Global and local excitation and inhibition shape the network dynamics for the control of movement and reward

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Global and local excitation and inhibition shape the network dynamics for the control of movement and reward.

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The cortico-striatal-thalamo-cortical (CSTC) pathway is a brain circuit that controls movement execution, habit formation and reward. Hyperactivity in the CSTC pathway is commonly observed in patients affected by obsessive compulsive disorder (OCD), a neuropsychiatric disorder characterized by the execution of repetitive involuntary movements. The striatum shapes the activity of the CSTC pathway through the coordinated activation of two classes of medium spiny neurons (MSNs) expressing D1 or D2 dopamine receptors. However, the exact mechanisms by which balanced excitation/inhibition (E/I) of these cells controls the network dynamics of the CSTC pathway remain unclear.

We used non-linear modeling of neuronal activity and bifurcation theory to investigate how global and local changes in E/I of MSNs regulate the activity of the CSTC pathway. Our findings indicate that a global and proportionate increase in E/I pushes the system to states of generalized hyper-activity throughout the entire CSTC pathway. Certain disproportionate changes in global E/I trigger network oscillations. Local changes in the E/I of MSNs generate specific oscillatory behaviors in MSNs and in the CSTC pathway. These findings indicate that subtle changes in the relative strength of E/I of MSNs can powerfully control the network dynamics of the CSTC pathway in ways that are not easily predicted by its synaptic connections.

Our most recent work is focused on introducing biophysical detail in the striatal component of our circuit, compatible with our data from optogenetic rodent models. To add the spiking temporal scale in the striatal nodes, we modified an existing model of clustering in globally coupled inhibitory networks, due to Golomb and Rinzel (1994). The striatal cells are then represented at the level of cell membrane dynamics, while the outside nodes are still represented by population firing rates (as in our original mode of the CSTC pathway). In this fashion, we can focus more specifically on understanding how factors internal and external to the striatal populations control striatal dynamics, without making the model analytically intractable.