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Understanding spike-time-dependent plasticity: A biologically motivated computational model

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Abstract

Long-term synaptic plasticity underlies many important learning processes in the brain. Recent physiological data have shown that the precise relative timings of pre- and post-synaptic neuron firings at a synapse determine both the direction of certain types of modification (potentiation or depression), and magnitude of this modification. We propose a neurophysiological mechanism by which this spike-time-dependent plasticity (STDP) could arise, and support this hypothesis using a model involving calcium dynamics. We show that, in addition to reproducing experimental data for paired spikes, the model can explain differences in experimentally observed STDP forms. We also demonstrate that the model provides a good match to recent data for the triplet and quadruplet paradigms, and that a simulated network of reciprocally connected neurons using this learning rule can store and recall a simple temporal sequence. In conclusion we make predictions from the model both on the plasticity effects of quadruple spike interactions and manipulations of concentrations of components involved at the synapse.

Keywords: Spike-time dependent plasticity; Learning; Memory; Sequence simulation; Calcium dynamics

1. Introduction

Long-term synaptic modification has been repeatedly shown to be an important mechanism for neural processing, effective in hippocampal memory acquisition, visual cortical processing and many other areas. The exact details of how this modification depends on the firing patterns of individual neurons are critical to our understanding of these processes.

An early pioneer of the study of memory, Donald Hebb, proposed that episodic memory requires a form of associative learning between stimuli, and that there must be a neural correlate of this associative process [8]. For neurons, this means that when one neuron (A) contributes to the firing of another neuron (B), the synaptic strength between A and B should be increased. This form of plasticity allows networks to act as auto-associators, storing patterns and recalling those patterns from fragments of the original—a form of data-based memory. Previous physiological studies have shown that so called "Hebbian learning" occurs in some biological neural networks. However, most existing studies focus on a time-symmetrical Hebbian modification (so that synaptic strength from A to B increases whenever A and B fire in close temporal proximity, but without dependence on the order of firing), such that neural coding effectively depends on overall firing rates.

1.1. Spike-time-dependent plasticity

Data from hippocampal and visual cortical neurons examining the effect of spike timing on changes in synaptic efficacy show that the precise timing of pre- and post-synaptic spikes is critical to the direction and magnitude of the change in synaptic strength produced by spike association [3,17], leading to the possibility of coding based on exact spike timings.

For the pyramidal cells studied, spike-time-dependent plasticity (STDP) leads to an increase in synaptic strength if the pre-synaptic spike at a synapse precedes the

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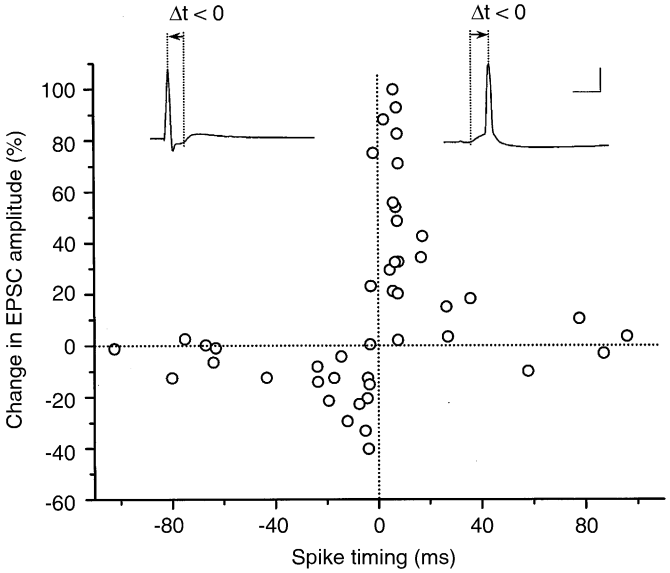

Fig. 1. Change in synaptic strength relative to inter-spike interval (taken from [3]).

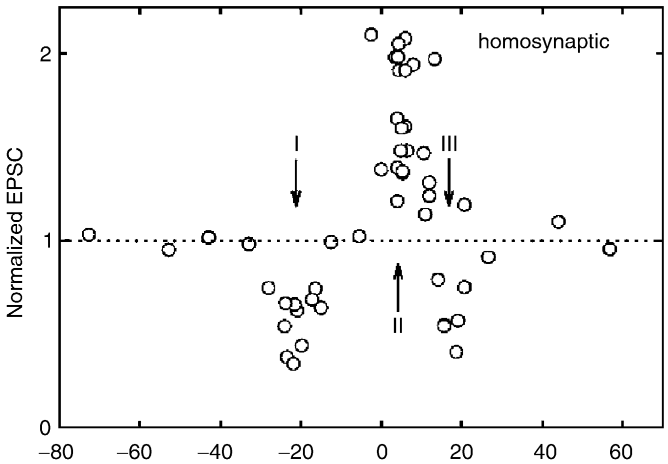

Fig. 2. Change in synaptic strength relative to inter-spike interval (taken from [17]).

post-synaptic neuron's firing, and a decrease in synaptic strength if the reverse is true (possibly with a late third phase of LTD—see below). The time window over which the change from negative to positive strength change occurs is very small (approx 5 ms—see Fig. 1).

1.1.1. *The differing forms of STDP*

The precise form of the dependence of plasticity on inter-spike interval is either not fully determined or varies depending on conditions. The form shown in [3] is different to that shown in [17] (see Fig. 2), but is very similar to that of [5] and a similar (although not identical) experiment on motor cortex [25]. Nishiyama et al. [17] show a significant third phase of LTD, with both early and late LTD (phases I and III to use the terminology in [17]) narrower temporally than the broad LTD in [3]. Froemke and Dan's [5] data (taken from pyramidal neurons from adult rat visual cortical slices) agree with that of [3].

Interestingly, the balance between LTP and LTD, reflected in the areas enclosed by the curves seems to be very even in both forms of STDP—this may be an important mechanism for stabilising overall neural activity.

The significant differences between these forms of causal STDP raise important questions. Are the differences significant, or do they arise simply because of variations in experimental technique? If they are significant, what effect does this have on mechanisms of neural information processing? While it is certainly the case that we can expect cells from different areas at different stages of growth to vary in plasticity behaviours (see [1] for several examples), we believe that it is important to explain these differences, since they are so crucial to our proper understanding of the brain areas concerned.

Additionally, other types of plasticity have similar forms to [17] involving causal plasticity, with an additional later depression phase. If we can explain the underlying principles behind these plasticity forms, we can greatly increase our understanding of plasticity processes in general. Because of this, we believe that our explanation of STDP as the interaction of competing potentiation and depression processes may be able to provide a simple explanation for at least some of the differing observed behaviours.

1.1.2. *Summary*

Because this phenomenon is such an important component of our understanding of the brain's operation at a very low level, it is vital that we understand how it works, and as such, we propose a possible mechanism based on competing calcium-dependent synaptic processes that can explain how STDP arises, and how differing forms might occur. We then develop a model of STDP based on this hypotheses and show that this model accurately reproduces experimental results, and that it allows a model network to store and recall a simple temporal sequence.

1.2. *Existing models of STDP*

An important similar model of STDP is given by Cooper et al. in [21]. This is based on the dynamics of Ca^{2+} inflow into the post-synaptic structure. Any synaptic modification is assumed to be caused solely by voltage-dependent NMDA receptors (NMDARs), changed by the pre-synaptic spike and separately by the back-propagated post-synaptic spike. These changes in Ca^{2+} concentration are then assumed to change the learning rate by means of a non-monotonic learning rate dependence on the Ca^{2+} strength (being negative for small Ca^{2+} and positive for large Ca^{2+}).

However, the resultant learning form is somewhat different from that of [3], having a significant late LTD phase when post-synaptic firing occurs more than 25 ms after a pre-synaptic spike (being therefore more like [17],