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Abstract Title:	I_h as a candidate mechanism for sliding the BCM modification threshold.
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Metaplasticity has been theoretically linked to the Bienenstock-Cooper-Munro (BCM) rule. In this process, however, a mechanism for the activity-dependent regulation of the modification threshold (θ) has remained an open question. In this simulation study of CA1 pyramidal cells, we used a modification of the calcium dependent hypothesis proposed by (Shouval et al, PNAS, 99(16), 2002) and show that the hyperpolarization-activated cation current I_h is capable of shifting θ by regulating intracellular calcium levels. Specifically, our results suggest that an up/downregulation of I_h leads to an increase/decrease in θ. Taken along with recent results that LTP/LTD-inducing stimulus up/downregulates I_h (Fan et al; Brager et al; SfN 2005), this suggests that LTP/LTD-inducing stimulus brings about an increase/decrease in θ. Observing this to match the requirement on θ within the BCM framework, we propose I_h as a candidate mechanism for regulating θ. Next, we show that an increase in the conductance of (i) AMPAR/NMDAR channel decreases θ; (ii) SK channel increases θ; (iii) KA channel regulates θ depending on I_h conductance. We then explored the implications of these results in terms of (i) non-uniform distribution of these channels in dendrites; (ii) independence of dendritic subunits in terms of locally regulated θ; and (iii) plasticity normalization across the stratum radiatum. Finally, extending the calcium dependent hypothesis to activity-dependent regulation of I_h, we propose a functional form for its dependence on average intracellular calcium. Based on theoretical and experimental observations along with physiologically relevant constraints, we postulate that this function takes a form similar to the one relating synaptic weight change to intracellular calcium. Using this, we explored the stability of the calcium dependent plasticity mechanism by subjecting it to various stimulus protocols. Our results suggest that I_h can act as a regulator of the BCM threshold, not by directly affecting synaptic currents, but by modulating dendritic excitability.

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