BNN Course: Synaptic Plasticity

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Freiburg, 20th July 2017

Reminder:

- git pull (To access new changes to the repository)
- Handouts (www.nest-simulator.org/introduction-to-pynest/)

Synaptic plasticity

- considered to be the biological substrate of learning and memory

Synaptic changes can be induced by specific stimulation conditions:

- presynaptic firing rates
- spike timing

Detailed biophysical models

- crucial to understand undelying biological mechanisms

Phenomenological models

- describe synaptic changes without reference to mechanism
- generally more tractable and less computationally expensive

Outline

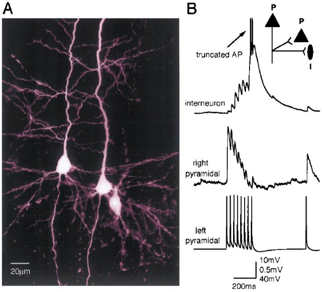
Basic experimental findings

Short-term plasticity
Long-term plasticity
Spike-timing dependent plasticity
Homeostasis

Phenomenological models of synaptic plasticity

Short-term plasticity (very briefly)
Spike-timing dependent plasticity (in detail)

Short-term plasticity

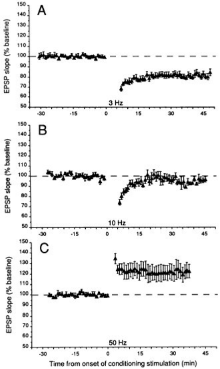


Markram et al. (1998)
Proc Natl Acad Sci USA. **95**(9):5323–5328

- sequence of eight presynaptic spikes at 20Hz evokes successively smaller (depression) or successively larger (facilitation) responses in postsynaptic cell
- same presynaptic neuron makes connections to different types of target neurons with different plasticity properties

- enables the synaptic efficacy to represent the history of presynaptic activity (e.g higher activity - faster depletion of resources - STD)
- change persists only for a few hundred milliseconds (amplitude of the postsynaptic response recovers to close-to-normal values within less than a second)

Long-term plasticity

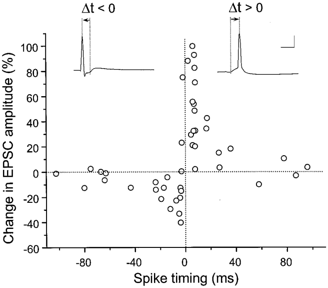


- 900 presynaptic stimulation pulses yield either persistent depression (LTD) or potentiation (LTP) depending on rate

  Dudek & Bear (1992)
  Proc Natl Acad Sci USA. **89**:4363–4367

- sensitive to the presynaptic firing rate over a time scale of tens or hundreds of seconds
- change can persist for more than one hour
- final stabilization on a time scale of hours
  (e.g. late phase of LTP reported by Frey & Morris, 1997)

Long-term plasticity

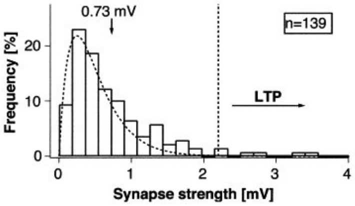


Bi & Poo (1998)
J Neurosci **18**:10464–10472

- LTP if presynaptic spike precedes postsynaptic spike by 10 ms
- LTD if order of spikes reversed
- repetitions of 60 pairs of spikes (single pair has no effect)

- depends on exact timing of pre- and postsynaptic spikes on time scale of milliseconds
- so called spike-timing dependent plasticity (STDP)

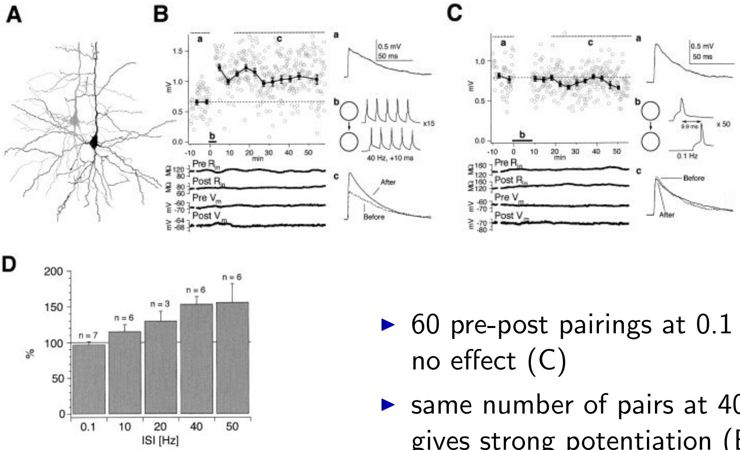
Spike-timing dependent plasticity (STDP)



Sjostrom et al. (2001) *Neuron*. **32**(6):1149–64

- synaptic strength (e.g. EPSP amplitudes) in data collected across several pairs of neurons reported to be unimodal

Spike-timing dependent plasticity (STDP)

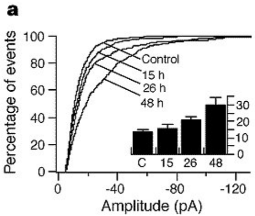


Sjostrom et al. (2001)
Neuron. **32**(6):1149–64

- 60 pre-post pairings at 0.1 Hz have no effect (C)
- same number of pairs at 40 Hz gives strong potentiation (B)

- depends on repetition frequency of pre-post spike-pairings

Homeostasis of synaptic efficacies

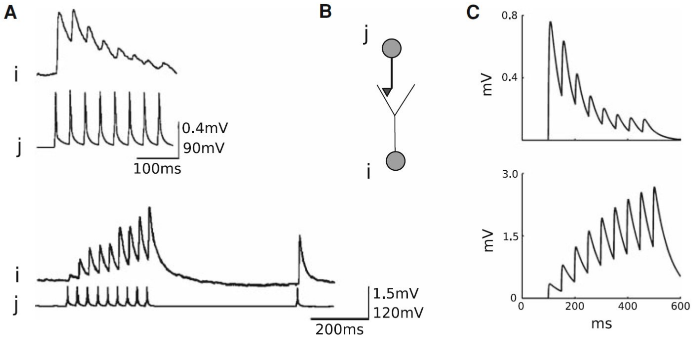


Turrigiano et al. (1998)
Nature. **391**:892–896

- chronic blockade of cortical culture activity increases amplitude of mEPSCs without changing their kinetics

- on a time scale of hours, rescaling of synaptic response amplitudes may occur
- useful to stabilize neuronal firing rates

Phenomenological models of short-term plasticity



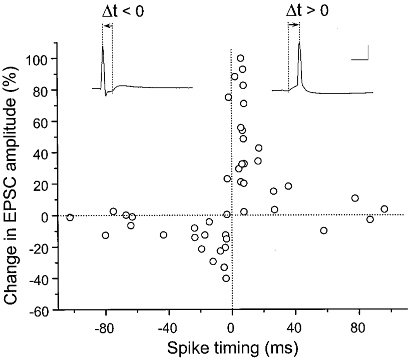
Left: experimental results adapted from Markram et al. (1998)

Right: simulation results using Markram-Tsodyks model

- fast synaptic dynamics firmly established in biological literature (Markram et al. 1998; Gupta et al. 2000)
- well-accepted models exist (Abbott et al. 1997; Tsodyks et al. 1998)

Phenomenological models of spike-timing dependent plasticity

What do we need to specify a pair-based model of STDP?



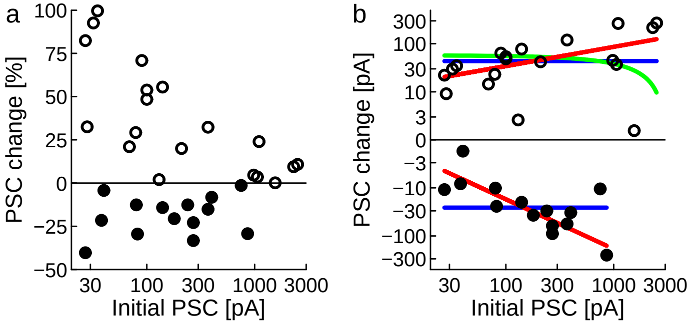
Bi & Poo (1998)
J Neurosci **18**:10464–10472

$$
\begin{aligned}
\Delta w_- &= -F_-(w)\exp\left(-\left|\Delta t\right|/\tau_-\right) \\
\Delta w_+ &= -F_+(w)\exp\left(-\left|\Delta t\right|/\tau_+\right)
\end{aligned}
$$

- weight dependence i.e. $F_\pm(w)$
- spike pairing scheme
- decomposition of synaptic delay into axonal and dendritic components
- asymmetry of STDP kernel $(\alpha = \frac{F_-}{F_+})$

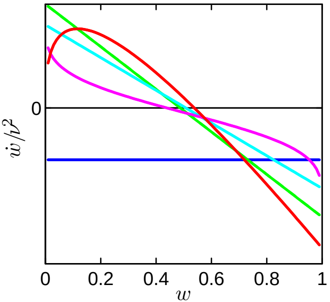
all of these aspects are important and should not be chosen arbitrarily

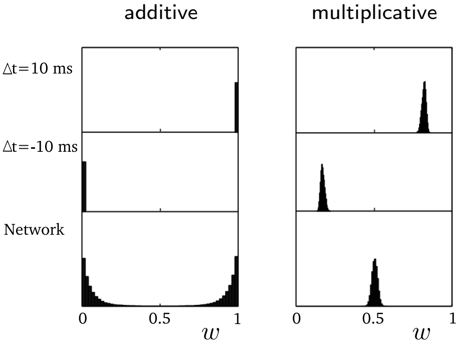
Models of STDP: Weight Dependence



- Reanalysis of Bi & Poo (1998) data shows that the commonly used additive model ($F_- = A_-, F_+ = A_+$) is not a good fit for the data.
- Depression data are best fit by a multiplicative/power law model ($F_- = A_- w^{-1}$)
- Potentiation data are best fit by a power law model ($F_+ = A_+ w^{\mu}$, $\mu = 0.4$)

Models of STDP: Distribution of synaptic weights





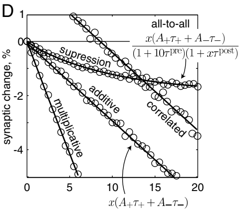
- all weight dependent models exhibit a stable fixed point and generate a unimodal distribution (multiplicative, power law, Gütig, Van Rossum)
- the additive model does not have a stable fixed point and produces a bimodal distribution

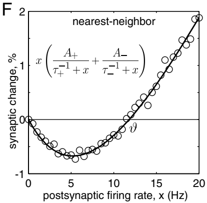
Models of STDP: Spike Pairing Scheme

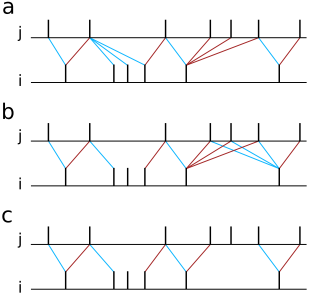
all-to-all...

... or nearest neighbour?

...but which nearest neighbour?



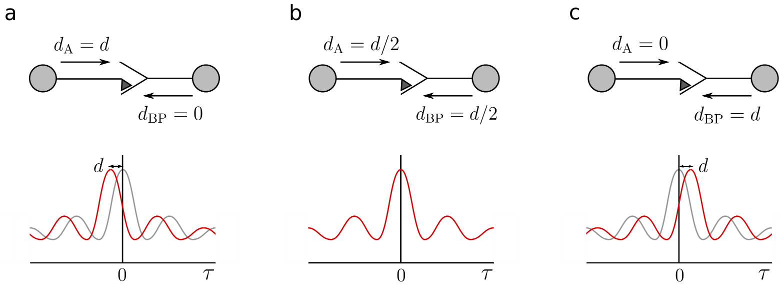




Izhikevich & Desai (2003) *Neural Comput* **15**:1511–1523

- there are many possible spike pairing schemes
- they can give qualitatively different results (e.g. Kempter et al. 2001; Morrison et al. 2007)
- see Burkitt et al (2004) for copious analysis

Models of STDP: Synaptic Delays

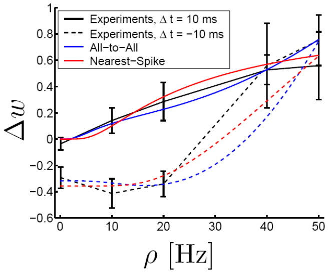


- The synaptic drift depends on the cross-correlation with respect to the synapse
- This is shifted from to the left or right of the (measurable) cross-correlation with respect to the soma depending on whether the synaptic delay is largely axonal (a) or dendritic (c)
- Therefore the same STDP rule coupled with the same network dynamics can give rise to either net depression, no change, or net potentiation

Models of STDP: Beyond Pair Effects

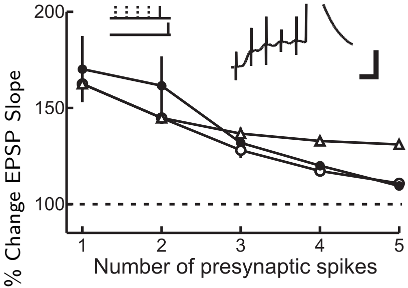
Many features of STDP cannot be explained by pair-based rules:

Frequency dependence



Data: Sjostrom et al. (2001) *Neuron* **32**:1149–1164
Modelling: Pfister et al. (2006) *J Neurosci* **26**:9673–9682
Modelling: Also see Clopath et. al (2010) *Front.Synap Neurosci.*, Shouval et. al (2010) *Front. Comp. Neurosci.*, Costa et al. (2015) *eLife*

Burst dependence



Data and modelling: Froemke et al. (2006) *J Neurophysiol* **95**:1620–1629

There is generally no cross-compatibility between models accounting for these features

Conclusions

- A modeller needs to specify a complete model despite lack of clear experimental evidence
- These choices can have profound consequences
- Almost all network modelling papers fail to consider whether the effects shown are artifacts of their specific choices
- There is so far no phenomenological model which accounts for all the nonlinearites exhibited by STDP
- For those who want to know more...

Biol Cybern (2008) 98:459–478
DOI 10.1007/s00422-008-0233-1

REVIEW

Biological Cybernetics

Phenomenological models of synaptic plasticity based on spike timing

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