

Balance between local and global connections enhance spatiotemporal complexity in a cortical network model

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Abstract

The cerebral cortex exhibits a rich repertoire of spontaneous activity patterns. The spatiotemporal patterns of the spontaneous activity depend on the state of the brain, which ranges from highly synchronized (e.g., sleep) to asynchronous states (e.g., awake). During NREM sleep, the brain activity is characterized by the presence of slow oscillations (SO) where neuronal circuits spontaneously switch between periods of neuronal sustained firing (Up states) and periods of silence (Down states) at approximately 1Hz [1]. Conversely, during wakefulness, the cerebral cortex is characterized by asynchronous activity where there is a suppression of the synchronized, low-frequency content. Therefore, an inherent property of the cerebral cortex is to transition between different brain states with different activity patterns. This is also the case in pathological studies, given that brain lesions can give rise to local synchrony, thus giving rise to different concurrent brain state [2]. A possible way to differentiate between these states is to study its spatiotemporal complexity. Several methods are available for estimating cortical complexity, but the perturbational complexity index (PCI) is commonly used in clinical studies [3].

While the change in complexity due to brain states, either physiological or pathological, is dynamic, complexity also relies on network connectivity. Theoretical studies have proposed that different structural connectivity patterns can give rise to different levels of emergent complexity. For example, the connectivity patterns of the cerebral cortex, such as a high density of connections and small world connectivity, are associated with high values of neural complexity [4]. These findings suggest that brain complexity depends on the

network functional structure, determining how neuronal activity propagates along the cortex [5]. By examining the relationship between network structure and emergent patterns, we can get a deeper understanding of brain function as well as develop new treatments for brain disorders. In the current study, we employed a two-dimensional network model of the SO observed in vitro cortical slices to explore the link between network structure and spontaneous and perturbed cortical spatiotemporal activity. The model, consisting of pyramidal cells (excitatory) and interneurons (inhibitory), captures the dynamics of these cells using Hodgkin-Huxley equations. Neurons are randomly distributed in a 50x50 mesh and locally interconnected through biologically plausible synaptic dynamics [6]. By manipulating a parameter (LRCp) that governs the probability of the excitatory cells forming long-rang connections with their postsynaptic contacts, we explored a large range of network structures, from segregated to fully integrated configurations and their resulting impact on the emergent network complexity.

Refefences

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