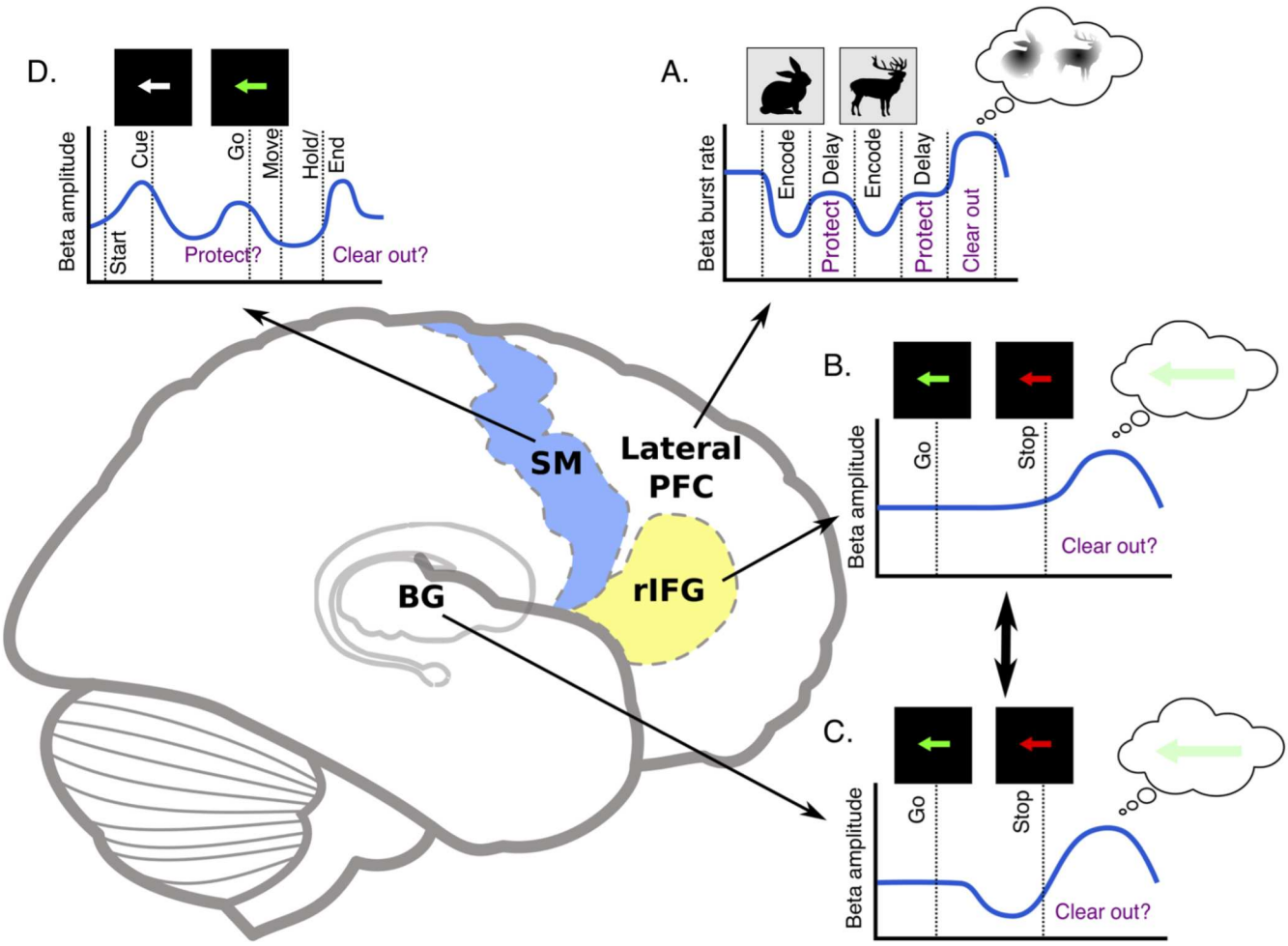
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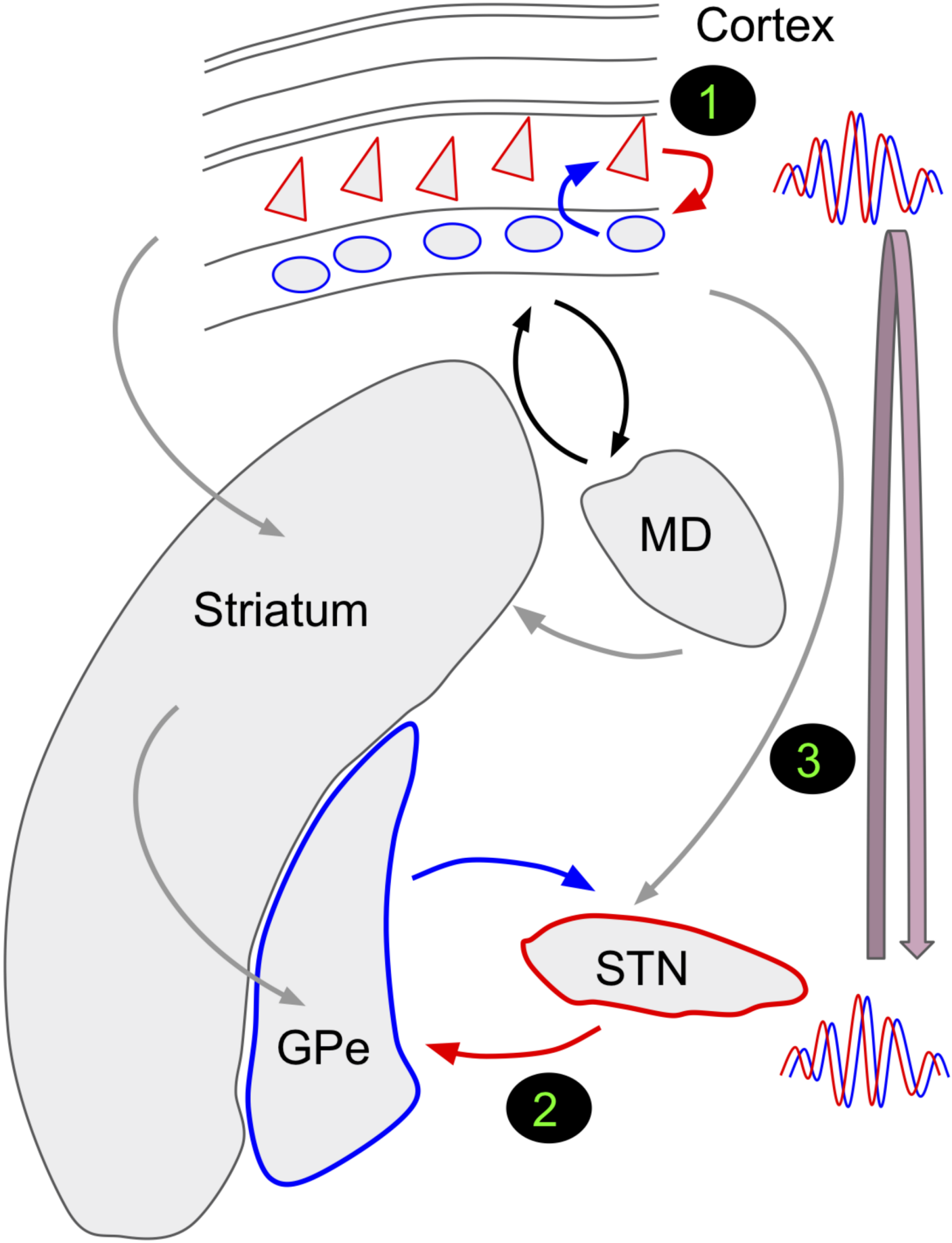
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