Modeling Parkinson's Neural Dysfunction via Coherence Decay

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Abstract

Parkinson's disease is characterized by not simply just a loss of dopaminergic neurons but also as a continuous degradation of neural signal transmission within motor circuits. This paper consists of the introduction of a theoretical model that integrates a coherence-decay function $C(t)$ into the Fitzhugh-Nagumo model to simulate this degradation of signal transmission. We draw on parallels from quantum decoherence and synaptic weaking and modify the original model by making the external input a time-dependent exponential decay. Through numerical and analytical analysis, we display the gradual transition from regular neuronal firing to silence, a hallmark of Parkinsonian neurodynamics. Parameter sweeps and conceptual results help validate the model's ability to emulate the various stages of neural decline. Lastly, we also explore its potential use in fields such as brain-machine interfaces along with strategies of dynamic neuromodulation. This work offers a minimal yet also extensible framework that links degenerative neural behavior to biophysical theory.

While this paper does present a novel, coherence-inspired modification to the classical Fitzhugh-Nagumo system, it also investigates the effectiveness of parameter tuning in capturing the many distinct electrophysiological profiles observed as Parkinson's progresses in patients. The tuned parameters include threshold sensitivity, recovery time constants, and coherence decay rate. The ability of this model to effectively transform from oscillatory to near-silent regimes without any requirement of complex circuitry or stochastic input highlights its strength for simulating neural dysfunction as a first-principles tool. Phase portraits coupled with time-series outputs support the predictions made theoretically, giving us visual confirmation of silencing of neurons driven by coherence decay.

The proposed system gives a foundational framework for studying the observed signal deterioration in other neurodegenerative conditions, beyond its immediate implications for Parkinson's. To combat the weaknesses of the current model, potential extensions include coupling multiple neurons, introducing noise-modulated coherence, or simulating treatment effects such as deep brain stimulation. The modified model invites further

interdisciplinary exploration which bridges theoretical physics, computational neuroscience, and clinical neuroengineering.

1 Introduction

1.1 Parkinson's Disease

Parkinson's disease – hereafter referred to as PD – is a neurological condition that causes increasing movement abnormalities such as issues with movement, tremor, stiffness, and decreased balance [1]. PD primarily affects neurons in the substantia nigra, cells which produce dopamine – a neurotransmitter which is extremely important for the regulation of voluntary motion via the basal ganglia circuit.

As the dopamine-producing nerve cells experience degradation, the signal transmissions to the striatum become increasingly more disrupted, ultimately leading to abnormal activity in regions such as the subthalamic nucleus and the globus pallidus interna, consequentially giving rise to excessive inhibition of the thalamus and diminished motor output [2].

At its core, this dysfunction is due to a breakdown of signal fidelity – the accuracy and efficiency at which electrical impulses propagate through neural circuits in the brain. This significant degradation calls for a more in-depth investigation with the use of theoretical models that capture signal disruption at the most fundamental level. Hence, in this paper, the author explores a modification of the Fitzhugh-Nagumo model using principles rooted in quantum coherence to conceptually show this degradation through the decay of coherence, offering a new perspective on the signal failure that takes place in Parkinsonian systems.

1.2 The Role of Signal Fidelity

In this section, building upon the previously defined term 'signal fidelity', the author will discuss its exact role in neuroscience and, in particular, PD. Expanding on a previously mentioned concept, in a healthy brain, neuronal communication not only relies on the presence of neurotransmitters but also on the proper and reliable propagation of oscillatory signals that coordinate activity across different regions of the brain.

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[1] National Institute of Neurological Disorders and Stroke, "Parkinson's disease," *national institute of neurological disorders and stroke*, Mar. 05, 2025. https://www.ninds.nih.gov/health-information/disorders/parkinsons-disease (accessed Jun. 10, 2025).

[2] G. Mandybur, "Parkinsons disease," *Mayfieldclinic.com*, 2018. https://mayfieldclinic.com/pe-pd.htm (accessed Jun. 10, 2025).

A method to measure this coordination is through spectral coherence – a method which essentially quantifies the temporal alignment of the activity of the aforementioned oscillatory signals. Although the extent of research regarding this coherence has mostly been studied in Alzheimer's disease – where the reductions of alpha-based coherence reflect the deterioration of the network connectivity in the cortex – its relevance extends to PD as well. A reduction in coherence acts as a signal of a breakdown in the brain's ability to accurately maintain consistent inter-regional communication, potentially mirroring the disrupted signal transmission often observed in Parkinsonian motor circuits [3].

This disruption is even more evident in studies of a therapeutic intervention of PD called 'Deep Brain Stimulation' (DBS). Generally, traditional DBS of high-frequence improves motor function, however, recent studies display the better effectiveness of patterned burst stimulations: they yield more significant reductions in bradykinesia. More precisely, the therapeutic efficacy of these patterned burst stimulations directly correlates with their ability to suppress beta-band oscillations. Beta-band oscillations are rhythms that are synchronized in PD abnormally and are known to disrupt flexible neural communication. Further emphasizing that PD is not simply a disease of loss of dopamine but of a deterioration in signal variability and precision, it must be noted that dopaminergic medication and conventional DBS suppress these beta rhythms [4].

1.3 The Need for a Theoretical Physics Approach

The findings from the previous two sections suggest that the motor symptoms of PD emerge not just from the loss of signal, but from the failure of the signals' ability to adapt, synchronize appropriately, or maintain integrity over time, or, in simpler terms, a loss of fidelity. Thus, modeling this degradation theoretically becomes important to understanding, and eventually intervening in, the core informational deficits that define Parkisonian circuits.

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[3] R. McMackin *et al.*, "Measuring network disruption in neurodegenerative diseases: New approaches using signal analysis," *Journal of Neurology, Neurosurgery & Psychiatry*, vol. 90, no. 9, pp. 1011–1020, Sep. 2019, doi: https://doi.org/10.1136/jnnp-2018-319581.

[4] W. M. Grill, "Temporal pattern of electrical stimulation is a new dimension of therapeutic innovation," *Current Opinion in Biomedical Engineering*, vol. 8, pp. 1–6, Dec. 2018, doi: https://doi.org/10.1016/j.cobme.2018.08.007.

2 Theoretical Background

2.1 The Hodgkin-Huxley Model

To better understand the Fitzhugh-Nagumo Model, it is essential to have a brief understanding of the model it attempts to simplify: the Hodgkin-Huxley Model (HH).

Hodgkin and Huxley characterized the time- and voltage-dependency of the ionic conductances that underpin an action potential in the squid giant axon using the voltage clamping methods, which were first devised by Cole (1949) and Marmont (1949) and then refined by Hodgkin et al. (1952). Furthermore, they created a coherent mathematical model that correctly predicted the action potential's waveform as well as a number of other physiological characteristics, including the refractory time, the action potential's axon propagation, anode break excitation, and accommodation. There are few quantitatively predictive theories in biology, and this study is notable for being one of the best attempts to combine computational and experimental methods to comprehend a crucial problem in neuroscience [5].

Hodgkin and Huxley described four equations in their model, and below is a scanned image from their original paper sourced from [6] (Figure 1):

Summary of equations and parameters

We may first collect the equations which give the total membrane current I as a function of time and voltage. These are:

$$I = C_M \frac{dV}{dt} + \bar{g}_K n^4 (V - V_K) + \bar{g}_{Na} m^3 h (V - V_{Na}) + \bar{g}_l (V - V_l), \quad (26)$$

where

$$dn/dt = \alpha_n (1 - n) - \beta_n n, \quad (7)$$

$$dm/dt = \alpha_m (1 - m) - \beta_m m, \quad (15)$$

$$dh/dt = \alpha_h (1 - h) - \beta_h h, \quad (16)$$

Figure 1 HH's Equations

While an extensive discussion of each term in these equations isn't central to the aims of this paper, it is important to foreground the model's visible complexity; HH consists of four

[5] D. A. Baxter and J. H. Byrne, "Dynamical Properties of Excitable Membranes," *Elsevier eBooks*, pp. 409–442, Jan. 2014, doi: https://doi.org/10.1016/b978-0-12-397179-1.00014-2.

[6] "Hodgkin & Huxley Equations," *www.sas.upenn.edu*. https://www.sas.upenn.edu/LabManuals/BBB251/NIA/NEUROLAB/APPENDIX/EQUAT.HH/HHEQUAT.HTM (accessed Jun. 11, 2025).

non-linear ordinary differential equations and a plethora of gating variables to model voltage-dependent ion channel dynamics. This complexity, though physiologically accurate, makes it computationally intensive for conceptual and large-scale explorations.

2.2 The Fitzhugh-Nagumo Model

To combat the complexity of HH, Fitzhugh derived a model which had a clear distinction between slow-timescale and rapidly evolving variables. m and v are both rapidly evolving variables, causing a faster change in potential and sodium channels activate faster. Meanwhile, n and h are both slow-timescale variables, i.e., the sodium channel deactivates slowly, and the potassium channel activates quickly [7].This separation motivated a model consisting of two variables:

$$C_m \frac{dV}{dt} = -g_K n^4 (V - V_K) - g_{Na} m_3^{\infty}(V)(0.8 - n)(V - V_{Na}) - g_L (V - V_L) + I_{appl}$$

$$n_w(V) \frac{dn}{dt} = n_{\infty}(V) - n$$

However, another observation due to Fitzhugh were the shapes of the V-nullcline and n-nullcline as are similar to those of a cubic function and an approximation of a straight line respectively, both being in the physiological range of the variables. This led to a polynomial reduction-based model:

$$\frac{dv}{dt} = v(v - \alpha)(1 - v) - w + I$$

$$\frac{dw}{dt} = \varepsilon(v - \gamma w)$$

The resulting dimensionless system can be interpreted as follows: v is the rapidly evolving variable representing potential, w is the slow-timescale variable representing the sodium-gating variable, and α, γ, and ε are constants with the following conditions: $0 < \alpha < 1$ and $\varepsilon \ll 1$, considering the slow kinetics of the sodium channel; these constants control the nullcline geometry and timescale separation. Moreover, I is the assumed constant input current. The equations above are what the Fitzhugh-Nagumo Model (FN) consist of [8].

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[7] A. J. Vollebregt, "The Fitzhugh-Nagumo model," *Research portal Eindhoven University of Technology*, 2004. https://research.tue.nl/en/studentTheses/the-fitzhugh-nagumo-model (accessed Jun. 12, 2025).

[8] C. Rummel, "Lecture 6 The FitzHugh-Nagumo Model," *Benesco*, Feb. 28, 2020. https://benesco.ch/seminar/lecture-notes-18-19-2/ (accessed Jun. 12, 2025).

2.3 Signal Propagation & Spiking Behavior

An analysis of the signal propagation and spiking behavior of FN is crucial to understand how we can modify the model with quantum coherence terms to model the decay in neural transmissions.

With two ion current dynamics—potassium and sodium ion flows—FN encompasses the whole temporal range of neural excitation and propagation. Despite being more biologically realistic than FN, HH only allows for the simultaneous observation of temporal projections of its four-dimensional phase trajectories. This has the drawback of preventing the observation of the model's solution through a single-phase portrait.

Moreover, FN's complete solution may be plotted in just two dimensions (Figure 2). As a result, the solution—that is, the states in which the system settles—is also shown, in addition to the temporal trajectory of activity (which is preserved from HH) (i.e., qualities not disclosed by showing the four dimensions of HH). Accordingly, FN not only captures the essential details of the excitation and propagation properties that contribute to single neuron spiking that HH does, but it also reveals nonlinearities and feedback that contribute to spiking activity when its complete solution is plotted on two dimensions [9].

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[9] L. H. Favela, "The dynamical renaissance in neuroscience," *Synthese*, Sep. 2020, doi: https://doi.org/10.1007/s11229-020-02874-y.

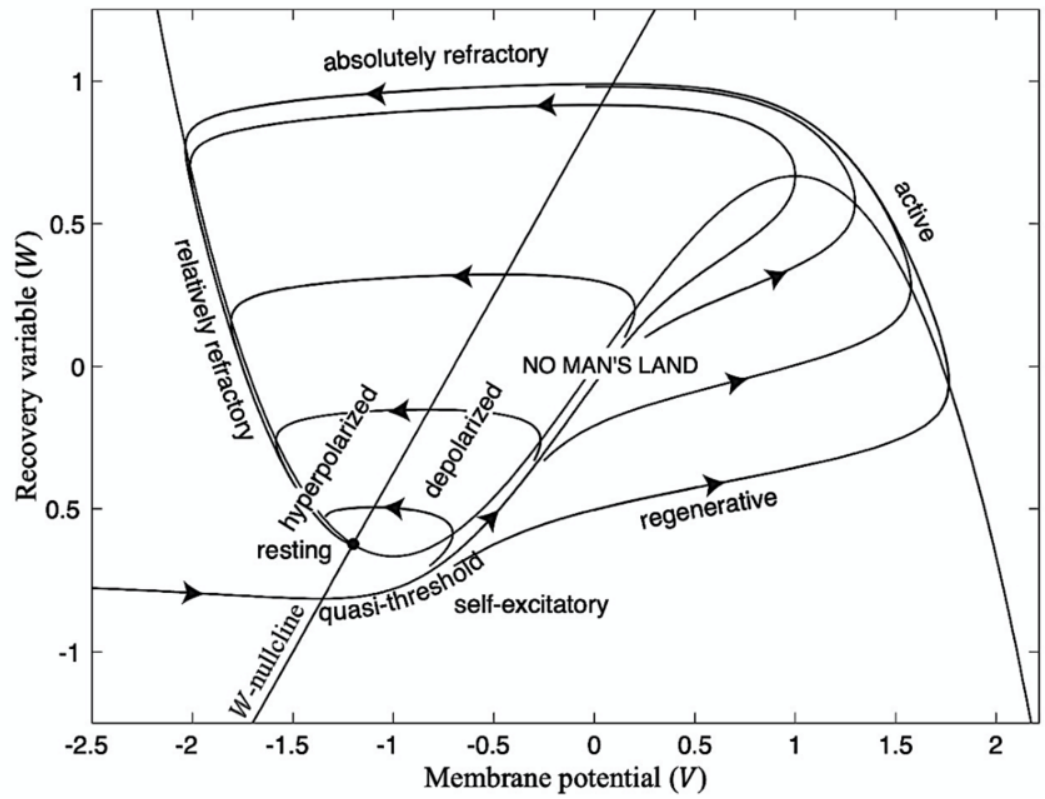

Figure 2 Phase Space Plot of FN

2.4 Limitations of Classical FN

The limitations of FN are highlighted by its inability to fully capture the multiple nuances of real neuron behavior. Moreover, due to it being constructed using ordinary differential equations, it doesn't account for spatial variations in the structure of neurons. Additionally, it doesn't account for observable behaviors such as bursts or adaptions due to its simplicity [10].

The model's assumption that signal fidelity is static, however, is its most significant drawback, especially when considering Parkinson's disease. The progressive loss of signal integrity seen in Parkinsonian neural circuits is not represented by any mechanism in the classical FHN model. It assumes that responsiveness and excitability won't change over time, which runs counter to PD's neurodegenerative progression. [11].

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[10] D. Cubero, J. P. Baltanás, and J. Casado-Pascual, "High-frequency effects in the FitzHugh-Nagumo neuron model," *Physical Review E*, vol. 73, no. 6, Jun. 2006, doi: https://doi.org/10.1103/physreve.73.061102.

[11] C. de Solages, B. C. Hill, M. M. Koop, J. M. Henderson, and H. Bronte-Stewart, "Bilateral symmetry and coherence of subthalamic nuclei beta band activity in Parkinson's disease," *Experimental Neurology*, vol. 221, no. 1, pp. 260–266, Jan. 2010, doi: https://doi.org/10.1016/j.expneurol.2009.11.012.

To address this in depth, this paper introduces a quantum coherence-inspired modification to the FN model. By implementing a time-dependent coherence decay term, the model aims to conceptually simulate the continuous deterioration of signal fidelity – capturing an aspect of cortical dynamics that most standard models tend to overlook.

3 Proposed Model Construction

3.1 Coherence Decay

In physics terms, the ability of a quantum mechanical system to produce interference patterns and sustain its quantum mechanical states steadily is known as quantum coherence, i.e., quantum states are well-defined and behave regularly and predictably in a coherent state. This makes it possible to precisely measure and regulate the system's quantum mechanical characteristics. However, incoherence is always the result of interactions with the environment, particularly in quantum physics. The system loses coherence and becomes decoherent as a result of these interactions, which cause phase shifts and uncertainty in the quantum mechanical states. As a result, in many quantum mechanical systems, quantum coherence is a fragile state that can only be sustained for brief periods of time [12].

This incoherence which leads to a decoherence system can be represented as a function of time as $C(t)$; in other words, we will define this decay in coherence as

$$C(t) = e^{-\lambda t}$$

where lambda represents the rate of coherence decay.

The coherence decay function used in this paper directly follows most quantum models of open systems in which the off-diagonal components of the decreased density matrix degrade exponentially, which is characterized by the decoherence of a quantum state [13]. Moreover, this form also appears in neuroscience where a normal single exponential model of postsynaptic currents was used to represent the synaptic connection between several

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[12] "Quantum Coherence," *www.cronologic.de*. https://www.cronologic.de/glossary/quantum-coherence (accessed Jun. 13, 2025).

[13] F. R. Klausen and S. Warzel, "Decoherence is an echo of Anderson localization in open quantum systems," *arXiv.org*, 2023. https://arxiv.org/abs/2310.09880 (accessed Jun. 13, 2025).

neuronal groups, particularly in a study titled ‘A Multi-Scale Computational Model of Excitotoxic Loss of Dopaminergic Cells in Parkinson's Disease’, amongst others [14].

3.2 Biological Interpretation of $C(t)$

As specified in the Introduction, the effective transmission across synapses progressively reduces due to the degeneration of dopaminergic neurons in PD. Thus, $C(t)$ serves as a proxy for synaptic-efficiency in a time-dependent context. This biological interpretation will be discussed in depth in and after 3.4.

It must also be explicitly stated that this paper does not imply that the human brain is a quantum computer; the use of an exponential decay function in this model is not meant to insinuate the existence of quantum coherence in biological contexts, however, it simply draws on mathematical parallels with observed signal fidelity degradation in quantum physics and the analogous loss of synaptic effectiveness observed in neurological disorders such as PD.

3.3 Modified Equations

As has been stated numerous times in this paper thus far, FN has multiple limitations, with the most glaringly obvious one when taken in the context of PD is the fact that it doesn’t account for degradation over time. To mitigate this error, we will introduce loss of coherence as a function of time as a multiplicative term in the pre-existing model as

$$\frac{dv}{dt} = v(v-\alpha)(1-v) - w + Ie^{-\lambda t}$$

$$\frac{dw}{dt} = \varepsilon(v - \gamma w)$$

In simpler mathematical terms, as the value of $-\lambda t$ increases, the input current reduces. In other words, input loses effectiveness as time increases. In this modified model, we have ensured that all the other variables remain unaffected so that we can isolate signal fidelity. It would also be right to explicitly state that this model is not a reinvention of the classical FN model, yet more of a biologically meaningful overlay that considers the decay in neural transmission effectiveness.

In the preceding section, we aim to examine its predicted dynamics and how the neuron’s responsiveness changes depending on our coherence function.

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[14] Vignayanandam Ravindernath Muddapu and V. Srinivasa Chakravarthy, “A Multi-Scale Computational Model of Excitotoxic Loss of Dopaminergic Cells in Parkinson’s Disease,” *Frontiers in Neuroinformatics*, vol. 14, Sep. 2020, doi: https://doi.org/10.3389/fninf.2020.00034.

3.4 Theoretical Predictions

To evaluate the behavior of our modified model, we must evaluate it for different rates of decay in Figure 3:

λ	**Meaning**	**Expected Behavior**
0	No decay	Standard, periodic spiking
Small	Slow decay	Gradual loss in spike frequency
Moderate	Noticeable decay	Spikes weaken and fade over time
Large	Rapid decay	Spiking gets suppressed quickly
$t \to \infty$	Input gone	System flatline

Figure 3 Theoretical Predictions of Our Modified Model in a Tabular Format

4 Conceptual Results

4.1 Behavior Over Time

For this section, we will divide the time into three separate time stages: Early, Middle, and Late.

4.1.1 Early Time

In this time stage, the coherence would be ~ 1. This implies that our model will follow classical FN behaviors where the neuron is responsive and all neural transmissions are completely effective.

4.1.2 Middle Time

Currently, the coherence is decaying. Here, the frequence of the neural transmissions will drop and there will be spikes in the amplitude. Moreover, the signal reliability would be inconsistent.

4.1.3 Late Time

At the stage, the coherence would be ~ 0. This implies that the input would be nearly over or majorly diminished and the neuron would be silent or firing erratically, depending on the value of w.

Figure 4 shows a graphical representation of the differences in these three time-stages of decay. We used the Desmos Graphing Calculator to plot three different waves where the red

wave represents Late Time, green wave represents Middle Time, and blue wave represents Early Time.

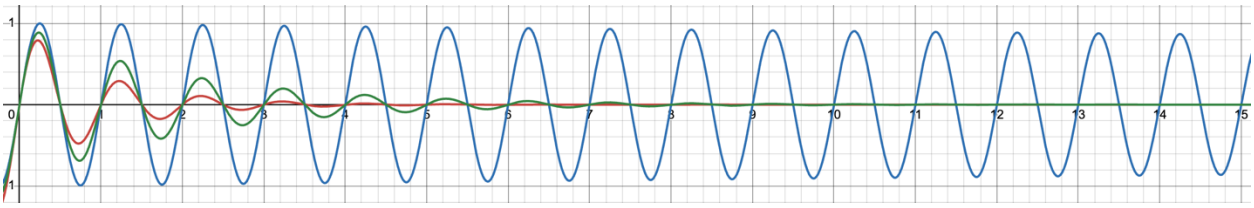

Figure 4 Graph of Neural Activity Over Time for Three Different Time Stages

We made use of three different example equations to simplify this process:
Early Time: $e^{-x}\sin(2\pi x)$
Middle Time: $e^{-0.5x}\sin(2\pi x)$
Late Time: $e^{-0.01x}\sin(2\pi x)$
where the sine function represents the spike wave and the variable x represents time.

4.2 Model vs Reality

In this section, we'll compare our modified model to the symptoms experienced by PD patients. As observed in Figure 5, neural activity initially replicates regular spiking behavior. However, as the coherence function decays over time, the input continuously loses its effectiveness, leading to a reduction in the frequency of spikes. Additionally, we can map this spiking behavior to symptoms observed by PD patients which we highlighted in the Introduction. The mapping has been done in Figure 5.

Model Behavior	**PD Symptom**
Reduced amplitude	Bradykinesia
Weakening spikes	Tremors
Complete silence	Rigidity & Freezing

Figure 5 Table Mapping Model Behavior to PD Symptoms

Admittedly, this model does not replicate the entire complexity of the basal ganglia-thalamocortical loops, it conceptually reflects the way reduced signal fidelity can cause impaired motor output – a key feature observed in Parkinsonian neurodynamics.

4.3 Parameter Variation

To gauge the robustness of the modified model, we adjusted several key parameters individually, including α, ε, γ, v, w, and λ and coded a python program for the evolution of v and w with time and a corresponding phase portrait (APPENDIX A).

Figure 6 shows the evolution with time of the v and w for each different parameter set as:
$\alpha = 0.3$,
$\varepsilon = 0.01$,
$\gamma = 1.0$,
$v(0) = 0.1$, and
$\lambda = 0.05$.

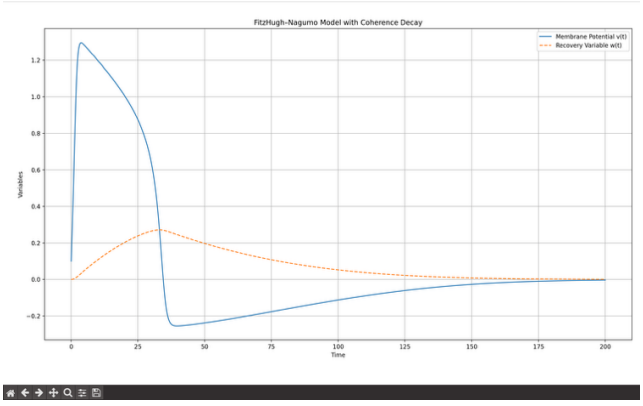

Figure 6 Evolution of v and w Over Time

As we observe Figure 6, the system shows an initial spike in v which is followed by a gradual decay to rest, staying consistent with our theoretically predicted results. Meanwhile, w mirrors the decay in activity; it rises briefly and then stabilizes gradually as the system approaches a non-oscillatory equilibrium.

Figure 7, on the other hand, plots w against v.

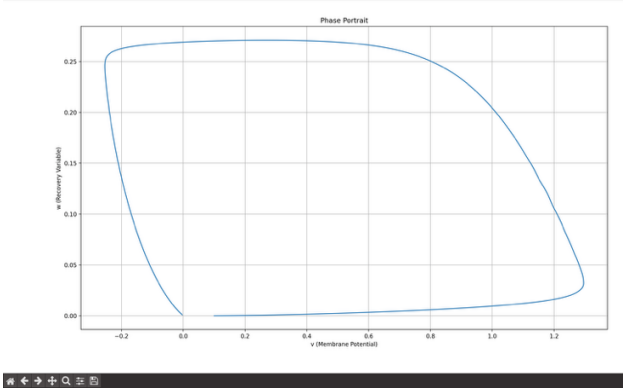

Figure 7 Graph of v against w

Unlike Figure 2, which displays a fixed space plot of FN, the path here spirals inwards and stops at one point. This confirms our prediction of the system's loss of periodic behavior due to the degradation of the input, reflecting the transition from excitation to silence. This absence of limit cycles which were present in Figure 2, Figure 6 highlights the suppression of spiking activity over the long term, which perfectly aligns with the paper's main objective

of simulating signal degradation which serves as an analog to that observed in Parkinsonian circuits.

4.4 Strengths vs Weaknesses

Below is a comparative table (Figure 8) which does a strength and weakness analysis of our modified model:

Strengths	Weaknesses
Makes use of a single, biologically interpretable decay parameter to accurately capture the progressive nature of PD.	Lacks network interactions, population-level behavior, and synchrony since it only models a single neuron.
Maintains mathematical simplicity.	The model does not capture feedback loops central to basal ganglia-thalamocortical dynamics.
Without requiring network-level complexity, our modified equations model a core feature of PD, temporal degradation.	The model assumes that coherence is smooth and deterministic, ignoring noise, nonlinearities, and more.
Offers a conceptual bridge between signal fidelity, and neurodegenerative signal failure.	While the model is interpretable, it lacks empirical validation.
It's a flexible framework, i.e., it can be extended to population behavior, stochasticity, or feedback in future models.	It cannot predict clinical symptoms or treatment responses such as those after DBS.

Figure 8 Strengths and Weaknesses of the Modified FN.

5 Future Directions

5.1 Physics-Led Understanding of Neural Degradation

Building on the framework of signal fidelity we introduced in this paper, future work may ground neural degradation in formal physics metrics. The free-energy principle, which is a dominant theory in neuroscience, proposes the unification of perception, action, and learning within a single framework which makes use of the minimization of energy. [15]

[15] K. Friston, "The free-energy principle: a unified brain theory?," *Nature Reviews Neuroscience*, vol. 11, no. 2, pp. 127–138, Jan. 2010, doi: https://doi.org/10.1038/nrn2787.

Meanwhile, extending our modified model to simulate Parkinsonian physiology could consist of the quantification of disease progression in terms of increases in entropy, which may call for a move of disease modeling from descriptive symptomatology to a theory based on physics, centered on informational thermodynamics.

5.2 Brain-Machine Interfaces

No doubt, as Brain Machine Interfaces (BMIs) rapidly increase in interfacing with clinical populations, ensuring that they account for concepts such as signal integrity degradation will be extremely critical. Researchers highlight that it is integral for BMI research to confront fundamental foundational questions about everything related to information in the brain, from encoding to maintenance [16]. Meanwhile, adaptive deep brain stimulation (aDBS) has displayed a direct correlation between the effectiveness of closed-loop systems with the ability to dynamically adjust to neural state changes in real time [17]. Future BMI design could incorporate coherence-based decoding algorithms that monitor the coherence function and adjust the input stimulation accordingly. This may potentially improve the reliability of signal translation in conditions such as PD.

5.3 Modeling Other Disorders

As specified in 4.4, the current model has multiple weaknesses of its own. For example, its single-neuron and deterministic. However, there is a scope for it to be expanded. Researchers discuss the utility of simple yet plausible neuron models which help simulate the diverse firing patterns [18]. We can build on this. The coherence decay concept may be immersed in networks of spiking neurons or even hybrid models. Additionally, the introduction of stochastic or dynamics dependent on feedback could allow our model to simulate recovery through medication and therapy or resilience through neuroplasticity. These further modifications may lead to dynamic simulations that more closely mimic real disease progression.

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Declarations

Ethical Approval

Not required since no living beings were affected in this study.

Funding

No funding was received for this paper.

Availability of Data and Materials

Data is provided within the manuscript.

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[6] "Hodgkin & Huxley Equations," *www.sas.upenn.edu*. https://www.sas.upenn.edu/LabManuals/BBB251/NIA/NEUROLAB/APPENDIX/EQUAT.HH/HHEQUAT.HTM (accessed Jun. 11, 2025).

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Appendix

Appendix A – Python Program Developed to Validate Model

```python
import sys
print(sys.executable)

import numpy as np
import matplotlib.pyplot as plt
from scipy.integrate import solve_ivp

alpha = 0.3
epsilon = 0.01
gamma = 1.0
I_ext = 0.5
lambda_decay = 0.05

def C(t, lambda_decay):
    return np.exp(-lambda_decay * t)

def fn_with_decay(t, Y):
    v, w = Y
    dvdt = v * (v - alpha) * (1 - v) - w + I_ext * C(t, lambda_decay)
    dwdt = epsilon * (v - gamma * w)
    return [dvdt, dwdt]

v0 = 0.1
w0 = 0.0
Y0 = [v0, w0]

t_span = (0, 200)
t_eval = np.linspace(*t_span, 2000)

sol = solve_ivp(fn_with_decay, t_span, Y0, t_eval=t_eval, method='RK45')

plt.figure(figsize=(12, 5))
plt.plot(sol.t, sol.y[0], label='Membrane Potential v(t)')
plt.plot(sol.t, sol.y[1], label='Recovery Variable w(t)', linestyle='--')
plt.title('Fitzhugh-Nagumo Model with Coherence Decay')
plt.xlabel('Time')
plt.ylabel('Variables')
plt.legend()
plt.grid(True)
plt.tight_layout()
plt.show()
```

This program was run, and screenshots were taken of the generated plots and are present in this paper as Figure 6 and Figure 7.