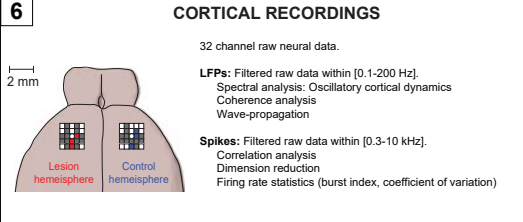
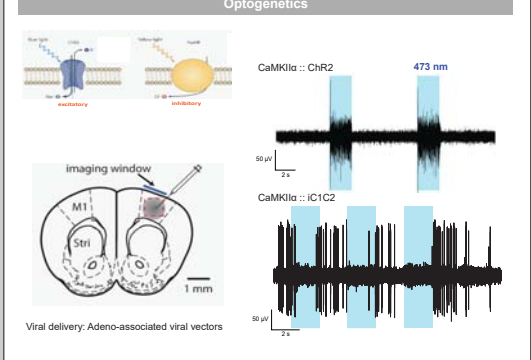
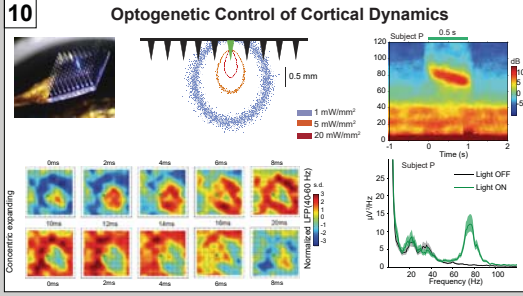
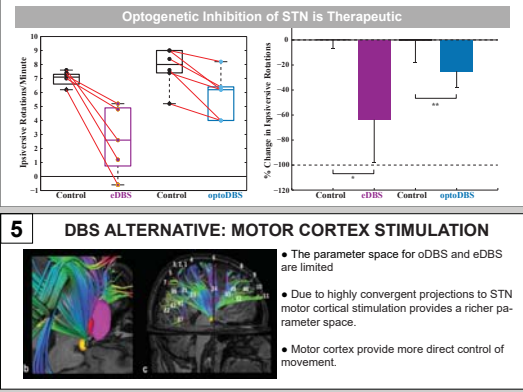
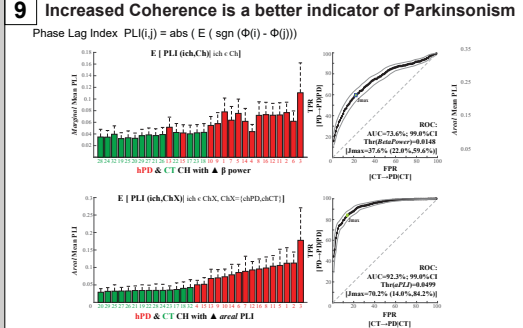
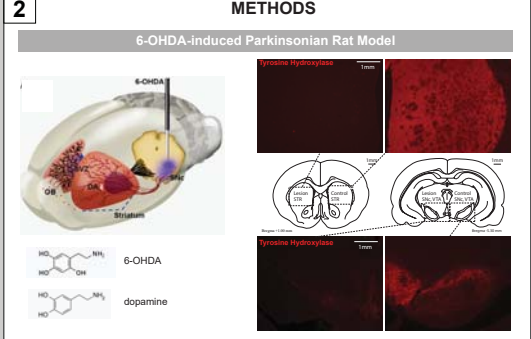
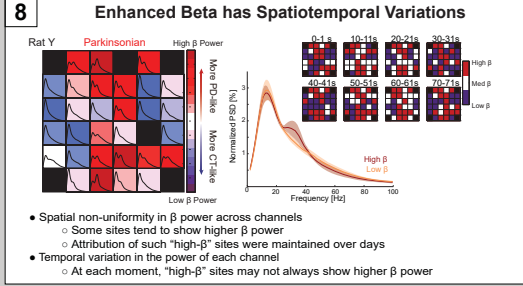
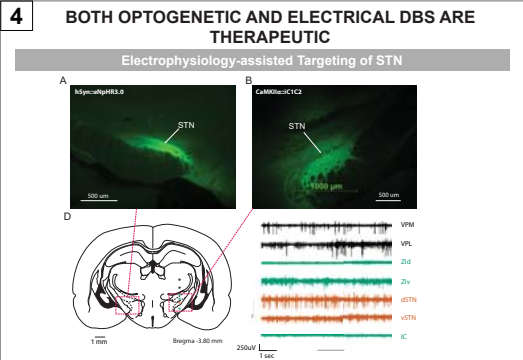
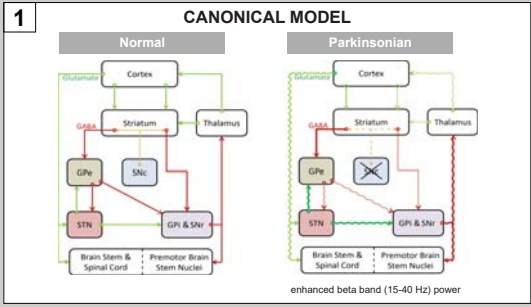
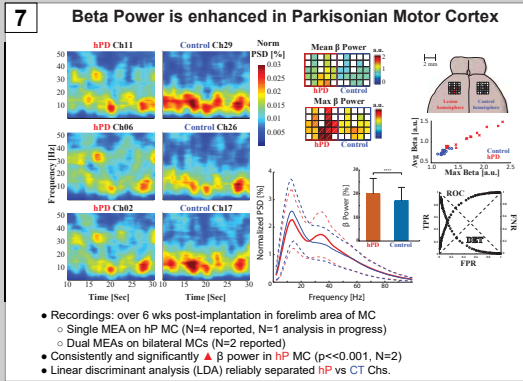
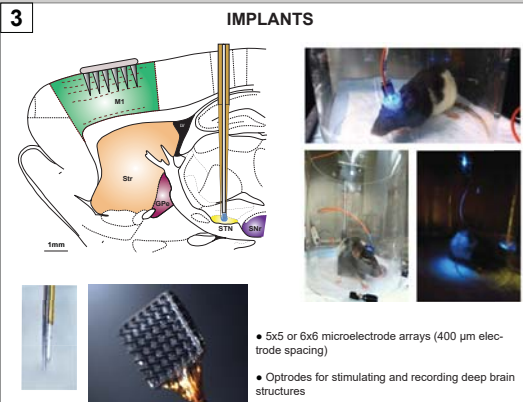


OPTOGENETIC MANIPULATION OF CORTICO-BASAL-GANGLIA SYSTEM AS A THERAPY FOR PARKINSON'S DISEASE.

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ABSTRACT

Parkinson's Disease (PD) is caused by the loss of dopamine (DA) neurons in the nigrostriatal pathway leading to severe motor deficits such as bradykinesia, muscle rigidity, tremor at rest and abnormal posture. Symptoms are accompanied by abnormal burst spiking activity in and synchronous beta oscillations (15-30 Hz) across the cortico-basal-ganglia system (CBGS, e.g. in motor cortex). Electrical deep brain stimulation (eDBS) is a standard treatment for severe PD, where high frequency (125Hz) electrical pulses are delivered to CBGS nuclei (e.g. the subthalamic nucleus, STN). eDBS alleviates and normalizes, to some extent, motor symptoms and the CBGS neural activity patterns, especially beta oscillations. However, how eDBS exerts its therapeutic effect is not known. We aim to use microelectrode arrays (MEAs, for motor cortex) and optrodes (for deep nuclei), together with optogenetic and electrical stimulation in the CBGS, to i) investigate neural mechanisms of therapeutic action of eDBS and oDBS (optogenetics-based DBS), and ii) ultimately develop a more targeted multi-site oDBS paradigm for better treatment of PD. A 6-OHDA lesion based hemi-Parkinsonian rat model is used as our animal model.



Future Directions

Testing therapeutic effect of cortical optogenetic stimulation in Parkinsonian rodents.
 Analysis of spiking data to identify disease induced changes in information processing and encoding.
 Modeling effects of optogenetic stimulation on cortical dynamics.