How do the basal ganglia control thalamocortical activity?

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The basal ganglia (BG) play a crucial role in controlling voluntary movements, and their dysfunction causes severe movement disorders. The BG receive inputs from a wide area of the cerebral cortices, and project back to the original cortices via the thalamus. Thus, for elucidating roles of the BG in voluntary movements, it is crucial to understand how their outputs control thalamocortical activity. The output nuclei of the BG, which convey processed information to the thalamus, are the internal segment of the globus pallidus (GPi) and substantia nigra pars reticulata (SNr), and they are composed of inhibitory GABAergic neurons. According to the well-accepted model, the GPi/SNr neurons continuously fire at high frequency, and thus continuously inhibit thalamic activity. When GPi/SNr activity is temporarily inhibited by striatal inputs, thalamic neurons are activated by disinhibitory mechanism. However, this disinhibition theory is based on studies of saccadic eye movements, and has not verified in limb movement control. There are some reports contradicting to the theory. The proportion of GPi neurons that increased their firings during forelimb movements was larger than that decreased their firings. Lesion of the GPi did not induce apparent motor deficits. In addition, recent studies on song birds strongly suggested that rebound excitation after GABAergic inhibition conveys information to the thalamus.

To clarify control mechanisms of thalamoacortical activity by GPi outputs, we recorded activity of thalamic neurons with GPi inputs and projection to the motor cortices in macaque monkeys. Based on our results, we will discuss how the GPi conveys processed information to the thalamus and motor cortices and controls movements.