2nd grade: Synaptic Plasticity

Summary: Neurons transmit signals through synapses, which are narrow junctions facing pre- and postsynaptic neurons. Synaptic plasticity is thought to be an elementary process for learning and memory, in which transient activation alters the efficacy of synaptic transmission for a long period of time. Two forms of synaptic plasticity are known: long-term potentiation (LTP) and depression (LTD) (Fig.1). Molecules involved in the synaptic transmission are AMPA type receptor (AMPAR), NMDA type receptor (NMDAR), Ca^{2+}, protein kinase CaMKII, and protein phosphatase calcineurin (CaN). The following mechanism shown in blue is the basis for LTP and LTD. The last event is the modification of AMPAR and NMDAR, thus forming a closed loop beginning at activation of AMPAR and NMDAR and finally returning to these receptors.

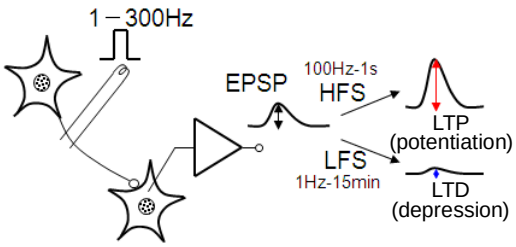

Fig.1 Synaptic plasticity

Release of transmitter → postsynaptic depolarization by AMPAR opening → activation of NMDAR → inward flow of Ca^{2+} → activation of CaMKII/CaN → modification of AMPAR/NMDAR

Cartoon and A-Cell model: Cartoon is shown at the center of Fig.2, which is block diagram of signaling.

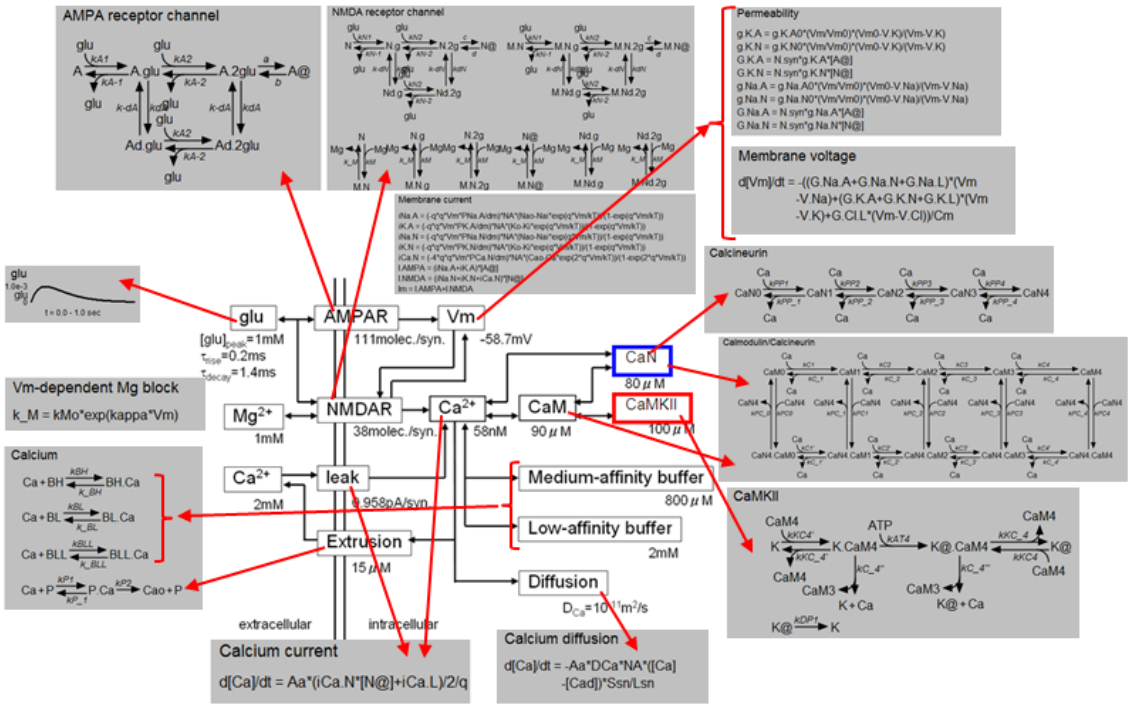

Fig.2 Block diagram of LTP/LTD and molecular mechanisms in each block in A-Cell

Reaction model in a single block (gray rectangles) is not simple. If you look carefully at each reaction, however, you can find that every reaction is that shown in documents of “Basic” A-Cell models. It should be noted that the starting point is a cartoon describing over all signal transduction mechanisms. Equations are used to describe membrane potential and current through ion channels. Transmitter molecules glutamate release is modeled by double exponential function using “Stimulation” of A-Cell.

References: Ichikawa, K., et al., Neurocomputing, 2007, 2055.
Ichikawa, K., Neuroinformatics, 2005, 49.
Ichikawa, K., Neurocomputing, 2004, 709.