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Activity-homeostasis preserves synaptic plasticity in Purkinje cell but calcium is not the activity-sensor

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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 [1]. We have recently shown [2] that it is possible to reproduce the complex activity of a Purkinje cell (PC) with very different combinations of ionic channel maximum conductances. However, if the global effect of homeostasis is starting to be understood, the detail of its machinery remains unknown. Some models [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 long-term memory [5]. To address this question, we generate 81 PC models, all having a similar electrophysiological activity and all different enough from each other in their conductance set. We demonstrate that, while the somatic membrane voltage is stable during complex spikes, the somatic calcium behavior is very variable from cell to cell, in agreement with experimental results [6]. Therefore calcium is a weak candidate for being an activity-sensor in this cell. Conversely, we show that the calcium signal in the spiny dendrites is very robust. To further test whether long-term depression (LTD) mechanisms are preserved for these different models, we use a PC spine model of calcium signal transduction pathways [7]. In all our models, conjunctive parallel fibers-climbing fiber activation leads to a sustained calcium release from internal stores, hence LTD induction is preserved.

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References

1. Marder E, Goaillard JM: **Variability, compensation and homeostasis in neuron and network function.** *Nat Rev Neurosci* 2006, **7:**563-574.
2. Achard P, De Schutter E: **Complex parameter landscape for a complex neuron model.** *PLOS Comput Biol* 2006, **2:**e94.
3. Liu Z, Golowasch J, Marder E, Abbott LF: **A model neuron with activity-dependent conductances regulated by multiple calcium sensors.** *J Neurosci* 1998, **18:**2309-2320.
4. LeMasson G, Marder E, Abbott LF: **Activity-dependent regulation of conductances in model neurons.** *Science* 1993, **259:**1915-1917.
5. Ito M: **Cerebellar long-term depression: characterization, signal transduction, and functional roles.** *Physiol Rev* 2001, **81:**1143-1195.
6. Swensen AM, Bean BP: **Robustness of burst firing in dissociated purkinje neurons with acute or long-term reductions in sodium conductance.** *J Neurosci* 2005, **25:**3509-3520.
7. Doi T, Kuroda S, Michikawa T, Kawato M: **Inositol 1,4,5-trisphosphate-dependent Ca2+ threshold dynamics detect spike timing in cerebellar Purkinje cells.** *J Neurosci* 2005, **25:**950-961.