Msc Thesis - Dynamics of adaptive neuronal networks.
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Postulates on modeling network plasticity

This document gathers the postulates on which modeling the interactions in a network of neurons will rely, under the influence of different types of plasticity. It condenses information from an initial reading list featured at the end of this document.

1. We distinguish between two types of dynamics: we study the dynamics of pulse-coupled networks *on* networks, and the dynamics *of* such networks is how they evolve over time.

2. The dynamics of the network occur on a different (slower) time-scale than the dynamics on the network. Both types of dynamics influence each other [1].

3. Neurons communicate through *synapses* with electrical and chemical signals, in the form of action potentials and neurotransmitters respectively. We will speak of the presynaptic neuron as the neuron that sends a signal and of the postsynaptic neuron as the neuron that receives a signal. When the membrane voltage of a presynaptic neuron reaches an internal threshold, the neuron spikes (or fires) and an electrical signal travels down the neuron axons [2]. At the synapse, the electrical signal is converted into a chemical signal in the form of a neurotransmitter release of the presynaptic neuron, upon which the postsynaptic neuron receives the neurotransmitters and constructs its own electrical signal [3]. Most neurons in the central nervous system use either the excitatory neurotransmitter glutamate (AMPA or NMDA) or the inhibitory neurotransmitter GABA [4, 5].

   As signals are converted from electrical to chemical and back, we will model the communication process as entirely electrical, consisting solely of action potentials. These will be modeled by a pulse-shaped curve, modulated by a dynamic variable of the neuron (for example the phase angle for the Theta neuron). The magnitude of the response of the postsynaptic neuron can be modeled by a coupling strength. The inhibitory/excitatory behavior can be modeled by allowing a negative/positive coupling strength between neurons [6–9].

4. The human brain is seen as a graph, with neurons as graph nodes, where the pre- to postsynaptic relation models a directional edge [10]. These edges are usually unidirectional though it can happen that the post- reconnects to the presynaptic neuron. We will model this by allowing the adjacency matrix of the network to be asymmetric. Axon-axon and dendrite-dendrite connections exist but will be ignored. [11]

5. Another electrical communication process occurs when current flows between neurons proportional to the difference in their membrane voltages. We will avoid modeling the two communication modes at the same time, due to tractability [7].

6. The process that allows neurons to adjust the strength of their synapses is called *synaptic plasticity*. Over time more (or less) neurotransmitters can be released (potentiation/depression). We will model this behavior by allowing the coupling strength to change over time, according to a rule. As we are interested in the long term effects on synchronization of the network, short-term potentiation and depression will not be considered [4].

7. One specific rule that determines the evolution of coupling strength over time is *spike-timing-dependent plasticity* (STDP), where the relative timing of action potentials from the pre- and postsynaptic neuron determine causality. This is a temporal interpretation of Hebbian learning [12, 13].

If the postsynaptic neuron fires right after the presynaptic neuron, then we will strengthen the coupling strength from post- to presynaptic neuron, and vice versa. The magnitude of change is modulated by an asymmetric biphasic learning window around pulses originating from the postsynaptic neuron. Asymmetric because the peak is not situated at 0 and the integral over the window is generally positive, biphasic because this allows both to strengthen and weaken coupling strengths [13]. Recently, triphasic learning windows have been used to account for when it takes too long for the postsynaptic neuron to fire, and thus to decorrelate the relation between neurons. These learning windows are curves that were fitted to experimental data of the cortex and the hippocampus [14].

This approach simplifies modeling the neuronal back-propagation, where another pulse is generated as an echo of the action potential which travels through the neuron dendrites (so, backwards). This behaviors is believed to adjust the presynaptic weights, though it is a controversial subject [13].

8. As the brain grows and learns we can observe neuronal plasticity, where new connections between neurons are made, or whole regions of the brain are remapped. This is (structural) plasticity on the level of the network topology, and can be modeled by allowing the adjacency matrix to change over time. However, it might be more practical to incorporate both synaptic and structural plasticity in a single coupling matrix, where connections between neurons are either a negative or positive number, and zero when two neurons are not connected. This is necessary to maintain the notion of the *degree* in a network - how many connections a neuron actually has. We can model this by either modifying current algorithms for STDP to have a stable equilibrium for a synaptic coupling strength of 0, or we can filter out the desired behaviour by saying that the registered coupling strength is thresholded to be zero close to zero. No sources have been found that incorporate this behaviour.

9. In recent years, criticism on STDP has been growing, as experimental data has shown that STDP is usually accompanied by homeostatic plasticity of the neuron excitability and the synaptic strengths. Processes like *intrinsic plasticity*, where one neuron's excitability changes over time as to self-regulate sensitivity to incoming action potentials, or *synaptic scaling*, where synapse characteristics are adjusted in unison to counteract positive feedback loops, have proven to stabilize the firing rate [14, 15]. We can model intrinsic plasticity by adjusting the neuron's excitability as the inverse of the firing rate: he more spikes that a neuron will receive, the less affected it is [16]. An observed phenomenon is that the excitability evolves together with the coupling strength, but that at the extremes this relation reverses [17, 18]. These types of plasticities should be relatively easy to implement but have no impact on the network topology.

10. Much time and effort of the work on computational neuroscience has been dedicated to computing with neurons: pattern recognition, image segmentation and other machine learning techniques have successfully developed [14, 16, 19]. We will concentrate our efforts on the effects of network topology and synchronization and will leave the computational applications aside.

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