Presentations (20 m talks)

Slow and fast pulses in neuronal cultures. A numerical approach to 1D excitatory systems.

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We study the wave-like propagation of activity in one dimensional neuronal networks, where the signal can travel only along one, well defined, path. The experimental implementation of this neuronal network consists of a long and thin strip whose surface is chemically prepared so that dissociated neurons dissected from prenatal or neonatal rats can attach, grow and connect with each other. The synaptic input basin for the attached neurons is on the order of the width of the line making this second dimension collapse, obtaining an almost ideal 1D model experimental system to study signal transmission.

Experimental results in 1-D cultures of hippocampal neurons from rats have shown the spontaneous generation of a slow, low amplitude pulse that precedes a high amplitude, fast pulse that propagates through all the system. Notably, this transition appears both with and without the presence of functioning inhibitory synapses. The focus of this talk is an unexplained feature of these experiments in 1D lines, namely, the transition from a slow and low amplitude pulse that lasts around a hundred ms and advances with a velocity around 55 mm/s to the high amplitude, faster pulse that propagates throughout the system at speeds on the order of 50 mm/s. We show via simulations that a simple integrate and fire model leads to a fast (under 10 ms) recruitment time scale, which would not be visible in the experiments. Therefore, the question arises regarding the mechanism which makes the slow pulse persistent even when inhibitory synapses are blocked. We propose to explain the slow pulses by increasing the complexity of the IF neuron model in a purely excitatory network with connectivity as close to the experiments as possible. First, we consider the network structure and connectivity as a basic element of the problem: therefore we will try to mimic as much as possible the neuronal network as it is given by the experimental evidence. Second, we start with the most simple model that does not work, and proceed to increase the complexity of the neuronal model to find the key ingredient that explains the synchronization and the slow-fast pulse transition.

This approach allows us to show that spike frequency adaptation is a fundamental ingredient for the initiation process of the pulse. The introduction of a slow variable produces spike frequency adaptation increases strongly the persistence of the transient activity before the emergence of the fast pulse up to temporal and spatial scales comparable with the experiments.

Finally, we demonstrate that proper levels of additive white noisy currents generate such pulses spontaneously. In other words, we fully reproduce the experimental dynamics when we use the appropriate basic neuron model, the correct experimental connectivity and introduce the presence of white noise in the soma dynamics.