LECTURE 5: HEBBIAN LEARNING

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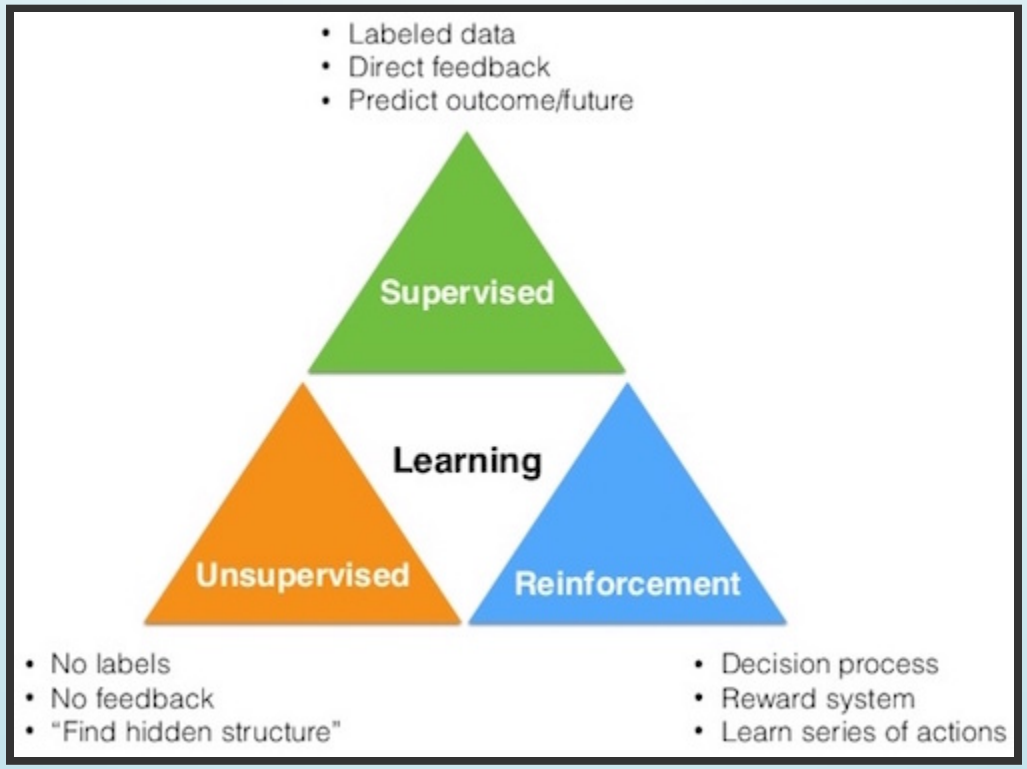
INTRODUCTION

- The steps to build and test a CCN model were the following:
- Step 1: define your units and what they do (e.g., LIF, AdEx)
- Step 2: define their connectivity (e.g., Synaptic models)
- Step 3: relate model activity to experimental measures (e.g., LFP model, decision making model)
- Regarding Step 2, we have talked only about fixed synapses
- In the following two classes we will cover synaptic changes (i.e., learning)

TYPES OF LEARNING

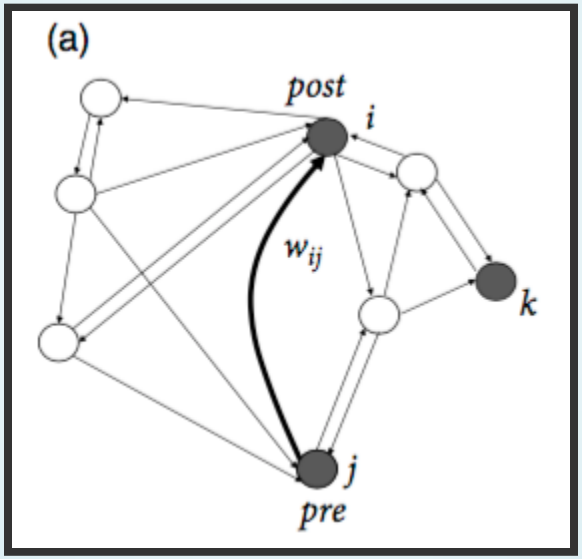
- The study of learning has been heavily influenced by machine learning
- In machine learning, a common classification of learning algorithms is:



- It’s not clear how useful this classification is in neuroscience, but it is commonly used
- This week we will talk about Hebbian learning
- Usually considered as a form of unsupervised learning
- Also considered the basis to implement more complex unsupervised learning algorithms (e.g., PCA, ICA) in the brain
- Next week we will talk about reinforcement learning

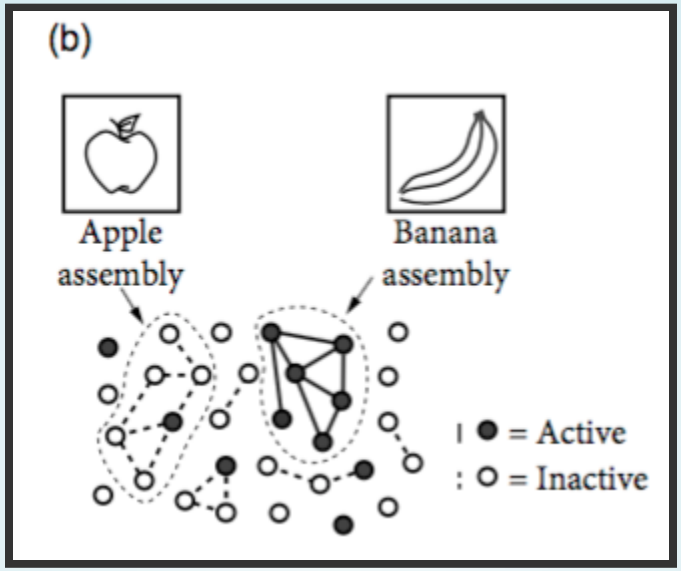
HEBBIAN LEARNING

- If neuron *j* repeatedly takes part in firing neuron *i*, then *j*'s efficacy to fire *i* (represented by w_{ij}) increases:



- All learning in which changes to w_{ij} depend only on correlated firing of *i* and *j* is known as Hebbian

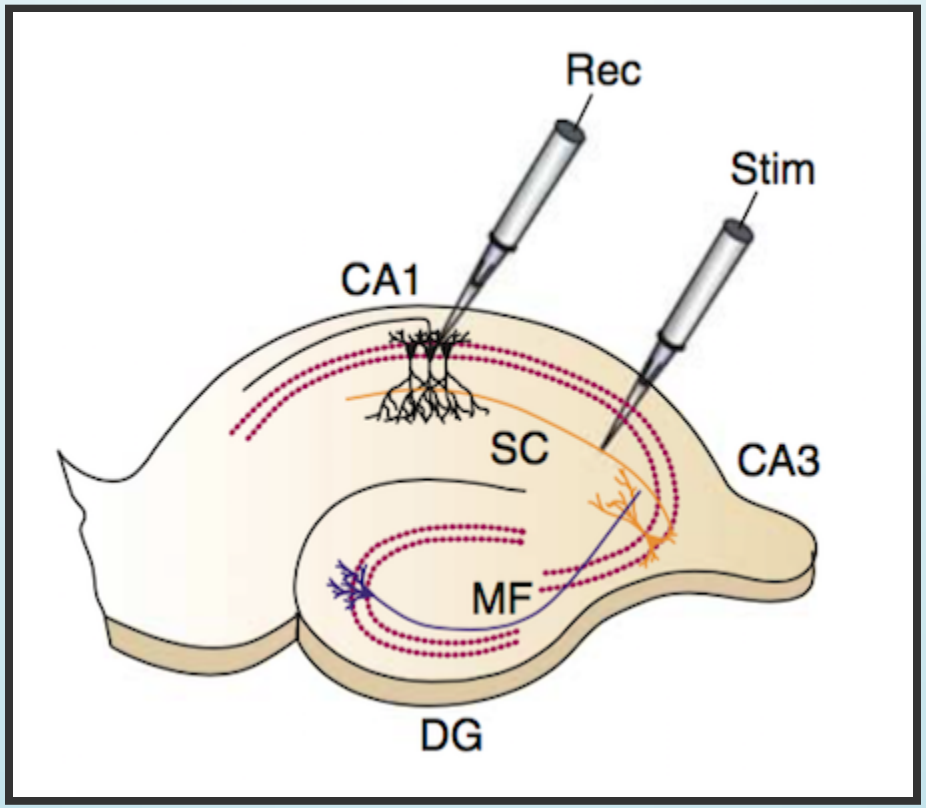
- Originally, Hebb's interest in this simple form of learning was as a basis for more complex representations:



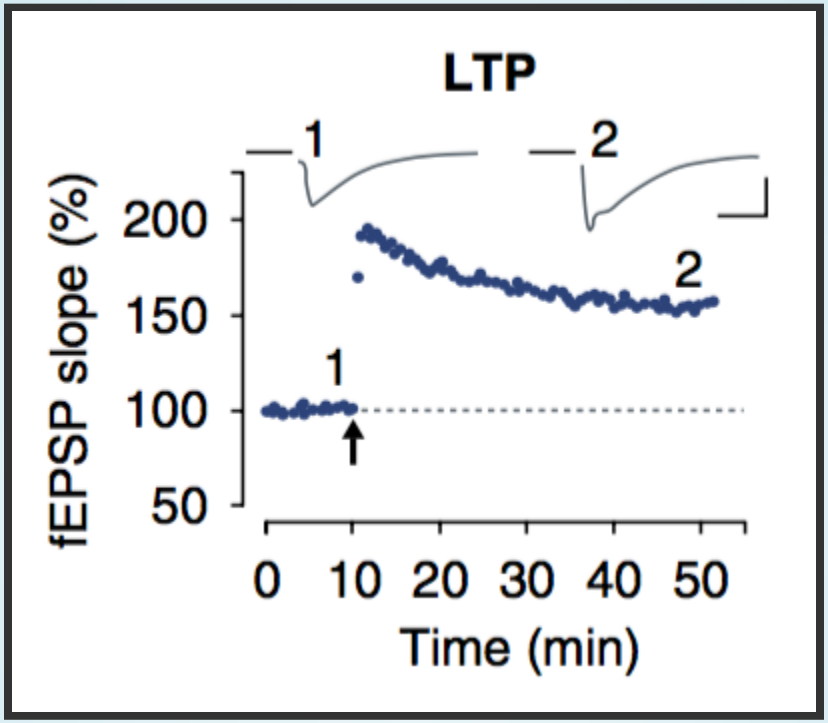
- Nowadays, too, Hebbian learning is not considered interesting on itself, but rather the basis for more complex forms of unsupervised learning

LTP AND LTD

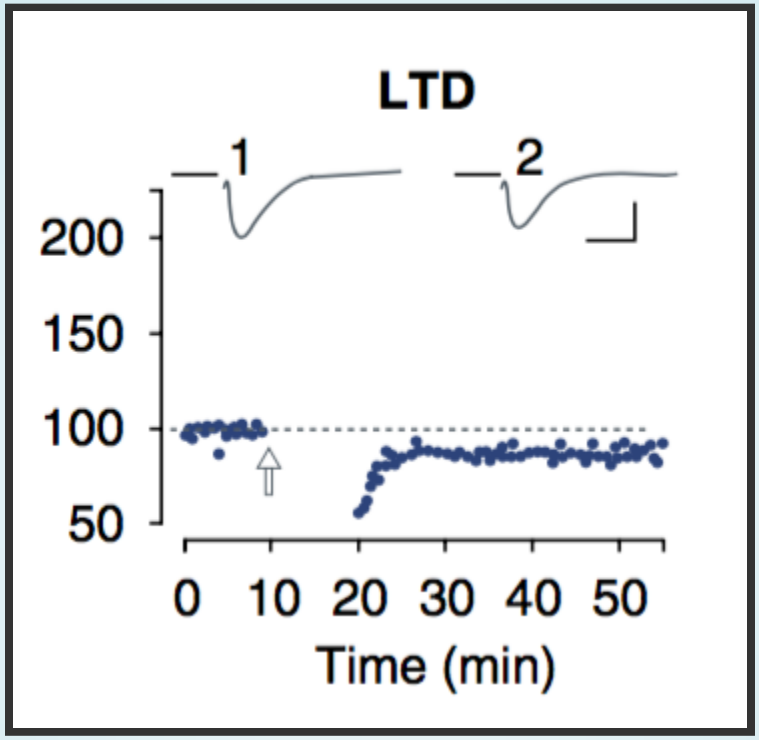
- For decades, LTP and LTD have been considered prime examples of Hebbian learning in the brain
- The experimental setup for a typical LTP/LTD experiment looks like this:



- High-frequency stimulation (e.g., 100Hz) produces LTP:



- Low-frequency stimulation (e.g., 5Hz) produces LTD:



FIRING RATE MODELS

- In a firing rate model, we only care about how often a neuron fires, not about the precise timing of each spike
- We can represent the firing rate of the presynaptic neuron by v_j
- We can represent the firing rate of the postsynaptic neuron by v_i
- The synaptic efficacy or weight is represented by w_{ij}
- There are many possible rules for updating w_{ij} that can be considered Hebbian rules:

1) Simple Hebbian rule

- $\frac{dw}{dt} = \gamma v_i \, v_j$
- Here the change in synaptic efficacy is proportional to the presynaptic firing rate times the postsynaptic firing rate
- γ is a learning rate, determining how much w_{ij} changes
- Note that if either v_i or v_j are zero, there is no learning
- In this model, v_i and v_j are *instantaneous* firing rates; that is, hypothetical variables representing how fast a neuron is firing at a single time t

- We could also propose a version of the model in which the firing rates are actual spiking activities in a given window of time (e.g., in an experimental trial)
- In this case, the change in synaptic efficacy is represented by Δw
- Problem: in this model, w_{ij} keeps growing forever
- The typical solution is to **add a bound**
- For example, w_{ij} is not allowed to be higher than w_{max}

- A **hard bound** is achieved by simply setting $\gamma = 0$ when $w_{ij} > w_{max}$
- A **soft bound** is achieved by slowly reducing the learning rate as the weight gets closer to w_{max}; that is, as a function of $w_{max} - w_{ij}$
- Typically, $w_{max} = 1$ and we get the following rule:
- $\frac{dw}{dt} = \gamma(1 - w_{ij})v_i v_j$
- Problem: in this model, w_{ij} keeps growing until it reaches w_{max}

- A typical solution is to **add a decay** or forgetting term:
- $\frac{dw}{dt} = \gamma(1 - w_{ij})v_i v_j - \gamma_0 w_{ij}$
- That is, we assume that the synapse efficacy is continuously being reduced, and decays back to zero in the absence of any correlated activity
- Something similar to this final version of the model is what you will usually find in simulations involving simple Hebbian learning
- Bounds and decay terms are common additions to all Hebbian learning rules, even though I'll omit them later (following Gerstner)

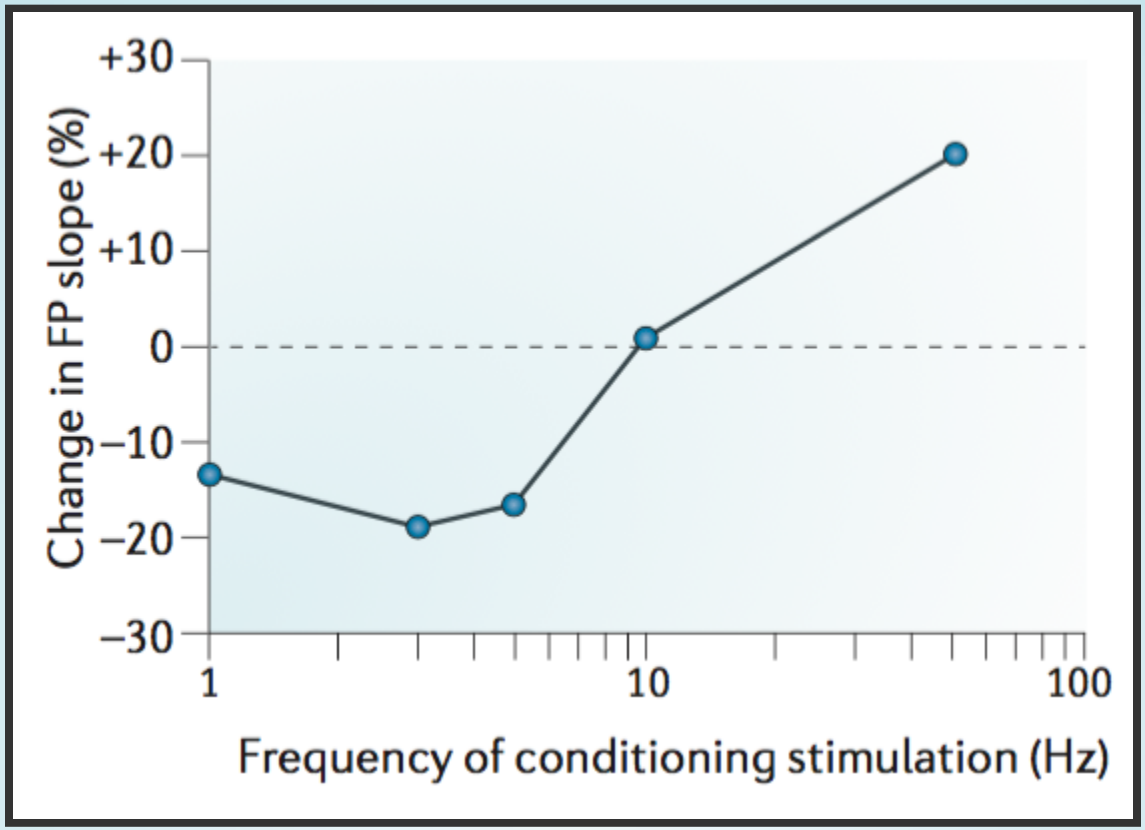
2) Oja's rule

- There's another way to constrain w_{ij}: **normalization**
- Only works when many presynaptic neurons j connect to a postsynaptic neuron i
- We want to constrain all w_{ij} to add up to some constant value C: $\Sigma_j w_{ij} = C$ or $\sqrt{\Sigma_j w_{ij}^2} = C$
- This means that if one synapse grows, the others decay, making learning "competitive"
- For this reason, its biological plausibility is not clear
- Oja's rule performs *normalization* implicitly:

$$\frac{dw_{ij}}{dt} = \gamma \left[v_i v_j - w_{ij} v^2 \right]$$

ACTIVITY THRESHOLDS

- Up to this point, we have included two **passive** ways in which synaptic efficacy can be reduced: by decay (forgetting) and normalization
- However, LTD is an **active** modification of synapses, in that it depends on pre- and post-synaptic activity
- When frequency of stimulation is continuously varied, we see a threshold between LTD and LTP:


+30
+20
+10
0
−10
−20
−30
Change in FP slope (%)
1
10
100
Frequency of conditioning stimulation (Hz)

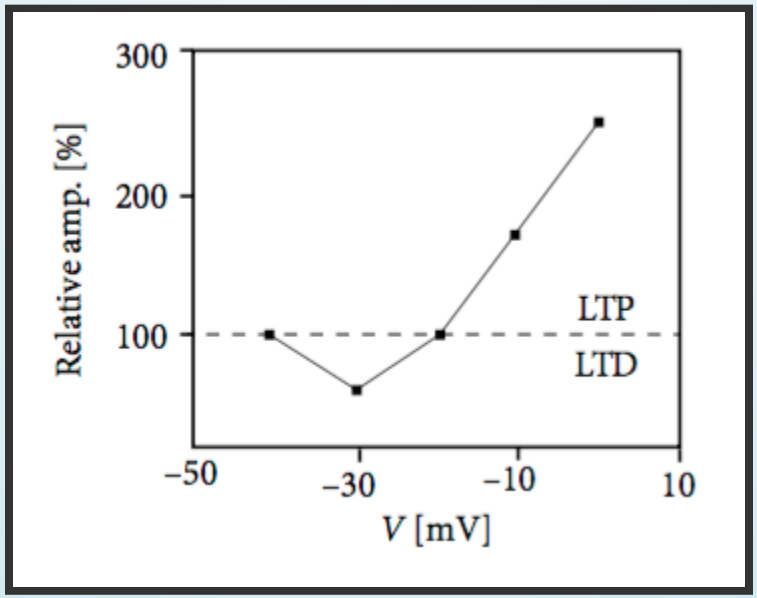
- Let's define a threshold on the presynaptic activity v_θ^{pre} and a threshold on the postsynaptic activity v_θ^{post}
- We can define a learning rule so that when presynaptic activity is below threshold, we see LTD:
- $\frac{dw_{ij}}{dt} = \gamma\left(v_j - v_\theta^{pre}\right)v_i$
- That is, when v_j is lower than v_θ^{pre}, the term in parentheses is negative and w_{ij} decreases
- We can also define a rule so that when postsynaptic activity is below threshold, we see LTD:
- $\frac{dw_{ij}}{dt} = \gamma\left(v_i - v_\theta^{post}\right)v_j$

1. Covariance rule

- We can also combine both of our threshold rules:
- $\frac{dw_{ij}}{dt} = \gamma\left(v_j - v_\theta^{pre}\right)\left(v_i - v_\theta^{post}\right)$
- If we set the thresholds to be the average activity of each neuron, or $v_\theta^{pre} = \left\langle v_j \right\rangle$ and $v_\theta^{post} = \langle v_i \rangle$, we get the covariance rule:
- $\frac{dw_{ij}}{dt} = \gamma\left(v_j - \left\langle v_j \right\rangle\right)(v_i - \langle v_i \rangle)$
- A neuron firing above average will contribute a positive term, whereas a neuron firing below average will contribute a negative term

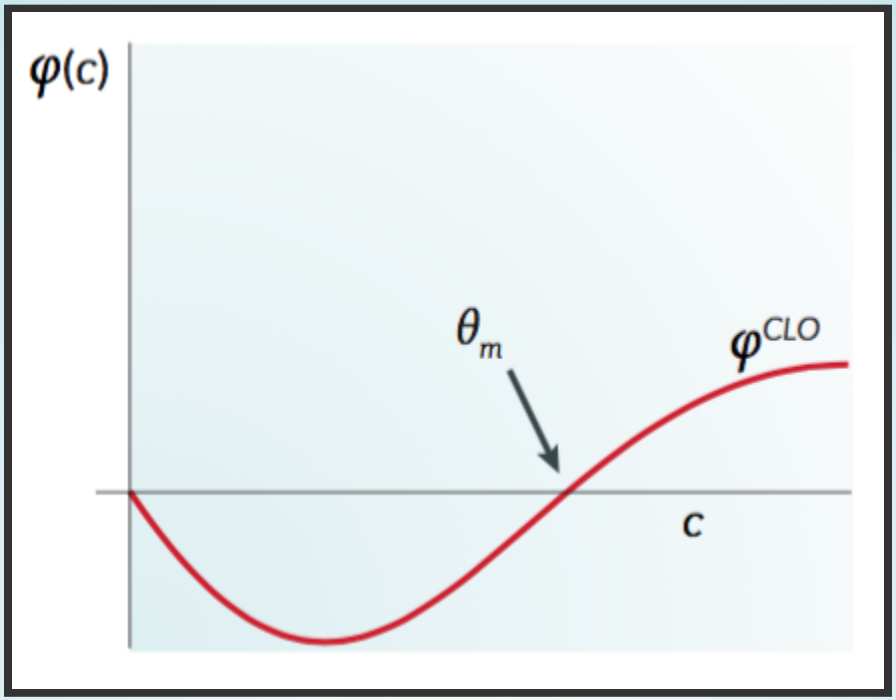
- If both neurons fire above average, we get $+ \times +$: LTP
- If one neuron fires above average, and the other below average, we get $+ \times -$ or $- \times +$: LTD
- The last case is problematic: if both neurons fire below average, we get $- \times -$: LTP
- A solution is to use the threshold in only one of the neurons
- As it turns out, this makes biological sense:

- Newer experiments have allowed to precisely control voltage in the postsynaptic neuron by injecting a current to it, while stimulating the presynaptic neuron
- In this case, it is observed that the variable that critically determines whether LTD or LTP is observed is the voltage of the postsynaptic neuron:

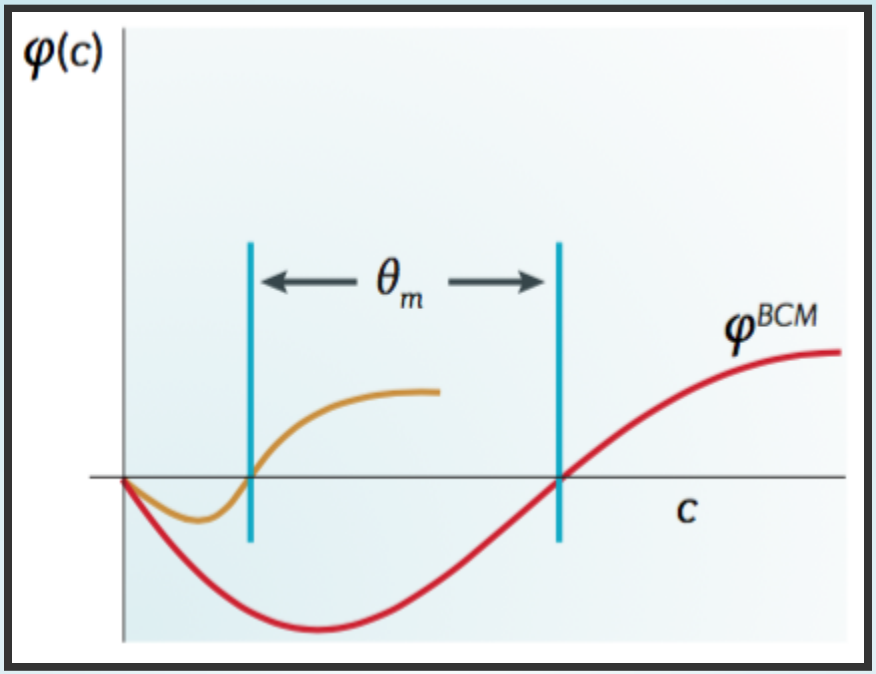


2. The BCM rule

- The BCM model proposes a threshold in the activity of the postsynaptic neuron only, and uses a non-linear function of $\left(v_i - v_\theta^{post}\right)$:
- $\frac{dw_{ij}}{dt} = \gamma \phi \left(v_i - v_\theta^{post}\right) v_j$
- In the original formulation of the model, $\phi \left(v_i - v_\theta^{post}\right) = v_i \left(v_i - v_\theta^{post}\right)$, which looks like this:

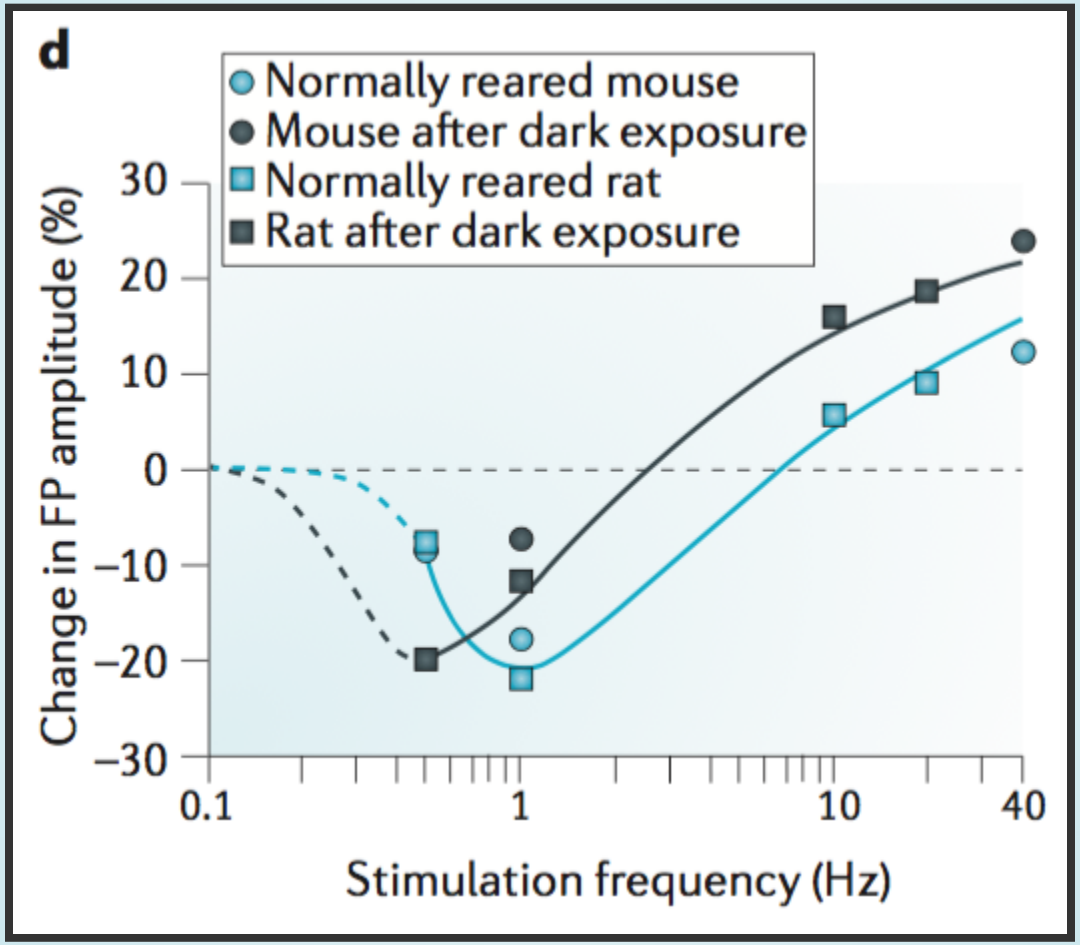


- With a fixed threshold, synapses may grow without bound or decay to zero
- The theory also allowed for changes in the threshold:



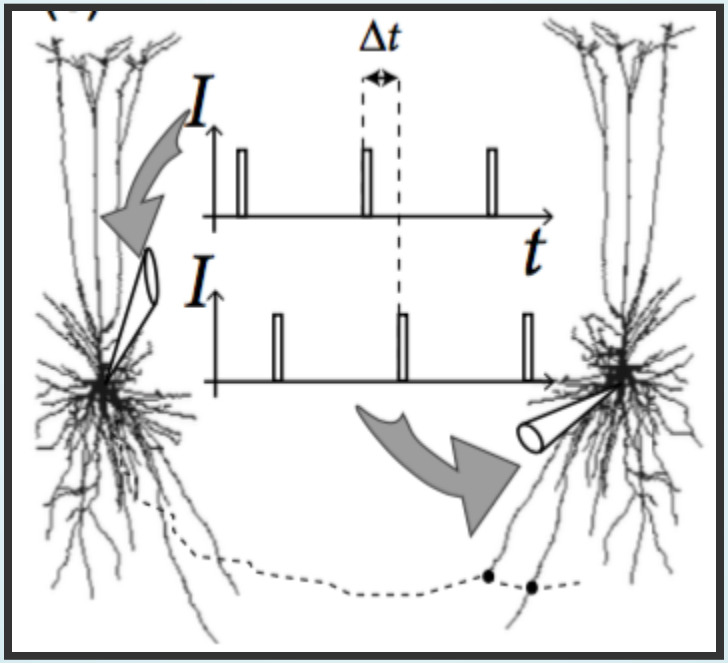
- The threshold changes as a function of $\langle v_i \rangle$
- This feature of the theory was confirmed by later experiments

- One experiment measured LTD and LTP in visual cortex, with normal or visually-deprived rats:



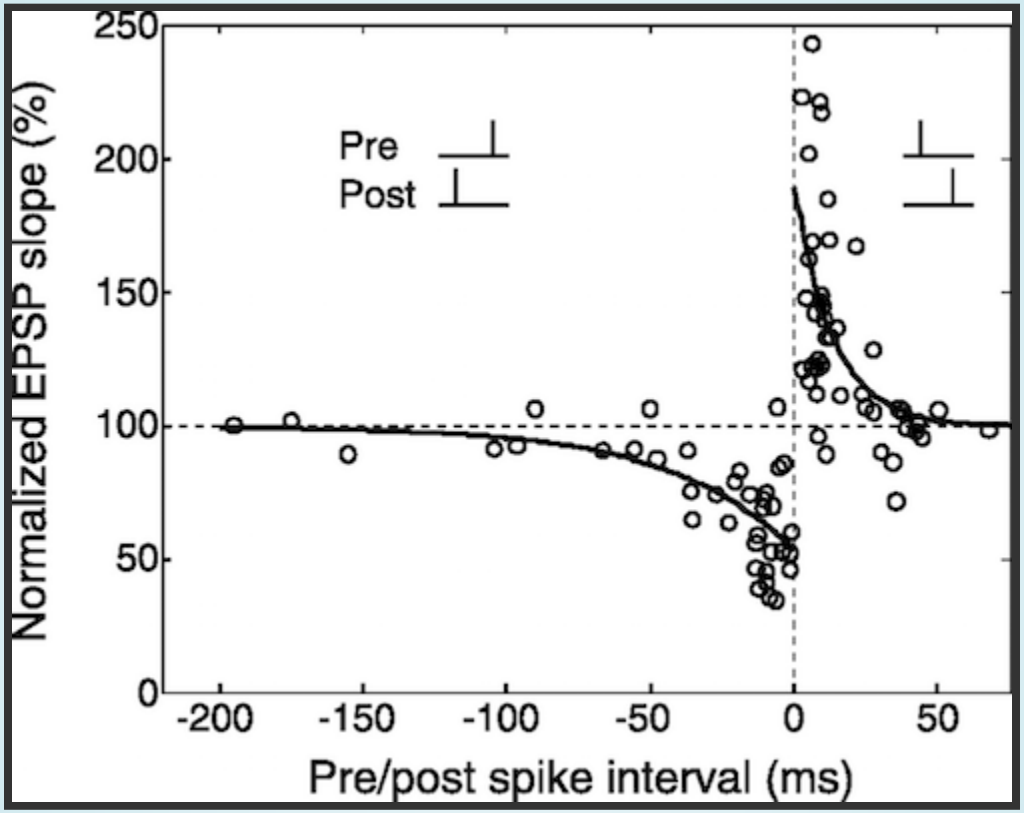
STDP

- An STDP experiment allows to control when spikes are produced in both the pre- and postsynaptic neuron:

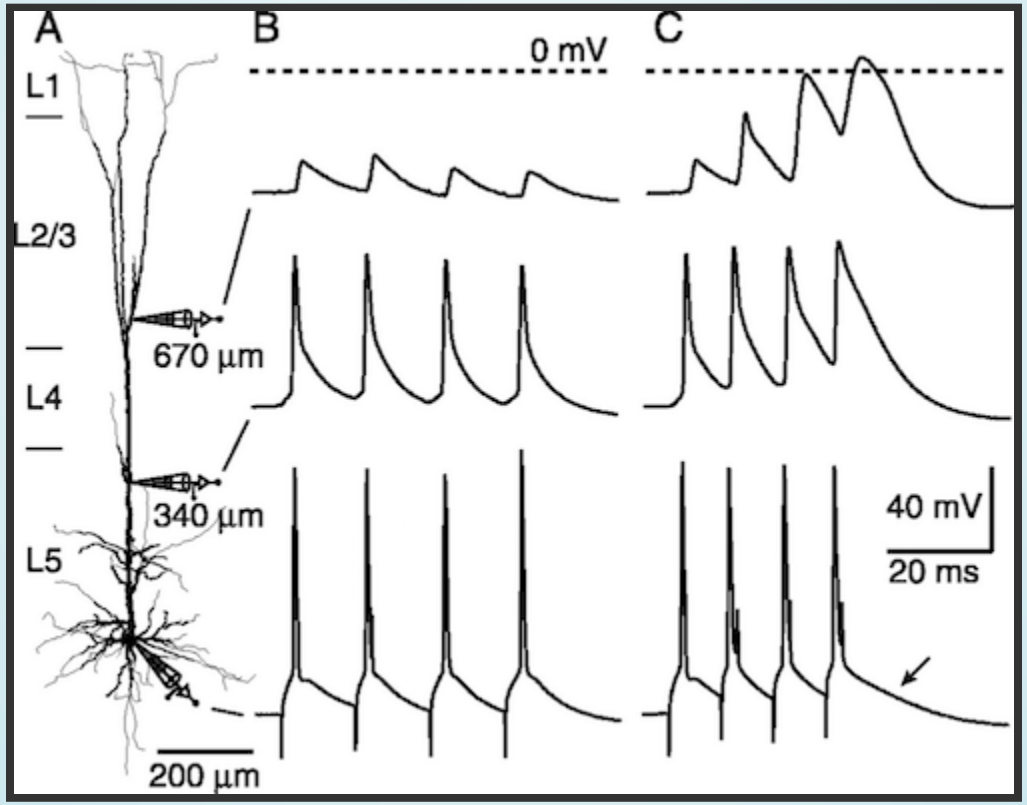


- It is possible to manipulate Δt: the time between pre- and post-synaptic spikes

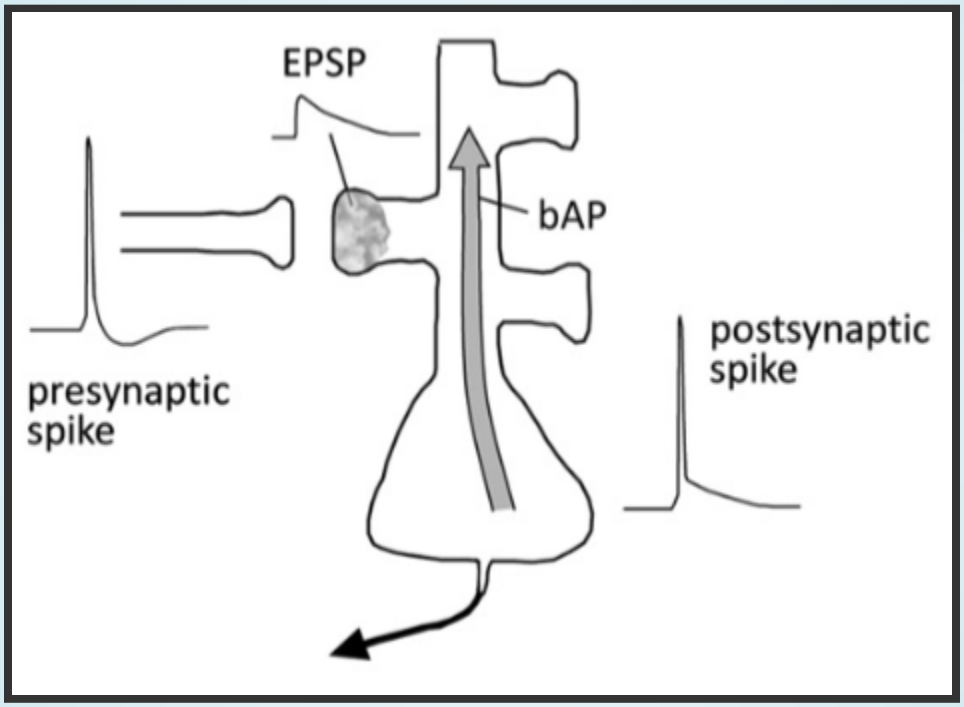
- Typical results look like this:



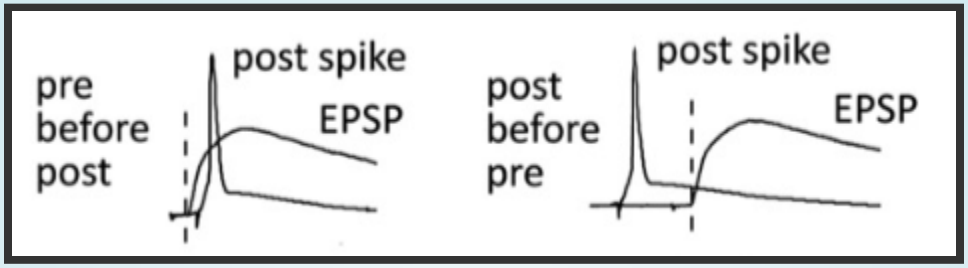
- STDP doesn't make any sense: why would an action potential in the postsynaptic neuron influence a synapse in a dendrite?
- Because the action potential back-propagates:



- The pre-synaptic spike produces an EPSP at the synapse
- The post-synaptic spike produces a back-propagating AP at the synapse



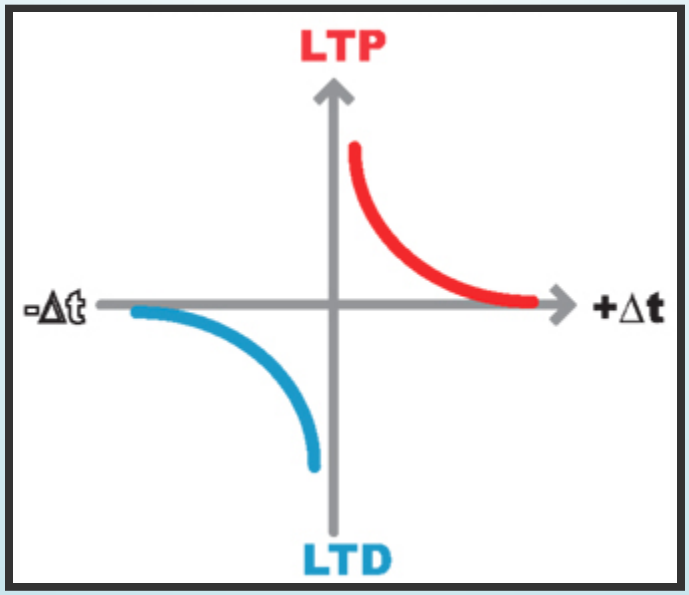
- Changing Δt changes the overlap of these two signals:



- Rate-based models of Hebbian learning cannot explain the spike-timing dependence observed in STDP

KERNEL MODELS OF STDP

- The most common models of STDP are phenomenological
- They use “kernels”: functions that represent the observed dependence of plasticity on spike timing
- For example, the **Pair-based kernel model** proposes two exponential kernels, one for LTP and one for LTD:

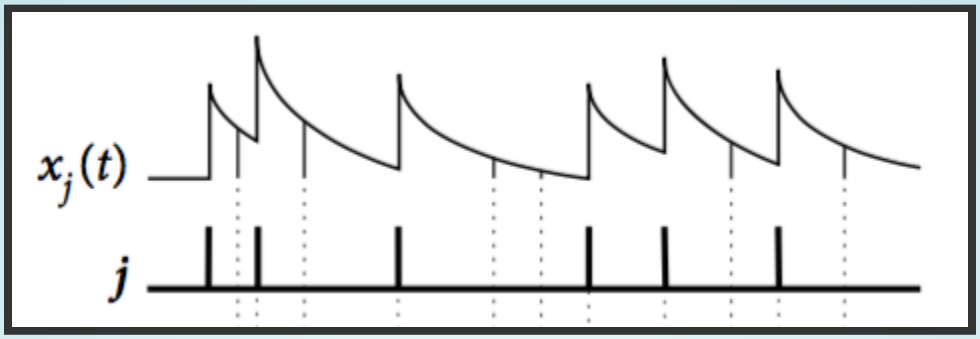


- Each of these kernels is an exponential function of Δt:
- $\exp\left(-\frac{|\Delta t|}{\tau_{\pm}}\right)$
- The only difference between the kernels is how fast they go toward zero, which depend on τ_+ and τ_-
- One implementation is to update the synaptic efficacy w_{ij} for every pair of pre- and post-synaptic spikes
- For example, if the presynaptic spike precedes the postsynaptic spike, we compute $\exp\left(-\frac{|\Delta t|}{\tau_+}\right)$
- Then we simply add the obtained value to w_{ij}, after multiplying it by a bound term $A_+(w)$

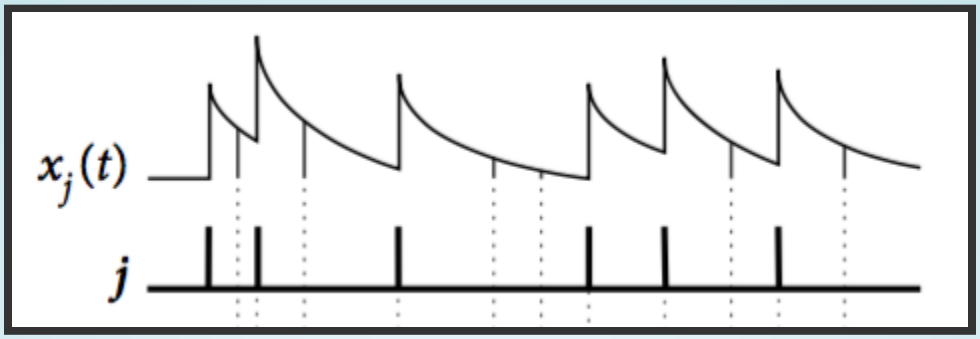
- This is summarized in the following equation:
- $\Delta w_+ = A_+(w) \cdot \exp\left(-\frac{|\Delta t|}{\tau_+}\right)$
- A similar equation describes updates when the postsynaptic spike precedes the presynaptic spike:
- $\Delta w_- = A_-(w) \cdot \exp\left(-\frac{|\Delta t|}{\tau_-}\right)$
- The main difference is that the LTD kernel should be used instead, and w_{ij} goes down

Convolution version

- The reason this is a “kernel” model is because it can be also described as a convolution
- The presynaptic spikes S_j can be represented as a function of time by several delta functions placed at the time of each spike: $S_j = \Sigma_f \delta\left(t - t_j^f\right)$
- This can be convolved with the LTP kernel: $W_+(s) = \exp\left(-\frac{s}{\tau_+}\right)$
- This gives us a new convolved variable, which is a function of time: $x_j(t)$:



- That is, in the convolution version of this model we have that:
- $x_j(t) = \int_0^\infty A_+\left(w_{ij}\right) W_+(s) S_j(t-s) ds$
- This “places” our LTP kernel at the time of each presynaptic spike



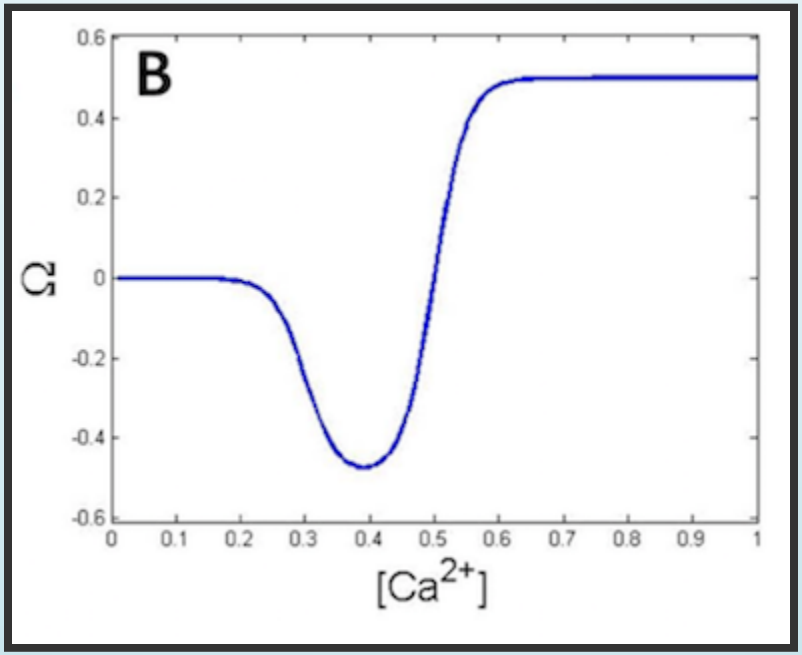
- To update our weights, at the time of each postsynaptic spike we nead to “read out” the value of $x_j(t)$ and add it to our weight
- This is done by multiplying $x_j(t)$ by the postsynaptic train: $S_i(t)x_j(t)$
- If we also convolve the postsynaptic spikes with the LTD kernel, obtaining $y_i(t)$, we can write a learning equation:

- $\frac{dw_{ij}}{dt} = -S_j(t)y_i(t) + S_i(t)x_j(t)$
- The red expression involves LTD
- The blue expression involves LTP
- The convolved variables x_j and y_i can also be implemented through differential equations, using the well-known "jump and decay" model:
- $\frac{dx_j}{dt} = \frac{x_j}{\tau_+} + \Sigma_f \delta\left(t - t_j^f\right)$
- $\frac{dy_i}{dt} = \frac{y_i}{\tau_-} + \Sigma_f \delta\left(t - t_i^f\right)$
- This is what Gerstner calls "local variables" implementation

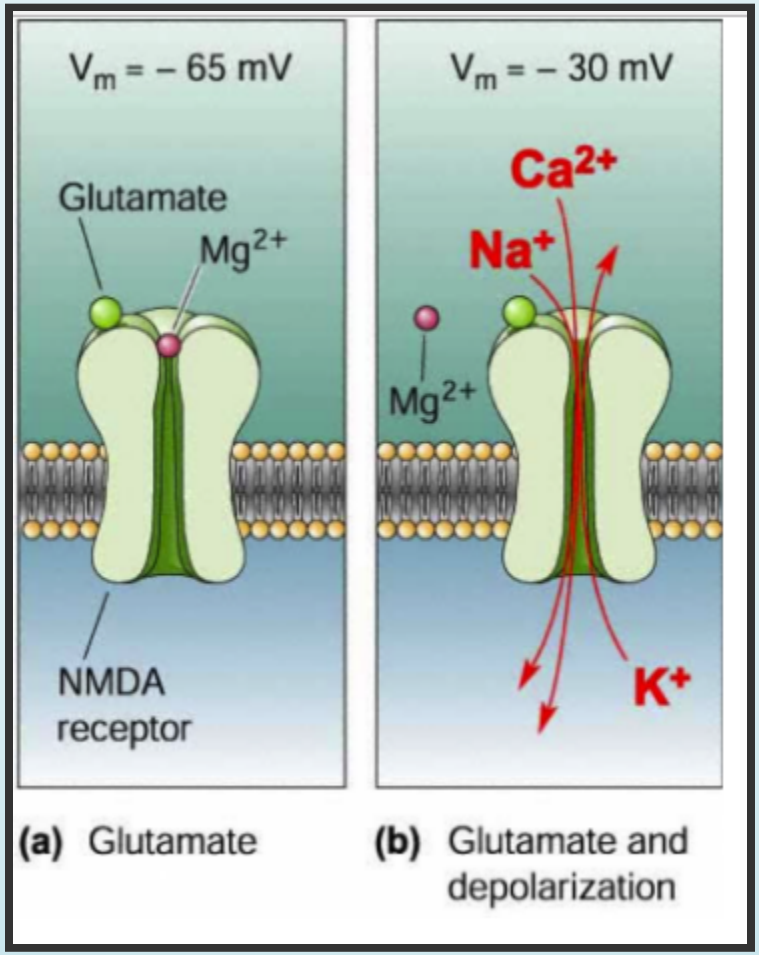
- While this “Pair-based” model cannot account for all the data, a “triplets-based” model (three convolved variables) does a better job
- An important advantage of the triplets model is that it “contains” the BCM model as a special case
- This suggests that STDP is a more “fundamental” phenomenon in synaptic plasticity, and that the STDP kernels summarize everything we know about the topic
- However, kernels and “local variables” do not have a biological interpretation
- The model is phenomenological: it only fits the results, but does not explain the mechanisms

THE CALCIUM HYPOTHESIS

- A different approach is to explain all forms of plasticity through a common mechanism
- The best candidate is the **calcium hypothesis**
- The idea is that obtaining LTD or LTP depends on the Ca2+ concentration at the synapse:

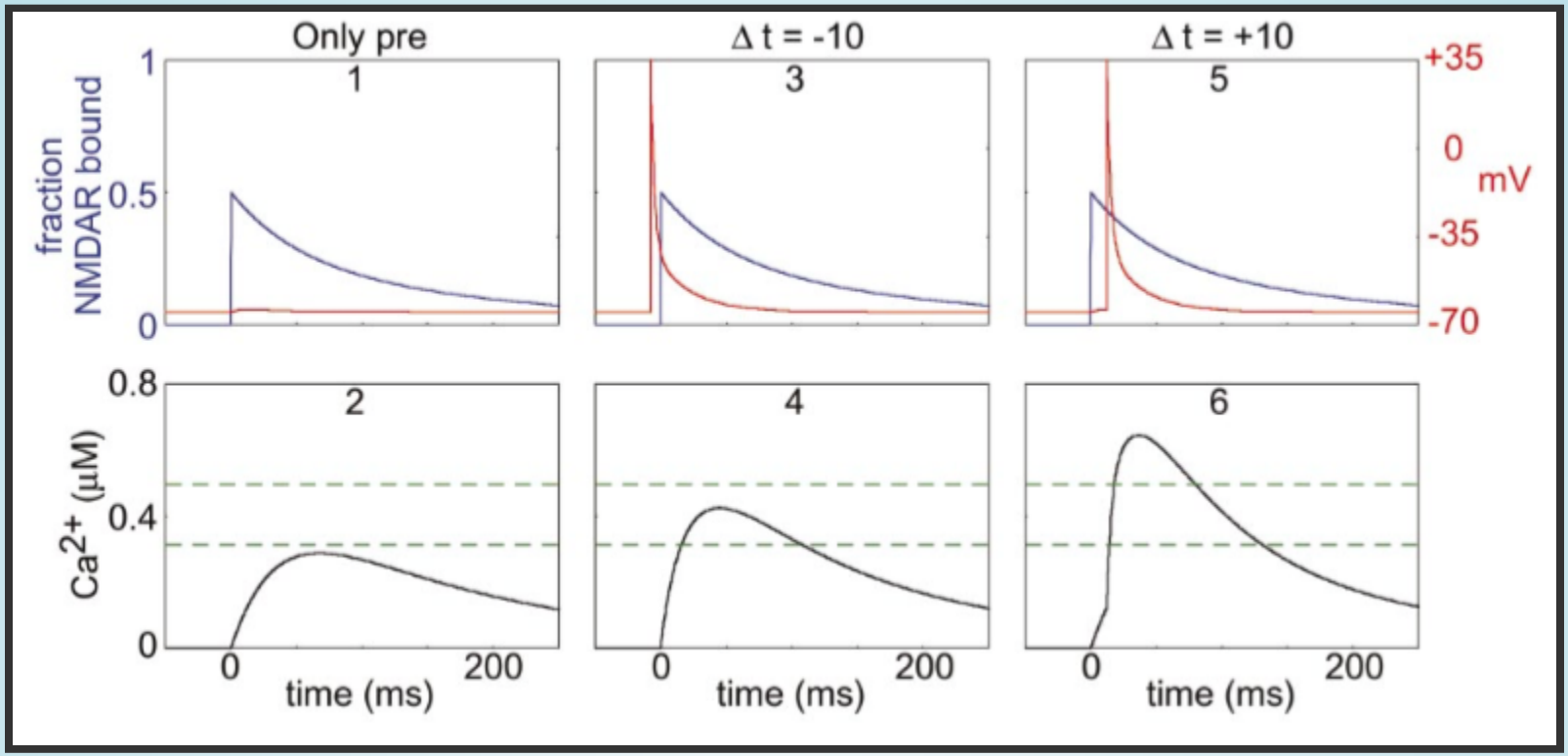


- We saw before that obtaining LTD or LTP depended on postsynaptic voltage
- The likely reason is that higher voltage produces release of the Mg2+ plug from NMDA channels:



- When the plug is off, a presynaptic spike releases glutamate that binds to the NMDA receptor
- The NMDA channel opens, and Ca2+ flows inside the cell
- Experiments have shown that Ca2+ concentration inside the cell influences LTD/LTP as proposed by the calcium hypothesis
- We have already seen how the conductivity of NMDA channels g_{NMDA} and ion currents through the channel I_{NMDA} can be modeled
- Once we have obtained the Ca2+ concentration at the synapse, or $[Ca]_{ij}$, we can use to implement a simple learning rule:

- $\frac{dw_{ij}}{dt} = \gamma\Omega\left([Ca]_{ij}\right)$
- Where the Ω function is as plotted earlier
- This learning rule can explain STDP because when the backpropagating AP can arrive at the synapse, it produces depolarization and release of the Mg2+ plug
- If there is a presynaptic spike before this, glutamate will be present and bind to the NMDA receptor
- If there is a presynaptic spike after this, the short-lived AP will be gone


Only pre
Δ t = -10
Δ t = +10
1
3
5
2
4
6
fraction NMDAR bound
1
0.5
0
+35
0
mV
-35
-70
0.8
0.4
0
Ca2+ (μM)
0
200
time (ms)

IN SUMMARY

- Rate-based models can explain basic results from LTP and LTD experiments
- Perhaps the most successful rate-based model is BCM
- Rate-based models cannot explain STDP
- Kernel models explain STDP and contain BCM as a special case
- However, they are phenomenological rather than mechanistic
- The Ca2+ hypothesis and related learning rule explains more data from LTP, LTD and STDP experiments

THAT'S IT!