

Dominique Debanne, Gaël Daoudal & Emilie Campanac

Bidirectional plasticity of synaptic integration in CA1 pyramidal neurons: learning rules and mechanisms

Neurobiologie des Canaux Ioniques, INSERM U464,
Institut Fédératif Jean Roche, Faculté de Médecine Secteur Nord,
Boulevard Pierre Dramard, 13916 Marseille cedex 20, France.

Tel: (+33) 4 91 69 87 45

Fax: (+ 33) 4 91 09 05 06

E-mail: debanne.d@jean-roche.univ-mrs.fr

Plasticity at synaptic contacts is generally considered as the cellular model for learning and memory. However, synaptic plasticity is not the exclusive mode of regulation of information processing in the brain. Persistent regulations of ionic conductances in some specialized neuronal areas such as the dendrites and the cell body could also modulate, in the long term, the propagation of neuronal information. In the area CA1 of the rat hippocampus, long term potentiation (LTP) is associated with a long-term enhancement of the EPSP-Spike coupling (E-S potentiation). E-S potentiation is partly independent of inhibitory synaptic transmission, can be reversed by protocols that induce long-term depression (LTD) and is thought to result from a long-lasting modification in intrinsic excitability (Daoudal et al. PNAS 2002). E-S potentiation and E-S depression are input specific and require NMDA receptor activation for their induction. E-S plasticity is functionally synergic with synaptic plasticity induced with protocols based on the modulation of the frequency of presynaptic stimulation or the timing between pre- and postsynaptic activity (STDP). Various currents shape dendritic EPSPs and thus, determine neuronal integration. Among them, the hyperpolarization-activated cationic current (I_h) attenuates EPSPs and controls dendritic excitability. We show here that I_h plays a critical role in the picrotoxin-insensitive component of E-S potentiation in the area CA1.