# Spike-timing-dependent synaptic plasticity can form "zero lag links" for cortical oscillations. [1]

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**Abstract**

We study the impact of spike-timing-dependent synaptic plasticity (STDP) on coherent gamma activity between distant cortical regions with reciprocal projections. Our simulation network consists of two areas and includes a STDP model reflecting efficacy suppression between pre/postsynaptic spike pairs as found in recent experiments during stimulation with spike trains (Froemke and Dan, 2002). We find that STDP in conjunction with common oscillatory input strengthens synapses with delays around multiples of the oscillation period. As a result, intrinsic excitatory interactions between the areas express in gamma waves with zero lag synchrony (instead of anti-phase synchrony without STDP). We discuss the impact of efficacy suppression on learning convergence and robustness.

*Key words:* axonal delays, synchronization, zero-phase-lag, efficacy suppression

## 1 Introduction

Neurophysiological experiments report that fast oscillatory activity (30-60Hz) can be synchronized in phase over distant cortical areas [3]. Synchronization of cortical rhythms has been ascribed to reciprocal cortico-cortical connections and influential cortex theories rest on the assumption that cortically mediated synchronization is the essential mechanism for dynamic binding and

[1] This work was partially supported by the MirrorBot project of the EU.

integration of cortical representations [2]. However, since the transmission delays between hemispheres can reach many tens of milliseconds ([12], Fig. 1B), nonlocal synchronization effects in cortex have been discussed controversially: Modeling studies have indicated that in-phase synchronization requires synaptic delays smaller than 1/3 or 1/4 of the oscillation period [10,9,6]. For 50Hz oscillations the zero-phase condition (delay $< 20/3$ ms) is obviously not met.

In a recent paper [7] we have investigated the effects of spike-timing-dependent synaptic plasticity (STDP, [8],[11]) in a pair of oscillating neuron pools with reciprocal couplings. With a delay distribution in the reciprocal connections taken from experiments [12], the pools in the model oscillated in anti-phase. But interestingly, STDP turned out to stabilize zero-lag synchronization in the network because it modifies synaptic strength dependent on the transmission delay. Synapses with delays around a multiple of the oscillation period become amplified while other synapses become weakened.

Simple models for STDP [11] were derived from neurophysiological experiments employing *single* pre- and postsynaptic spike pairs to estimate the modification function of synaptic strength (cf. Fig.1C). However, for a neuronal network in oscillatory mode the synapses are exposed to pre- and postsynaptic *spike trains* rather than isolated spike pairs. Recent experiments assessed STDP induced by natural pre- and postsynaptic spike trains [4]. Changes induced by an isolated spike pair turned out to become suppressed by preceeding spikes in the same pair of neurons. Froemke and Dan have derived from their data a more realistic model of STDP where each spike is assigned an efficacy which depends on the interval from the preceding spike. We use this model in a simulation of two reciprocally connected neuron populations and characterize its effects on the synchronization of fast oscillatory activity.

## 2 Methods

In a simulation model we examine the interaction of two reciprocally connected cortical areas (Fig. 1A) each consisting of three neuron populations of size $15 \times 15$ (cf. [7,5]). We use standard integrate&fire neurons [5]. All parameters are the same as in our previous study [7], except the algorithm for STDP.

All connections are topographically organized ($25 \times 25$ kernels). The probability of a synapse between two neurons is $p = 0.5$, and all synapses have initially the same strength. We chose random synaptic delays $s + \gamma d + \sigma N_{0,1}$ with base delay $s$, distance $d$ between the neurons and $\sigma^2$ the variance of a Gaussian $N_{0,1}$ (local connections: $s = 0.8$ms, $\gamma = 0.2$ms and $\sigma = 0$; inter-areal connections: bimodal delay distribution restricted to 2-50ms with $s_{1/2}$=5ms/8ms, $\gamma_{1/2} = 0$, $\sigma_{1/2}$=4ms/40ms to approximate data in [12]; see Fig. 1B). For the ratio be-

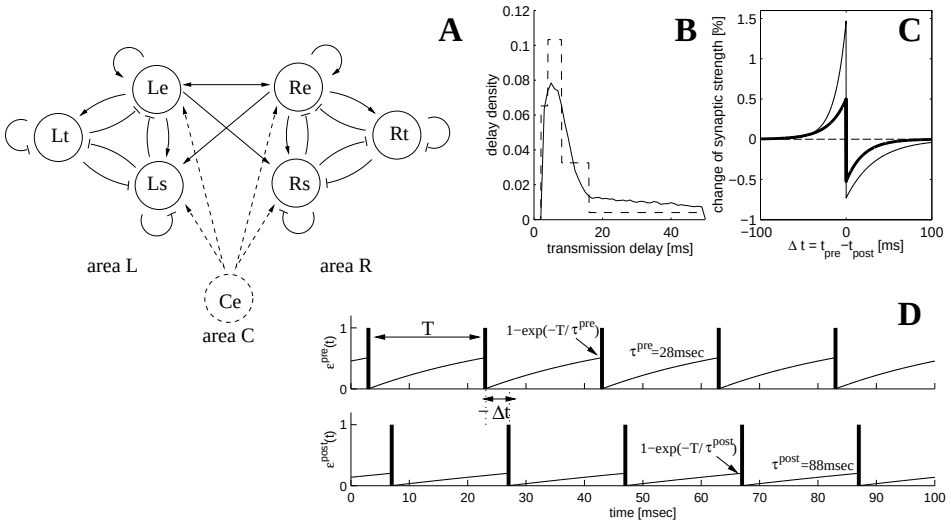


Fig. 1. A: Network model of two reciprocally connected areas, each consisting of three neuron populations (cf. [5], $\rightarrow$ exc., $\dashv$ inh.). A third area C can deliver common input to areas L and R to force in-phase synchronization during STDP. B: Distribution of the inter-areal conduction delays in the model (solid) and in rabbit inter-hemispheric connection of primary visual cortex (dashed) as measured from antidromic latencies (modified from [12]) C: Modification function of STDP with two exponentials, $A_+ \exp(-\Delta t/\tau_+)$ for $\Delta t$<0 and $A_- \exp(-\Delta t/\tau_-)$ for $\Delta t$>0. Parameters as in [11,7] (thin line, $A_+$=0.5%,$\tau_+$=20ms,$A_-$=0.525%,$\tau_-$=20ms) or [4] (thick line, $A_+$=1.47%,$\tau_+$=13.3ms,$A_-$=0.73%,$\tau_-$=34.5ms) D: Suppression of spike efficacy according to [4] during oscillatory activity. A spike at time $t_s < t$ leads to a efficacy recovery $\epsilon^{\mathrm{pre}}(t) = 1 - \exp(-(t-t_s)/\tau^{\mathrm{pre}})$ at the presynaptic site and $\epsilon^{\mathrm{post}}(t) = 1 - \exp(-(t-t_s)/\tau^{\mathrm{post}})$ at the postsynaptic site. Parameters $\tau^{\mathrm{pre}} = 28$ms and $\tau^{\mathrm{post}} = 88$ms as estimated for the *additive* model in [4].

tween maximal local and inter-areal excitation we assumed a value of ten [1]. Long-range connections on inhibitory neurons were modeled almost as strong as the connections on excitatory neurons (balanced regime in [7]).

We implemented STDP in the inter-areal projections including the spike efficacy suppression mechanism proposed in [4]: Synaptic modification caused by the i-th presynaptic and j-th postsynaptic spike is $\Delta w_{ij} = \epsilon_i^{\mathrm{pre}} \cdot \epsilon_j^{\mathrm{post}} \cdot F(\Delta t_{ij})$ where F is the modification function (Fig. 1C), and $\epsilon_i^{\mathrm{pre}}/\epsilon_j^{\mathrm{post}}$ are the pre-/postsynaptic spike efficacies. The spike efficacy of a neuron is zero immediately after a spike and subsequently relaxes exponentially to 1 (Fig. 1D). In [4] two model variants are proposed both explaining the neurophysiological data equally well. In the *additive* model the $\Delta w_{ij}$ of each spike pair are added, whereas in the *multiplicative* model factors $1 + \Delta w_{ij}$ are multiplied. Here we focus on the additive model, however, we expect no major changes for the multiplicative model. To compare the more realistic STDP model with common simple STDP algorithms, we have also simulated STDP without suppression of spike efficacy using the same parameters as in previous studies [11,7].

## 3 Results

We have examined *three* different models. *SMA00*: no spike efficacy suppression, modification function (Fig.1C) of [11]. *SMA00/FD02*: modification function of [11], spike efficacy suppression of [4]. *FD02*: modification function and efficacy suppression of [4]. Before STDP we always observed anti-phase oscillations [7]. Fig.2 shows the temporal evolution of the synaptic strength distributions (vs. conduction delays) during STDP and synchronized (forced by common input from area C, see Fig.1A) oscillatory activity. In all three models the synapses with delays around a multiple of the oscillation period become amplified, while other synapses are weakened so that the final synaptic distributions (after 100s of learning) supported autonomous zero-phase synchronization (i.e., without common input). The learning phases required, however, varied: In the SMA00 model we observed autonomous zero-phase synchronization already after 2-3s [7], while the models with efficacy suppression required 20-50s (SMA00/FD02) and 10-20s (FD02).

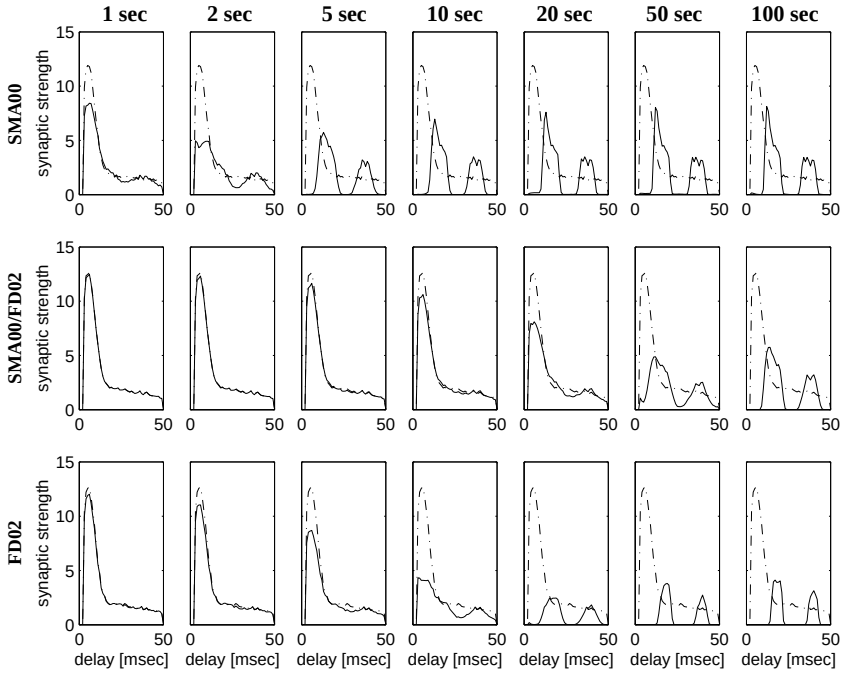


Fig. 2. Impact of STDP on the synaptic strength distribution during oscillations for the 3 models (see text): Each row corresponds to one model and shows the synaptic strength distributions (vs. conduction delay) after 1s, 2s, 5s, 10s, 20s, 50s, 100s of STDP learning. Top row: SMA00 model (no spike efficacy suppression). Middle row: SMA00/FD02 model (with spike efficacy suppression). Bottom row: FD02 model (with spike efficacy suppression). Dash-dotted lines show initial distributions.

In Fig.3 one can compare convergence rates and shape differences of the resulting synaptic delay distributions. The distance measure used is the (non-delayed) cross correlation with a final distribution (after 100s). Fig. 3A shows

the correlations with the final distribution of the corresponding model. One can observe that spike efficacy suppression reduces the convergence rates. Further, in models with spike efficacy suppression different STDP curves for spike pairs (Fig. 1C) can lead to quite different rates of convergence (Interestingly, the FD00 model with smaller amplitudes in the STDP curve converges faster). Fig. 3B displays correlations with the final distribution of model SMA00. One can see that the two models with the same STDP curves for spike pairs (SMA00 and FD02/SMA00) converge to a very similar synaptic distribution just at quite different rates. The FD02 model, however, converges to a synaptic distribution of another shape.

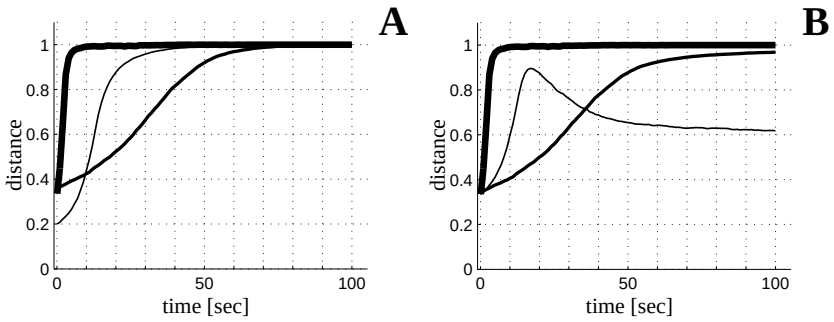


Fig. 3. Convergence of the distribution of synaptic strength (vs. conduction delay) during oscillatory activity and STDP. Each line corresponds to the distance (vs. time) between two synaptic strength distributions measured as the scalar-product (normalized to $[0;1]$) for the corresponding histogram-vectors (cf. Fig.2). Different line stiles correspond to the three models: Thick line: SMA00 model (no spike efficacy suppression). Medium line: SMA00/FD02 model. Thin line: FD02 model. (The latter two models include spike efficacy suppression parameters as estimated in [4].) A: Distances measured relative to the respective distribution after 100s. B: Distances measured relative to the SMA00 distribution after 100s.

## 4 Conclusion

This study addressed the question whether spike-timing-dependent synaptic plasticity (STDP) can account for zero-phase synchronization of fast oscillatory activity found even in distant cortical areas. Transmission delays for inter-areal cortical transmission can be so large that they cause anti-phase synchronization in simulation models, conflicting with the physiological observations. In an earlier paper [7] we have demonstrated that STDP, as found during stimulation with isolated spike pairs [8,11], can establish zero-phase synchrony in networks with realistic transmission delays. In recent experiments Froemke and Dan studied STDP induced by spike trains and found interactions between spike pairs involved: preceeding spikes can suppress the efficacy of spike pairings. For networks in oscillatory mode such effects have clearly to be taken into account and called the result of [7] into question.

The simulation experiments in this paper show that a more realistic model of STDP –including efficacy suppression– can still explain the expression of zero-phase synchrony between distant cortical areas. Oscillatory input in distant neuron populations strengthens synapses with delays close to multiples of the oscillation period, while others are weakened. Thus, time-dependent plasticity form intrinsic excitatory interactions resulting in coherent gamma-band activity, so to say, "zero lag links". Efficacy suppression slows the learning convergence down, but on the other hand it makes zero lag links, once formed, less vulnerable with respect to incoherent network activity at high rates.

The described function of STDP is important in the context of theories proposing synchrony of fast oscillatory activity as a means for temporal binding [2]. In the light of our model distributed gamma oscillations in the cortex indicate excitatory influences –links– between regions. Such long-range influences rely on previous learning and they set in not immediately (since fast synapses are depressed). Links become effective depending on local coherence and are specific with respect to neural populations as well as oscillation rhythms.

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