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

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Abstract

Multi-electrode recordings revealed that fast oscillatory (30–60 Hz) spike activity is often synchronized with zero phase lag, even for recording sites in distant cortical areas. We show in this simulation study that the dominance of zero-phase correlations is inconsistent with the long conduction delays measured between distant areas: For realistic delays, reciprocally connected neuron populations exhibit anti-phase rather than zero-phase correlations. We show that this inconsistency can be removed by taking into account spike-timing-dependent synaptic plasticity (STDP), as found in experiments (Science 275 (1997) 213). We demonstrate that STDP can weaken fast excitatory feedback and strengthen slower feedback with delays in the range of one oscillation period. This yields stable zero-lag oscillations, even for realistic delays.

Keywords: Synaptic plasticity; STDP; Axonal conduction delays; Synchronization; Gamma oscillations; Zero-phase-lag

1. Introduction

Neurophysiological experiments revealed evidence for long-range synchronization of fast (30–60 Hz) oscillatory activity. For example, oscillations at 50 Hz were synchronized between the two hemispheres of cat primary visual cortex apparently due to the inter-hemispheric connection via the corpus callosum [4,8]. Simulation studies of reciprocally connected neuron populations have shown that zero-lag oscillations occur only

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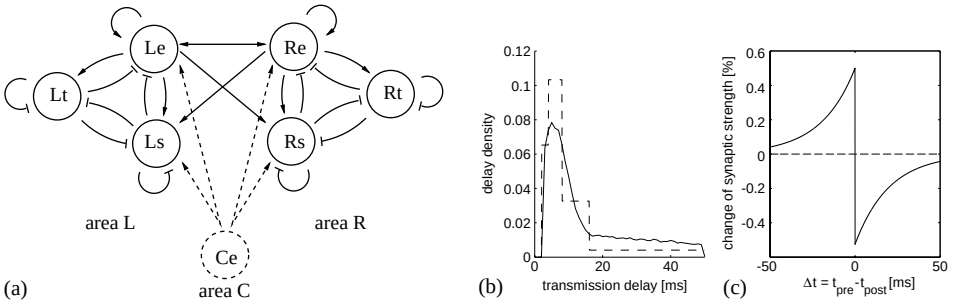

Fig. 1. (a) Network model of two reciprocally connected areas, each consisting of three neuron populations (cf. [5], → exc., ⊣ inh.). In some simulations a third area C delivered common input to areas L and R. (b) Distribution of the inter-areal conduction delays in the model (solid) and in rabbit inter-hemispheric connection of primary visual cortex (dashed) as measured from antidromic latencies (modified from [12]) (c) Modification function of STDP (parameters as in [11]).

if the mean conduction and synaptic delays are not larger than a third or a quarter of the oscillation period [10,9,6]. This implies for the example above (oscillation period 20 ms) that the mean delays for the interhemispheric connection of the primary visual cortex of cat must not exceed values of about 5–7 ms. However, conduction delay measurements have shown that the delay distribution reaches up to delay values of many tens of milliseconds ([12], Fig. 1b).

In this paper we investigate how spike-timing-dependent synaptic plasticity (STDP) can stabilize zero-lag synchrony of gamma-oscillations even at distant cortical sites. Recent experiments have shown that synaptic learning depends asymmetrically on the timing of spike pairs ([7], Fig. 1c): If a postsynaptic spike follows the presynaptic one, the synapse is strengthened, while the synapse is weakened by the inverse order. In an oscillatory regime as in the example above this has the following consequences: If two neurons from distant areas fire synchronously and the delays between the neurons are 5 ms then the postsynaptic spike precedes for about 5 ms the presynaptic spike. Consequently, this should weaken all the synaptic connections with small delays (e.g. < 10 ms) and strengthen only those with long delays (e.g. 10–20 ms).

In a simulation study we assess 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, cf. [5]). The neuron model in this study is a standard spiking neuron similar to [5].

All connections are topographically organized (25×25 kernels). The probability of a synapse between two neurons is $p = 0.5$. We chose random synaptic delays $s + \gamma d + \sigma N_{0,1}$ 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 ($s_{1/2} = 5$ ms/8 ms, $\gamma_{1/2} = 0$, $\sigma_{1/2} = 4$ ms/40 ms) restricted to 2–50 ms to approximate the values found by Swadlow ([12], see Fig. 1b).

For the ratio between maximal local and inter-areal excitation we assumed a value of ten. This assumption agrees with the ratio between local and inter-areal connection densities estimated by neuroanatomical methods [3]. STDP was implemented for the inter-areal connection of excitatory neurons with 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 so-called *balanced* regime both connections have strengths corresponding to about a tenth of local excitatory connections, while in the '*dominant-excitation*' and '*dominant-inhibition*' regimes the connections on inhibitory and excitatory neurons are weakened to a hundredth, respectively.

In the first experiment we examined the model without any synaptic plasticity. Activity from the two areas oscillates (about 50 Hz) with anti-phase for the balanced and dominant-excitation regimes (Fig. 2 top row) as expected from the large delays [10,9]. Only in the dominant-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 repeated the simulations with activated STDP (Fig. 2 mid row). The results concerning in-phase vs. anti-phase are the same as before. The behavior of the neural dynamics is additionally embossed by the synaptic dynamics. The STDP changes synaptic efficacy as a function of transmission delay. In the case of anti-phase oscillations (balanced and dominant-excitation regimes) fast synapses with delays < 10 ms and also slower synapses with delays of about 20–30 ms or 40–50 ms are strengthened while other synapses are weakened. For in-phase oscillations (dominant-inhibition regime) the situation is complementary: the fast synapses (< 10 ms) are weakened, and synapses with delays corresponding to one or two oscillation periods (10–20 ms or 30–40 ms) are strengthened.

To examine whether in-phase oscillations can be stabilized by STDP with the more realistic balanced and dominant-excitation regimes, we conducted a third experiment where we synchronized the two areas during activated STDP using common input from area C (Fig. 2 bottom row). After inactivating common input, in-phase oscillations occur even in the balanced and dominant-excitation regime.

Fig. 3 shows the temporal evolution of the synaptic strength distribution during activated STDP (and common input) for the balanced regime. After about 5 s the

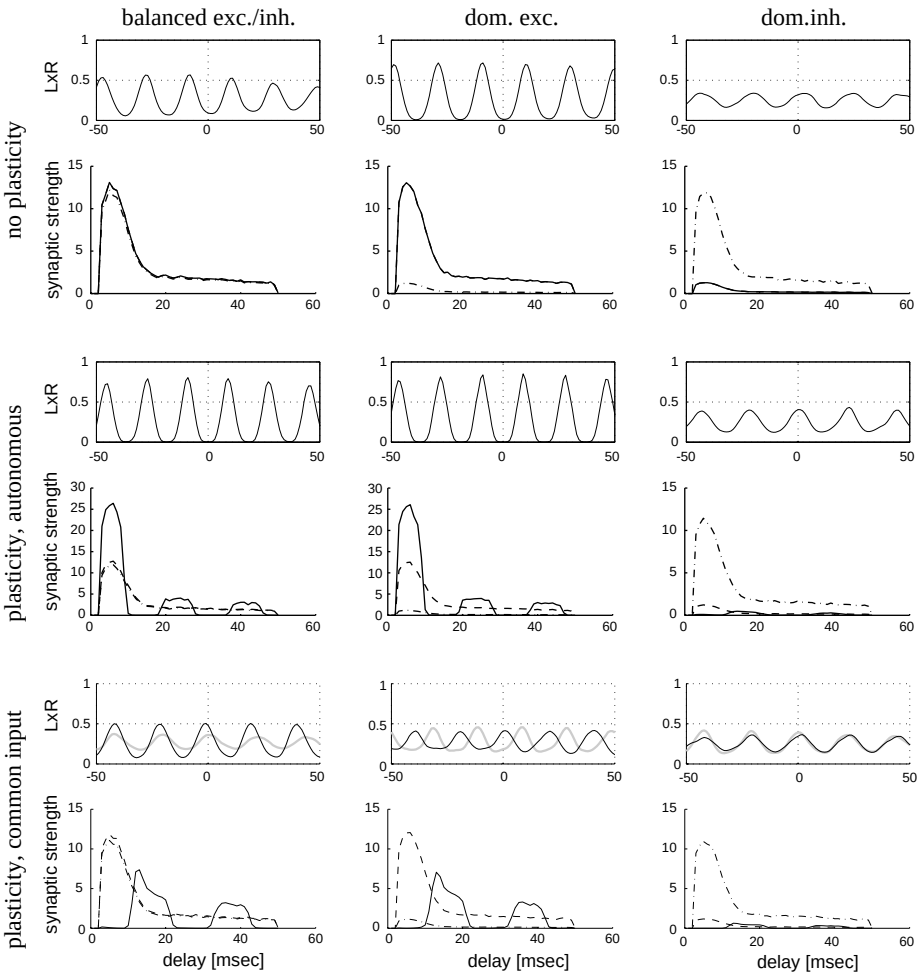

Fig. 2. Simulation results for the three regimes (balanced, dominating excitation, dominating inhibition). Upper row: no synaptic plasticity. Middle Row: Plasticity, but no common input from area C. Bottom row: Plasticity and common input from area C. The correlograms (thin black line) are computed from 5 s recordings (populations Le and Re) after 20 s of activated plasticity (for middle and bottom rows). Thick gray lines (bottom row) correspond to results after only 2 s of activated plasticity (cf. Fig. 3). Distribution of synaptic strength over delays are shown for inter-areal connections between excitatory neurons before (dashed) and after plasticity (solid), and for connections from excitatory to inhibitory neurons (dash-dotted).

distribution approaches its final shape and further changes are minor. For the balanced regime already 2 s of activated STDP are sufficient to obtain in-phase correlations (cf. Fig. 2 bottom row). In the dominant-excitation regime 3–4 s of STDP were necessary (data not shown).

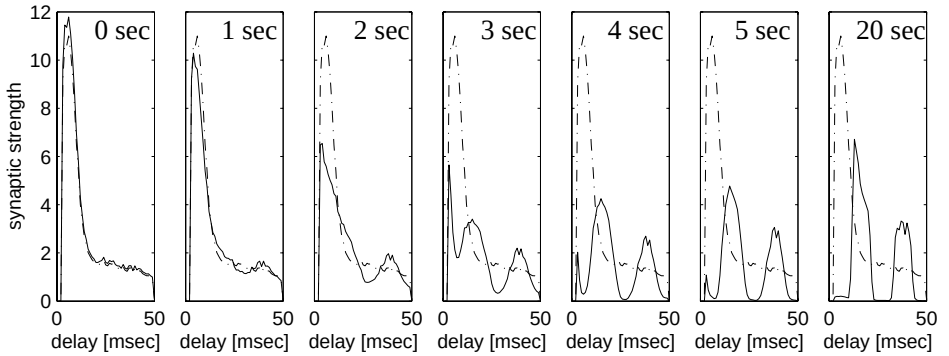

Fig. 3. Impact of STDP on synapses of different delays during oscillations. The plots show the temporal evolution of the distribution of synaptic strength (vs. conduction delay) for a simulation (balanced exc./inh.) with oscillations synchronized with zero-phase lag forced by common input. The histograms show distributions corresponding to inter-areal connections between excitatory neurons (solid), and from excitatory to inhibitory neurons (dash-dotted), and are computed (from left to right) $0, 1, 2, 3, 4, 5$, and 20 s after beginning of STDP.

4. Conclusion

The standard explanation for experimentally found zero-lag synchronization of fast activity over long distances is to assume excitatory-to-excitatory connections with sufficiently small conduction delays [10,9]. However, the simulation experiments in this paper show that the distribution of delays that has been experimentally measured between distant areas [12] supports anti-phase rather than zero-lag synchronization.

Previous simulations showed that also long axonal delays can support inter-areal zero-lag synchronization if the delays are close to multiples of the oscillation period [6]. Here we showed how common input in connection with spike-timing-dependent synaptic plasticity [7] can shape the experimentally determined broad distribution of conduction delays [12] in order to support long-range synchronization. In a network model implementing the learning rule of [11] the medium-latency synapses (e.g. < 10 ms) that would produce anti-phase oscillations are weakened, while synapses corresponding to slower axons with delays in the range of one or even two oscillation periods (e.g. 20 or 40 ms) can be strengthened. This effect is complementary to other proposed mechanisms of long-range synchronization (e.g. [2,13,1]).

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