Normal and pathological activity patterns of the subthalamo-pallidal network model during cortical slow wave activity

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It is well known that one of the most important functions of the basal ganglia is motor information processing and that Parkinson's disease is a movement disorder arising from degeneration of dopamine neurons in the basal ganglia. Also, neural activity patterns of neurons in the basal ganglia, particularly subthalamic nucleus (STN) neurons and external segment of globus pallidus (GPe) neurons, are closely related to such a behavioral function; neurons exhibit irregular and tonic spiking activity in the normal condition whereas those do synchronous and rhythmic one in the pathological condition. Although such an activity pattern transition could be attributed to chronic dopamine depletion, it is still unclear what factor affected by the dopamine depletion has an impact on the activity pattern transition.

To address this issue, we conducted numerical simulations of a computational model of STN-GPe network that was built based on recent findings of cellular properties, synaptic properties, connectivity between the nuclei and so on. We here focused on activity patterns of the STN-GPe network during cortical slow wave activity (~1Hz) under the normal and pathological conditions. We investigated the impacts of (i) changes in cellular and synaptic properties, (ii) changes in synaptic strengths (LTP/LTD) and (iii) changes in cortical and/or striatal inputs to the nuclei, which are caused by dopamine depletion. Our simulation results showed that the increased striatal inputs had the most impact on STN-GPe phase-locking to the cortical slow wave activity, suggesting that prevention of excessive striatal activity is effective to relieve the pathological patterning of STN-GPe activity.