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# GABAergic control of backpropagating action potentials in striatal medium spiny neurons

Johannes Hjorth*[1], Misha Zilberter[2], Rodrigo F Oliveira[3], Kim T Blackwell[3] and Jeanette Hellgren Kotaleski[1,2]

Address: [1]School of Computer Science and Communication, Royal Institute of Technology, AlbaNova University Centre, 106 91 Stockholm, Sweden, [2]Department of Neuroscience, Karolinska Institute, 171 77 Stockholm, Sweden and [3]School of Computational Sciences and the Krasnow Institute for Advanced Study, George Mason University, Virginia 22030-4444, USA

Email: Johannes Hjorth* - hjorth@csc.kth.se

* Corresponding author

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## Introduction

Experiments have demonstrated the ability of action potentials to actively backpropagate in striatal medium spiny (MS) neurons, affecting the calcium levels in the dendrites [1-3]. Increased calcium levels trigger changes in plasticity [4,5], which is important for learning and other functions [6]. Studies in the hippocampus have shown that GABAergic input can modulate the backpropagation of action potentials from the soma to the distal dendrites [7]. The MS neurons receive both proximal feedforward GABAergic inhibition from fast spiking interneurons (FS), and distal feedback inhibition from other neighbouring MS neurons. In the present study the effect of these GABAergic inputs on the dendritic calcium dynamics is investigated.

## Model

A previously published MS model [8] was reimplemented in GENESIS. The dendritic axial resistance and sodium conductances have been modified to better fit experimental results [1-3]. For example, in the modified model, backpropagation of dendritic action potentials requires sodium channel activation, and fails if those channels are blocked [1]. The MS neuron was activated by simulated AMPA/NMDA and GABAergic synaptic inputs [8], or by somatic current injections.

## Computational investigation

In this study we compare the effect of FS and MS synaptic inhibition on the backpropagation of action potentials in the MS model. Preliminary results suggest that GABAerigic inputs in distal dendrites can decrease the backpropagating action potential, and thus can reduce dendritic calcium levels, even though the cell is still spiking in the soma. These findings might suggest that feedback inhibition can control how prone the neighbouring MS neurons will be to plastic changes.

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