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Does striatum support competitive dynamics? A test of this hypothesis using a biologically realistic model of the striatal microcircuit

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Introduction

The striatum is the man input structure of the basal ganglia and consists principally of medium spiny neurons (MSNs). The remaining neurons comprise several species of interneuron, including the GABAergic fast spiking interneuron (FSIs). Both neuron species are highly interconnected (including a network of gap junctions between the FSIs) and both are modulated by dopamine. Understanding this complex microcircuit is therefore very challenging. Previous computational hypotheses have suggested that the inhibitory collaterals between MSNs lead to a strong competitive dynamic [1]. In contrast, Koós and Tepper [2] suggest that feed-forward inhibition from the FSIs is the dominant force in the control of MSNs. We have developed a detailed, biologically constrained model of the striatal microcircuit aimed at resolving these issues and discovering the computations performed in this critical brain area.

Methods

The model incorporates dopamine modulated MSNs and FS interneurons, and we used a novel technique in computational anatomy to develop realistic connection statistics for all known pathways in this circuit. The response of the model to realistic in vivo background input was analyzed using a novel multiple spike-train analysis technique to find groups of synchronized neurons (as observed experimentally). Predicated on the hypothesis that the basal ganglia is performing action selection, we then used these groups to define "channels" in a series of selection experiments. We hypothesized that, if there were naturally emerging clusters of MSNs in the network, these might serve to compete well with each other. We repeated these experiments with channels comprised of randomly selected neurons.

Results

Using realistic parameter values for the input glutamatergic spike trains, and for the GABAergic and gap junction conductances, we found little evidence for selection in the model. Removing the FSI input to the MS neurons also failed to reveal any competition via the MSN collaterals, suggesting that FSN input was not imposing another dynamic. Varying the level of dopamine in the simulation also failed to show any significant change in the networks selective ability. Increasing the conductance of the MSN collateral synapses by a factor of ten, however, did force the network to show signs of competition between competing channels. No significant difference was observed when using channels of randomly selected neurons, compared to channels defined by the multiple spike-train analysis method. We conclude that the striatum does not support competitive dynamics using the circuits comprising MSNs and FSIs within a range of realistic parameter settings.

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