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Modulation of synaptically induced burst strength and spike onset timing by inactivating K_{IR} currents in medium spiny neurons

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Background

The membrane potential of striatal medium spiny neurons (MSNs) fluctuates between down- and up-states. In ventral striatum, inward rectifying potassium (K_{IR}) currents in 40% of MSNs inactivate [1]. The significance of this property is not clearly known. We describe a computational study investigating how synaptic integration is influenced by K_{IR} current inactivation.

Methods

Two MSNs were modeled using NEURON, one equipped with non-inactivating K_{IR} currents (henceforth, "Cell A") and the other with inactivating K_{IR} currents (henceforth "Cell B") and their behaviors were compared in response to trains of inputs activating NMDA, AMPA and GABA synapses. Injected current inputs were then used to investigate the mechanisms underlying the observed differences.

Results

It was observed that the behavior of these two types of cells were different in several ways. For instance, Cell B when compared with Cell A (i) had a more depolarized mean down-state potential (+2.8 mV); (ii) had a mean burst strength (number of action potentials fired from an up-state) higher by 35% for same strength synaptic input (Figure 1A); (iii) showed noticeable differences in strength-duration curve; (iv) had a mean spike onset earlier by 14% for a given number of spikes fired from the up-state (Figure 1B). It was also found that while the

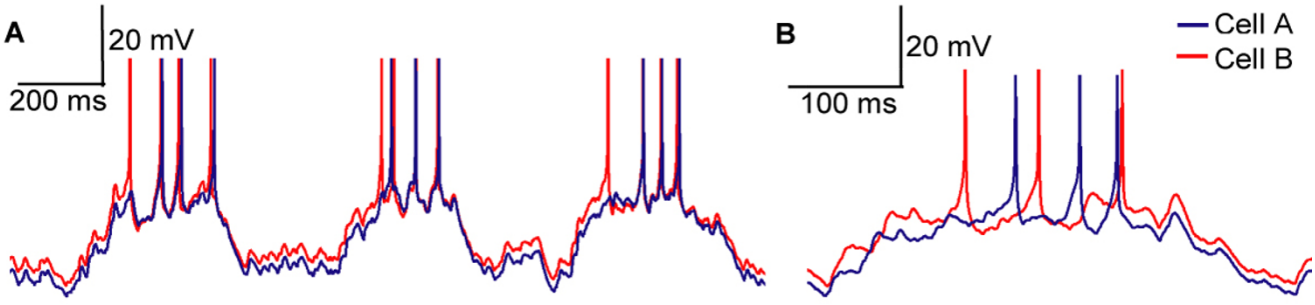

Figure 1
Membrane response of Cell B to synaptic input compared with that of Cell A (action potentials chopped at -20 mV). **A**, Burst strength is higher for Cell B for a synaptic input of given strength. **B**, Spike onset latency is lower for Cell B for a given number of spikes fired from the up-state.

higher input resistance offered by Cell B is responsible for the earlier spiking onset, the lower permeability to potassium ions underlies the enhanced burst strength.

Discussion

Our model demonstrates the facilitatory effect of K_{IR} current inactivation on MSN excitability in response to synaptic inputs. In view of the reports that dendritic intracellular calcium levels depend closely on burst strength as well as the spike onset time in MSNs [2], our findings suggest that K_{IR} current inactivation may significantly modulate synaptic plasticity as well.

References

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