Plasticity

Long term plasticity, the changes in synaptic strength that appear to result from activity in the pre- and post-synaptic neuron, is thought to be the main mechanism that supports learning and development in the brain.

Synaptic plasticity

Synaptic plasticity usually refers to the long-term changes in synapse strength, an long term increase in synaptic strength is called *long term potentiation* of LTP, a decrease is called *long term depression* or LTD. It is believed that synapses respond to their pre- and post-synaptic activity, so that the changes depend on the behavior of the pre- and post-synaptic neurons. It is not known in detail what rules govern this plasticity, it seems different neurons have different plasticity rules.

The closest thing to an overall rule was formulated by Hebb in 1949 when he said [1]:

> Let us assume that the persistence or repetition of a reverberatory activity (or 'trace') tends to induce lasting cellular changes that add to its stability. [...] When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased.

In other words, if one neurons tends to cause another to fire, the synapse from the first to the second will get stronger. In artificial neural networks the nodes, modelling neurons, often lack spiking dynamics and so have a continuous state or rate variable; since *Hebbian plasticity* often plays a role in artificial neural networks it is often applied to a rule that strengthens synapses between neurons that are active at the same time, that is, the explicit causal structure is ignored in favor of

> Neurons that fire together wire together.

This leads to a plasticity rule

$$\delta w_{ij} = \eta x_i x_j \tag{1}$$

where w_{ij} is the strength of the synapse from neuron i to neuron j, x_i and x_j are the states of the two neurons and η is a learning rate. Another version is

$$\delta w_{ij} = \eta(x_i - \theta)(x_j - \theta) \tag{2}$$

where θ is a threshold, this allows negative changes, when $x_i > \theta$ and $x_j < \theta$, or visa versa.

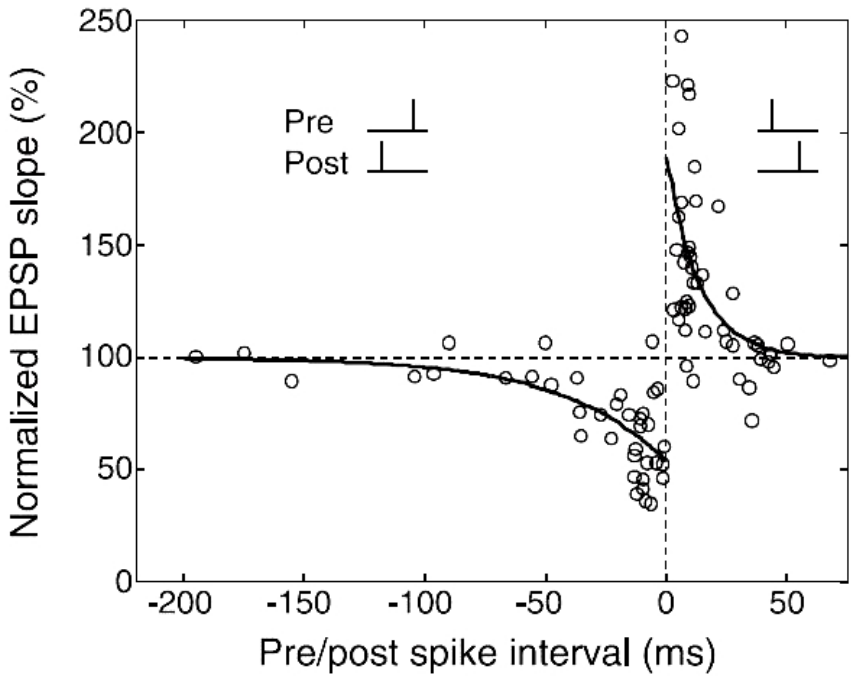

Figure 1: Spike timing dependent plasticity. This shows the change in synapse strength as a function of the timing gap between the pre- and post-synaptic spike.

Spike-timing dependent plasticity

The late nineties saw a revival of interest in causal, spike-timing dependent plasticity (STDP). A series of papers pointed to experimental evidence for timing effects in plasticity [2, 3, 4, 5, 6] including a definitive demonstration of a STDP changes *in vitro* in [3], the observation of asymmetric STDP *in vivo* in developing Xenopus in [7] and a clear graph of the time dependence of plastic changes *in vitro* in [8].

The famous graph of STDP is shown in Fig. 1. This shows measurements of plastic changes made in an *in vitro* preparation. Electrodes are inserted into two synaptically connected cells and currents are used to cause both to spike periodically with a gap of δt between the pre- and post-synaptic spikes. This causes the synaptic strength to change, if the pre-synaptic spike precedes the post-synaptic spike the synapse gets stronger with the degree of strengthening depending on the size of $|\delta t|$, the bigger the gap the smaller the effect with a roughly exponential profile. The opposite is observed if the pre-synaptic spike arrives after the post-synaptic spike has left, in this case the synapse gets weaker, again the size of the effect falls like an exponential as $|\delta t|$ gets bigger.

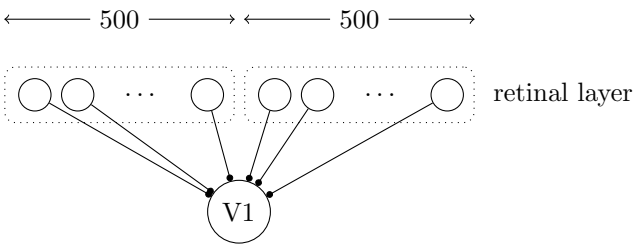

Figure 2: The STDP of Song and Abbott. 1000 input neurons, refered to as retinal neurons, feed forward to a single V1 neuron. The retinal neurons are divided into two groups: the first 500 and the second 500, the two groups provide noisy output, these give the input to the V1 neuron. The inputs from neurons in the same group are correllated, meaning they are more likely to be similar to each other in their activity than neurons from different groups.

There are lots of caveats to be added to this, it is quite an artificial situation, in vitro with periodic spiking; since the changes are only tracked over a show period it isn't clear whether the changes are additive

$$w \to w + \delta w \tag{3}$$

or multiplicative

$$w \to \lambda_w w \tag{4}$$

However, it does give a striking picture of how STDP might work.

In fact, an example of how STDP might support unsupervized learning was given in [9, 10]. A toy model is introduced with multiple neuron inputs feeding forward to a single integrate-and-fire neuron. The inputs are divided into two groups and given a correlation structure so that inputs in the same group are more likely to spike at roughly the same time. This is sketched in Fig. 2. The synapses are then adjusted according to a simple STDP rule. It is seen that one of the two groups 'wins out', its synapses get stronger while the synapses corresponding to the other group gets weaker. Basically if one group, by chance, is slightly more likely to cause the post-synaptic neuron to spike than the other, then the post-synaptic spikes are more likely to occur after the pre-synaptic spikes for that group, so the synapses will get stronger, increasing the effect.

References

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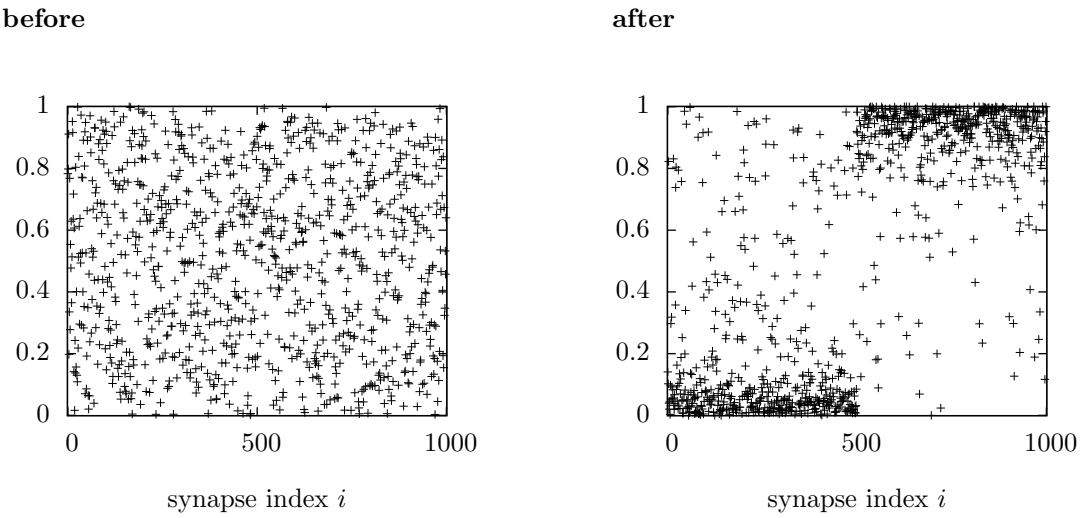

Figure 3: Synapse strengths before and after in Song and Abbotts simple model. At first they are all random, after the STDP has had an effect the synapses from one of the two groups have approached their maximum value, the others are near zero.

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