Novel aspects of synaptic and non-synaptic hippocampal plasticity

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Cognitive processes such as learning and memory require functional modifications within neural circuits which involve reorganization of existing synaptic connections and modulation of its strength. In addition to synaptic plasticity, neurons can significantly enhance information storage capacity by scaling dendritic and somatic excitability. However, the mechanism of the latter is poorly understood. The activity of several proteases in extracellular matrix has recently emerged as a key signal to drive various forms of neuronal plasticity. By combining electrophysiology and pharmacology tools, we recently showed that activity of matrix metalloproteases may modulate NMDARs, intracellular Ca^{2+} level and expression of immediate early genes during episodes of enhanced neuronal activity. This in turn altered not only synaptic plasticity but also endogenous neuronal excitability and neuronal output in the time scale of minutes. Moreover, we discovered that in striking contrast to apical dendrites, synaptic plasticity induced at basal dendrites of hippocampal pyramidal neurons required neither the function of protease activated receptor 1 (PAR1), integrin signaling, intracellular PKC kinase nor Rho GTPases activity. Thus, a picture emerges were extracellular proteases activity modulation of neuronal plasticity is synapse-specific and determines the neuronal output. Considering that beyond hippocampus, also cortical pyramidal neuron wiring is specific to dendritic tree, these novel discoveries may allow to model the temporal integration of signals (inputs) and neuronal output upon episodes of enhanced neuronal activity.