NMDA Receptor Dynamics Enable Single-Neuron Working Memory in Spiking Neural Networks

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Spiking Neural Networks (SNNs) are studied both as energy-efficient machine learning algorithms and biologically grounded models of neural computation. Reconciling these perspectives requires architectures that are both trainable and neurophysiologically plausible. This motivates exploring single-neuron mechanisms as a substrate for short-term working memory.

The NMDA receptor has been hypothesized to support such mechanisms in the brain. To test this hypothesis, we compare our NMDA-based SNN, which adheres to neurophysiological constraints, to a standard LIF-based SNN with learnable temporal parameters. Our model is a small-scale, two-layer network of fewer than 100 excitatory neurons, along with subcellular components. The soma is modeled using LIF dynamics, while dendritic activity and receptor dynamics are modeled as linear and non-linear systems of ordinary differential equations (ODEs). To isolate single-neuron memory mechanisms, we restrict the network to a feedforward architecture.

Our contributions are twofold. On the one hand, we perform bifurcation and stochastic analyses to identify bistable regimes. This results in closed-form conditions on the synaptic weights that induce bistability in the NMDA dynamics, which we use to initialize the NMDA model parameters. On the other hand, we empirically evaluate working memory. To this end, we design a delayed classification task where models are trained to classify sequences from the Spiking Heidelberg Digits (SHD) dataset. After training, we evaluate each model's capacity to accurately classify sequences after increasingly long, silent delay periods. The model parameters are optimized using surrogate gradient methods in conjunction with population-based training (PBT) for hyperparameter search.

We find that, while the baseline has superior accuracy when no delay is present, our NMDA model consistently outperforms it as the delay period exceeds the length of the input sequence. Furthermore, an ablation study using a linearized version of the NMDA dynamics eliminates this effect, indicating that the non-linear NMDA dynamics are crucial for the observed behavior. Notably, the baseline achieves competitive performance by adapting its temporal parameters to regimes that deviate from neurophysiological observations, whereas our model operates under fixed, biologically constrained dynamics. These results suggest that NMDA receptor dynamics provide a local, neuron-level memory mechanism that is complementary to recurrence and learned temporal parameters. As such, we provide support for the main hypothesis as well as a step toward extending SNN memory capacity beyond prior architectural and optimization-based approaches.