

Poster presentation

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A mechanism for achieving zero-lag long-range synchronization of neural activity

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How can two distant neural assemblies synchronize their firings at zero-lag even in the presence of non-negligible delays in the transfer of information between them? Neural synchronization stands today as one of the most promising mechanisms to counterbalance the huge anatomical and functional specialization of the different brain areas. However, and albeit more evidence is being accumulated in favor of its functional role as a binding mechanism of distributed neural responses, the physical and anatomical substrate for such a dynamic and precise synchrony, especially zero-lag even in the presence of non-negligible delays, remains unclear.

Here we propose a simple network motif that naturally accounts for zero-lag synchronization of spiking assemblies of neurons for a wide range of temporal delays. We demonstrate that when two distant neural assemblies do not interact directly but relaying their dynamics via a third mediating single neuron or population and eventually achieve zero-lag coherent firing. Extensive numerical simulations of populations of Hodgkin-Huxley neurons interacting in such a network are analyzed. The results show that even with axonal delays as large as 15 ms the distant neural populations can synchronize their firings at zero-lag in a millisecond precision after the exchange of a few spikes. The role of noise and a distribution of axonal delays in the synchronized dynamics of the neural popu-

lations are also studied confirming the robustness of this sync mechanism. The proposed network module is densely embedded within the complex functional architecture of the brain and especially within the reciprocal thalamocortical interactions where the role of indirect pathways mimicking direct cortico-cortical fibers has been already suggested to facilitate trans-areal cortical communication.

In summary the robust neural synchronization mechanism presented here arises as a consequence of the relay and redistribution of the dynamics performed by a mediating neuronal population. In opposition to previous works, neither inhibitory, gap junctions, nor complex networks need to be invoked to provide a stable mechanism of zero-phase correlated activity of neural populations in the presence of large conduction delays.

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