Part 3

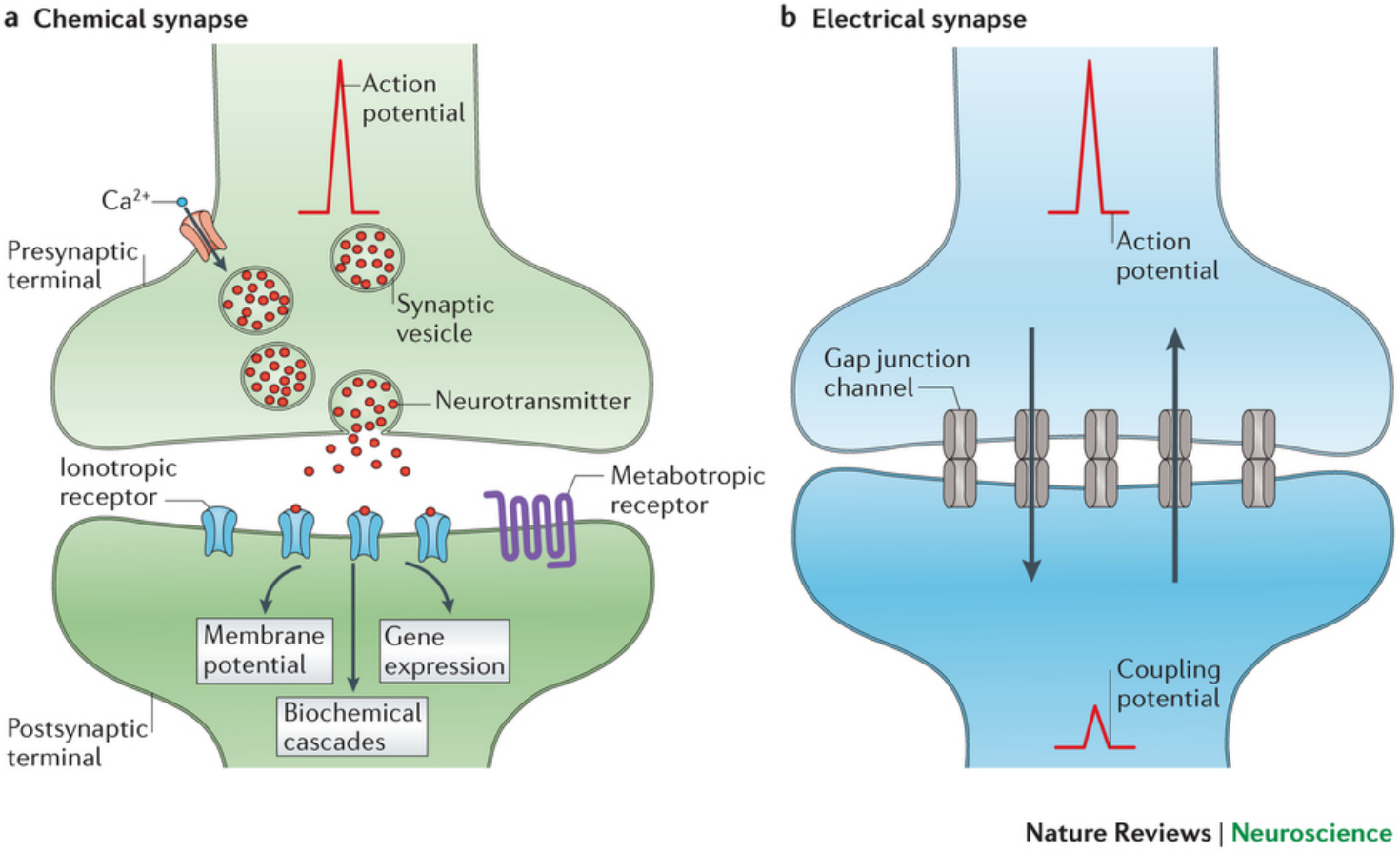
Synaptic models

Purpose of synaptic modeling

- To capture the following facts:

1. Some neurons have stronger and more lasting influences over a given neuron than others
2. Some of these influences are excitatory (increase the likelihood of spike emission) while some are inhibitory (decrease this likelihood)
3. The strength of the influence of a neuron over another one changes over time as a function of the activities of both neurons (synaptic plasticity)

Types of synapses



Pereda, 2014

Types of synapses

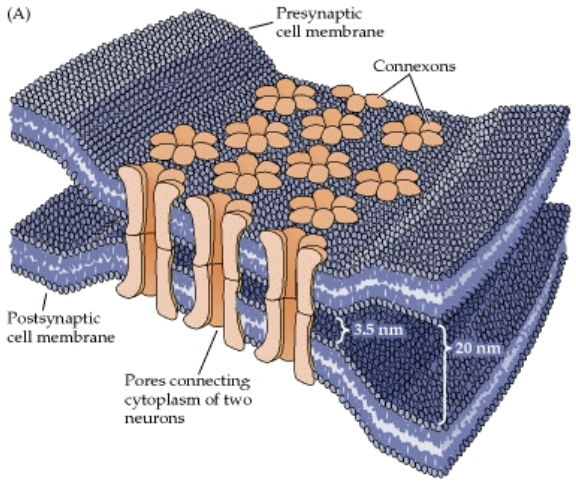
Electrical

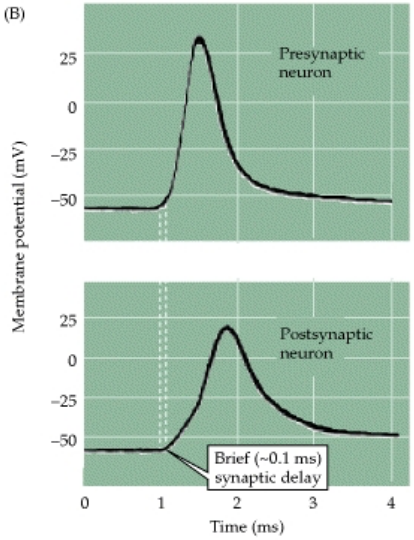
- 2 neurons linked together by gap junctions
- Rapid communication
- Bidirectional communication
- Excitation/inhibition at the same synapse
- Occur between neurons and glia

Chemical

- Signal transduction
- Excitatory or inhibitory
- Slower communication
- Unidirectional communication
- More plastic

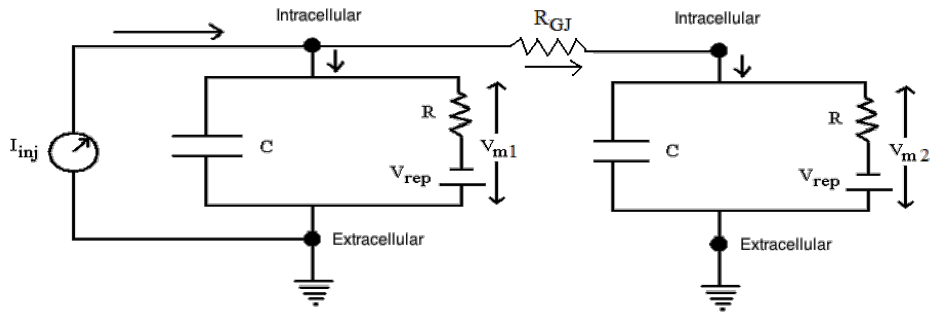
Electrical synapses





An action potential in the presynaptic neuron causes the postsynaptic neuron to be depolarized within a fraction of millisecond

Neuroscience. 2nd edition.
Purves D, Augustine GJ, Fitzpatrick D, et al., editors.
Sunderland (MA): Sinauer Associates, 2001



$$V_2 = kV_1$$

$$I_{GJ} = G_{GJ}\left(V_1 - V_2\right)$$

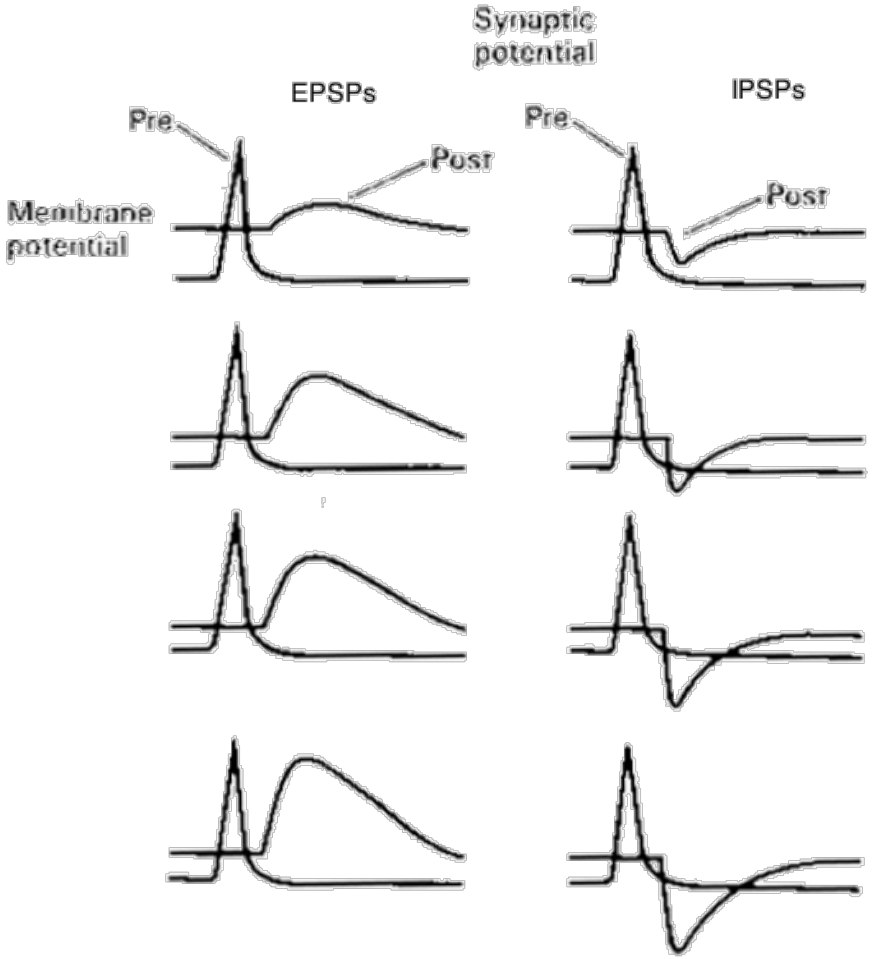
Gap junction coupling can be modeled by a single resistance connecting the 2 cells (see equivalent circuit to the left). The corresponding equations are given below the figure. The cell-cell coupling coefficient is *k* and the transjunctional current is I_{GJ}

Chemical Synapses

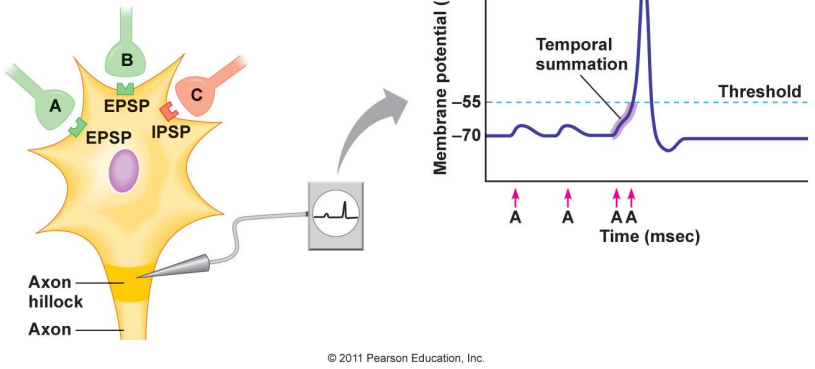
- Excitatory or inhibitory
- Ionotropic (fast) and metabotropic (slow)
- Many neurotransmitters, but the most common in the cortex are:
  - Glutamate (usually excitatory)
  - γ-aminobutyric acid (GABA) (usually inhibitory)
- Dynamics depends on receptor type:
  - Glutamate receptors: AMPA/Kainate and NMDA
  - GABA receptors: $GABA_A$ and $GABA_B$
- Short-term and long-term synaptic plasticity

Postsynaptic potentials

- Excitatory postsynaptic potential (**EPSP**): Transient depolarization of the postsynaptic membrane by presynaptic release of neurotransmitter
- Inhibitory postsynaptic potential (**IPSP**): Transient hyperpolarization of the postsynaptic membrane by presynaptic release of neurotransmitter



Postsynaptic potentials summation



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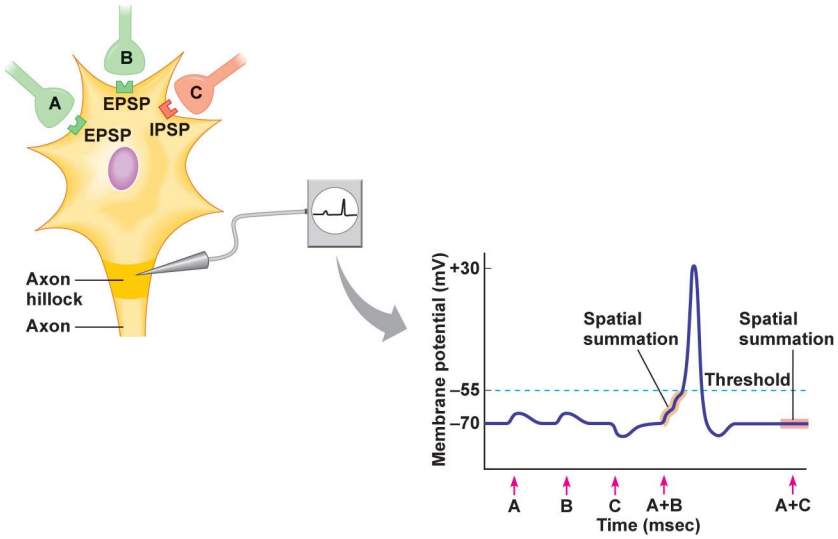

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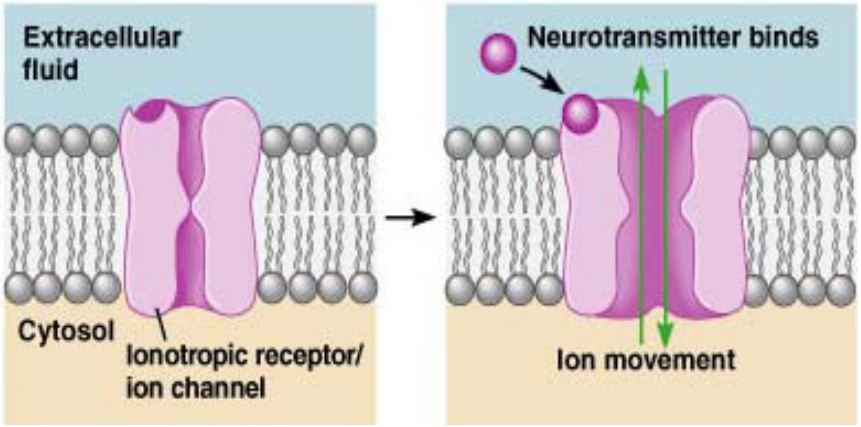
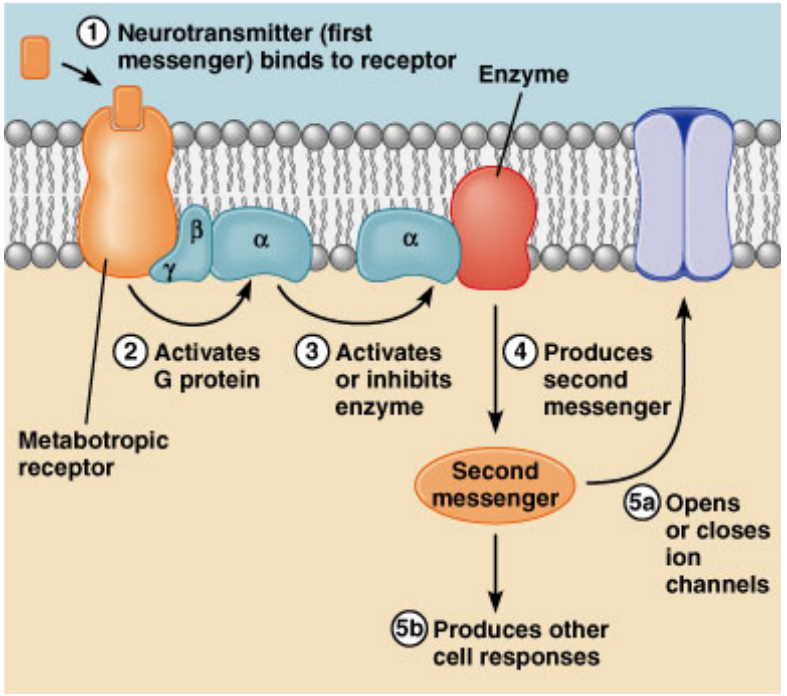
Temporal summation:
(spikes from the **same** cell arriving at successive times)

Spatial summation:
(spikes from **different** cells arriving at the same time)

Ionotropic and Metabotropic Synapes



Ionotropic: fast



Metabotropic: slow

Synaptic receptors

Glutamate

- Ionotropic
  - AMPA/Kainate: early EPSP
  - NMDA: activated when cell is already depolarized (late EPSP)

GABA

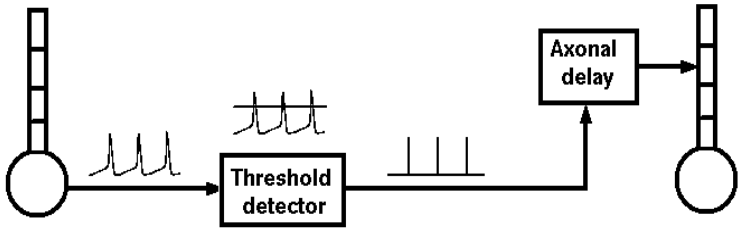
- Ionotropic
  - $GABA_A$
- Metabotropic
  - $GABA_B$

Synaptic models

- There are many models, the most common in network models assumes a **synaptic current**

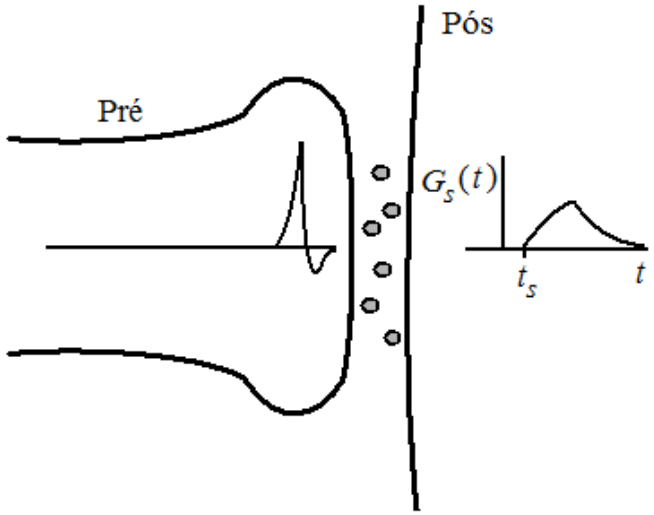
$$I_{\text{syn}} = g(t)\left(V_{\text{post}} - V_{\text{rev}}\right)$$

- V_{rev} = −75 mv (inhibitory synapses) and V_{rev} = 0 (excitatory synapses)
- $g(t)$ = synaptic conductance of **postsynaptic** cell
- Synaptic delays can also be introduced

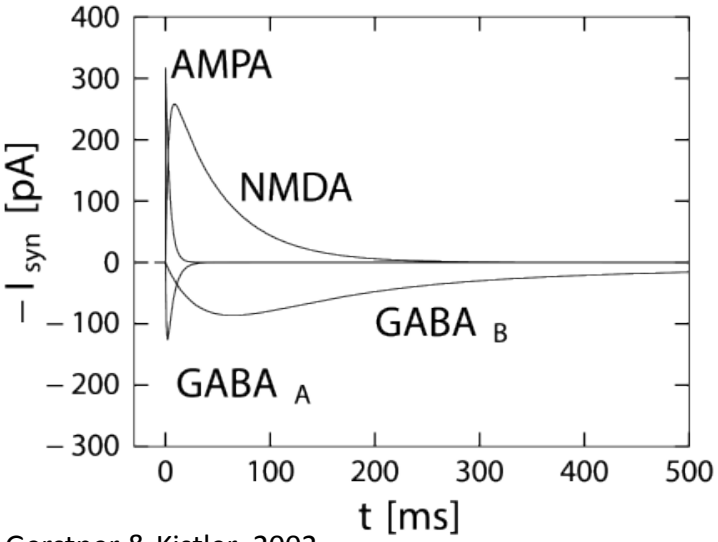


Synaptic conductance

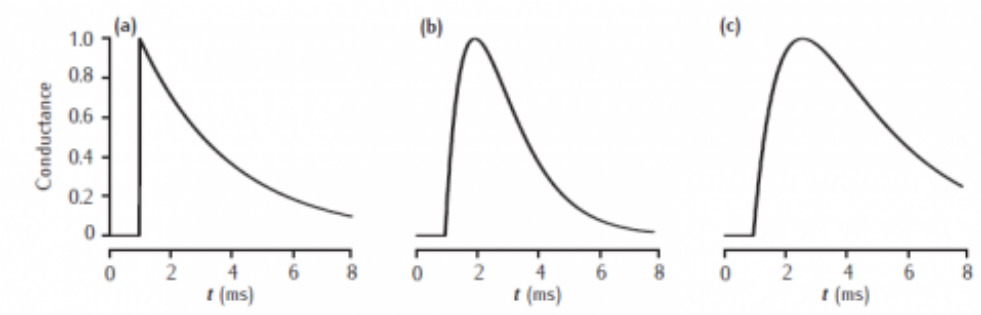
- The time course of $g(t)$ can be modeled by kinetic equations but in general **fixed** time course functions are used



Fixed functions used to model $g(t)$



Gerstner & Kistler, 2002



(a) $$g(t) = \bar{g} \exp\left(-\frac{t - t_j}{\tau}\right)$$

(b) $$g(t) = \bar{g} \frac{t - t_j}{\tau} \exp\left(-\frac{t - t_j}{\tau}\right)$$

(alpha function)

(c) $$g(t) = \bar{g} \frac{\tau_1 \tau_2}{\tau_1 - \tau_2} \left(\exp\left(-\frac{t - t_j}{\tau_1}\right) - \exp\left(-\frac{t - t_j}{\tau_2}\right)\right)$$

Synaptic inputs in the LIF model
(also valid for other IF models)

- Current-based model: Each presynaptic spike generates a postsynaptic **current** pulse in neuron *i*

$$\tau \frac{dV_i}{dt} = -V_i + RI_i^{syn}(t)$$

$$I_i^{syn}(t) = \sum_j w_{ij} \sum_f \alpha\left(t - t_j^{(f)}\right)$$

Fixed function

- $t_j^{(f)}$ are the spike times of presynaptic neuron *j*
- w_{ij} is the synaptic efficacy (weight) of the synapse from neuron *j* to neuron *i*

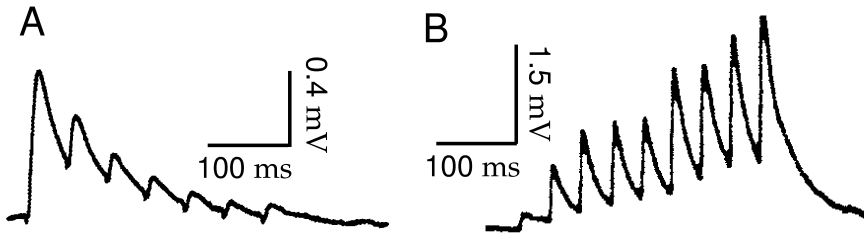
Synaptic inputs in the LIF model
(also valid for other IF models)

- Conductance-based model: Each presynaptic spike generates a **change in the conductance** of the postsynaptic membrane with time course $g(t - t_j^{(f)})$

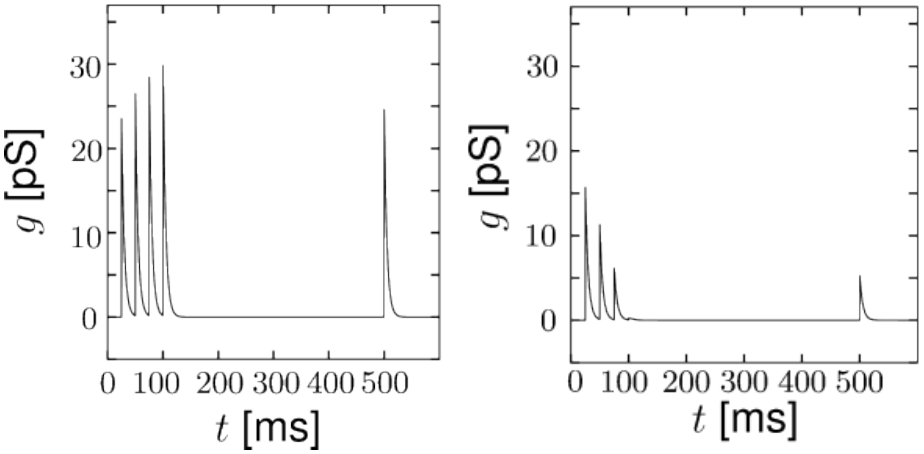
$$\tau \frac{dV_i}{dt} = -V_i + RI_i^{syn}(t)$$

$$I_i^{syn}(t) = -\sum_j w_{ij} \sum_f g\left(t - t_j^{(f)}\right)(V_i(t) - V_{rev})$$

Short-term plasticity



Experimental recordings showing depression (A) and facilitation (B) of excitatory cortical synapses in a slice of rat somatosensory cortex. A. Markram & Tsodykes, 1996. B. Markran et al., 1998



Simulation of facilitation (left) and depression (right): sequence of 4 presynaptic spikes followed by a 5th spike 400 ms after (Gerstner & Kistler, 2002)

- Synaptic conductance given by

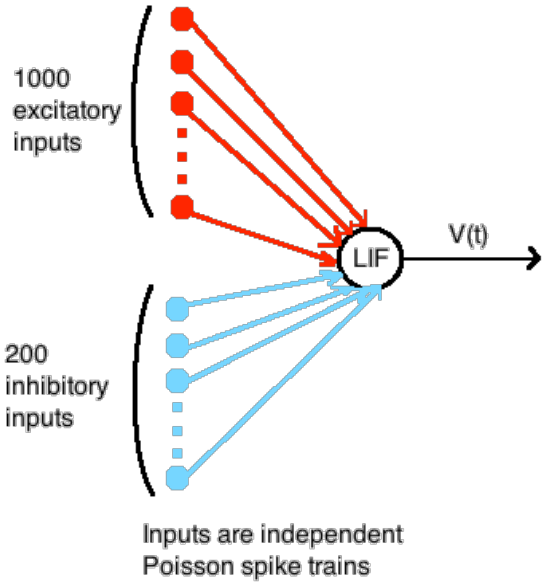
$$g(t) = \bar{g} p_s p_{rel}$$

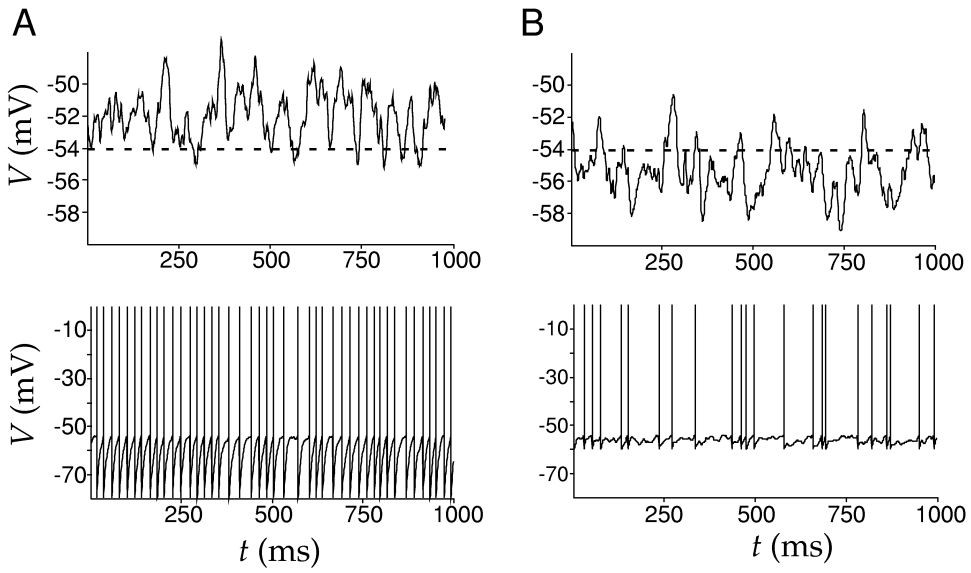
- p_s = probability that a postsynaptic channel opens given that a transmitter was released by presynaptic neuron → modeled by an α function
- p_{rel} = probability that a transmitter is released by presynaptic neuron following the arrival of an action potential
- p_{rel} is affected by synaptic **facilitation** and **depression**

$$\tau_p \frac{dp_{rel}}{dt} = p_0 - p_{rel} + f_F \left(1 - p_{rel}\right) \sum_j \delta\left(t - t_j\right)$$

$$\tau_p \frac{dp_{rel}}{dt} = p_0 - p_{rel} - f_D p_{rel} \sum_j \delta\left(t - t_j\right)$$

Excitatory/Inhibitory synaptic balance





$$I^{ex}_{syn\_tot} > I^{in}_{syn\_tot}$$

Regular spiking

$$I^{ex}_{syn\_tot} \approx I^{in}_{syn\_tot}$$

Irregular spiking

Upper panels: Spikes not put by hand (V is allowed to change according to input)

Lower panels: Spikes generated in the usual way

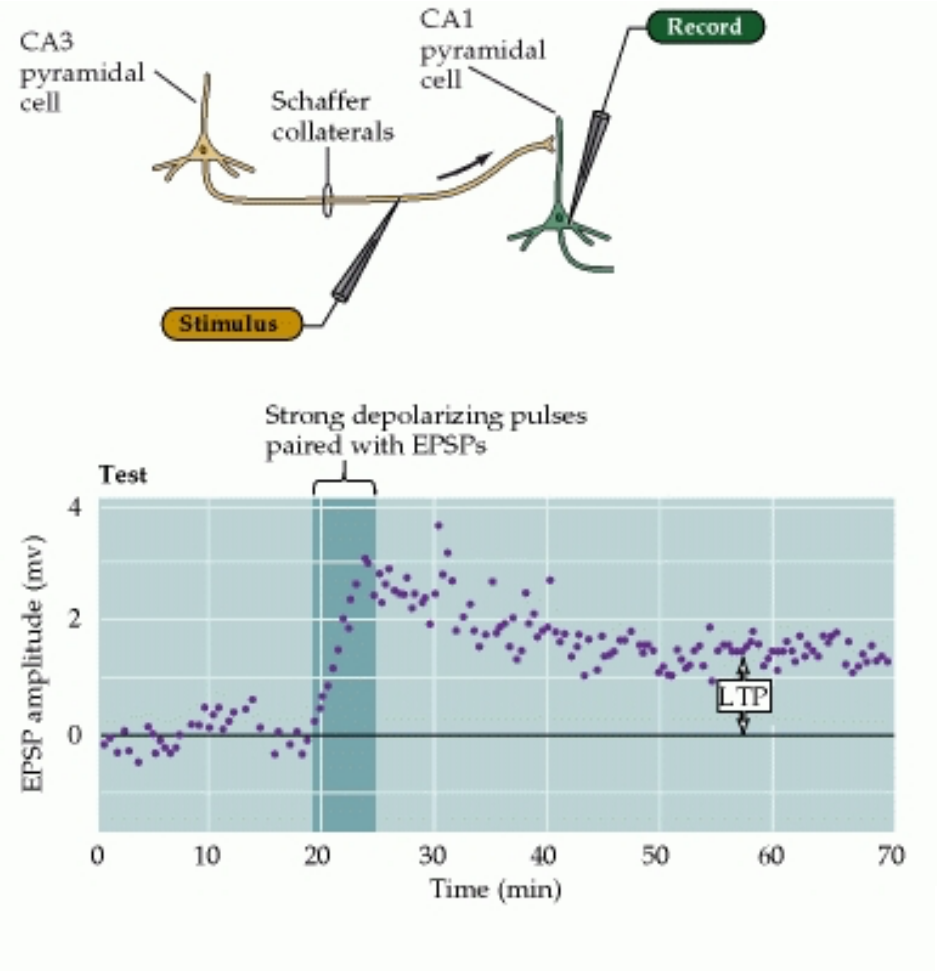
To keep firing rates from differing too greatly between the two cases, the value of reset voltage is higher in B than in A

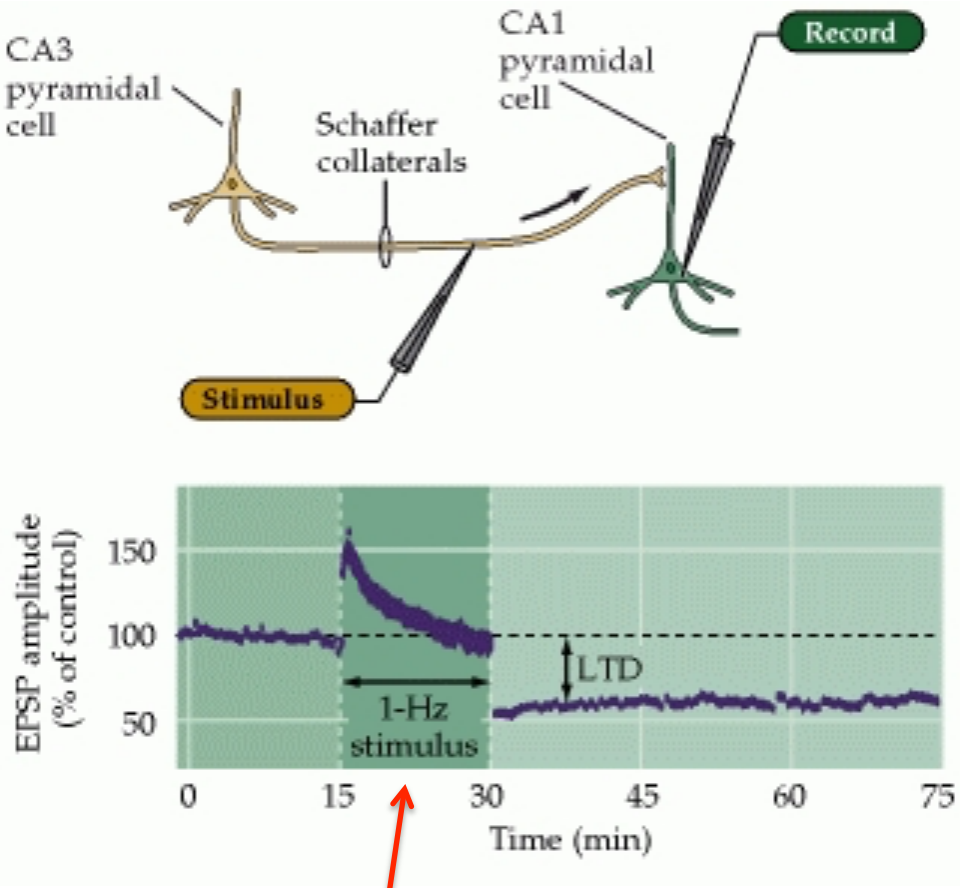
Dayan & Abbott (2001)

Activity-dependent synaptic plasticity

- Widely believed as the basic phenomenon underlying learning and memory
- Hebb (1949): if input from neuron A often contributes to the firing of neuron B, then the synapse from A to B should be strengthened
- More recently, Hebb's suggestion has been generalized to include decreases in strength arising from repeated failure of neuron A to be involved in the activation of neuron B

LTP and LTD



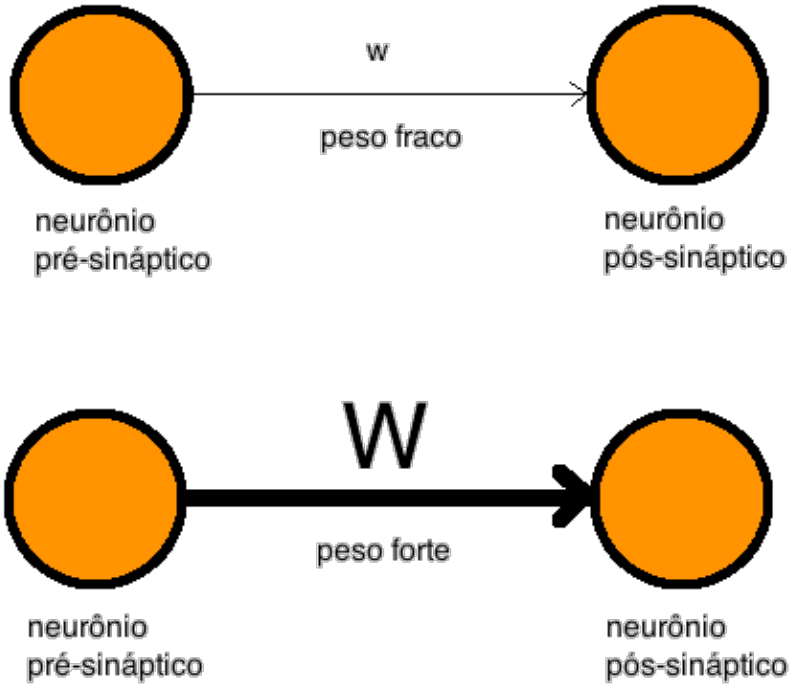


Low-frequency stimulation

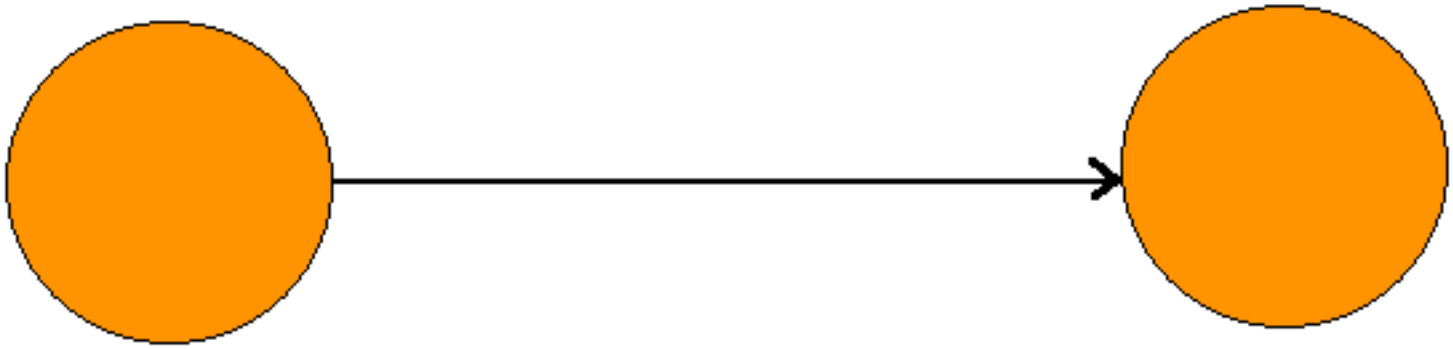
Purves et al. (2001)

Synaptic weight (w_{ij})

- Simplest synaptic model, mostly used by firing rate models but good to illustrate synaptic plasticity rules

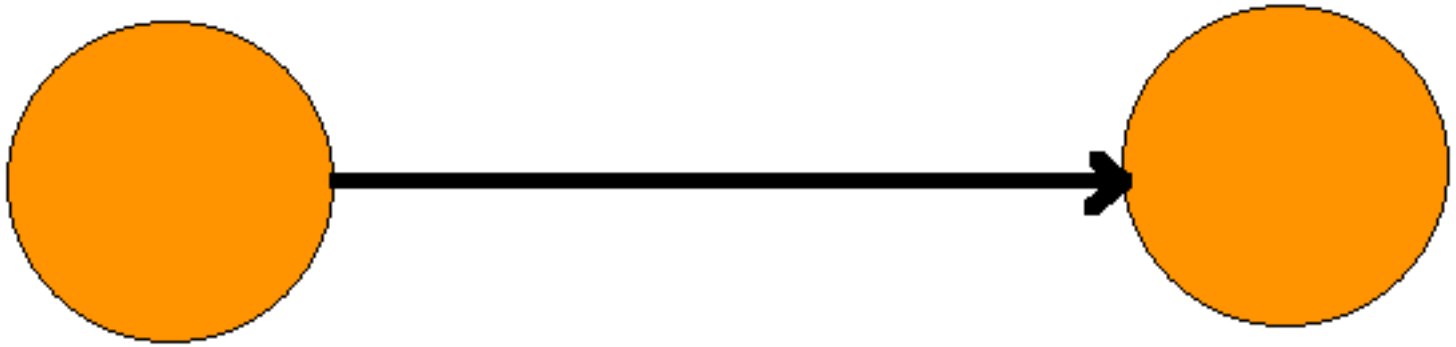


Hebbian plasticity



Hebbian plasticity

Hebbian plasticity



Hebbian plasticity

Hebbian plasticity

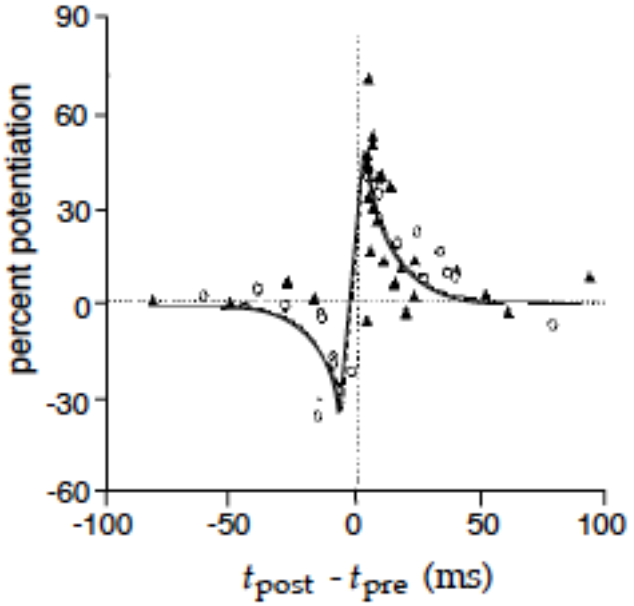
Hebbian plasticity

Hebbian plasticity

Neurons that fire together wire together

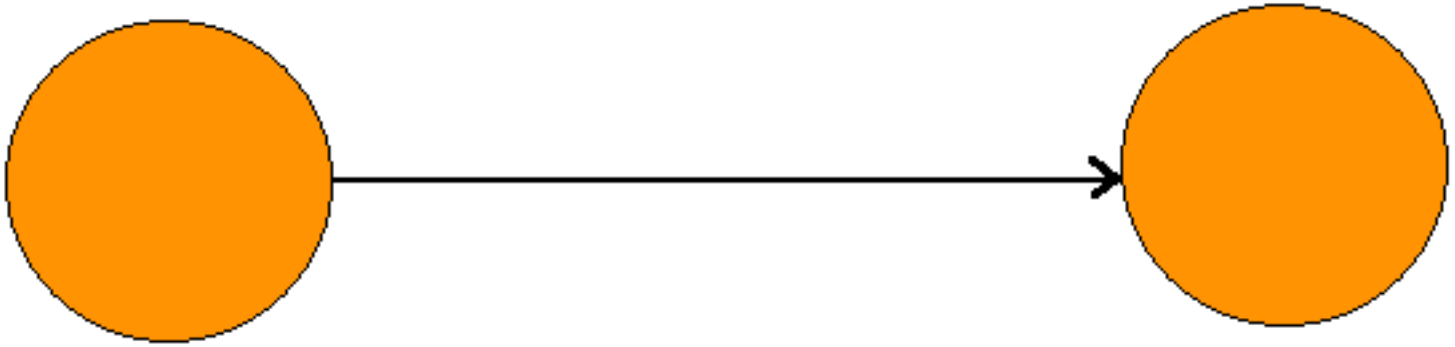
Spike-timing dependent plasticty (STDP)

- The relative timing of pre- and postsynaptic affects the sign and amplitude of the activity-induced changes in synaptic efficacy



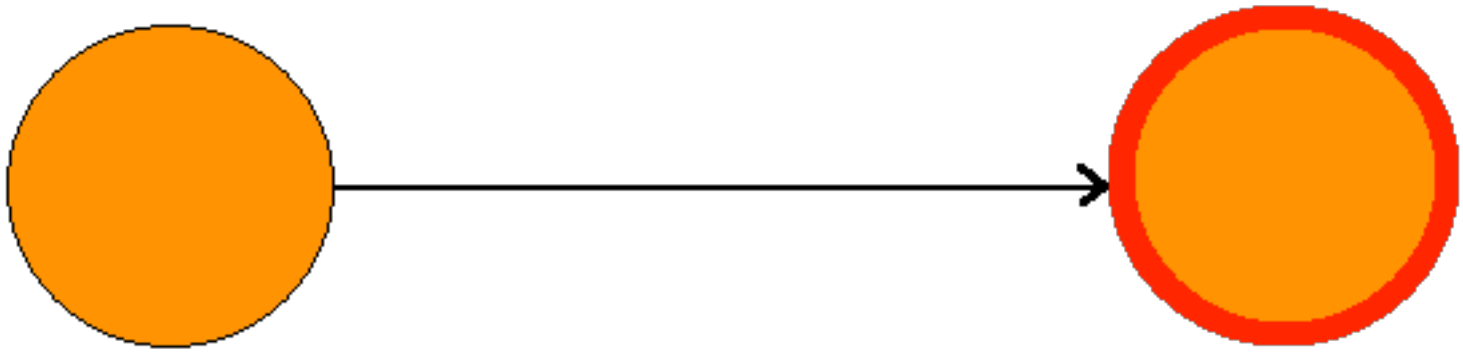
Zhang et al., 1998 (Fig. 8.2B from Dayan & Abbott, 2001)

Spike-timing dependent plasticity



Spike-timing dependent plasticity

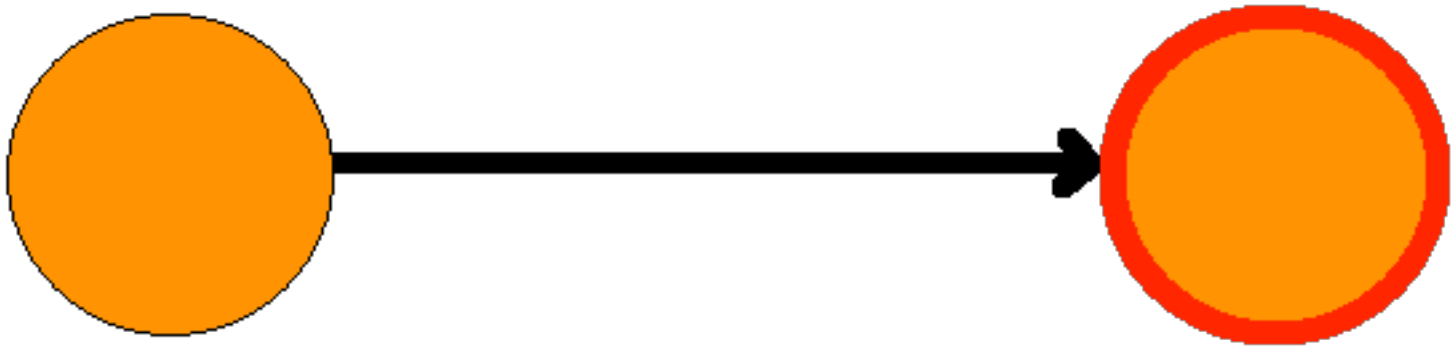
Spike-timing dependent plasticity



Spike-timing dependent plasticity

Spike-timing dependent plasticity

Spike-timing dependent plasticity



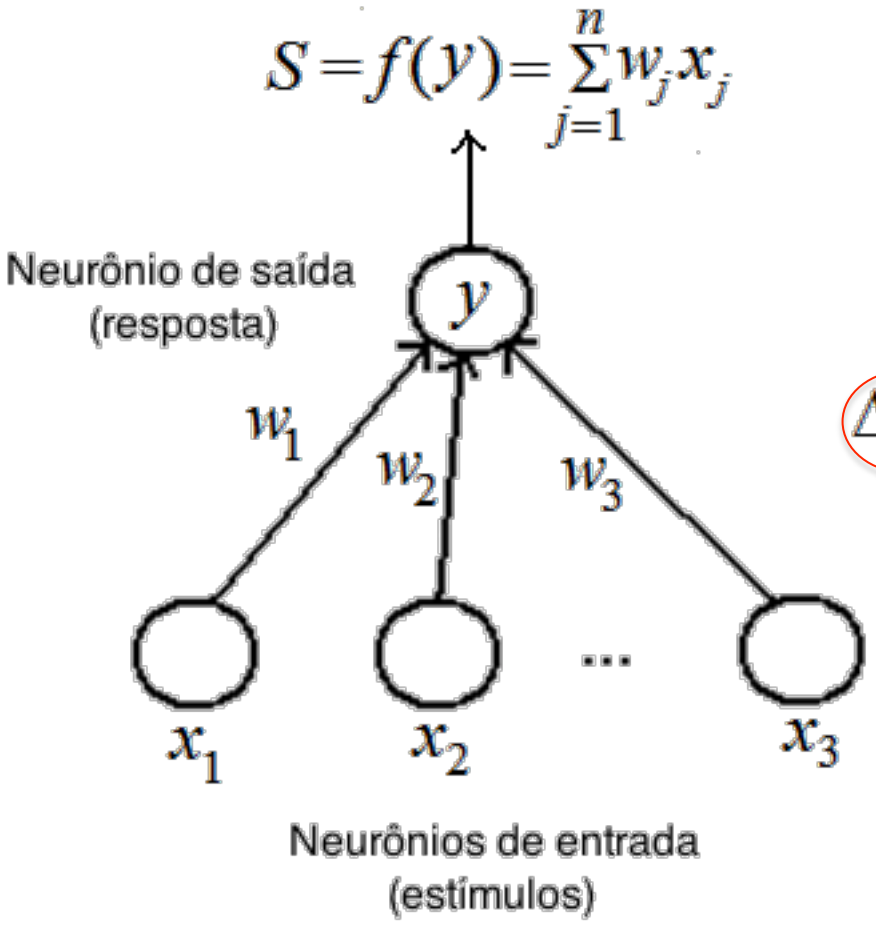
Spike-timing dependent plasticity

Models of long-term plasticity

$$I_{\text{syn}} = \overline{g} \cdot s(t) \cdot \left(V_{\text{post}} - V_{\text{rev}} \right)$$

- Changes in $\overline{g}$ (to model incorporation or removal of channels)
- Implemented by hand or through a **learning rule**
- The time-scale of $\overline{g}$ changes is much slower than the one of membrane potential dynamics

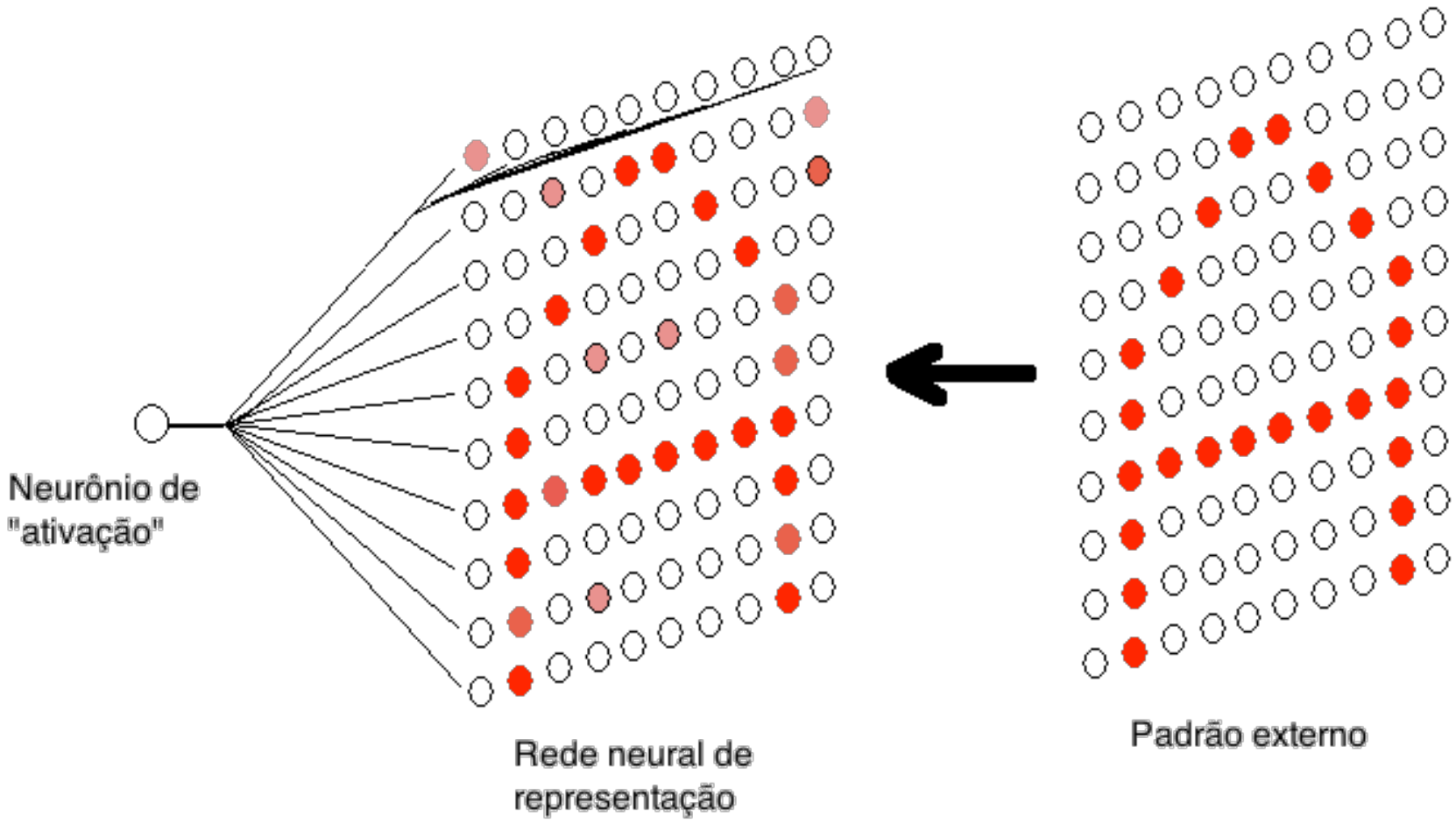
Example of learning rule (Hebb's rule)

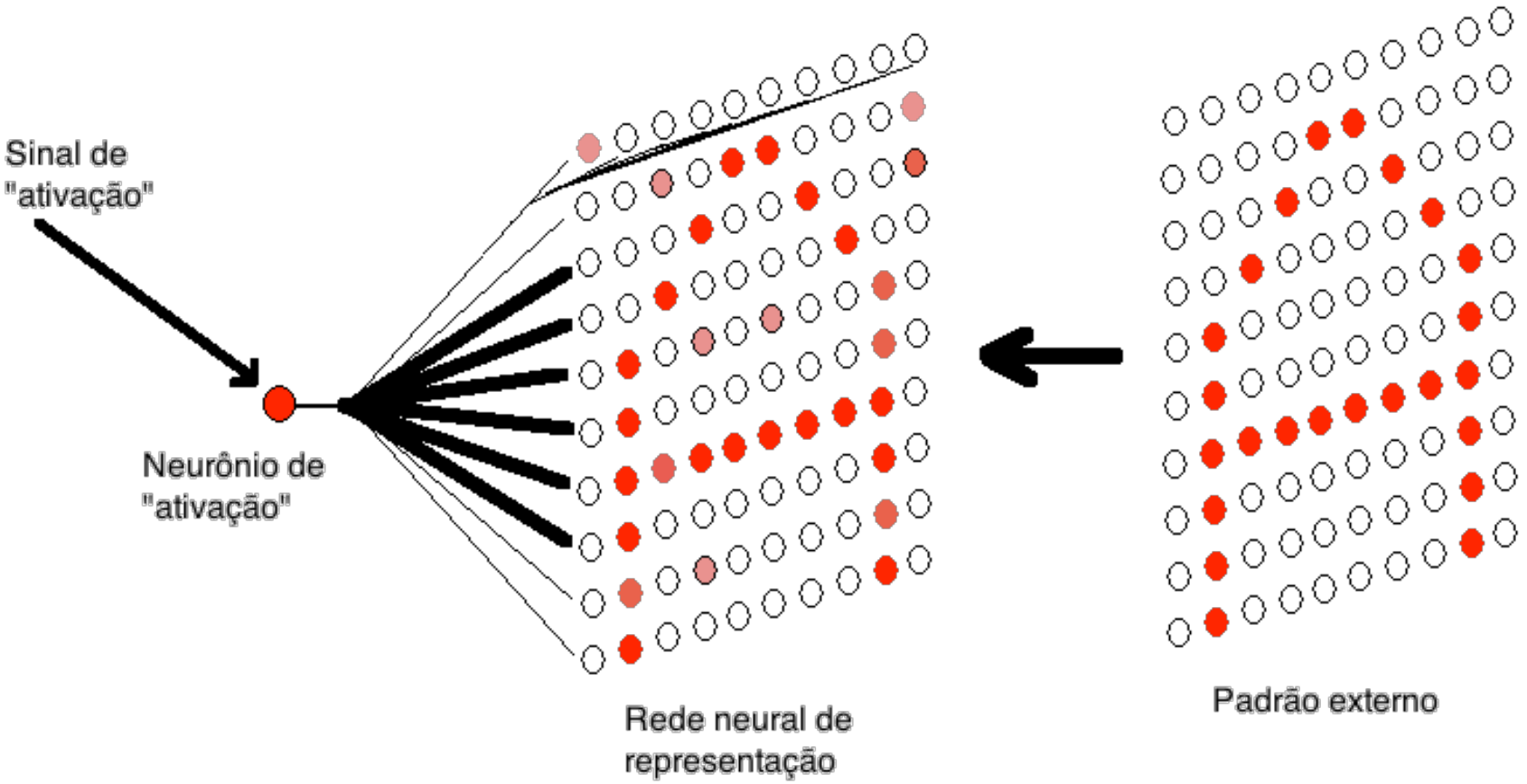


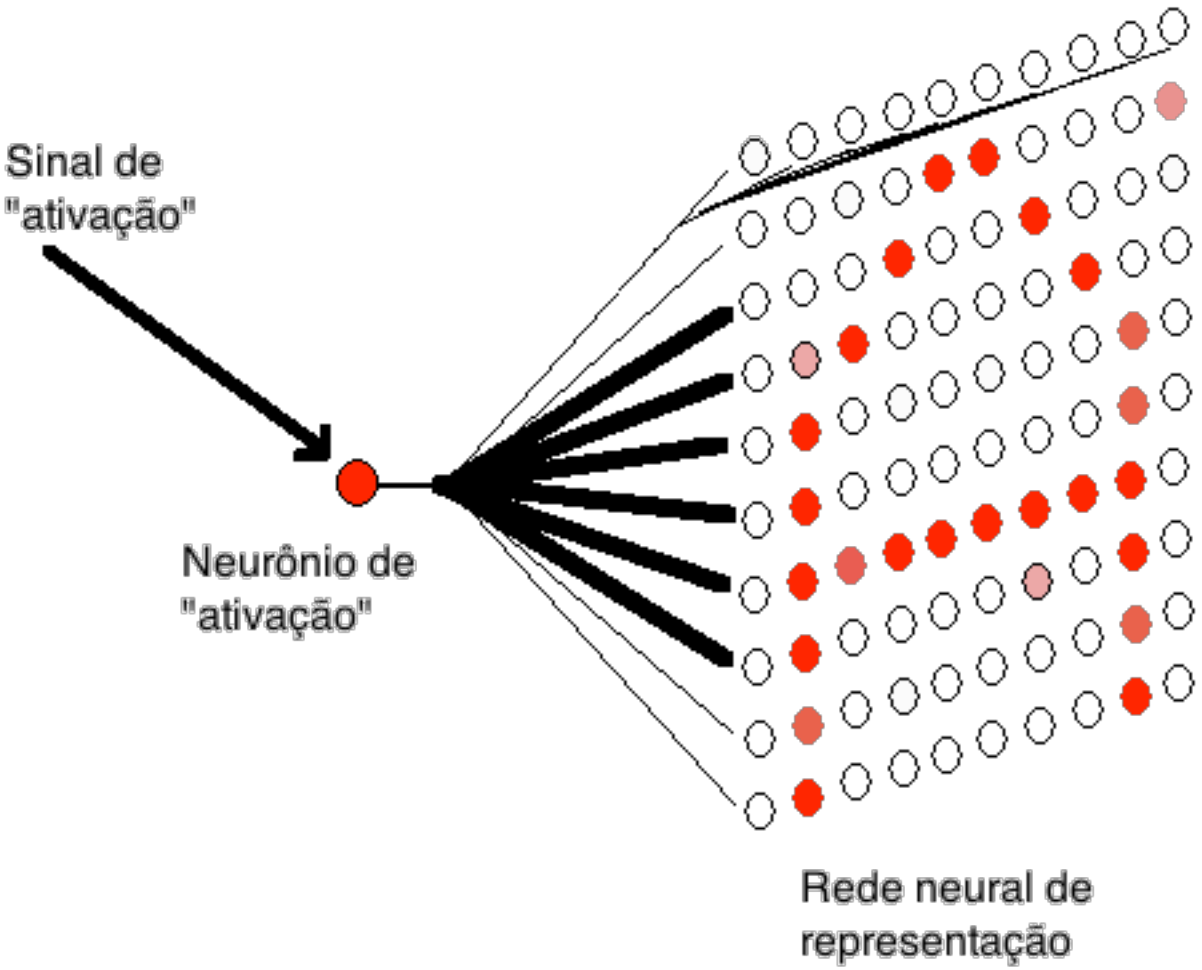
$\Delta w_i = \beta S\, x_i$

Change in *i*th synaptic weight (Hebb's rule)

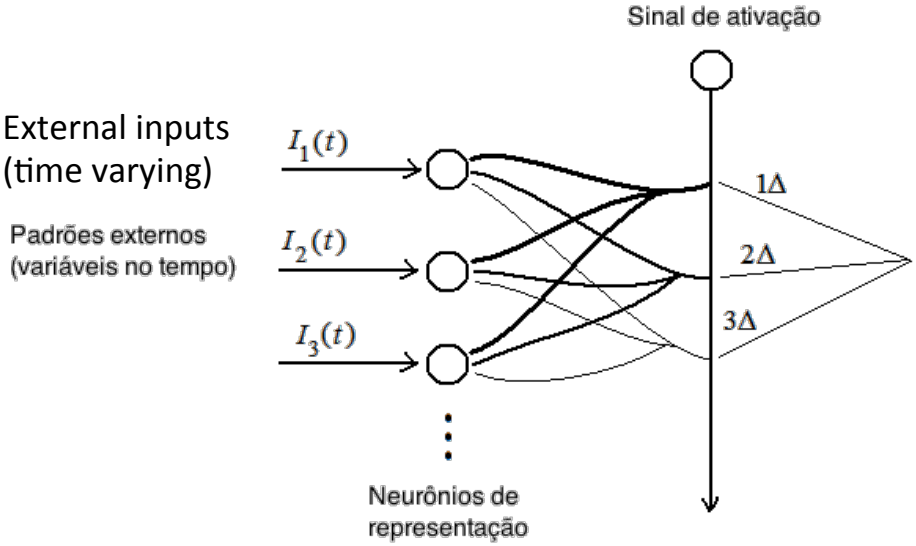
β = learning rate;
S = activity of postsynaptic neuron;
x_i = activity of *i*th presynaptic neuron


Neurônio de
"ativação"
Rede neural de
representação
Padrão externo


Sinal de
"ativação"
Neurônio de
"ativação"
Rede neural de
representação
Padrão externo


Sinal de
"ativação"
Neurônio de
"ativação"
Rede neural de
representação

Other implementations



Time intervals at which the activation neuron sends signals to representation neurons