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Master thesis : Implementation of heterosynaptic plasticity in biological neuron models and application in the context of allodynia

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Implementation of heterosynaptic plasticity in biological neuron models and application in the context of allodynia

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Abstract

Synaptic plasticity, defined as the modification of synaptic strength, plays a very important role in many mechanisms such as memory or learning. There are several types of synaptic plasticity, the best known and most studied being homosynaptic plasticity where a synapse strength is modified by its own activity. However, this type of plasticity cannot explain all phenomena and other types of plasticity are needed. Heterosynaptic plasticity is defined as changes in the synaptic strength induced by the activity of adjacent synapses. Until now, only scarce data, either experimental or computational, have been generated to study heterosynaptic plasticity. Yet, this type of plasticity is necessary, especially to study pain-related phenomena.

Pain may be triggered by various causes. Moreover, following an injury, one can notice an increase in sensitivity to touch on and around the wound. Thus, a caress, no matter how gentle, will cause a sensation of pain. This phenomenon is known as allodynia. Studies have shown that central sensitization, an increase in the excitation of synapses in the spinal cord, has a role to play in the induction of allodynia. Both homosynaptic and heterosynaptic plasticities are involved.

The aim of this thesis is to establish a new model of heterosynaptic plasticity with the subsidiary goal of modeling allodynia. To do so, we started with two homosynaptic plasticity models (calcium-based and pair-based spike-timing dependent plasticity (STDP)) to which we added a heterosynaptic dimension by modeling two presynaptic neurons and one postsynaptic neuron. In the calcium-based model, this has been done through the integration of a new parameter α, intervening when presynaptic neurons spike, which could represent the distance between two presynaptic neurons or the amount of calcium diffusing (or being released) through the postsynaptic neuron. In the pair-based model, this has been done through the integration of two new parameters α, governing the potentiation, and A_{het} governing the depression of synaptic weights. Both parameters intervene when the presynaptic neurons spike. We studied the dependency between α and A_{het} which shows that when they are independent of each other, a phenomenon of pruning, the mechanism by which some neuronal connections are eliminated after some time, can be inferred with the right set of parameters. Our new calcium-based heterosynaptic models were evaluated in the same experimental conditions previously reported by Chistiakova et al. and we were able to reproduce a Mexican hat pattern where the induction of homosynaptic long-term potentiation (LTP) provokes weaker LTP at the closest neighbor synapses, long-term depression (LTD) at further neighbor synapses and no modification at the furthest neighbor synapses. Finally, we were able to customize our new models to reproduce a mechanism of heterosynaptic central sensitization causing allodynia. However, our models have shown a certain fragility that may be related to suboptimal physiological modeling.

In conclusion, our new models introduce for the first time two new parameters namely α and A_{het} which, in our view, could contribute to better model heterosynaptic plasticity. However, further work will be needed to flesh out our models in the future.