γ frequency synchronization in a local cortical network model

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1 Summary

Synchronized gamma frequency oscillation (20 − 70Hz) in cortical networks is considered to play important roles in higher brain function such as cognition. Although the neural mechanism of such synchronous oscillation has not been fully clarified, it appears likely that synchronous oscillation in fast-spiking (FS) interneuron network and fast rhythmic bursts in the gamma frequency range of chattering neuron are promising candidates for generating synchronous gamma frequency oscillation.

In our previous study (Nomura et al.), we theoretically investigated the synchronous spiking phenomena of FS interneuron network. FS interneurons have specific types (Kv3.1/3.2 type) of the delayed potassium channel, which differ from the conventional Hodgkin-Huxley (HH) type potassium channel in several aspects. It was recently revealed that the Kv3.1/3.2 voltage-gated K^+ channels play a crucial role in creating a characteristic feature of FS interneurons, that is, the appearance of sustained high-frequency trains of brief action potentials with little spike frequency adaptation. It was also found that FS interneuron network is one of the major GABAergic inhibitory networks in the neocortex. Nearby pairs of FS interneurons are often interconnected simultaneously by electrical synapses (gap junctions) and chemical (GABAergic) synapses, the latter of which are sometimes bi-directional. Modeling a network of FS interneurons incorporating Kv3.1/3.2 K^+ channel (Erisir

et al.), so as to elucidate the different roles played by gap junctions and GABAergic synapses in the emergence of synchronous firing, we studied how spikes of two weakly coupled FS interneurons are synchronized or desynchronized by each of the two synaptic transmissions. The results are summarized below:

- Two identical electrically-coupled FS interneurons fire synchronously at arbitrary firing frequencies, unlike similarly coupled HH neurons that show frequency-dependent synchronous and anti-synchronous firing states.

- Introducing bi-directional $\mathrm{GABA_A}$ receptor-mediated synaptic connections into an FS neuron pair tends to induce an anti-synchronous firing states.

- An FS neuron pair connected simultaneously by a gap junction and a uni-directional GABAergic synapse achieves near synchronous firing state. In contrast, an FS neuron pair connected simultaneously by a gap junction and a bi-directional GABAergic synapse allow both a synchronous and anti-synchronous firing state at physiologically plausible range of the conductance ratio between electrical and chemical synapses with physiologically acceptable value of the decay time constant of the GABAergic synaptic current (< 5ms).

- A large-scale network of FS interneurons connected by all-to-all gap junctions and GABAergic synapses shows similar bi-stability (synchronous state and 2-cluster state) in the range of gamma frequencies ($20 - 70$Hz). A similar large-scale network except that 70% of GABAergic synapses are uni-directional and the rest are bi-directional shows bi-stability between synchronous and non-phase-locked state.

- An excitatory input can switch the state of the interneuron network between synchronous firing and non-phase-locked firing.

One of the modulatory excitatory input to FS interneurons is an ensemble of pyramidal neurons projecting to the FS interneurons in the local cortical circuitry. A particular class of neocortical pyramidal neurons, i.e., chattering (CH) neurons, shows gamma-frequency rhythmic bursting. Thus, CH neurons may exert a synchronous and powerful excitatory drive on the interneuron network at the gamma frequencies. Recently, Aoyagi et al. have proposed a computational model of chattering neurons. In their model, one of the characteristic features of CH neurons, a hump-like depolarizing afterpotential (DAP), is mediated by $\mathrm{Ca^{2+}}$-dependent cationic current, and fast rhythmic bursts (FRB) of doublet/triplet spikes in the entire range of gamma-band frequency can be generated through an enhancement of a DAP. The firing pattern during FRBs is primarily determined by a $\mathrm{Ca^{2+}}$-dependent cationic current and a small-conductance $\mathrm{Ca^{2+}}$-dependent potassium current. The synchronous firing properties of CH neuron networks are summarized below:

- The coherent level of synchrony in a pair of weakly-coupled chattering neurons can be modulated by intensity of the external input and/or the properties of the calcium-dependent cationic current.
- A change in the bursting mode systematically modulates the coherence of neuronal firing. In a two-neuron network, anti-synchronous firing is abruptly replaced by synchronous firing whenever the neuron pair make a transition to a higher bursting mode, i.e., from doublet firing to triplet firing. Similarly, asynchronous firing is replaced by synchronous firing in a uniformly connected large-scale network of chattering neurons when they enter into a higher bursting mode.

In the above two studies, both network of FS interneurons and that of CH neurons exhibit synchronous spiking in the gamma-band. However, in the local cortical circuitry, pyramidal neurons and interneurons may cooperatively modulate their firing states such as firing frequency and coherence level. In this modeling study, we investigate how regular-spiking (RS) pyramidal neurons and FS interneurons synchronize at the gamma frequency. Particularly, the possible role of CH neurons in the synchronization is studied. We use FS interneuron model and CH neuron model mentioned above, and as for RS neuron, we adopt the same model as CH neuron except that the cationic current is absent from the CH neuron model. At first, we investigate synchronization of a small network model consisting of a RS neuron and an FS interneuron. These studies are conducted analytically by means of the phase reduction method. For a large network model, we calculate coherence level through numerical simulations. Then, we study synchronization of a small and large network consisting of RS neurons, CH neurons and FS interneurons.

2 References

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