

Controlling oscillations in Parkinson's disease

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AI method use:

I used **NotebookLM (Google)** for the initial discussion and brainstorming around the paper. Then I switched to **Gemini** for the coding side, mainly for setting up the networks using the NEST documentation and helping with plotting. For text editing, I relied on **Grammarly Pro**.

Methods used

The network was simulated using the NEST^[1] simulator. It comprised two populations modeled as Leaky Integrate-and-Fire (LIF) neurons: 300 excitatory neurons (STN) and 500 inhibitory neurons (GPe).

Table 1: Neuron Parameters

Parameter	Value	Description
C_m	30.0 pF	Membrane capacity
E_L	-70.0 mV	Leak reversal potential
V_{th}	-60.0 mV	Spike threshold
V_{reset}	-70.0 mV	Reset potential
τ_{ref}	2.0 ms	Refractory period
τ_m	20.0 ms	Membrane time constant
$\tau_{syn,ex}$	1.0 ms	Excitatory synapse time constant
$\tau_{syn,in}$	10.0 ms	Inhibitory synapse time constant

Network connections were established using a fixed indegree rule. To maintain realistic baseline activity, all neurons received independent background noise from a **Poisson generator (800 Hz)** with weights of 40 pA (to GPe) and 65 pA (to STN).

Table 2: Synaptic Connectivity

Source	Target	Indegree	Weight (pA)	Delay (ms)
GPe	STN	30	-15.0	5.0
STN	GPe	50	30.0	5.0
GPe	GPe	30	-5.0	2.0
STN	STN	50	50.0	2.0

Three distinct network states were simulated.

Table 3: Simulation Conditions

Condition	Description	Input Specifications
Baseline	Healthy state	Background Poisson noise only.
Parkinsonian	Increased striatal inhibition	Poisson Generator → GPe Rate: 800 Hz Weight: -5.0 pA
DBS	Deep Brain Stimulation	DC Generator → STN Amplitude: -180.0 pA

Table 4: Analysis Metrics

Metric	Method
Spiking Activity	Raster plots & Rate histograms
Spectral Analysis	Spectrogram & Power Spectral Density (PSD)
Auto-correlation	Auto-correlation Function (ACF)

Analysis

2.1 Baseline simulation

After connecting the spike detector to the STN and GPe populations, I ran a sanity check to see the simulation of the baseline.

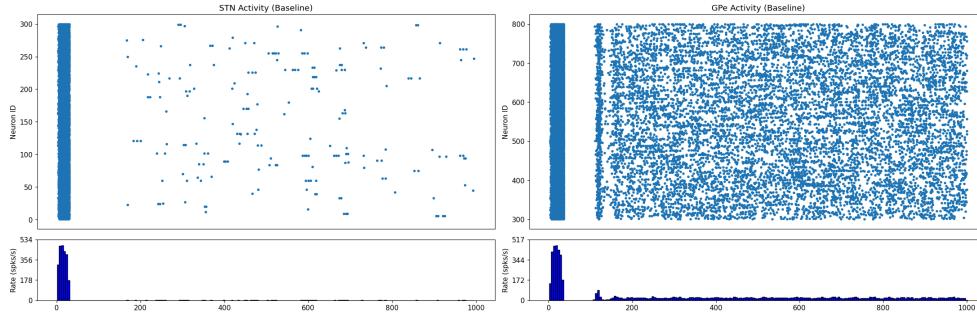


Figure 1: Baseline firing activity in STN (left) and GPe (right). No clear oscillations are visible.

Questions

Q: What is the role of the Poisson generator in this model?

It models uncorrelated spike trains that provide external background input to both STN and GPe neurons. This is done to produce realistic, aperiodic baseline firing. It is also later used to simulate inhibitory striatal input to GPe neurons.

Q: What biological phenomenon is it equivalent to?

Uncorrelated background spiking activity, basically baseline neural noise.

Q: Why do we use a Poisson random process to model this input?

Because its mathematical properties match the statistics of random, uncorrelated spikes seen in real neural activity.

Q: Is the input excitatory or inhibitory?

It can be both, depending on the source and target. External input to STN and GPe is excitatory, while striatal input to GPe is inhibitory.

2.2 Increased input to the GPe (Parkinsonian state)

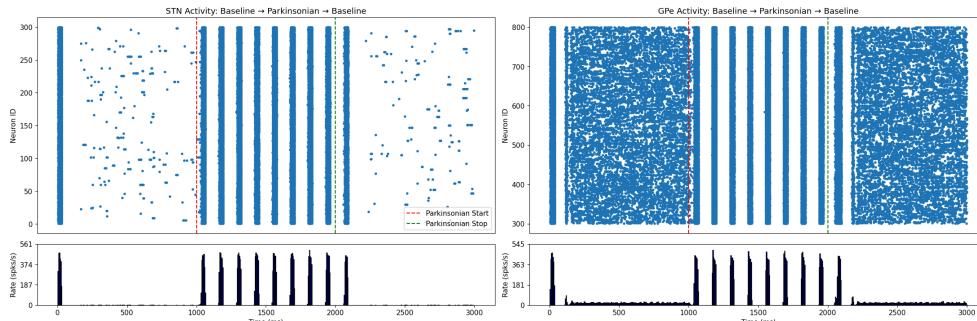


Figure 2: Activity changes after increasing inhibition to the GPe. The red line marks the onset of extra inhibition.

Q: Is this equivalent to increasing excitation or inhibition? What biological phenomenon does this model?

Here we increased **inhibition** to the GPe. Since the GPe normally inhibits the STN, suppressing GPe means the STN becomes disinhibited and starts firing more. This excites the GPe, and the oscillatory loop begins.

This step models **dopamine depletion in Parkinson's disease**. Dopamine normally prevents excessive striatal inhibition of the GPe. When dopamine is lost, the striatum becomes overactive and strongly inhibits the GPe and this causes some motor symptoms.

2.3 Deep Brain Stimulation (DBS)

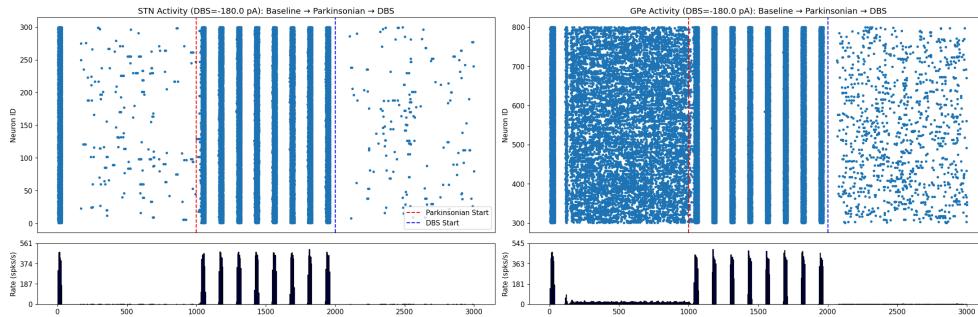


Figure 3: Timeline showing transitions from Healthy → Parkinsonian → DBS-treated state. The blue dashed line marks DBS onset

Figure 3 shows the full transition. DBS involves implanting an electrode into the STN that delivers high-frequency electrical pulses. As shown in the raster plots, this breaks the pathological synchronized oscillations and produces a more random firing pattern.

However, under DBS the overall spiking activity is lower than in the healthy baseline. This is because the disease mechanism is still present and the GPe is still inhibited by striatal input. The GPe remains relatively sparse because STN excitation is not strong enough to completely overcome the Parkinsonian inhibition. So the biggest difference in all three stages can be seen in the GPe activity. Going from dense random firing to oscillation to random but sparse firing.

Plotting results

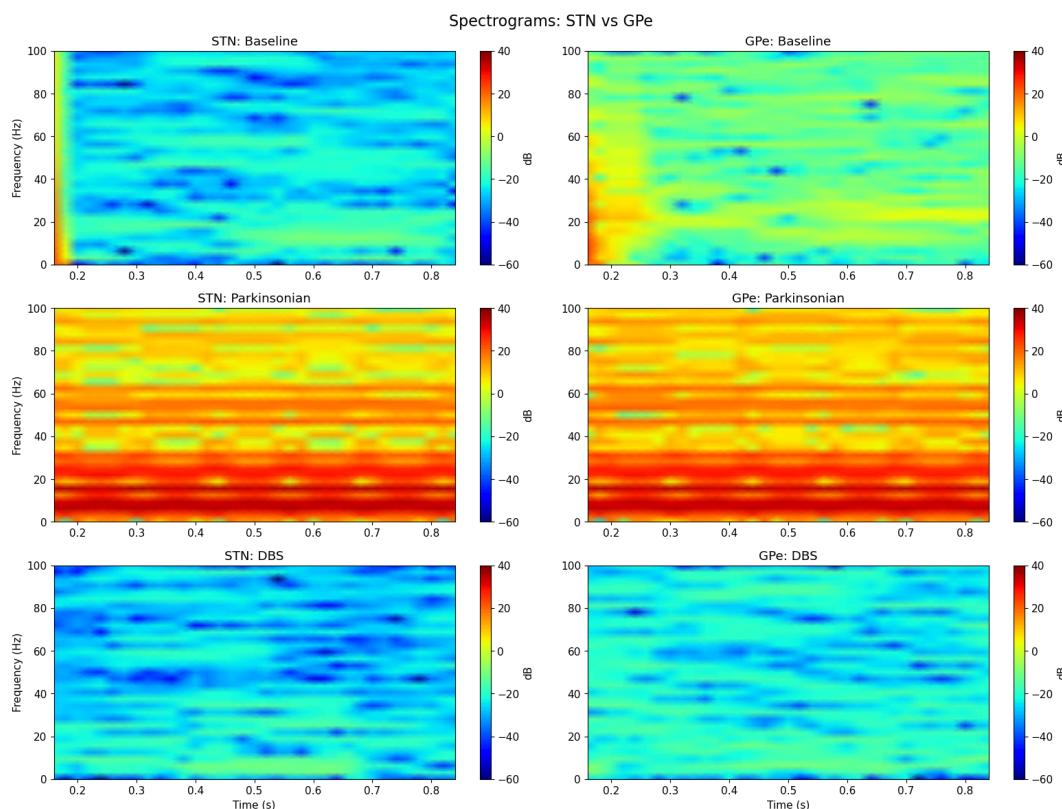


Figure 4: Spectrogram evolution for STN and GPe across conditions.

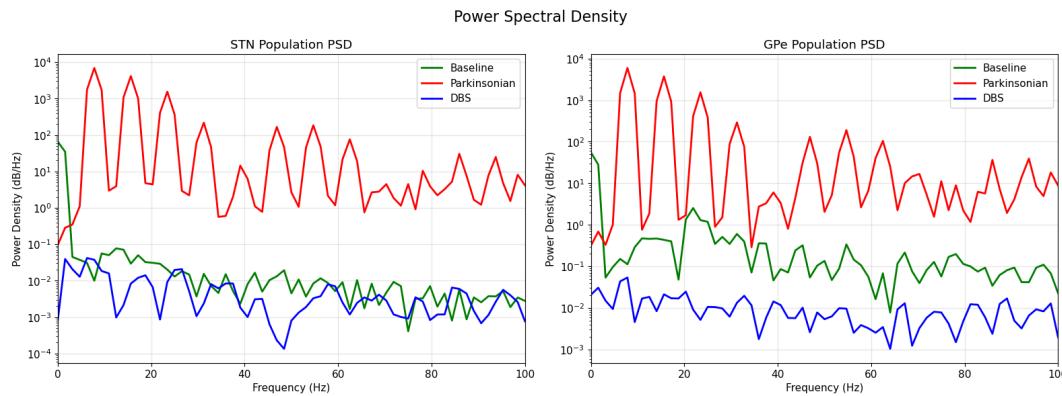


Figure 5: Power Spectral Density (PSD) comparison across baseline, Parkinsonian, and DBS states.

Oscillations:

Oscillations appear in the Parkinsonian condition. In the spectrogram (Figure 4), this shows up as kind of strong horizontal band of high power. The frequency lies roughly between 5–30 Hz, corresponding to the beta band.

1/f spectral component:

Our small network cannot show a 1/f component. Large populations produce it through many asynchronous spikes that create broad, long-range fluctuations, but in our model individual spikes dominate and mask any aperiodic background. Our variables are also very deterministic, with fixed weights, so the activity forms clean oscillations rather than noise. Finally, we compute the PSD from population firing rates, but to see a 1/f component we would need to use synaptic currents.

The spectrogram values were taken from around **t = 1000 ms** with a bin size of **5 ms**, giving 200 data points. The window size was **64 samples**, which corresponds to about **320 ms (64/200)**. Larger windows reduce time resolution but improve frequency precision, while smaller windows do the opposite. We used an overlap of **60 samples**, so the window shifts by 4 samples each time, giving much smoother plot.

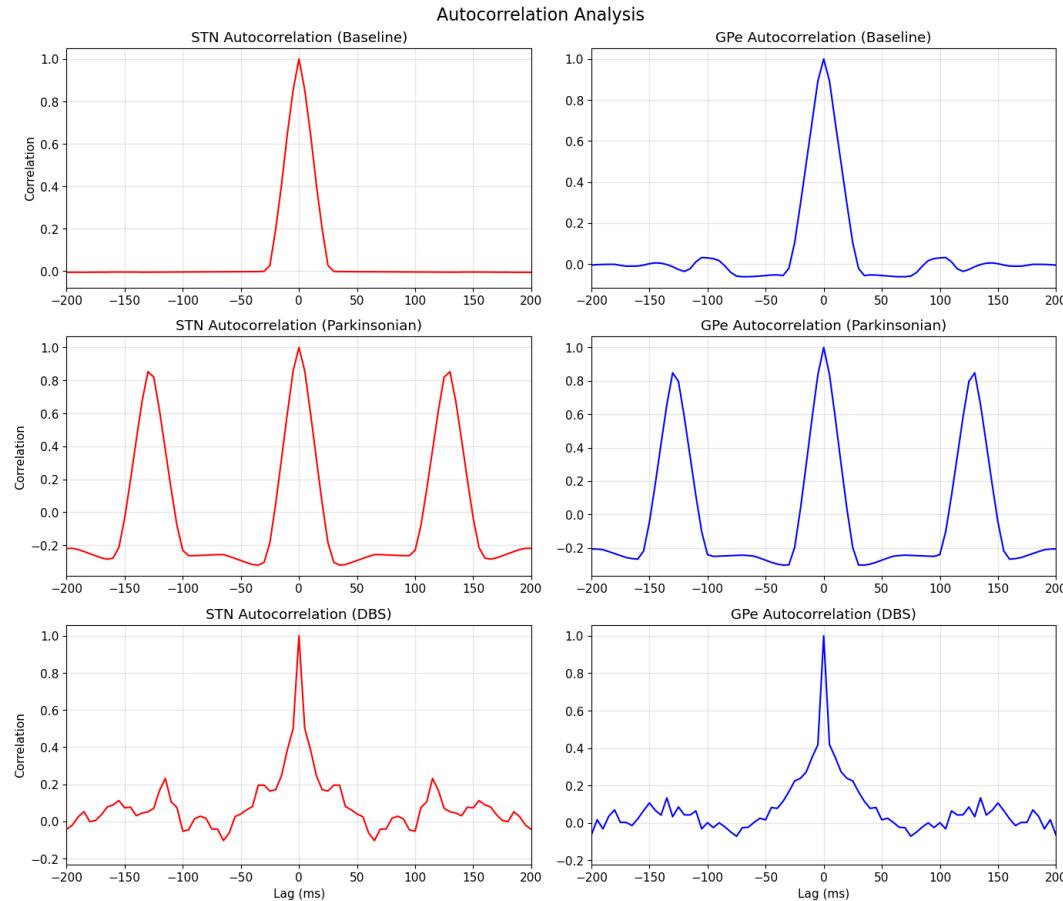


Figure 6: Autocorrelation functions for baseline, Parkinsonian, and DBS states.

The autocorrelation function detects repeating patterns even in noisy signals. It works by shifting the signal against itself and measuring similarity. This always produces a strong peak at lag 0.

The interesting result is in the Parkinsonian condition, where clear repeating peaks appear at around ± 125 ms. This implies a period of about 125 ms. Under DBS, these peaks mostly disappear.

Using $f=1/T$ with $T \approx 125$ ms, the oscillation frequency is about **8 Hz**. Clinically, PD is associated with oscillations in the **12–30 Hz beta band**^[2], so this is slightly lower but still consistent.

The amplitude is about **0.9**, which indicates strong periodicity.

Bonus 1: Effect of changing DBS amplitude

Since the DBS amplitude is negative, it delivers a type of pulse that disrupts STN activity. If the negative value becomes smaller, the stimulation may not be strong enough to break the unwanted rhythm. If it becomes too negative, it can quiet the STN more than needed and cause side effects.

I tested amplitudes of **-200, -90, and +90 pA**.

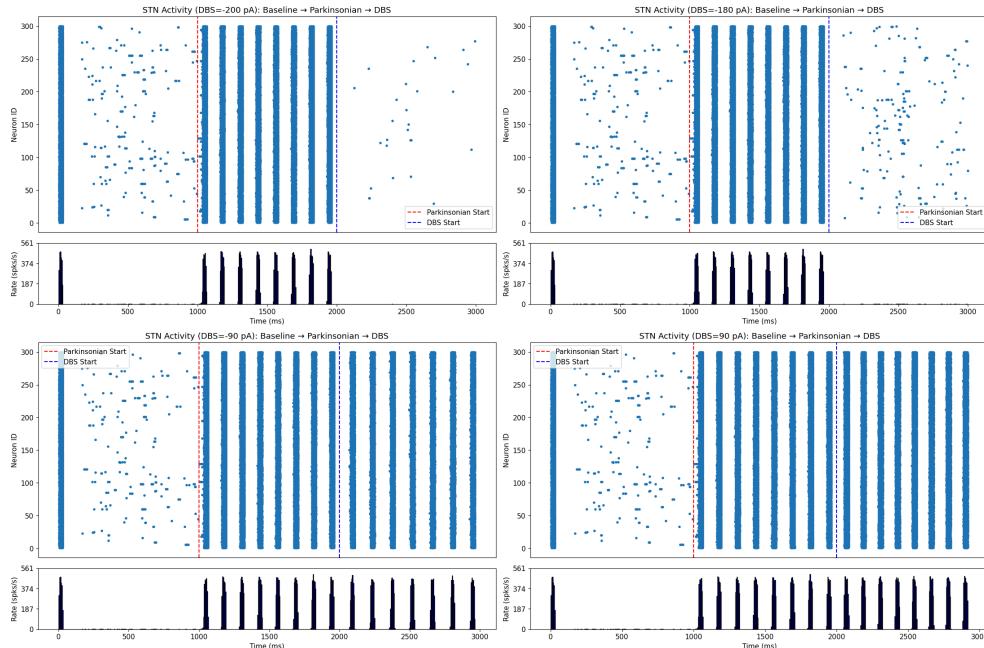
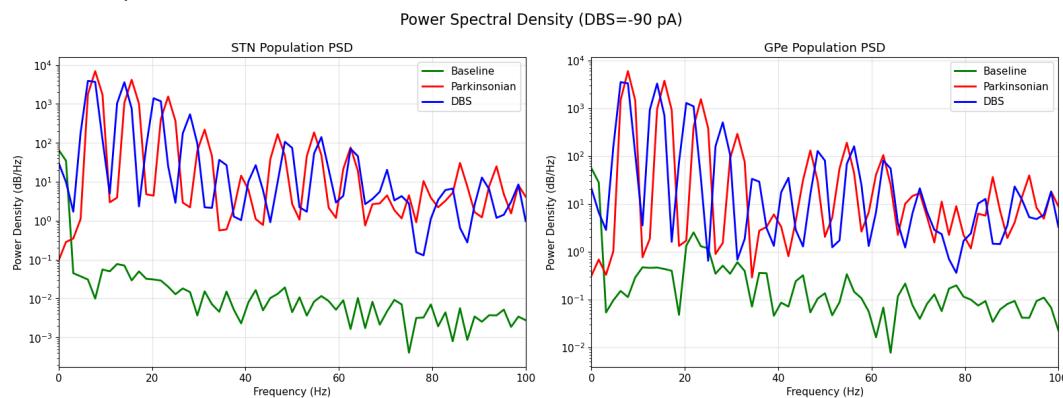


Figure 7: Timeline plots for different DBS amplitudes.

From Figure 7 we can see that at -200 the STN activity with DBS treatment is not oscillatory but is much more sparse which can have other negative side effects. Higher values than -180 on the other hand forms oscillations.

Next, I compared the PSDs for +90 and -90:



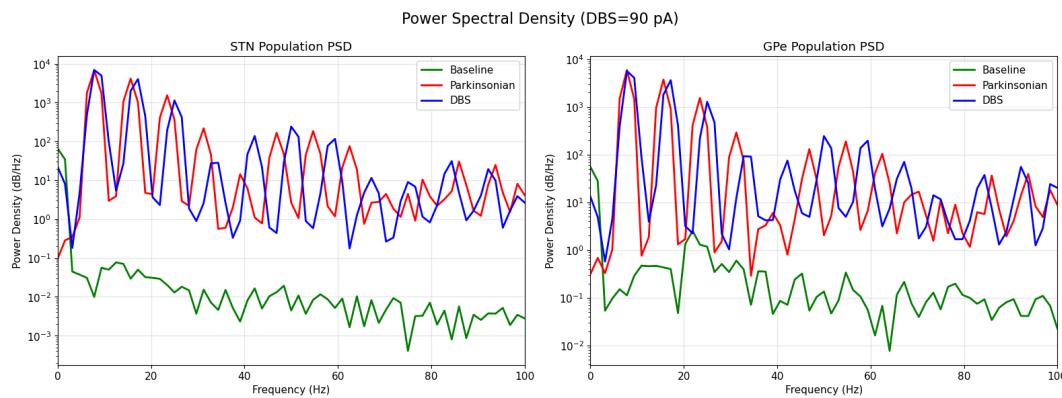


Figure 8: PSD comparison under different DBS amplitudes.

With **+90 pA**, the blue line is slightly higher than with **-90 pA**, meaning this setting actually enhances oscillations rather than suppressing them.

Bonus 2

Part 1: Making oscillations worse

To exaggerate oscillations, I increased the stimulation amplitude to **300 pA**. My theory is that positive amplitude can make the oscillations worse because this type of pulse can excite nearby pathways instead of calming them, which strengthens the unwanted rhythm instead of weakening it.

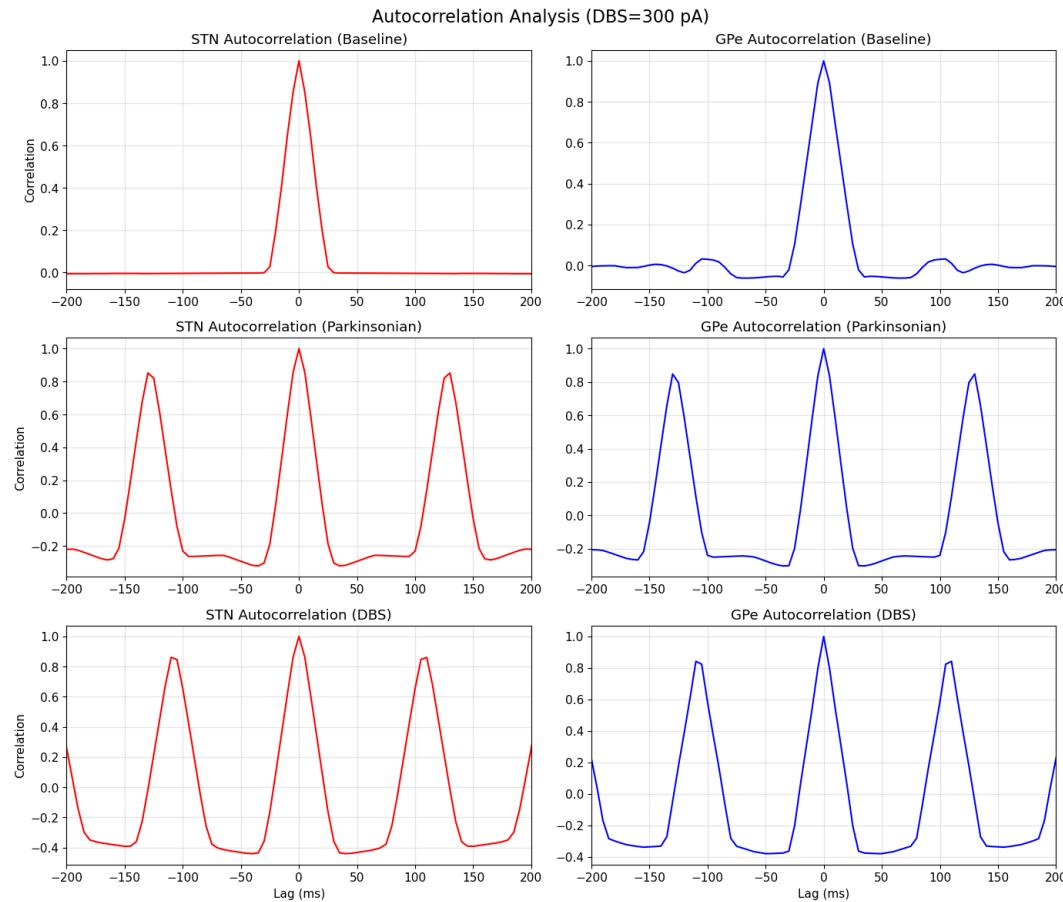


Figure 9: Autocorrelation functions with strong stimulation (300 pA).

Here, the oscillation peaks period shortens, indicating higher-frequency oscillations.

Part 2: Restoration Strategy

Is the DBS-treated state the same as baseline?

No.

In the baseline state, GPe fires relatively strongly while STN is quieter. Under DBS, pathological oscillations disappear, but at the cost of suppressing overall network activity. We can see it most clearly in GPe where in DBS the spiking activity is really sparse

Strategy to restore a more normal state

The main issue is that the GPe is still strongly inhibited by striatal input in the Parkinsonian state. That is why we gonna add excitatory input to GPe.

After several trials of different values, I found a balance by using:

- Inhibitory input to STN: **-30 pA**
- Excitatory input to GPe: **+90 pA**

This combination produced firing rates and spectra close to the healthy baseline. I arrived at these values through experimentation, knowing that the STN likely needed less inhibition and the GPe needed more excitation.

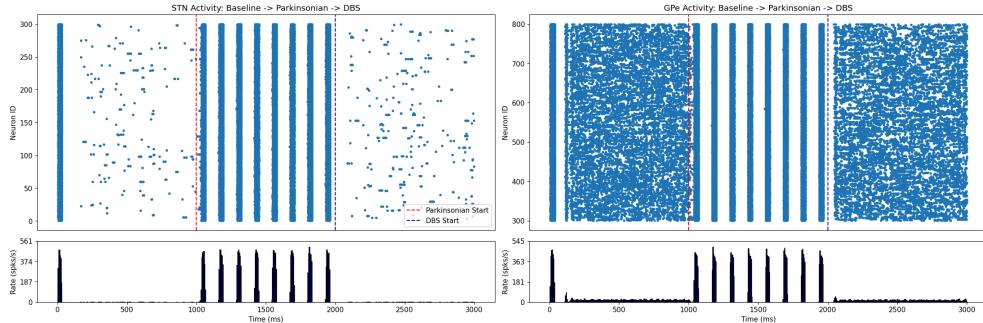


Figure 10: Raster plots for the “restored” condition.

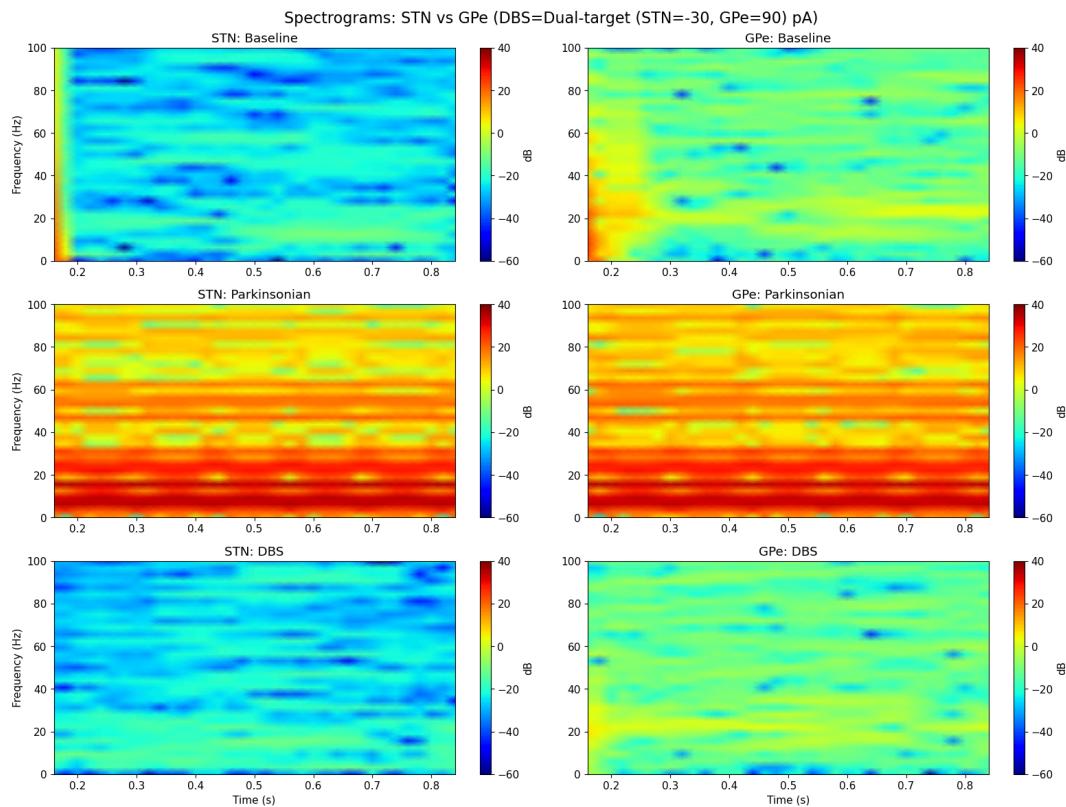


Figure 11: Spectrogram for the restored condition.

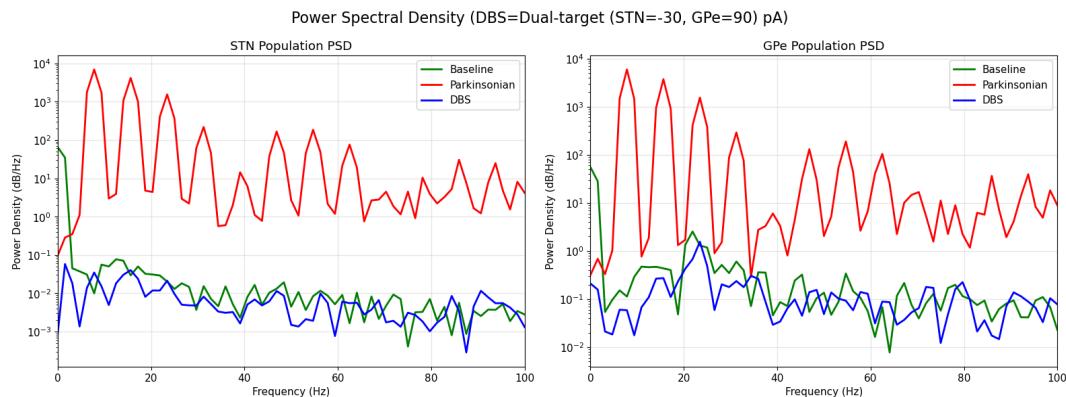


Figure 12: PSD comparison including the restored condition.

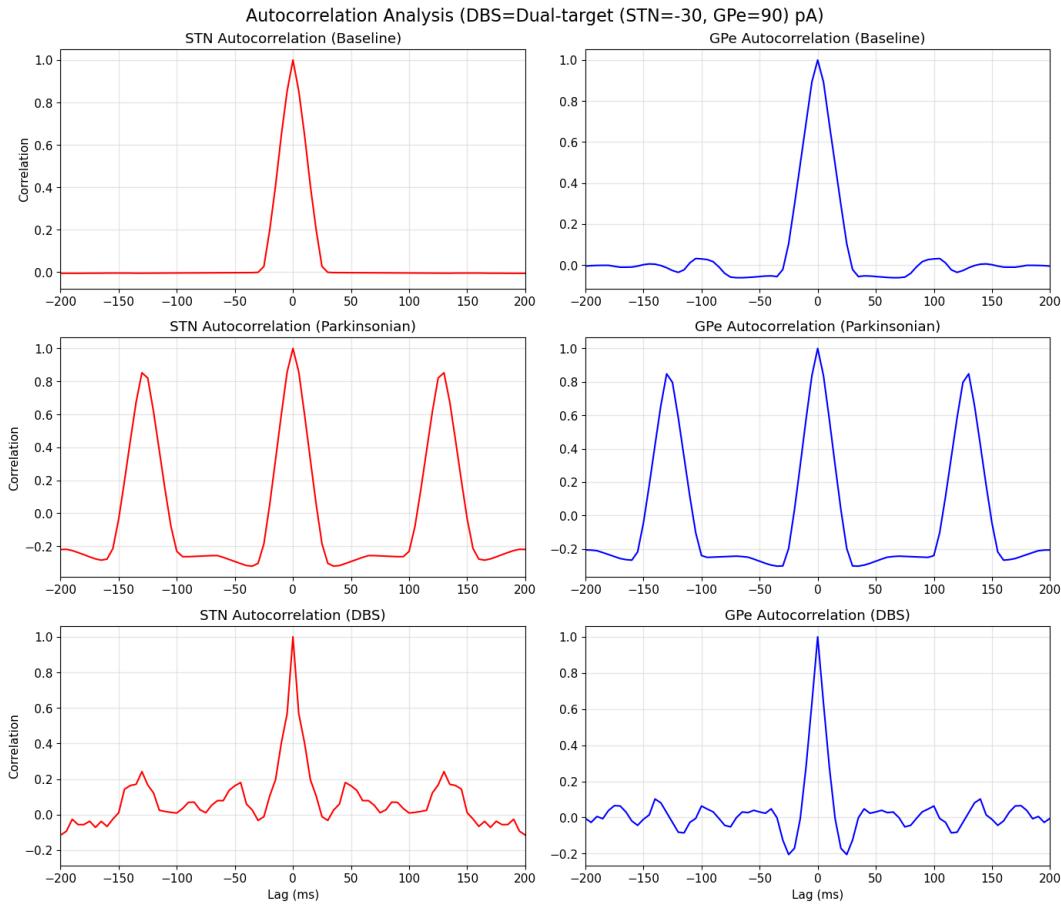


Figure 11: Spectrogram for the restored condition.

The autocorrelation is not completely flat, but it no longer shows strong oscillatory structure and looks closer to the baseline than DBS state. All other spectral measurement exhibits better behavior than with DBS simulation.

```

--- Testing amplitude: -180 pA ---

Dec 11 15:06:02 SimulationManager::set_status [Info]:
  Temporal resolution changed from 0.1 to 0.1 ms.

Dec 11 15:06:02 NodeManager::prepare_nodes [Info]:
  Preparing 805 nodes for simulation.

Dec 11 15:06:02 SimulationManager::start_updating_ [Info]:
  Number of local nodes: 805
  Simulation time (ms): 3000
  Number of OpenMP threads: 1
  Not using MPI

Dec 11 15:06:03 SimulationManager::run [Info]:
  Simulation finished.

```

Figure 12: Screenshot of final model initialization

Comparison

The main takeaway is that striatal inhibition acts as a trigger for Parkinsonian oscillations, which is consistent with previous work. Our results also show that DBS can suppress oscillations, but often at the cost of reduced firing rates, which may relate to clinical side effects.

One major difference is network size. The reference study used about 3,000 neurons, while our model used 800, which likely explains some slightly different results such as why our oscillations appeared around 8 Hz instead of higher beta-band frequencies.

In this work, we also explored restoration strategies by adding excitatory input to the GPe and adjusting STN inhibition.

A key limitation of this model is that the connectivity is random, whereas real neural circuits are highly structured and region-specific.

Finally, DBS here was modeled as a constant signal. In clinical practice, adaptive DBS^[3] is now used, where stimulation is only applied when pathological oscillations are detected, reducing side effects and improving battery life.

1. https://nest-simulator.readthedocs.io/en/v2.18.0/getting_started.html ↵
2. A. Kumar. The role of inhibition in generating and controlling Parkinson's disease oscillations in the basal ganglia. *Frontiers in Systems Neuroscience*, 2011. URL: <https://doi.org/10.3389/fnsys.2011.00086>. ↵
3. Little, S., Pogosyan, A., Neal, S., Zavala, B., Zrinzo, L., Hariz, M., Foltyneie, T., Limousin, P., Ashkan, K., FitzGerald, J., Green, A. L., Aziz, T. Z., & Brown, P. (2013). Adaptive deep brain stimulation in advanced Parkinson disease. *Annals of neurology*, 74(3), 449–457.
<https://doi.org/10.1002/ana.23951> ↵