Pallidal gap junctions – Triggers of synchrony in Parkinson's disease?

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An unusual high level of synchrony and low-frequency oscillations have been measured in the basal ganglia of patients with Parkinson's disease (PD). Notably, while neural activity of the healthy external globus pallidus (GPe) shows almost no correlations between pairs of neurons, prominent synchronization in the β frequency band arises after dopamine depletion. Although these changes in network activity are often assumed to underly the motor symptoms of PD, it is not clear yet how and where they arise.
We introduce pallidal gap junctional coupling as a possible mechanism for failure of GPe-desynchronization after dopamine depletion. With confocal imaging, we showed the presence of the neural gap junction protein Cx36 in the human GPe, including an increase of Cx36 in PD patients compared to controls. Dopamine has been reported to decrease the conductance of gap junctions in different areas of the brain, making dopamine depletion a possible candidate for an increased influence of gap junctional coupling in PD.
To see what effect electrical coupling in the GPe could have, we incorporated gap junctions in a conductance-based model of the basal ganglia. Inhibitory synapses of the GPe were able to desynchronize the network, leading to irregular firing. When gap junctions of low conductance are added, this desynchronizing effect was even boosted. However, when gap junctional coupling became too strong, the network synchronized. We further tested what effects increased gap junctional coupling has on the transmission of β oscillations from both cortex and striatum to downstream basal ganglia nuclei.
In conclusion, we hypothesize that strong gap junctional coupling in the GPe disturbs the self-desynchronization in this nucleus and renders the GPe susceptible to synchronize with incoming low-frequency oscillations.