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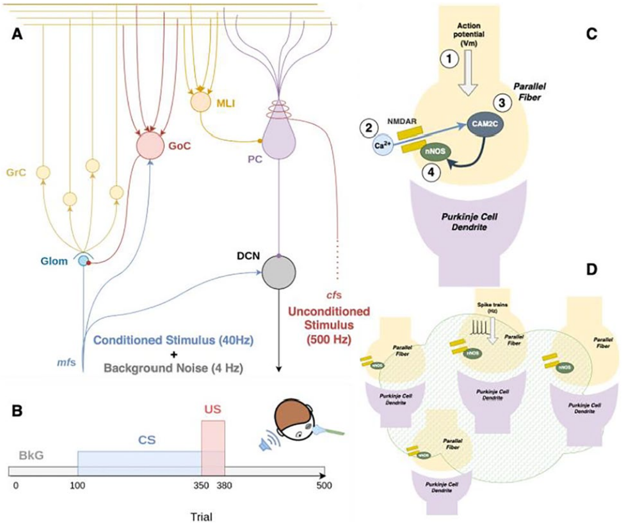

Figure 1: Spiking neural network with NODS mechanism. (A) SNN of the cerebellum microcircuit, with the different populations and detail of CS, US and Background Noise stimuli. (B) One trial of the EBCC protocol with timing of the stimuli. (C) The NO production mechanism at a single synapse. (D) NO as volume transmitter at different pf-PC synapses.

P269 Modeling Calcium-Mediated Spike-Timing Dependent Plasticity in Spiking Neural Networks

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Introduction

Calcium dynamics serve as bridge between neuronal activity and synaptic plasticity, orchestrating the biochemical cascades that determine synaptic strengthening (LTP) or weakening (LTD) [1]. Extending the work of Graupner and Brunel [2], Chindemi and colleagues recently introduced a data-constrained model of plasticity based on postsynaptic calcium dynamics in the neocortex [3]. The model has been developed for NEURON simulations capturing diverse plasticity dynamics with a single parameter set across pyramidal cell-types. In this work, we translated Chindemi’s model to a spiking neural network by implementing a point neuron model and a unified synapse, testing it across various calcium-concentration scenarios.

Methods

We developed our model using NESTML [4], an open-source language integrated with NEST [5] simulator, enabling the application of our models to diverse neural networks. The implemented neuron was built upon the existing Hill-Tononi (HT) model, which already incorporates detailed NMDA and AMPA conductance dynamics [6]. As in Chindemi, the synapse was instead based on the Tsodyks -Markram (TM) stochastic synapse model [7], allowing to manipulate vesicle release probability. Following paired pre-and post-synaptic activity calcium-dependent processes influence synaptic efficacy at both sides. Our implementation extends these established components to create a comprehensive framework that captures the relationship between calcium dynamics and synaptic plasticity while maintaining computational efficiency for network-scale simulations.

Results

We first validated our model for the TM stochastic synapse paired with HT modifications to account for calcium currents postsynaptic neuron. Then, we connected two neurons and stimulated either the pre - or post - synaptic neuron directly, creating respectively NMDA and VDCC calcium currents. Next, we tested the paired activation of pre- and post- synaptic neurons at varying time intervals. The results of these simulation s are comparable with the ones of Chindemi et al. Finally, we adjusted LTD and LTP thresholds to match calcium signal properties of pyramidal neurons across different cortical layers. Our simpler point neuron model successfully replicated findings obtained with multicompartmental models while maintaining computational efficiency.

Discussion

Our work implements calcium-dependent plasticity into an efficient model for spiking neurons. We validated that our point neuron approach reproduces the complex calcium dynamics and plasticity outcomes across different stimulation patterns. By maintaining the ability to capture layer-specific plasticity with adjusted LTP/LTD thresholds, we preserve biological accuracy while reducing computational demands. Our efficient implementation of calcium-dependent plasticity possibly enables large-scale spiking neural network simulations to study how synaptic mechanisms affect network functionality.

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P270 Subthreshold extracellular electric fields alter how neurons respond to naturally occurring synaptic inputs in temporal interference stimulation

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Introduction

Temporal interference (TI) stimulation enables noninvasive and spatially selective neuromodulation of deep brain structures [1,2]. This approach exploits the nonlinear response of neurons to electric fields by delivering multiple kHz-range oscillations, which interfere and generate an effective low-frequency envelope only at the target site [1,2]. This mechanism allows for selective activation of deep neuronal populations without affecting the overlying tissue. Recent studies have successfully applied this stimulation to the human hippocampus, showing significant effects on memory function [3, 4]. Despite its potential for clinical applications, the neural mechanisms underlying TI-induced effects remain poorly understood.

Methods

We used a biophysically accurate computational neuron model to investigate how subthreshold electric fields influence neural activity in the CA1 hippocampal pyramidal neurons. These neurons receive inputs from Schaffer collaterals, known to play an integral role in memory formation. To replicate this connectivity, we implemented AMPA and NMDA synapses at the proximal apical dendrites, with synaptic activity driven by hippocampal CA3 activity recorded *in-vivo*. The model neuron was placed in a uniform electric field, simulating the effects of an externally applied field between two conducting plates.

Results

Consistent with previously published modelling results [4], we observed that the electric field strength required to elicit action potentials grew with increasing carrier frequency. Moreover, the subthreshold electric field strength also depended on the orientation of the model neuron in the electric field, requiring higher amplitude when the neuron was perpendicular rather than parallel to the direction of the electric field. Following an long-term potentiation (LTP) induction protocol, the subthreshold stimulation affected the synaptic weight distribution by altering the spike timing, firing frequency, and inter-spike interval patterns. A similar effect was observed with naturally occurring synaptic inputs.

Discussion

In summary, our model shows that subthreshold electric fields alter how neurons respond to naturally occurring synaptic inputs by affecting underlying long-term synaptic plasticity processes. The impact of TI on synaptic plasticity may underlie its effects on memory enhancement, observed in human experiments. The stimulation efficacy is partly determined by the neuron orientation in the electric field, as not all neurons are affected equally. Since our study focuses on single-neuron processes, further research is needed to explore network-level effects.

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P271 Accelerated cortical microcircuit simulations on massively distributed memory

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Introduction

Comprehensive simulation studies of dynamical regimes of cortical networks with realistic synaptic densities depend on compute systems capable of running such models significantly faster than biological real time. Since CPUs still are the primary target for established simulators, an inherent bottleneck caused by the von Neumann design is frequent memory access with minimal compute. Distributed memory architectures, popularized by the need for massively parallel and scalable processing for AI workloads, offer an alternative.

Methods

We introduce extensible simulation technology for spiking networks on massively distributed memory using Graphcore's IPUs (https://www.graphcore.ai). We demonstrate the efficiency of the new technology based on simulations of the microcircuit model by [1] commonly used as a reference benchmark. The model represents 1~mm² of cortical tissue,