Calcium, synaptic plasticity and intrinsic homeostasis in Purkinje neuron models

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Activity homeostasis designates bio-mechanisms that regulate the activity of a neuron through the dynamic expression of ion channels or synapses \cite{1}. We have recently reproduced the complex electrical activity of a Purkinje cell (PC) with very different combinations of ionic channel maximum conductances \cite{2}, suggesting that a large parameter space is available to homeostatic mechanisms. Some models \cite{3,4} have hypothesized that one such mechanism could work via the regulation of the average cytoplasmic calcium concentration. While this hypothesis is attractive for rhythm generating neurons, it raises many questions for PCs since in these neurons calcium is supposed to play a very important role in the induction of synaptic plasticity \cite{5}. To address this question, we generated 148 new PC models. In these models the somatic membrane voltages are stable, but the somatic calcium dynamics are very variable, in agreement with experimental results \cite{6}. Conversely, the calcium signal in spiny dendrites is robust. Using a PC spine model of calcium signal transduction pathways \cite{7}, we demonstrate that the induction of long-term depression is preserved for all models. We conclude that calcium is unlikely to be the sole activity-sensor in this cell but that there is a strong relationship between activity homeostatis and synaptic plasticity.

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