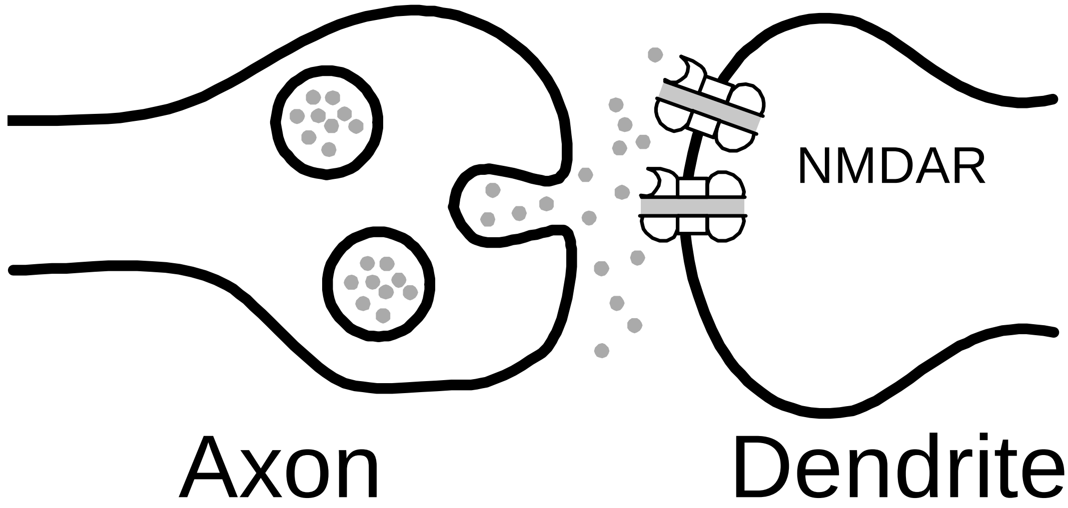
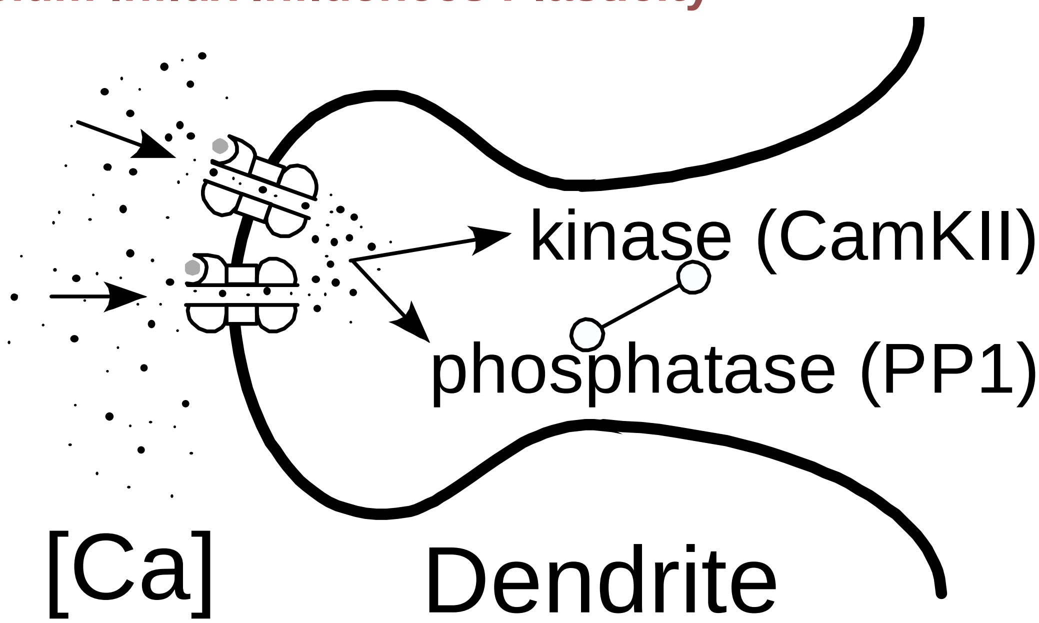
Synaptic Plasticity-I: Spike Timing-Dependence



An axon terminal releases the neurotransmitter glutamate at an excitatory synapse

Activity drives changes in the efficacy of synapses .

Calcium Influx Influences Plasticity

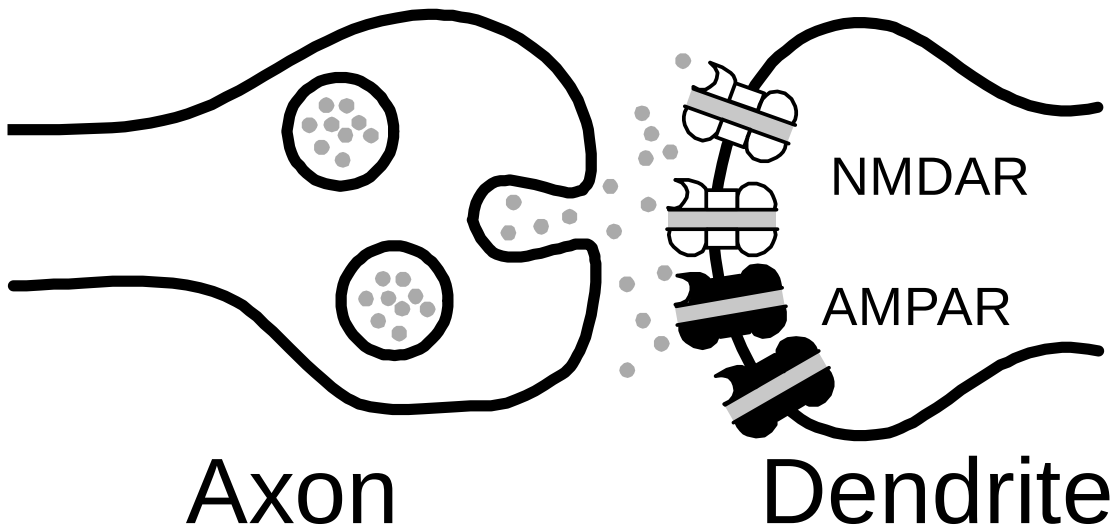


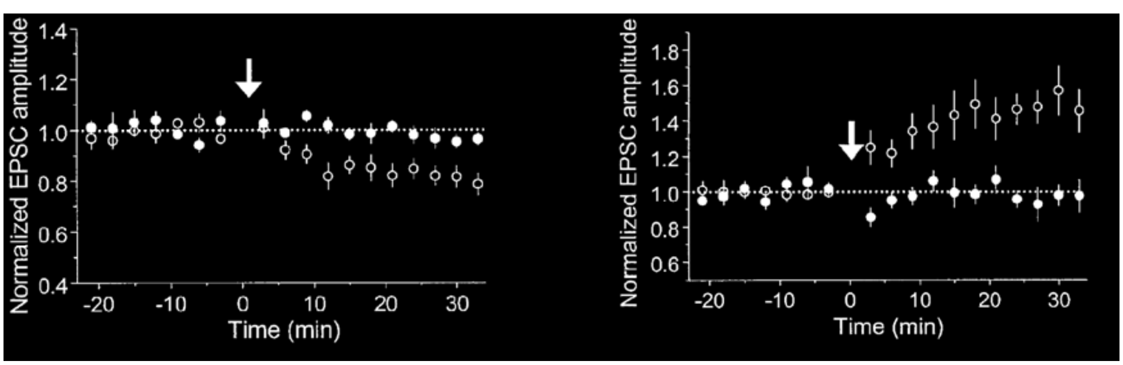
Calcium influences plasticity by activating a kinase pathway and a phosphatase pathway

When high calcium levels activate the kinase pathway (CamKII), the synapse undergoes long-term potentiation (LTP).

When moderate calcium levels activate the phosphatase pathway (PP1), the synapse undergoes long-term depression (LTD).

Potentiation inserts AMPARs





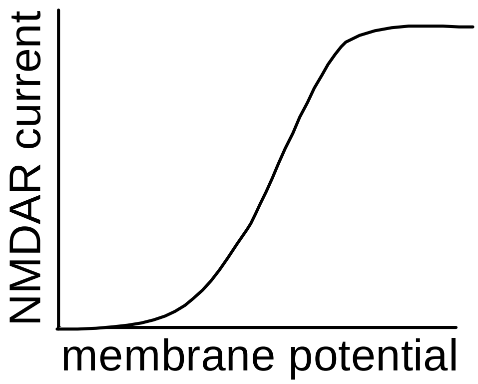
When a synapse is potentiated AMPARs are inserted into its membrane.

CamKII inserts AMPARs into the synapse, whereas PP1 removes them.

What determines calciums influx?

4 of 10

NMDARs' Part



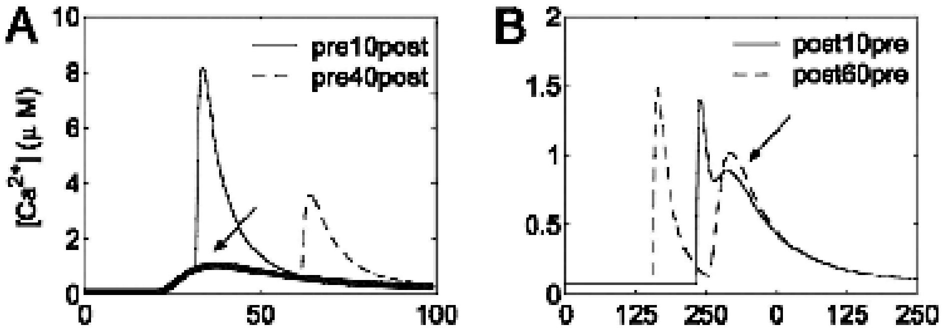
NMDARs gate current depending both on glutamate concentration and membrane potential.

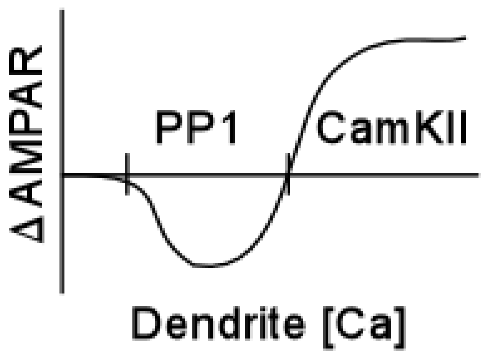
NMDARs allow calcium (and other cations) to flow into the synapse depending on the membrane potential, gating the level of calcium influx and the resulting plasticity.

How does timing influence NMDARs and calcium?

5 of 10

The Role of Spike Timing

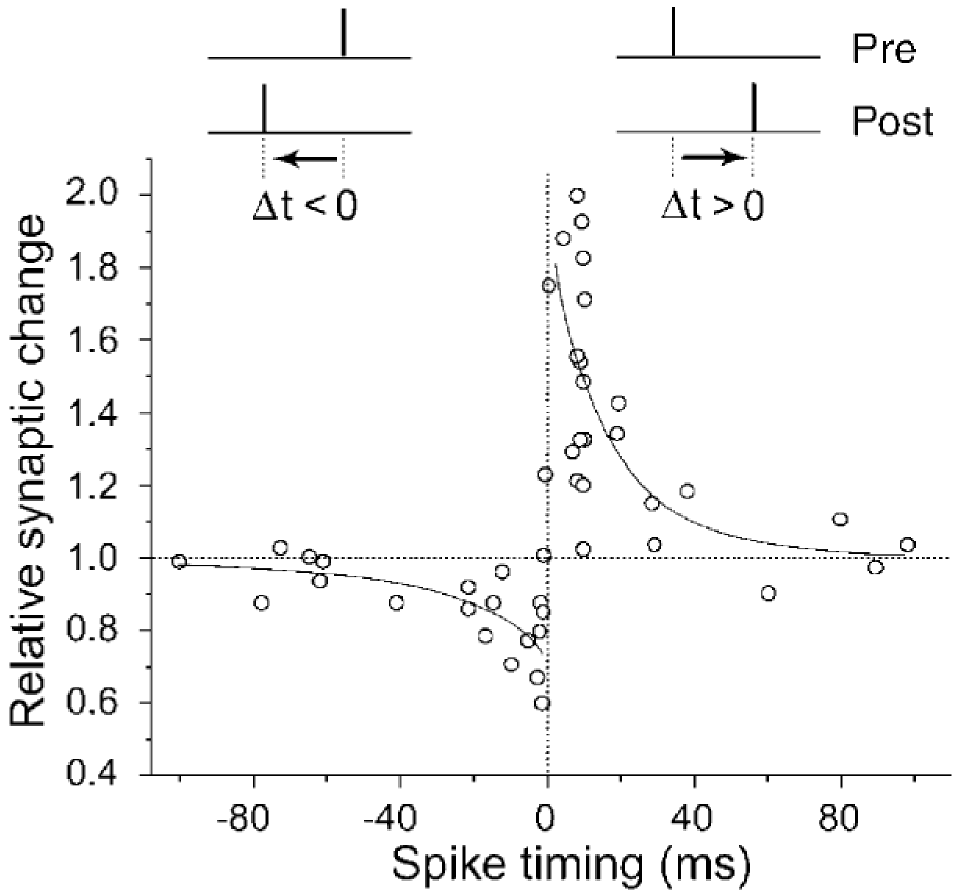




When presynaptic spike's precede postsynatic spikes calcium levels rise higher than when the order is reversed.

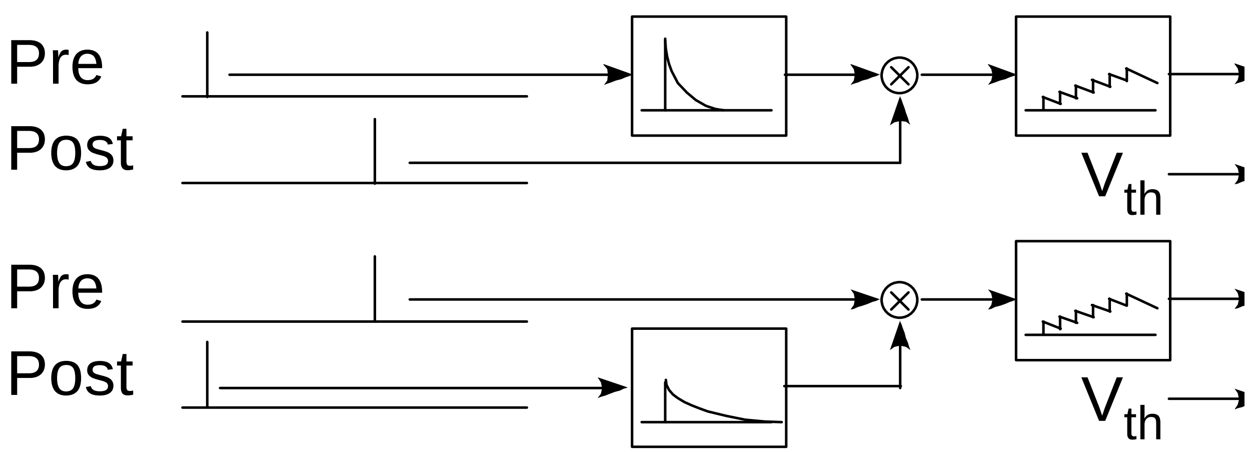
Pre-before-post pairings result in high calcium, which drives LTP.
Post-before-pre pairings result in moderate calcium, which drives LTD.

Spike Timing-Dependent Plasticity



Pre-before-post pairings result in LTP and Post-before-pre pairings result in LTD.

A Model of STDP



The LTP and LTD implementation rely and decay elements and integrators.

When a presynaptic spike occurs, a delay element is activated. When a subsequent postsynaptic spike occurs it samples the delay.

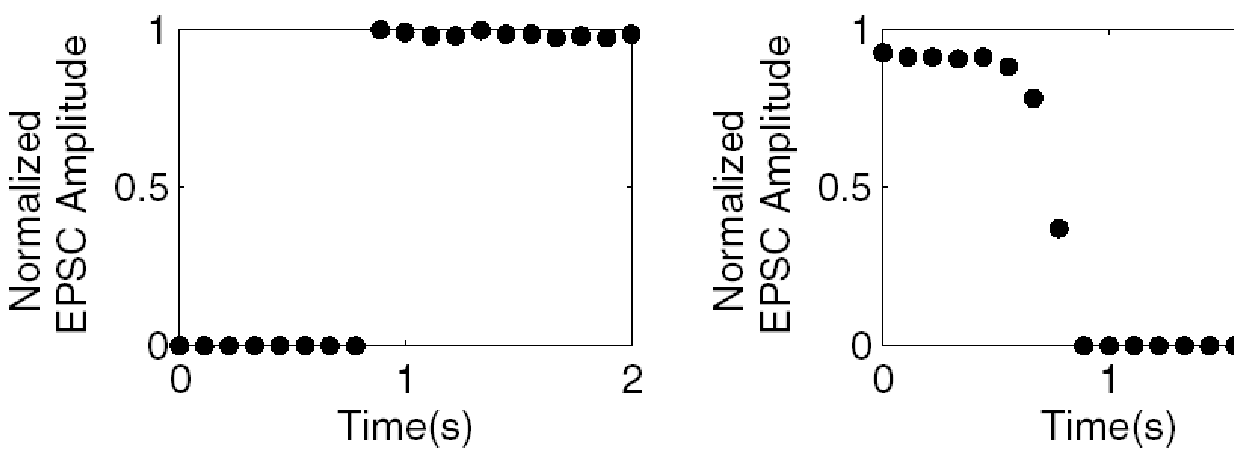
The closer the two are together the larger the increment of the integrator element. If the integrator reaches a threshold, the synapse undergoes LTP.

LTD works the same but the roles of the pre- and post- synaptic spikes are reversed.

A state-holding element (binary) remembers which when high last LTP or LTD and determines if AMPARs should be present at the synapse.

8 of 10

LTP and LTD



LTP and LTD insert or remove all AMPARs from a synapse.

9 of 10

STDP Equations

$$LTP = n\,Decay[tdiff] - (n-1)\,L_{int}$$

If we drive the STDP synapse repeatedly with a constant pre-before-post pairing, we can determine how many pairings, **n**, (at a constant rate) are required to cause **LTP** to exceed its threshold, **Vth**.

Decay[tdiff] is the decay elements response to a pairing tdiff apart.

L_{int}; is the integrator's leak per cycle (at a given constant pairing rate).

The same equations apply for depression, although the parameters may change.

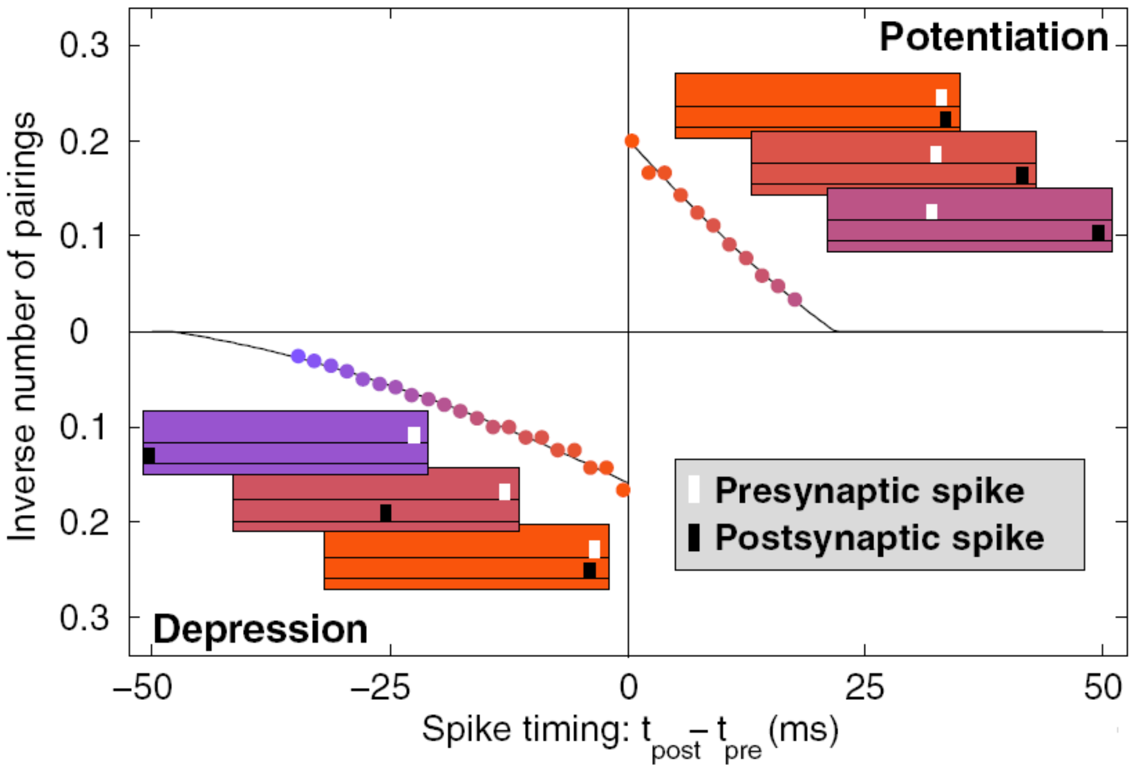
We model **Decay[tdiff]** as a quadratic:

$$\text{Decay[tdiff]} = \left(1 - \frac{\text{tdiff}}{\text{te}}\right)^2, \ \text{tdiff} < \text{te}$$

$$n = \frac{1}{\alpha \left(1 - \frac{\text{tdiff}}{\text{te}}\right)^2 - \beta}, \quad \frac{1}{n} = \alpha \left(1 - \frac{\text{tdiff}}{\text{te}}\right)^2 - \beta$$

10 of 10

Spike Timing-Dependent Plasticity



Pre-before-post pairings result in LTP and Post-before-pre pairings result in LTD.