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Spike timing-dependent plasticity induces non-trivial topology in the brain

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We study the capacity of Hodgkin-Huxley neuron in a network to change temporarily or permanently their connections and behavior, the so called spike timing-dependent plasticity (STDP), as a function of their synchronous behavior. We consider STDP of excitatory and inhibitory synapses driven by Hebbian rules. We show that the final state of networks evolved by a STDP depend on the initial network configuration. Specifically, an initial all-to-all topology evolves to a complex topology. Moreover, external perturbations can induce co-existence of clusters, those whose neurons are synchronous and those whose neurons are desynchronous. This work reveals that STDP based on Hebbian rules leads to a change in the direction of the synapses between high and low frequency neurons, and therefore, Hebbian learning can be explained in terms of preferential attachment between these two diverse communities of neurons, those with low-frequency spiking neurons, and those with higher-frequency spiking neurons.

Keywords: plasticity, synchronization, network*PACS:* 87.10Hk, 87.19.lj, 87.19.lw**1. Introduction**

Neuroplasticity, also known as brain plasticity or brain malleability (Strong et al., 1998; Brenner et al., 2000), refers to the ability of the brain to reorganize neural pathways in response to new information, environment, development, sensory stimulation, or damage (Draganski et al., 2004; James, 1890; Lashley, 1923). The term neuroplasticity was firstly introduced in 1948 by neuroscientist J. Konorski in a work (Konorski, 1948) that showed the associative learning as a result of the adaptation of the brain to external stimuli. In 1949, D. O. Hebb, in his book entitled “The Organization of Behavior” (Hebb, 1949), proposed a plasticity rule, today known as Hebb’s rule.

Scientific advances in neuroimaging and in noninvasive brain stimulation have provided insights to understand better neuroplasticity. Learning-induced structural alterations in gray and white matter have been documented in human brain (Dayan & Cohen, 2011). Draganski and collaborators (Draganski et al., 2004) used whole-brain magnetic-resonance imaging to observe learning-induced neuroplasticity. They verified structural changes in areas

of the brain associated with the processing and storage of complex visual motion. Lu and collaborators (Lu et al., 2003) demonstrated that neuroplasticity is affected by environmental stimuli. In addition, neuroimaging studies have showed alterations of neuroplasticity in depression, namely depressive disorder may be associated with impairment of neuroplasticity (Fuchs et al., 2004).

Aiming at understanding the fundamental mechanisms behind plasticity, Popovych and collaborators studied the effect of noise on synchronous behavior in globally-coupled spiking Hodgkin-Huxley neurons with spike timing-dependent plasticity (STDP) and excitatory synapses (Popovych et al., 2013; Borges et al., 2016). STDP networks have nodes that adapt their synaptic strength according to some rule based on their spike timings (Gilson et al., 2010; Markram et al., 2011; Markram et al., 2012). Abarbanel and Talathi (Abarbanel & Talathi, 2006) studied a neural circuit responsible for recognizing interspike interval sequences by means of STDP of inhibitory synapses. Similar results, though using different kinds of neural models, have been reported earlier by Kalitzin and collaborators (Kalitzin et al., 2000), where it was shown that coherent input can enhance synapses inducing high connectivity, whereas mutually anti-correlated inputs to individual neurons wea-

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