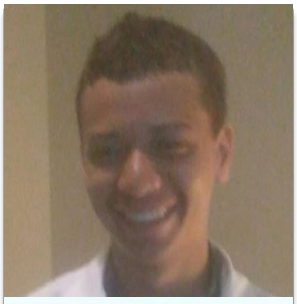

SUBTHALAMIC NUCLEUS DEEP BRAIN STIMULATION REDUCES PATHOLOGICAL INFORMATION TRANSMISSION TO THE THALAMUS IN A RAT MODEL OF PARKINSONISM

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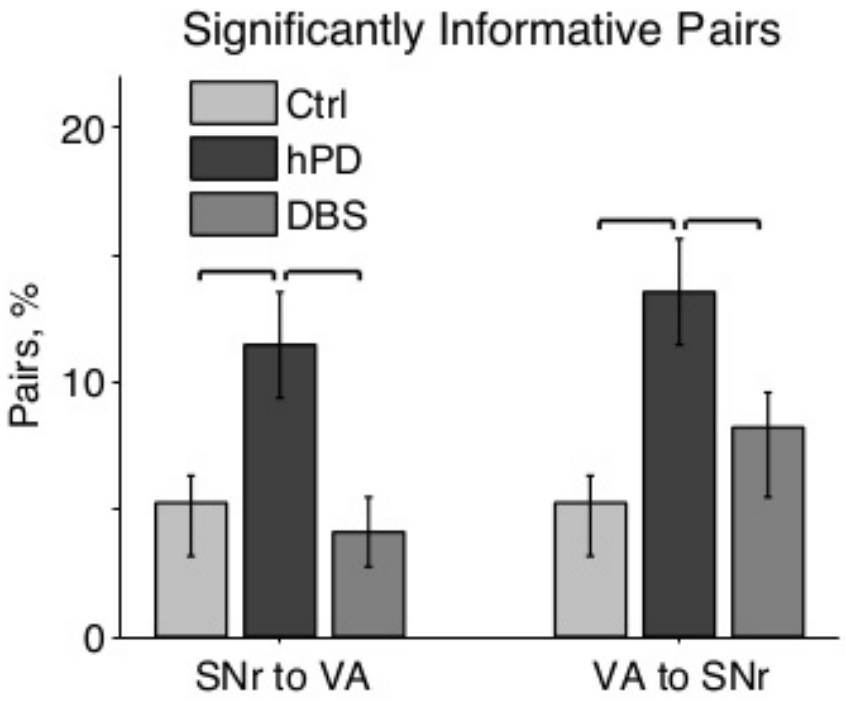
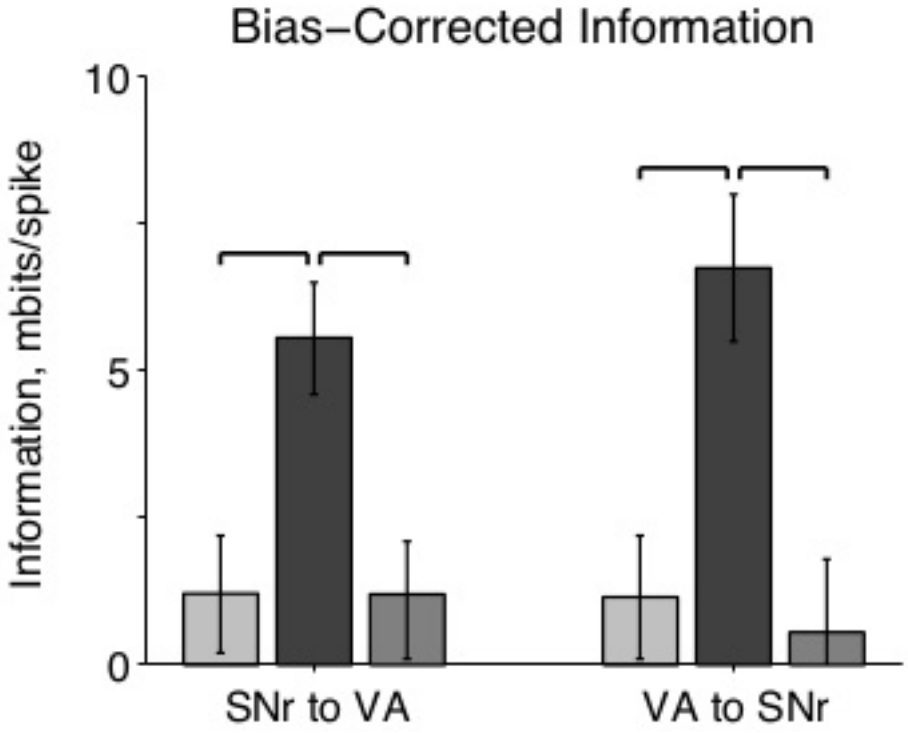
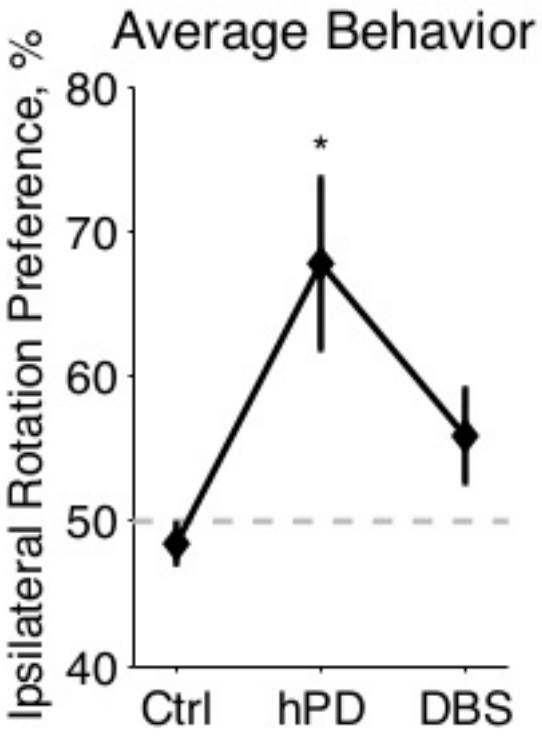
Parkinsonian motor symptoms result from a degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNc). Deep Brain Stimulation (DBS) is used to partially treat these motor symptoms. However, the mechanisms of DBS are unclear. The Dorval lab hypothesizes that the motor symptoms of Parkinson's disease (PD) are associated with increased information transmission between the substantia nigra reticulata (SNr) and the ventral anterior thalamus (VA). To test this hypothesis, seven Long-Evans rats were implanted with stimulating and recording arrays and a cannula. Healthy behavioral and electrophysiological data was subsequently collected. After data collection, 6-hydroxydopamine (6-OHDA) was injected unilaterally to induce a dopamine lesion in the SNc. Using a hemiparkinsonian (hPD) rat model, electrophysiological and behavioral data was collected in both the hPD and DBS conditions. Histological techniques were subsequently performed to verify lead location and neural tissue damage. Upon analysis, it was found that information transfer between SNr and VA significantly increases in the orthodromic and antidromic directions in the hPD state. It is possible that this extraneous information is the underlying cause of motor symptoms in PD. The increase of information is rectified through the use of DBS. This finding suggests that the increase in information results in a loss of information channel independence. Furthermore, DBS partially this effect by decreasing extraneous information which increases information channel independence.




Bias–Corrected Information
Information, mbits/spike
10
5
0
SNr to VA
VA to SNr


Average Behavior
Ipsilateral Rotation Preference, %
80
70
60
50
40
*
Ctrl
hPD
DBS