

Mission Synaptech: Save Dr. Z's Mind – Report

Reference Paper->

Title: Canonical Microcircuits for Predictive Coding

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This paper outlines the organization of canonical cortical microcircuits and proposes their function in predictive coding.

Neuron Types

The simplified microcircuit modeled in this simulation includes:

Excitatory neurons (E-cells):

Modeled as regular spiking pyramidal neurons. These form the bulk (~80%) of the network and propagate signals across the cortex.

Inhibitory neurons (I-cells):

Modeled as fast-spiking interneurons, such as basket cells. They account for ~20% of the network and help maintain stability through inhibition.

Connectivity Patterns

The network follows biologically-inspired connectivity rules:

E→E: Sparse, weakly connected to propagate excitation.

E→I: Strong and dense to quickly recruit inhibition.

I→E: Moderately strong to suppress runaway excitation.

I→I: Inhibitory loops to control interneuron activity.

Connections are probabilistic, e.g., a 10–20% chance of forming a synapse between any two neurons.

Neuron Models

The neurons are modeled using the Izhikevich model, which balances biological realism with computational efficiency. The model captures:

Regular spiking (pyramidal cells)

Fast spiking (interneurons)

Spike timing and adaptation

Computational Functions

This microcircuit performs several key computational functions:

Excitatory-Inhibitory Balance: Maintains network stability and dynamic range.

Spike-Timing Dependent Plasticity (STDP): Synaptic weights adjust based on timing of spikes, enabling learning and memory formation.

Predictive Coding: Based on the Rao & Ballard paper, cortical microcircuits may continuously compare top-down predictions with bottom-up inputs, generating error signals.

STDP Synaptic Plasticity

STDP is implemented as follows:

If a pre-synaptic spike occurs just before a post-synaptic spike, the connection is strengthened (long-term potentiation).

If a post-synaptic spike occurs before the pre-synaptic, the connection is weakened (long-term depression).

This timing-based learning rule mimics Hebbian learning in biological neurons.

Simulated Threat: Corticon

In our scenario, Corticon disrupts the inhibitory neurons' functioning, leading to hyperexcitation in the network.

Conclusion

This simulation shows how cortical circuits achieve balance and learn through plasticity. The paper also highlights how disruption in inhibitory control (as caused by Corticon) can result in network failure — mirroring pathological conditions like epilepsy, schizophrenia, or in our case, Dr. Z's compromised neural state.