MSc in Computational Cognitive Neuroscience





Competition And Cooperation through Spike Timing Dependent Plasticity as Driving Forces in Self-Organised Quasi-Criticality.

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Introduction

Critical phenomena emerge during phase transitions, where systems shift states. Evidence suggests the brain operates near a critical point [1], optimising information transmission and storage [2]. How the brain remains at this point is still a question at large. Our research question investigates the homeostatic mechanism for self-organised quasi-criticality.

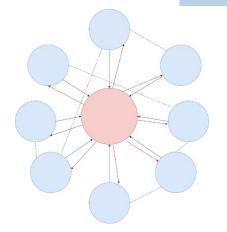
A promising candidate for this mechanism is synaptic plasticity, which has shown promising applications in self-organised, critical neural networks [3,4]. Spike Timing Dependent-Plasticity (STDP) can organise a network towards metastable states [4]. Previous work has also shown that when at the critical point, spiking neural networks exist in a balance of competitive and cooperative environments [5].

We, therefore, hypothesise that networks are capable of selforganisation towards criticality through mechanisms of cooperation and competition, facilitated through STDP.

- Fig.1: Our network consists of a total of 1000 neurons, 800 being excitatory and 200 inhibitory.
- To simulate the spiking of a neuron we use the Izhikevich model.
- Excitatory neurons are split into modules (blue) of 100 with an edge density of 0.1. Inhibitory neurons exist in an inhibitory pool (red).
- Focal connections (blue arrows) exist from excitatory to inhibitory; with every four neurons from an excitatory module connecting to the same neuron from the inhibitory pool
- Diffusion connections (red arrows) connect from the inhibitory pool to every other neuron in the network.
- Thus, one module may inhibit all others.
- Random weak connections (dashed lines) are established between modules.
- Each connection/synapse is subject to increase or decrease according to STDP.

Methods

Fig 1: Schematic diagram of the network



Results

- We define p_{local} as the probability of an intermodular synapse firing. We define p_{gloabl} as the mean of all p_{local} . We expect p_{gloabl} to stabilise near intermediate values, as this aligns with [4].
- We will conduct a one-way ANOVA to compare the probability of intermodular synapse firing between our network with synaptic plasticity and a network without any synaptic learning rules.
- We will use OID [5], a state-of-the-art information-theoretic technique for dynamical systems, to confirm that this process is caused by upward causation.

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