Spike-timing-dependent plasticity (STDP) and its relation to differential Hebbian learning
Overview over different methods

**Machine Learning**
- Anticipatory Control of Actions and Prediction of Values
  - REINFORCEMENT LEARNING
    - example based
    - Dynamic Prog. (Bellman Eq.)
    - δ-Rule
    - Eligibility Traces
    - Monte Carlo Control
    - SARSA
    - Q-Learning
    - TD(λ)
      - often λ = 0
    - TD(1)
    - TD(0)
  - δ-Rule
  - Rescorla/Wagner
  - Neur. TD-formalism
    - Neur. TD-Models
      - ("Critic")
    - ISO-Learning
    - ISO-Model of STDP
    - Actor/Critic technical & Basal Gangl.
    - Correlation based Control (non-evaluative)
    - ISO-Control

**Classical Conditioning**
- Anticipatory Control of Actions and Prediction of Values
  - Un-Supervised Learning
  - example based
  - δ-Rule
  - Eligibility Traces
  - Monte Carlo Control
  - SARSA
  - Q-Learning
  - TD(λ)
    - often λ = 0
  - TD(1)
  - TD(0)
  - Hebb-Rule ("slow")
  - Hebb-Rule ("fast")
  - Differential Hebb-Rule ("fast")
  - STD-P-Models biophysical & network
  - ISO-Model of STDP
  - Biophys. of Syn. Plasticity
    - Dopamine
    - Glutamate
  - Neuronal Reward Systems (Basal Ganglia)
  - ISO-Control

**Synaptic Plasticity**
- Correlation of Signals
  - UN-SUPERVISED LEARNING
    - correlation based
    - LTP (LTD = anti)
  - Differential Hebb-Rule ("fast")
  - STD-P-Models biophysical & network
  - ISO-Model of STDP
  - Biophys. of Syn. Plasticity
    - Dopamine
    - Glutamate
  - Neuronal Reward Systems (Basal Ganglia)
  - ISO-Control

**Evaluation over different methods**

- You are here!
Differential Hebb Learning Rule

\[ \frac{d}{dt} \omega_i(t) = \mu u_i(t) V'(t) \]

Simpler Notation

- \( x = \) Input
- \( u = \) Traced Input

Early: “Bell”

Late: “Food”
Defining the Trace

In general there are many ways to do this, but usually one chooses a trace that looks biologically realistic and allows for some analytical calculations, too.

\[ h(t) = \begin{cases} 
  h_k(t) & t \geq 0 \\
  0 & t < 0 
\end{cases} \]

EPSP-like functions:

**\(\alpha\)-function:** \( h_k(t) = te^{-at} \)

**Dampened Sine wave:** \( h_k(t) = \frac{1}{b} \sin(bt) e^{-at} \)

**Double exp.:** \( h_k(t) = \frac{1}{\delta} (e^{-at} - e^{-bt}) \)

This one is most easy to handle analytically and, thus, often used.
Differential Hebbian Learning

\[
\frac{d}{dt} \omega_i(t) = \mu u_i(t) v'(t)
\]

Produces asymmetric weight change curve
(if the filters \( h \) produce unimodal „humps“)

\[
v(t) = \sum \omega_i(t) u_i(t)
\]
Spike-timing-dependent plasticity (STDP): Some vague shape similarity

Synaptic change %

Pre follows Post: Long-term Depression

Pre precedes Post: Long-term Potentiation

Weight-change curve
(Bi&Poo, 2001)
Hebbian learning

When an axon of cell A excites cell B and repeatedly or persistently takes part in firing it, some growth processes or metabolic change takes place in one or both cells so that A’s efficiency ... is increased.

Donald Hebb (1949)
Conventional LTP

Symmetrical Weight-change curve

The temporal order of input and output does not play any role
Plastic Synapse

NMDA/AMPA Postsynaptic: Source of Depolarization

The biophysical equivalent of Hebb’s postulate

Pre-Post Correlation, but why is this needed?
Plasticity is mainly mediated by so called N-methyl-D-Aspartate (NMDA) channels. These channels respond to Glutamate as their transmitter and they are voltage depended:
Biophysical Model: Structure

Hence NMDA-synapses (channels) do require a (hebbian) correlation between pre and post-synaptic activity!

Source of depolarization:

1) Any other drive (AMPA or NMDA)
2) Back-propagating spike
3) Dendritic Spike
Local Events at the Synapse

Current sources “under” the synapse:

- Synaptic current
- Currents from all parts of the dendritic tree
- Influence of a Back-propagating or dendritic spike
Membrane potential:

\[ C \frac{d}{dt} V(t) = \sum_i \left( \omega_i + \Delta \omega_i \right) g_i(t)(E_i - V) + \frac{V_{\text{rest}} - V(t)}{R} + I_{\text{dep}} \]

Weight

Synaptic input

Depolarization source

On „Eligibility Traces“

Pre-syn. Spike

BP- or D-Spike

On „Eligibility Traces“

\[ \omega \]

\[ \sum \]

\[ V^* h \]
Model structure

• Dendritic compartment

• Plastic synapse with NMDA channels
  Source of Ca\(^{2+}\) influx and coincidence detector

• Source of depolarization:
  1. Back-propagating spike
  2. Local dendritic spike
Plasticity Rule
(Differential Hebb)

Instantenous weight change:

\[ \frac{d}{dt} \omega(t) = \mu c_N(t) F'(t) \]

- Presynaptic influence
- Glutamate effect on NMDA channels
- Postsynaptic influence
NMDA synapse - Plastic synapse

\[
\frac{d}{dt} \omega(t) = \mu c_N(t) F'(t)
\]

Pre-synaptic influence

Normalized NMDA conductance:

\[
c_N = \frac{e^{-t/\tau_1} - e^{-t/\tau_2}}{1 + \eta [Mg^{2+}] e^{-\gamma V}}
\]

NMDA channels are instrumental for LTP and LTD induction (Malenka and Nicoll, 1999; Dudek and Bear, 1992)
Depolarizing potentials in the dendritic tree

Dendritic spikes
(Larkum et al., 2001
Golding et al, 2002
Häusser and Mel, 2003)

Back-propagating spikes
(Stuart et al., 1997)
The time course of the $[\text{Ca}^{2+}]$ concentration is important in defining the direction and degree of synaptic modifications. (Yang et al., 1999; Bi, 2002)

Filter $h$ is adjusted to account for steep rise and long tail of the observed Calcium transients induced by back-propagating spikes and dendritic spikes (Markram et al., 1995; Wessel et al., 1999)

Filtered Membrane potential = source of depolarization

Filtered Membrane potential = $V(t) \ast h(t)$

$\frac{d}{dt} \omega(t) = \mu c_N(t) F''(t)$

$\frac{dV}{dt} \sim \sum_i g_i(t)(E_i - V) + I_{dep}$

Source of depolarization
What triggers LTP/LTD: The role of Ca\(^{2+}\)

Differential threshold hypothesis
(Artola and Singer, 1993; Lisman 1989)

LTD: low intrinsic [Ca\(^{2+}\)] threshold
LTP: higher intrinsic [Ca\(^{2+}\)] threshold

Problems:
STDP

post before pre:
Mg$^{2+}$ is already removed
NMDAR opens (a slow process!)
little Ca$^{2+}$ influx

pre before post:
NMDAR is already open
Mg$^{2+}$ is then removed
much Ca$^{2+}$ influx

pre long before post:
NMDAR starts to close
Mg$^{2+}$ is then removed
little Ca$^{2+}$ influx
⇒But no late LTD window found ??
temporal development of Ca\textsuperscript{2+} matters (Ca-gradient)!
STDP

post before pre:
Mg\(^{2+}\) is already removed
NMDAR opens (a slow process!)
SLOW Ca\(^{2+}\) influx

pre before post:
NMDAR is already open
Mg\(^{2+}\) is then removed
FAST Ca\(^{2+}\) influx

pre long before post:
NMDAR starts to close
Mg\(^{2+}\) is then removed
FAST (but little) Ca\(^{2+}\) influx
⇒ no late LTD window found
Some more physiological complications!

Modeling Ca\(^{2+}\) pathways

\[ I_{NMDA} + \text{Back-propagating spike} \]

\[ \text{Ca}^{2+} \text{ concentration and gradient} \]

Calmodulin

\[ \text{Ca}^{2+}/\text{CaM Kinase II} \]

\[ \text{Calcineurin} \]

\[ \text{Phosphorylation} \]

\[ \text{AMPA receptors} \]

\[ \text{Dephosphorylation} \]

Synapse gets stronger=LTP

Synapse gets weaker=LTD
Plasticity Rule
(Differential Hebb)

**Instantaneous weight change:**

\[
\frac{d}{dt} \omega(t) = \mu c_N(t) F'(t)
\]

Presynaptic influence
Glutamate effect on NMDA channels

Postsynaptic influence
NMDA synapse - Plastic synapse

\[
\frac{d}{dt} \omega(t) = \mu c_N(t) F'(t)
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Pre-synaptic influence

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$$F(t) = V(t) * h(t)$$

$F(t)$ is the filtered membrane potential, $V(t)$ is the source of depolarization, and $h(t)$ is the low-pass filter.
Weight Change Curves
Source of Depolarization: Back-Propagating Spikes

Back-propagating spike

Weight change curve

NMDAr activation

Back-propagating spike

$T = t_{Post} - t_{Pre}$

$\Delta t < 0$  $\Delta t > 0$
Weight Change Curves

Source of Depolarization: Dendritic Spike

Dendritic spike

Weight change curve

NMDAr activation

Dendritic spike

$T = t_{Post} - t_{Pre}$
Local Learning Rules

The same learning rule:

Hebbian learning for distal synapses

Correlations beyond ±30 ms are mostly random

Differential Hebbian learning for proximal synapses

Saudargiene et al Neural Comp. 2004
Figure 1. Dendritic Excitability Creates a Switchable, Spatial Gradient of Plasticity in L5 Pyramids

(Left) Short bursts of somatic spikes elicit bAPs that fail to backpropagate fully to distal dendrites. The result, as shown in Sjöström and Häusser (2006), is LTP of synchronously active proximal synapses, but LTD or no plasticity at distal synapses. (Middle) Cooperative activation of additional synapses depolarizes the dendrite and boosts bAP propagation into distal dendrites. This cooperativity serves as a switch to enable distal LTP (Sjöström and Häusser, 2006). (Right) Plasticity also varies with firing mode of these neurons: when bAPs are coupled with strong distal input, bAP-activated calcium spikes (BACs) are evoked in the apical tuft, which enables robust LTP (Kampa et al., 2006).
Biologically inspired Artificial Neural Network algorithm which implements local learning rules: Circuit Diagram Representation

Site-specific learning using the same learning rule
An example Application: developing velocity sensitivity
The diagram illustrates the changes in weight over different group numbers for both STDP and LTP conditions. The left graph shows weight changes under STDP, with line colors indicating different weight values. The right graph shows weight changes under LTP, with similar line colors. The bottom graphs provide a closer look at the weight distribution for each group number, with bars indicating the weight range for each group.
After learning the cell becomes sensitive to stimulus velocity $1/\text{vel.}$
Temporally local Learning
Self-Influencing Plasticity
BP Spike: Before At sameTime After
DS-Spike Only

LTD dominates

LTP dominates

LTD weakly dominates

DS before BP = Causal

BP before DS = Acausal

BP Spike: Before At sameTime After the DS-spike

LTP weakly dominates

Displacement BP vs DS spike

LTP dominates

T zero-crossing

LTD dominates
Local DS-Spike only

Cluster 1

DS- and BP-Spike

Cluster 2

Somatic Firing Threshold passed

weak hebbian learning

pronounced STDP
Why might this make sense??

Single phase learning will lead to weight growth regardless
Calcium protocols

From the viewpoint of the cell these two peaks are almost of equal quality (height and rise phase). Hence chances for LTP and LTD are equal.
Solution?

long-lasting low frequency stimulation

short burst-like high frequency stimulation

Look at the scaling and compare to last slide!