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

3  
4                    R Schmidt<sup>1</sup>, M Herrojo Ruiz<sup>2,3</sup>, BE Kilavik<sup>4</sup>, M Lundqvist<sup>5</sup>, P Starr<sup>6</sup>, AR Aron<sup>7</sup>  
5  
6

- 7    1.        Department of Psychology, University of Sheffield  
8    2.        Department of Psychology, Goldsmiths University of London  
9    3.        Center for Cognition and Decision Making, Institute for Cognitive Neuroscience, National  
10        Research University Higher School of Economics, Moscow, Russian Federation  
11    4.        Institut de Neurosciences de la Timone (INT), UMR 7289, CNRS, Aix-Marseille Université,  
12        Marseille, France  
13    5.        The Picower Institute for Learning and Memory, Department of Brain and Cognitive  
14        Sciences, Massachusetts Institute of Technology  
15    6.        Department of Neurosurgery, University of California San Francisco  
16    7.        Department of Psychology, University of California San Diego  
17

18    **Correspondence:**

19    Robert Schmidt  
20    Department of Psychology  
21    University of Sheffield  
22    [robert.schmidt@sheffield.ac.uk](mailto:robert.schmidt@sheffield.ac.uk)

23    or

24    Adam R Aron  
25    Department of Psychology  
26    University of California San Diego  
27    [adamaron@ucsd.edu](mailto:adamaron@ucsd.edu)

28  
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37 **Abstract**

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

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## 56 **Introduction**

57 Since the first descriptions of sensorimotor rhythms (Berger, 1929) many researchers have pondered  
58 the functional role of beta (~13-30Hz). These oscillations are often prevalent during stable postures  
59 and rare during movement, and some researchers have proposed that they indicate a brain state of  
60 ‘neuronal activity equilibrium’, or alternatively, at a more functional level, a state of ‘status quo’ or  
61 akinesia (e.g. Jasper and Penfield, 1949; Engel and Fries, 2010; Khanna and Carmena, 2017). These  
62 neural and functional descriptions fit well with the exaggeration of beta in Parkinson’s disease, with  
63 its symptoms of rigidity and slow movement (Hammond et al., 2007). However, several experimental  
64 findings do not seem readily compatible with these ideas. This has led to proposals that sensorimotor  
65 beta also has a functional role in sensorimotor integration, temporal anticipation, and confidence in  
66 expectations (Kilavik et al., 2013; Torrecillos et al., 2015; Tan et al., 2016). Furthermore, beta is also  
67 observed *outside* of the sensorimotor system. For example, beta occurs in prefrontal cortex (PFC)  
68 during executive control of action (Swann et al., 2009; Ruiz et al., 2011; Wessel et al., 2013), working  
69 memory (Lundqvist et al., 2016; Miller et al., 2018) and preventing distraction (Hanslmayr et al.,  
70 2014; Zavala et al., 2017); and they increase in the basal ganglia in relation to sensory cues (Leventhal  
71 et al., 2012) and the encoding of sequence boundaries (Herrojo Ruiz et al., 2014). In this article, we  
72 address similarities across studies, aiming towards the larger goal of integrating these observations  
73 under a common rubric for beta.

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

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

86

## 87 **Prefrontal beta for controlling contents of working memory**

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

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

103 We speculate that the intermediate and strong beta increases have different functional roles.  
104 The intermediate elevation of beta during the delay period relative to the low levels seen at encoding  
105 and read-out may serve to protect the current working memory contents from interference. Indeed,  
106 human studies have shown increases of prefrontal beta when subjects must filter out distractors  
107 (Zavala et al., 2017) or prevent encoding (Hanslmayr et al., 2014). In contrast, the strong level of beta  
108 at the end of the trial might reflect a ‘clear out’ of the working memory content. It’s noteworthy that  
109 this ‘beta rebound’ clear-out in PFC was specific to recording sites that carried working memory  
110 information during the trial (Lundqvist et al., 2018); i.e. it was not merely motor-related. This opens  
111 up the intriguing possibility that, in sensorimotor cortex, the so-called post-movement beta rebound  
112 could serve a similar function for motor plans (discussed below). Overall, these studies suggest that  
113 beta bursting, originating in deep layers of PFC (Bastos et al., 2018), might explain how information  
114 is regulated during encoding, retention, read-out, and working memory reallocation (Lundqvist et al.,  
115 2018; Miller et al., 2018).

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

124

### 125 **Prefrontal–basal-ganglia beta for stopping action and thought**

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

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

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

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

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

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

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

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

181

### 182 **Sensorimotor beta: amplitude, frequency and beta bursts**

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

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

203 In order to compare beta modulations across different studies, it is important to first  
204 understand the underlying relationship between the LFP and neuronal spiking activity. Many studies  
205 have shown that sensorimotor LFP beta at least partly reflects local activity, with the spikes of  
206 inhibitory interneurons and pyramidal tract neurons locking to the phase of beta (Murthy and Fetz,  
207 1996; Donoghue JP, 1998; Baker et al., 1999 ; Jackson et al., 2002; Denker et al., 2011; Canolty et  
208 al., 2012; Confais et al., 2019). However, whether there is also an intrinsic relationship between the  
209 amplitude of beta oscillations and neuronal spike rates has been controversial (Canolty et al., 2012;  
210 Rule et al., 2017). A recent study resolved this issue (Confais et al., 2019), by showing that spike rates  
211 and beta amplitude have no intrinsic correlation, but are both modulated by external factors, such as a  
212 behavioral task.

213 A different issue is that the term 'beta' is broad and actually involves several types of  
214 oscillations in distinct frequency bands (Kopell et al., 2011). First, in parkinsonian rats, slow and fast  
215 beta seem to take different routes through cortical and basal ganglia circuits (West et al., 2018) and  
216 see for human evidence: (Lopez-Azcarate et al., 2010). Second, in the human, some evidence suggests  
217 beta frequency is effector specific, with frequencies  $>20$  Hz associated with lower limbs and  
218 frequencies  $<20$  Hz with upper limbs (Pfurtscheller et al., 2000; Neuper and Pfurtscheller, 2001).  
219 Third, in the macaque monkey, two beta bands, at  $\sim 20$  and  $\sim 30$  Hz, are present in motor cortical LFPs  
220 (Kilavik et al., 2012), and phase-locking analysis of neuronal spiking activity suggest both bands have  
221 at least partly a local origin within motor cortex (Confais et al., 2019). Whereas those particular  
222 studies found similar modulations of both bands with behavioral context and movement direction,  
223 other work found that pre-stimulus beta frequencies  $<20$ Hz were positively correlated with reaction  
224 times, while higher beta frequencies ( $>20$  Hz) were negatively correlated (Zhang et al., 2008;  
225 Chandrasekaran et al., 2019). One interpretation is that lower beta ( $<20$  Hz) is 'anti-kinetic' (Engel  
226 and Fries, 2010), while higher beta band ( $>20$  Hz) reflects attention and anticipation (Saleh et al.,  
227 2010; Fujioka et al., 2012; Kilavik et al., 2012; Kilavik et al., 2014).

228 Finally, in addition to amplitude and frequency changes in beta, the duration, distribution and  
229 onset of beta bursts influences different properties of the movement. It was suggested that changes in  
230 beta bursts before movement was related to 'specifying the movement goal' while fewer bursts and  
231 later bursts after an error were related to 'error evaluation and monitoring' (Little et al., 2018).  
232 However, those results are perhaps also compatible with a 'protection of the current state' function  
233 before movement (also see Shin et al., 2017) and, after movement error, a reduced and delayed 'clear-  
234 out' to 'buy time' to learn. We note, however, that Torrecillos et al (2015) showed reduced post-  
235 movement beta power also for errors that do not induce motor adaptation, suggesting the reduced beta  
236 power instead reflects the saliency of the error, irrespective of whether the motor plan should be  
237 preserved or updated. Other recent findings are from reward-dependent motor learning (Sporn et al.,  
238 2018). That study showed that a phasic, post-reward, increase in the rate of long beta bursts (duration  
239  $> 500$  ms) attenuated the update in predictions about the rewarded movement goal (also see Tan et al.,



240 2016). While further work is needed to integrate these new proposals for post-movement and post-  
241 feedback sensorimotor beta, these studies highlight the usefulness of analyzing features such as  
242 duration, rate, and timing of beta bursts to better understand sensorimotor function.

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

252

### 253 **Mechanisms of generating beta: basal ganglia and cortex**

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

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

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

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

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

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

296 The coordination of cortical and basal ganglia beta might orchestrate cognition and  
297 movement. One option is ‘top-down’ communication, in which beta is generated in the cortex and  
298 then propagates to the basal ganglia. This might reflect a situation in which cortical circuits use beta  
299 to maintain stimulus information in working memory (see above), and exert control on subcortical  
300 structures to protect them against interference. In contrast, ‘bottom-up’ communication could  
301 potentially generate beta in the STN-GPe network due to (non-oscillating) sensory and motor inputs,  
302 including ramping activity in STN (Mirzaei et al., 2017). This beta could affect reaction times and  
303 propagate through cortex via the mediodorsal thalamus. Finally, beta could be generated in the cortex  
304 and in the basal ganglia separately, perhaps relying on a shared input signal, e.g. increased excitation  
305 from the mediodorsal thalamus, to both areas (**Figure 2**). This might open a privileged  
306 communication channel between cortex and basal ganglia (Fries, 2015), so that spiking activity  
307 related to working memory or stopping can be processed across these circuits. Even though this  
308 remains speculative at this point, some evidence for bidirectional communication involving beta in  
309 cortical and basal ganglia circuits has been found in humans with Parkinson’s disease (Lalo et al.,  
310 2008).

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

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### **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.

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

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

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

365

## 366 **Conclusions**

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

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**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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