

Introduction

Parkinson's Disease is a neurodegenerative disorder of the basal ganglia. The main cause for Parkinson's Disease is the depletion of dopamine, a neurotransmitter. The basal ganglia contains four major nuclei: the substantia nigra, the subthalamic nucleus, the external globus pallidus, and the striatum. These nuclei communicate with each other by the use of neurons.

This experiment specifically focuses on the external globus pallidus (GPe) and the dorsal striatum (dStr).

- Communicate with each other through a class of neurons called spiny projection neurons (SPNs).
- Both indirect and direct spiny projection neurons.
- Direct spiny projection neurons have a D1 receptor while indirect spiny projection neurons have a D2 receptor.

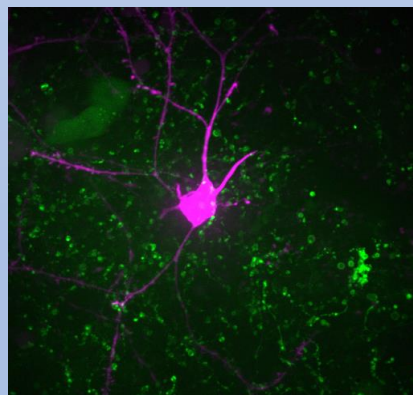
Materials and Methods

Mice

- Transgenic Mice
 - Npas1-Cre;D2-GFP
- Control group left without injection
- Experimental group injected with 6-OHDA

Cell Recordings

- Whole-cell patch clamping
- Confocal Microscopy
- Fluorescent Dye and Protein Fluorescence



Data Analyses

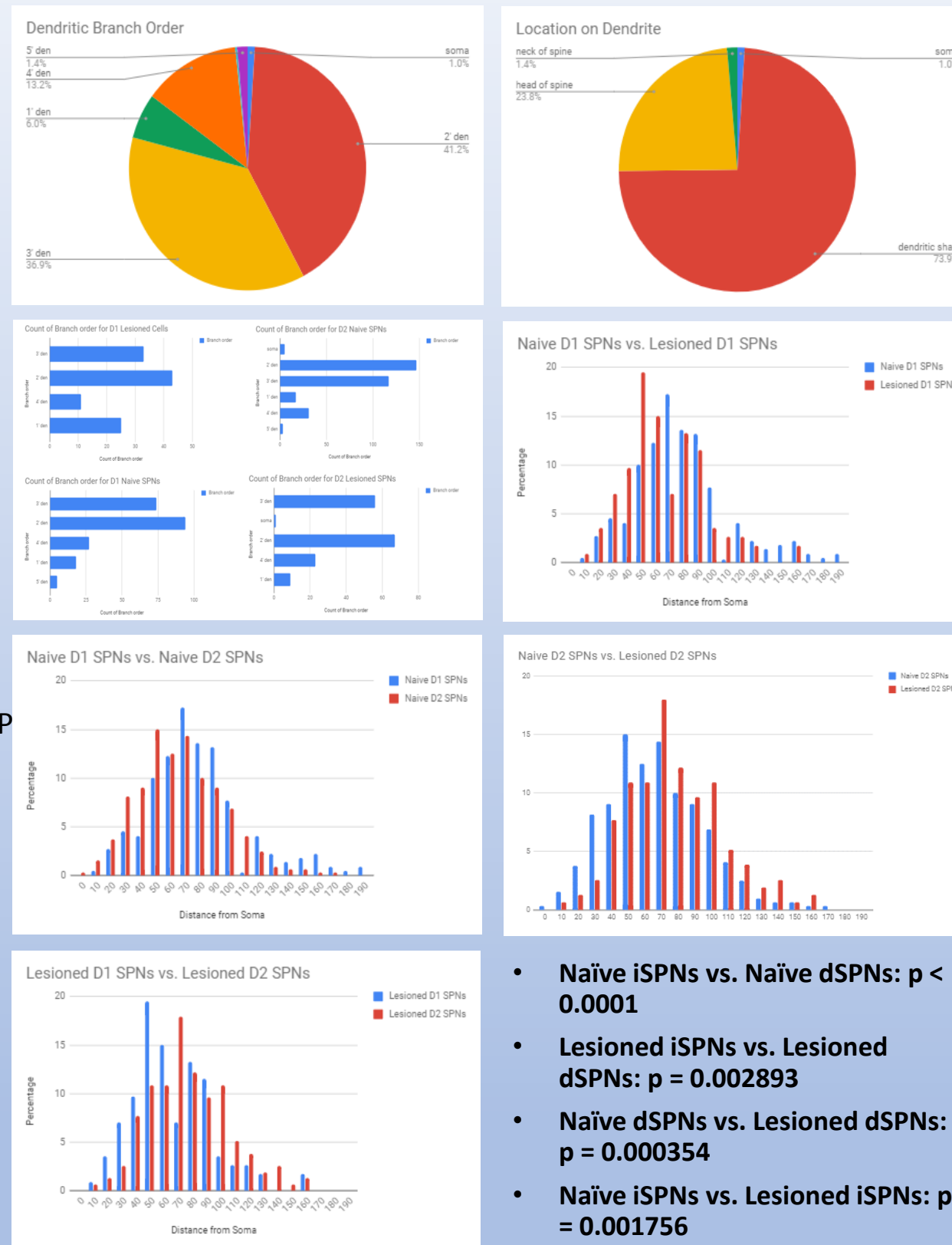
- Whole-cell patch clamping
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Cell Specific Control of the Pallidostriatal Pathway

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Results



Conclusion

- Most synapses occurred on 2' and 3' dendrites
 - This stayed constant throughout each group of SPNs
- Most synapses occurred on the dendritic shaft
 - This stayed constant throughout each group of SPNs
- The frequency of synapses was unaffected.
 - The average frequency for each group ranged between 6-7 contacts per cell.
- The projections of the iSPNs are increased in the lesioned model.
 - The depletion of dopamine strengthened the indirect pallidostriatal pathway.
- The projections of the dSPNs are diminished in the lesioned model.
 - The depletion of dopamine weakened the direct pallidostriatal pathway.

Future

- Understanding the interactions of the basal ganglia will allow for improved treatment strategies.
 - Inhibition of cortical input of iSPNs by Npas input further explains hypokinetic movement in the Parkinsonian model.
- Possibilities for treatment include designer drugs, stem cell therapy, and improved surgery techniques.
 - We've found potential therapeutic targets for use while treating the disease.
 - More research on the GPe and dStr as well as the other nuclei must be done.

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