Synaptic plasticity, conduction delays, and inter-areal phase relations of spike activity in a model of reciprocally connected areas

Andreas Knoblauch [a] Friedrich T. Sommer [a]

[a] *Department of Neural Information Processing*
University of Ulm, Oberer Eselsberg, D-89069 Ulm, Germany
email: (knoblauch,fritz)@neuro.informatik.uni-ulm.de

Abstract

Neurophysiological experiments reveal zero-phase correlations of fast oscillatory (30-60Hz) spike activity even between distant cortical areas. Simple models of reciprocally connected neuron populations can explain zero-phase oscillations only by assuming small axonal delays, i.e. smaller than a quarter of the oscillation period (5ms for 50Hz). Such assumption, however, is incompatible with conduction delay measurements between areas reporting delays in the order of tens of milliseconds.

As a possible process for stabilizing zero-lag oscillations we propose asymmetric spike-timing-dependent synaptic plasticity, as found in experiments (Markram et al. 1997). Our modeling study shows that synchronization weakens fast excitatory feedback, but strengthens slower feedback with delays in the range of one oscillation period. This yields stable zero-lag oscillations, even with realistically large inter-areal conduction delays.

Key words: synaptic plasticity, axonal conduction delays, synchronization, gamma oscillations, zero-phase-lag

1 Introduction

Neurophysiological experiments revealed considerable evidence for long-range synchronization of fast (30-60Hz) oscillatory activity. For example, oscillations at 50Hz were synchronized between the two hemispheres of cat primary visual cortex apparently due to the inter-hemispheric connection via the corpus callosum [3,8]. Simulation studies of reciprocally connected neuron populations have shown that zero-lag oscillations occur only if the mean conduction and synaptic delays are not larger than a third or a quarter of the oscillation period

[10,9]. This implies for the example above (oscillation period 20ms) that the mean delays for the interhemispheric connection of the primary visual cortex of cat must be not larger than about 5-7ms. This contradicts to experimental evidence that a large amount of the delays lies in the tens of milliseconds ([12], Fig. 1C).

In this paper we investigate the role of spike-timing-dependent synaptic plasticity in the emergence of zero-lag gamma-oscillations at distant cortical sites. Recent experiments have shown that synaptic learning takes place in a rather asymmetric fashion ([6], Fig. 1B) : If a postsynaptic spike follows the presynaptic one, the synapse is strengthened, while the synapse is weakend by the inverse order. In an oscillatory regime as in the example above this has the following consequences: If two neurons from the two distant areas fire synchronously and the delays between the two neurons are 5ms then the presynaptic spike follows the postsynaptic spikes for about 5ms. Consequently, this should weaken all the synaptic connections with small delays (e.g. < 10ms) and strengthen only those with long delays (e.g. 10-20ms).

In a simulation study we investigate the dependency of inter-areal phase relations on an asymmetric learning rule and large conduction delays in more detail. We find that the specific strengthening of synapses with long conduction delays in the range of the oscillation period may contribute to zero-phase synchronization of neural activity from distant areas.

2 Methods

We examine the interaction of two reciprocally connected areas (Fig. 1A). Each area consists of three populations of 15×15 neurons (one population excitatory, the other two inhibitory, only one of them receiving extra-areal synaptic input). As neuron model we use simple spiking neurons similar to the PTNR10 model of [5] with similar parameters as in [4].

All connections are topographically organized with kernel sizes 25×25. The probability of a synapse between two neurons is generally $p = 0.5$. Synaptic delays are chosen randomly as $s + \gamma d + \sigma N_{0,1}$ with base delay s, distance d between the neurons and σ^2 the variance of a Gaussian $N_{0,1}$. For local connections we used $s = 0.8$ms, $\gamma = 0.2$ms and $\sigma = 0$. For inter-areal connections we used a bimodal delay distribution ($s_1 = 5$ms, $\gamma_1 = 0$, $\sigma_1 = 4$ms and $s_2 = 8$ms, $\gamma_2 = 0$, $\sigma_2 = 40$ms) restricted to values between 2ms and 50ms to approximate the values found by Swadlow (Fig. 1B).

Standard values for synaptic strengths of the inter-areal connections were a tenth of the strength of local connections [2]. Synaptic learning was imple-

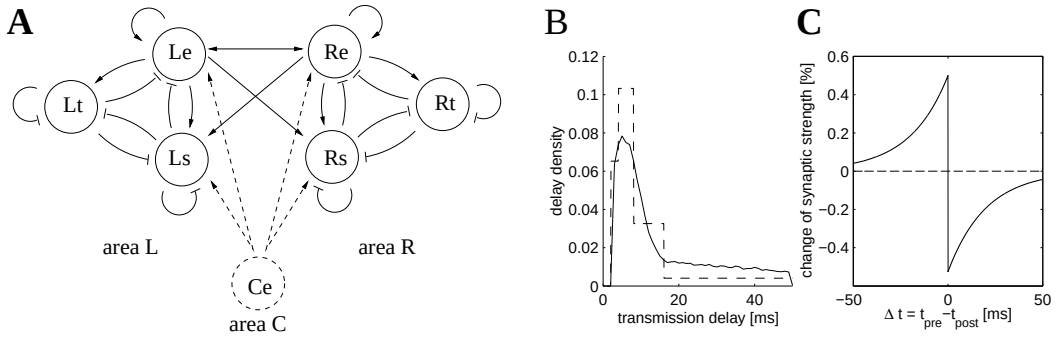

Fig. 1. A: The network model consists of two reciprocally connected areas. Each area consists of three neuron populations. Excitatory populations (Le,Re) have extra-areal projections onto excitatory and inhibitory (Rs,Ls) populations. In some simulations a third area C delivered common input to areas L and R. B: Distribution of the inter-areal transmission 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: The modification function of spike-time-dependent synaptic plasticity (parameters as in [11]).

mented for the inter-areal connection of excitatory neurons using the same parameters as described in [11] (see Fig. 1C).

3 Results

We examined three initial conditions for the strength of inter-areal connections on excitatory and inhibitory neurons in our model: In the 'balanced' regime both connections have strengths corresponding to a tenth of local excitatory connections. In the 'dominant-excitation' regime the connection on inhibitory neurons are weakend to a tenth. And in the 'dominant-inhibition' regime the connections on excitatory neurons are weakend to a tenth.

The first experiment examined the model without any synaptic plasticity. Activity from the two areas oscillates (about 50Hz) with anti-phase for the 'balanced' and 'dominating-excitation' regime (Fig.2 top row) as expected from the large delays ([10,9]). Only in the 'dominating-inhibition' regime the two areas oscillate in phase as in the experiments. However, dominating inhibition seems to be rather unrealistic. Most previous simulation studies even ignored the presence of synapses on inhibitory neurons.

In a second experiment we introduced spike-timing-dependent synaptic plasticity for the inter-areal connection of excitatory neurons (Fig.2 mid row). The results concerning zero-phase vs. anti-phase are the same as before. The plasticity changes synaptic efficacy as a function of transmission delay. In the case of anti-phase oscillations fast synapses are strengthened, while for zero-

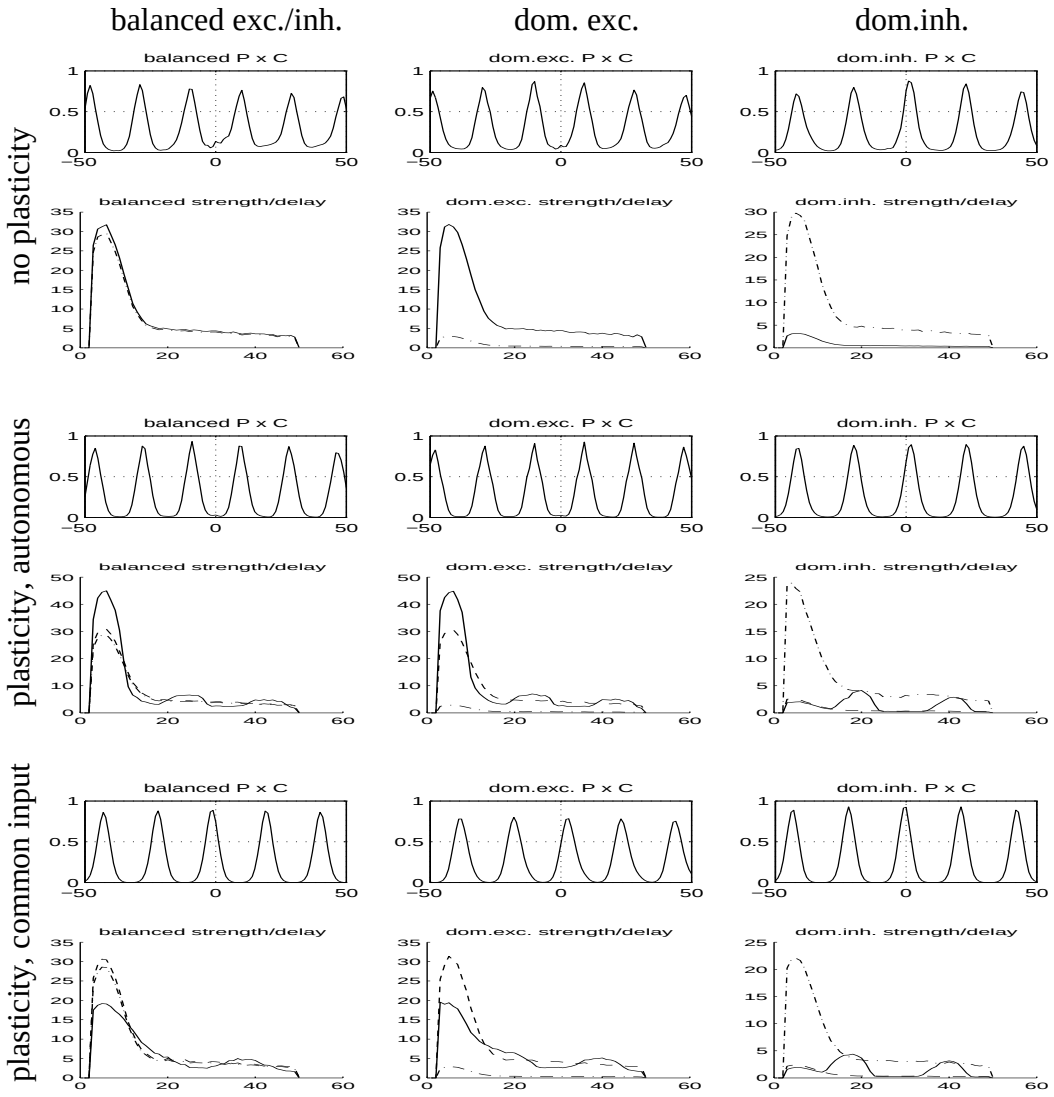

Fig. 2. Simulation results for the three regimes (balanced, dominating excitation, dominating inhibition). Upper row: no synaptic plasticity. Middle Row: Plasticity, but no common input from area C. Bottom row: Plasticity, and common input from area C during the first 48 of simulation. The correlogram is from the last 1s of the 5s simulation. Cross correlograms are computed from spike activity of populations Le and Re. Distribution of synaptic strength over delays are shown for connections of excitatory on inhibitory neurons before (dashed) and after learning (solid), and for connections of excitatroy on inhibitory neurons (dash-dotted).

phase oscillations the fast synapses are weakend, and synapses with delays corresponding to one (and even two) oscillation periods are strengthened.

To examine if zero-phase oscillations can be stabilized by synpatic learning with the more realistic 'balanced' and 'dominating excitation' regimes, we conducted a third experiment where we synchronized the two areas during the first 4 seconds of the simulation using common input from area C (Fig.2 bottom row). Now zero-phase oscillations are displayed even in the 'balanced'

and 'dominant excitation' regime.

4 Conclusion

The standard explanation for experimentally found synchronization of fast activity over long distances is to assume excitatory-to-excitatory connections with sufficiently small conduction delays. This is inconsistent with the finding of mean delays larger than requested theoretically ([12,10,9]).

We suggest that spike-timing-dependent synaptic plasticity [6] in combination with broadly distributed conduction delays [12] may establish zero-phase-synchronization over long distances. The learning rule given in [11] weakens the medium-latency synapses (e.g. $5 - 15$ms) that would produce anti-phase oscillations. On the other hand, synapses corresponding to either very fast axons (e.g. < 5ms) or slower axons with delays in the range of one oscillation period (e.g. $15 - 25$ms) are strengthened.

There are several other possible mechanisms for long-range synchronization (e.g. [1,13]). However, the mechanism suggested here avoids the 'dominant-inhibition' regime which usually is considered unrealistic, and the resulting solution is more energy-efficient compared to other solutions [9,13] because it avoids massive clashing of excitation and inhibition.

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