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Local circuit model of the subthalamo-pallidal network for the generation of parkinsonian oscillations

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Parkinson's disease is psychomotor dysfunctions caused by degeneration of nigral dopaminegic neurons. Increasing evidence suggests that abnormally synchronized oscillatory activity in the basal ganglia contributes to the expression of parkinsonian motor symptoms. Experimental works demonstrated that neurons in the subthalamic nucleus (STN) and the internal (GPi) and external segments of the globus pallidus (GPe) of dopamine-depleted animals exhibit enhanced oscillatory burst discharges in 8-15 Hz, and suggest that interactions between STN and GPe generate these pathological oscillations [1]. However, the biophysical and neuronal circuitry mechanisms for the generation of these abnormal oscillations by the dopamine depletion remain poorly understood. We addressed this problem by computer simulations of spiking neuron models of the local STN-GPe circuitries with the biophysical properties of neurons and synaptic connections [2-4]. These simulations revealed that the connection patterns between STN and GPe neurons, the high maximal conductance of GPe-to-STN synapses, a type of short-term plasticity at the glutamatergic synapses of GPe neurons, and the reduced baseline firing activity of GPe neurons were critical for generation of oscillatory burst discharges in 10-15 Hz. These latter two conditions were consistent with experimental evidence [4-6]. Characterization of the mechanisms underlying these oscillations suggest that a moderate level of STN post-inhibitory rebound excitation shapes the 10-15 Hz oscillatory bursts, and that strengthened GPe-STN connectivity and reduced autonomous firing of GPe neurons are critical for the induction of the STN rebound excitation. These results suggest that the physiological and structural changes in the local subthalamo-pallidal circuits caused by the dopamine depletion underlie the induction of the 10-15 Hz parkinsonian oscillations in the STN.

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