Poster presentation

Open Access K_{IR} current inactivation modulates dendritic calcium in medium spiny neurons John Eric Steephen* and Rohit Manchanda

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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]. K_{IR} current inactivation appears to alter spike frequency and onset during upstates [2]. However, it is not known whether these translate into significant changes in calcium dynamics in the dendrites. We describe a computational study investigating how this inactivation influences dendritic calcium transients.

Methods

Two MSNs were modeled using NEURON, one equipped with non-inactivating K_{IR} currents (henceforth, "noninK_{IR}") and the other with inactivating K_{IR} currents (henceforth "inK_{IR}") and their dendritic calcium transients were compared in response to injected current and synaptic inputs. Measurements were made from the distal dendrites.

Results

It was observed that dendritic calcium transients were significantly enhanced by K_{IR} current inactivation (Figure 1). For instance, inK_{IR} cell when compared with non-inK_{IR} cell had dendritic calcium transient peaks higher by as much as 93% in response to an injected current of 0.25 nA in the distal dendrite (Figure 1A). Though this difference decreased with higher currents, still the calcium peak remained higher for the inK_{IR} cell by at least 10% (Figure 1B). The enhanced calcium influx (by up to 51%) was present even when the injected currents were matched for firing frequency. Similar results were obtained with synaptic inputs.

Discussion

The facilitatory effect of KIR current inactivation on dendritic calcium influx appears to be mediated through action potential firing frequency and their timing. In view of the reports that dendritic intracellular calcium levels influence cortico-striatal input plasticity [3], our findings suggest that K_{IR} current inactivation may significantly modulate synaptic plasticity.

References

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