A Possible Role of Neural Gap Junctions in Parkinson's Disease Pathology
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Background and Hypothesis:
The pathology of Parkinson's disease (PD) is characterized by modified behavior of neuronal networks in the basal ganglia after depletion of dopamine. PD states show bursting neural activity and high synchronization among neurons as well as altered oscillations in local field potentials. These network modifications are thought to be directly related to PD motor symptoms such as tremor, akinesia and bradykinesia. However, it is still a matter of debate what triggers and stabilizes the pathological behavior and how deep brain stimulation (DBS) influences it.
We hypothesize that neural gap junctions are involved in this remodeling. Their possible dependence on dopamine could contribute to the effects of L-Dopa medication.

Methods and Tissues Used:
We investigate the occurrence of neural gap junctions by immunohistochemistry and confocal imaging in rat and human post-mortem tissue, including tissue from the subthalamic nucleus, putamen and globus pallidus. The distribution of the protein connexin-36 and its distributions in PD and control tissue can indicate neural gap junctions and their modification in PD.
The observed Cx36 levels serve as an input for a computational simulation of gap junctions in the basal ganglia.