Simulating Synaptic Depression to Infer Properties of Synaptic Transmission

Rono Xia[1], Meiyu Zhong[2], Joseph Ramirez[3], Mona Miraftab[4], and Mayank Mehta[5]

[1]UCLA, CASB 187 Capstone
[2]W. M. Keck Center for Neurophysics

Abstract

Modeling neural systems at the level of individual synapses contributes to the understanding of learning and memory and improves our understanding of the neural code, which in turn depends on synaptic transmission properties. This work uses a phenomenological model of short-term synaptic plasticity implemented in Python that accounts for synaptic depression. The model captures postsynaptic response based on neurotransmitter release probability in depression-dominated synapses, circumventing the complexity of modeling Ca^{2+} dynamics. We find that the postsynaptic neuron reaches a stationary value during presynaptic firing, and its amplitude decreases inversely with input frequency. This suggests synaptic depression may serve a frequency-filtering role.

1 Introduction

The brain's circuitry is composed of synaptic connections between neurons. Fast chemical synapses enable communication via neurotransmitter release triggered by Ca^{2+} influx through voltage-gated channels. Synaptic plasticity modifies these connections and contributes to functions such as learning and memory [1]. Long-term plasticity occurs over minutes to years, while short-term synaptic plasticity (STP) occurs over milliseconds, affecting neurotransmitter release probability [2].

Experimental data shows that repeated presynaptic spikes influence postsynaptic potentials (PSPs) on short timescales [3]. Synapses may exhibit depression or facilitation depending on how their responses change with closely timed inputs [4]. Most synapses are shaped by a combination of both mechanisms [5].

To better understand the neural code, we simulate synaptic transmission with plasticity using a simplified phenomenological model based on release probability, avoiding the full complexity of Ca^{2+} kinetics [6]. The model reproduces the depression-dominated behavior of Layer V pyramidal neurons from patch clamp data [7].

2 Method

Phenomenological Model of Synapse

The model assumes finite neurotransmitter resources in three states: effective (E), inactive (I), and recovered (R). Each presynaptic spike moves a fraction of recovered resources to the effective state.

The dynamics are governed by:

$$\frac{dR}{dt} = \frac{I}{\tau_{\text{rec}}} - U_{SE} \cdot R \cdot \delta(t - t_{AP}) \tag{1}$$

$$\frac{dE}{dt} = -\frac{E}{\tau_{\text{inact}}} + U_{SE} \cdot R \cdot \delta(t - t_{AP}) \tag{2}$$

$$I = 1 - R - E \tag{3}$$

ODEs are solved using Python's `odeint` for both uniform and Poisson spike trains. Parameters such as U_{SE} (release probability), τ_{rec} (recovery), and τ_{inact} (decay) are derived from empirical data.

3 Results

The model captures synaptic depression across a variety of input conditions. Stationary excitatory postsynaptic potentials (EPSPs) are reached under constant firing and decrease with increasing input frequency, demonstrating an inverse relationship.

Varying U_{SE} reveals its control over both transient and stationary components of PSP. High U_{SE} leads to faster depression and higher stationary EPSPs. The model reflects the concept of a "limiting frequency" beyond which postsynaptic response saturates [7].

4 Discussion

This simple model reproduces key properties of synaptic depression. It supports the idea that synaptic depression imposes a frequency limit on information transmission and enables transient sensitivity to firing changes. High U_{SE} leads to faster, more dynamic responses, suggesting temporal coding mechanisms may arise from short-term plasticity.

References

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