Effects of basal ganglia on cortical computation: a hybrid network/field model

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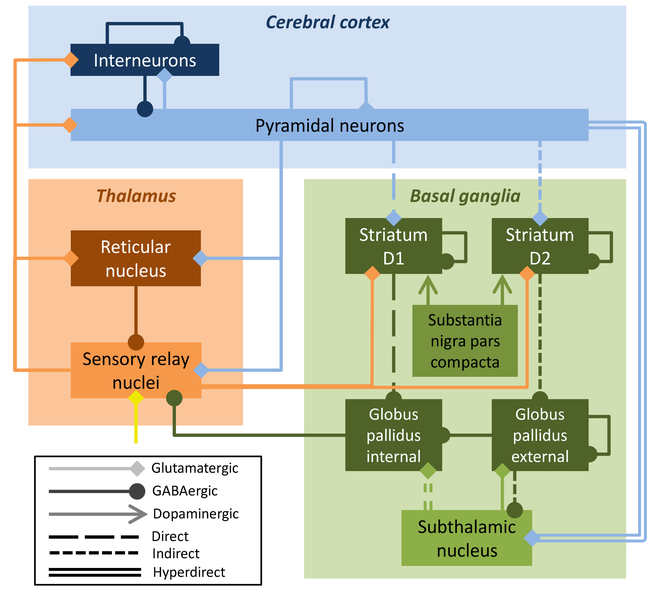
1. Introduction

Deficits in the basal ganglia can lead to motor disorders such as Parkinson's disease. But motor commands originate from the cortex, so it is important to understand how the basal ganglia influence cortical dynamics and computation, and how this influence becomes pathological in Parkinson's disease. To explore these questions, we developed a hybrid network/field model of the thalamocortical (TC) + basal ganglia (BG) system.

2. Methods

We used the outputs from four different neural field models – white noise, TC only, TC + BG, and TC + BG with reduced dopamine (DA) – as the input to a 2000-cell network of integrate-and-fire neurons, then explored how the different inputs affected the dynamics of the network.



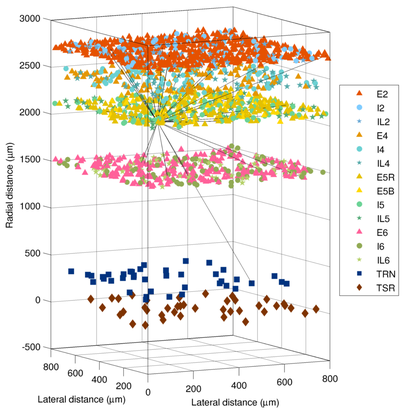


Fig. 1: Structures of the field model (top) and network model (left). The field model has 10 populations and 28 connections across the cortex, thalamus, and basal ganglia. The network model has 15 cell types distributed across 6 cortical layers and the thalamus, for 2000 cells and 60,000 connections in total.

E=excitatory pyramidal;
I=inhibitory interneuron;
L=low-threshold spiking;
R=regular firing;
B=bursting;

TSR=thalamic sensory relay;
TRN=thalamic reticular nucleus.

3. Results

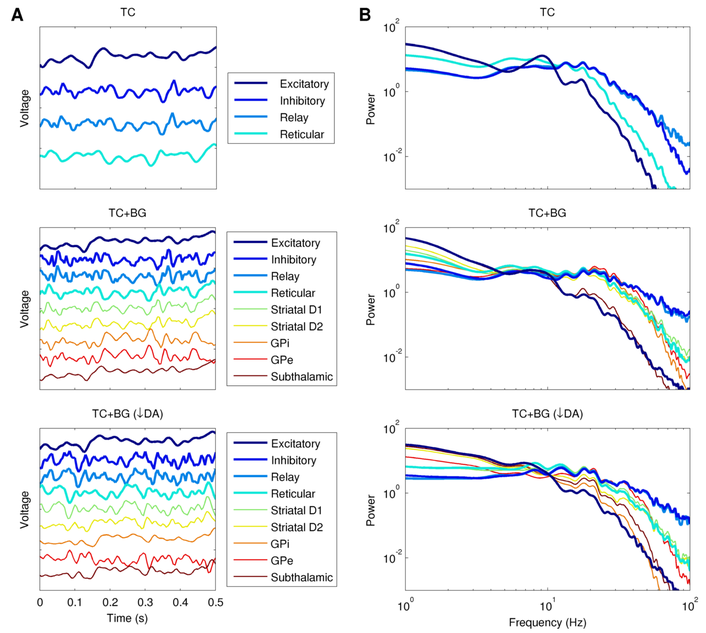


Fig. 2: Dynamics of the field models (white noise not shown). **A.** Local field potential (LFP) time series, showing phase relationships between populations. Activity in the internal and external segments of the globus pallidus are normally in phase, but in Parkinson's disease, the former entrains to the subthalamic nucleus instead. **B.** LFP spectra. Healthy basal ganglia nuclei spectra follow the sensory relay nucleus spectrum from 10–40 Hz, but this is also disrupted in Parkinson's disease.

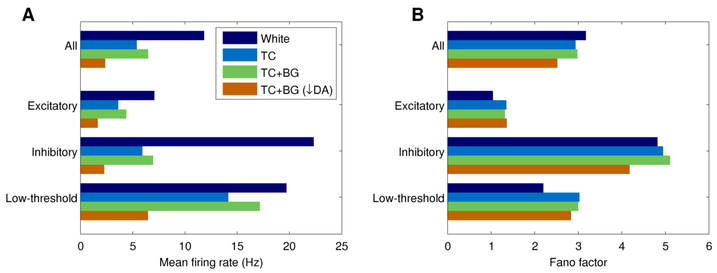


Fig. 3: Network firing and variability. **A.** Mean firing rates. Overall, the model with reduced dopamine had considerably lower firing rates, which results from excessive inhibition of the thalamic sensory relay nuclei. **B.** Fano factors. Excitatory and inhibitory cells show below and above average spiking variability, respectively.

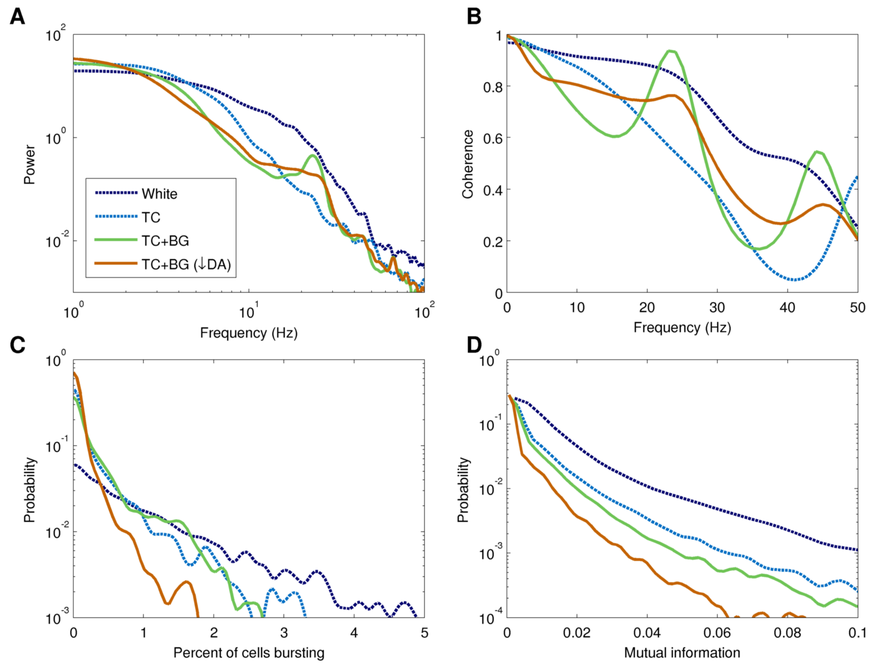


Fig. 4: Characteristics of the network model as driven by each field model. **A.** Power spectra. The pronounced peak at ~20 Hz in the basal ganglia model is weakened and broadened with reduced dopamine. **B.** Coherence. The healthy basal ganglia model has the most pronounced features, while the white noise model has the least. **C.** Neuronal burst frequency. **D.** Mutual information. Reduced burst frequency and mutual information in Parkinson's disease are partly due to reduced firing rate.

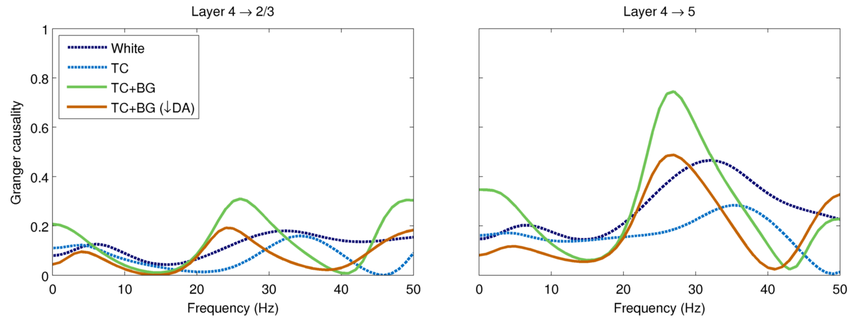


Fig. 5: Spectral Granger causality in each of the models. The healthy basal ganglia model shows strong "input" causality (layer 4→2/3) below 5 Hz and from 20–35 Hz; these are attenuated with reduced dopamine (left panel). The "output" causality (layer 2/3→5, not shown) is also attenuated below 10 Hz. These effects combine to significantly reduce the total causality from layer 4→5 in Parkinson's disease (right panel).

4. Discussion

- Loss of dopamine significantly changes the phase relationships between basal ganglia nuclei (Fig. 2).
- Our model predicts a larger reduction in cortical firing rates in Parkinson's disease (Fig. 3) than is seen experimentally, suggesting that homeostatic mechanisms may exist that partly offset this effect.
- The loss of Granger causality (Fig. 5) from the main input layer of the cortex (layer 4) to the main output layer (layer 5) may explain some features of Parkinsonism, especially bradykinesia.
- In summary, the brain's large-scale oscillatory environment strongly influences the information processing that occurs within its subnetworks.

Reference

van Albada SJ, Robinson PA (2009). Mean-field modeling of the basal ganglia-thalamocortical system. *J Theor Biol.* **257**:642–688.

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Further information

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