Hyper-synchronicity in simplified neural networks: modelling the emergence & propagation of focal seizures in the brain

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October 2025

1 Abstract

2 Introduction

Seizures occur when excessive, abnormal or synchronous neuronal activity in the brain impair brain function which, depending on the area in the brain, can cause a loss of consciousness, muscle stiffening and spasms (tonic-clonic) and in some cases death (status epilepticus).

Epilepsy is a condition marked by the random onset of seizures in the brain. Refractory epilepsy, defined as seizures which do not respond to ≥ 2 anti-epilpectic seizures [1]. These account for 30-40% of all epipletic cases yet 80% of the cost in the US [2].

Fatalities in seizures are typically attributed to status epilepticus: status epilepticus occurs when the duration of a seizure has exceeded 5 minutes and is especially a cause for concern due to the neuronal death within the brain - caused by the neurotoxicity of the high concentration of certain neurotransmitters - as well as increasing the likelihood of cardiac arrest & arythmia and stroke. This is fatal in $10 - 22\%$ of cases of status epilectus [3].

Holistically, epilepsy - affecting x people around the world (citation please) & being one of the most prevelant and rapidly rising neurological disorders around the world - accounts for a loss of x disability-adjusted life years (DALYs) per $100,000$ [4].

Seizures are a complex neurological disorder with many different factors leading to its formation. One such factor is the hyper-synchronisation of neuronal activity in the brain. This is caused by the balance of excitatory (gluatamate) and inhibitory (γ-aminobutyrate acid (GABA)) neurotransmitters in the brain (E/I balance): the higher concentration of glutamate allows for a higher frequency firing of action potentials along axons, which increases the likelihood of this further propagation. [5]

Current drugs aim to provide symptomatic relief by affecting the ion channels in the axons and dendrites to restore the E/I balance. However, given that this is merely symptomatic relief and does not give further light into how these signals can synchronise and propagate from focal seizures (occurring in one region of the brain) to generalised seizures (across the whole brain), computational models have worked in conjunction with biochemistry to non-invasively explore this relationship [6] as this could help with treatments for refractory epilepsy.

I aim to explore how synchronisation in neuronal activity emerges in the brain and how this serves as the carriage for its propagation from focal to generalised seizures.

Computational models cited in the literature often present a significant barrier to entry: their reliance on sophisticated maths can obscure the core intuitive principles of seizure dynamics for a non-specialist audience and could make it difficult for this to be truly interdisciplinary. This includes students, researchers from adjacent fields like clinical medicine or molecular biology, and the growing community of independent scientists. For collaboration to flourish in tackling a multi-dimensional problem like refractory epilepsy, there is a need for conceptual frameworks that translate these technical findings into more accessible, intuitive models.

In response to this need, this paper introduces a highly abstracted model of seizure propagation: sacrificing biological detail in favour for making an accessible model by representing the brain as a network of simple excitable units (with the soma and dendrites are represented by nodes; axons and axon terminals are represented by edges). The primary goal of this abstraction is to demonstrate how random, local hyper-excitability of neurons (as well as global parameters) can lead to large-scale synchronisation within the network - characteristic of focal seizure.

Visualising how certain parameters affect this initial propagation (and later for generalised seizures and sustained generalised seizures (status epilepticus)) will make the entrance into this kind of research easier.

3 Background

4 Model

5 Discussion

6 Conclusion

7 References

[1] 10.1016/j.seizure.2021.06.028

[2] 10.1016/j.seizure.2021.06.028

[3] https://epilepsysociety.org.uk/sites/default/files/2020-08/Chapter37Neligan2015.pdf

[4] https://pmc.ncbi.nlm.nih.gov/articles/PMC11669576/

[5] 10.1016/j.lfs.2020.118826

[6] 10.1016/j.seizure.2012.08.012

8 Biography

Reuben Okotie, 18, attending Bishop's Stortford College (in Bishop's Stortford, Hertfordshire).

Reuben has an interest to study general engineering with a focus on bioengineering and computational neuroscience, using this topic as an introduction to computational approaches in this field.

- 3D Design A-Level on a posture correction device
- Wrote blog entry on whether the design of cities can promote social cohesion
- Deputy Head of School & Academic Prefect at school

He has achieved a Gold in Computational Physics Challenge (2025)