

## **Dynamical Origins of Parkinsonian Biomarkers: Bifurcations and Spectral Signatures in a Minimalist Model of the Subthalamic Nucleus**

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### **Abstract:**

The synchronization of neural activity in the beta band (13–30 Hz) coupled with high-frequency oscillations (HFO, >200 Hz) represents a distinct dynamical signature of Parkinson's disease. While often studied through complex multi-population circuit models, the nonlinear mechanisms governing the emergence of this cross-frequency coupling remain obscured by biological detail. In this talk, I will present a minimalist computational framework that isolates candidate bifurcation structures underlying these pathological states.

Using a reduced model of the subthalamic nucleus incorporating spike-frequency adaptation and recurrent coupling, we identify two distinct dynamical routes to pathological synchronization. Through systematic numerical bifurcation analysis, we show that these routes map onto generic transitions in the regime phase space: Route 1 involves a network-driven transition from tonic firing to synchronized bursting, while Route 2 arises from the synchronization of intrinsically bursting neurons. We demonstrate that these seemingly disparate regimes are topologically connected in the system's phase space, accessible through the variation of a single excitability parameter. Notably, we observe that the pathological state characterized by strong beta-HFO coupling coincides with the emergence of canard-like or mixed-mode oscillation solutions in the population firing rate dynamics.

Crucially for diagnosis, these dynamical trajectories leave distinct fingerprints on the power spectral density of the local field potential. We identify specific spectral morphologies, characterized by localized power suppression preceding high-frequency peaks, that serve as observable proxies for the underlying population state. I will conclude by discussing our ongoing work to leverage these theoretical insights for model validation against human intracranial data. By bridging the gap between nonlinear theory and clinical observation, we aim to develop robust dynamical markers for patient-specific diagnosis and adaptive neuromodulation.