Gamma band synchronization and communication between delay-coupled neuronal populations

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In neuronal systems, firing synchronization is of great importance in information transmission between brain areas.

It is known that synchronized spikes increase their impact to a given target neuron[1]. Analogously, synchronized electrical oscillations emerging from the activity of thousands of neurons can increase the functional connectivity between neural assemblies by coherently coordinating their firing dynamics.

According to the communication-through-coherence hypothesis of Pascal Fries[3] synchronization between neuronal oscillations of different cortical areas might offer a mechanism for selecting the route of information. In order to communicate effectively the synaptic delay, the phase difference and the frequency of the oscillatory LFPs of the two areas should match. Neurophysiological and modeling results suggest that neuronal populations influence one another most strongly if they oscillate at zero phase lag[4]. However, zero lag synchrony between neuronal ensembles can only be achieved by mechanisms that are able to compensate for the axonal delays involved in the propagation of the spikes. Given that propagation times between different cortical regions, including interhemisferic areas[5] and not only short-range interactions within a cortical area, could be of several tens of milliseconds[6], zero lag synchronization seems implausible. Interestingly, Vicente et al.[7] showed that zero- lag synchronization can be achieved despite long axonal delays when two neuronal populations interact via a third population, which can be associated to the thalamus.

In this work, we have studied the influence of the synaptic delay on the synchronization of neural populations oscillating in the gamma range. To this aim we have modeled two neural populations using conductance-based models for both excitatory and inhibitory cells. We have reproduced particular features of cortical dynamics such as the coexistence of irregular firing at the single-neuron level with collective oscillations at the population level, emerging from the synaptic recurrent connections. The emergent rhythmicity is achieved by a balance between the excitatory and inhibitory synaptic currents[2] and can be explained by periodic changes of the excitability of the network, i.e. periodic modulation of the distance to threshold. We have both the multi-unit activity (MUA) and local field potential (LFP) to characterize the global activity.

A key requirement of the CTC hypothesis is the existence of a constant phase difference between the oscillations that reliably allows a stationary binding between networks, favoring communication. Therefore, we have first quantified phase coherence between the oscillatory activity of the two delayed coupled populations at varying mean axonal delays. In order to assess whether different synchronization scenarios boost communication, we have next used information theory to quantify the response of one population (the receiver) to a varying external input impinging on the other population (the emitter). To this purpose, we have increased the external firing rate of the background synaptic activity affecting a neuronal subpopulation of the emitter network and we have computed the LFP and MUA power spectrum of the receiver network. So we quantify how well the power spectrum of LFP and MUA conveys information about which stimulus is being applied to the receiver population.

In summary, we have performed extensive simulations with a conductance-based neuronal network model to characterize the effect of long conduction delays in the synchronization patterns of two coupled networks. Phase coherence and mutual information has been evaluated in terms of frequency in order to provide a wide analysis of the complex dynamics of neuronal networks, which are far from being harmonic oscillators.

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