Effects of short-term synaptic plasticity on the dynamics of a conductance-based neuronal network model
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The spontaneous activity of cortical circuits is organized in UP/DOWN states consisting in an alternation of firing periods (UP states) and silent periods (DOWN states), succeeding during synchronized brain states. Although the functional implications of these states have been extensively studied both experimentally and computationally [4, 5, 6], it is still not clear which mechanisms underlie the high- and slow-frequencies of these oscillations and how the dynamics switches from one frequency to the other.

Goal of this work is to analyse the role of different stages of short-term synaptic plasticity (both facilitation and depression) not only on the dynamics of a network, but also on the dynamics of the excitatory and inhibitory conductances.

For this purpose, we used a bio-inspired network model made up of a population of 320 multicompartment neurons (around 80% excitatory and 20% inhibitory) containing different membrane channels modelled according to the Hodgkin-Huxley formalism [1]. We have added both facilitation and depression mechanisms to the network according to the formulation described in [2].

Depending on the level of depression, the dynamics of the network displays two different regimes, either (i) UP and DOWN states or (ii) tonic activity [3]. By plotting the instantaneous firing rate (IFR) of excitatory versus inhibitory neurons, our results show that, without depression, the model seems to present a relaxation-oscillation limit cycle; the presence of a saddle at the origin (vanishing IFRs) causes the DOWN state, which is maintained for a couple of seconds by the spontaneous dynamics. As depression increases the limit cycle passes approaches a saddle-focus point corresponding to low IFRs; this phenomenon induces longer UP states. Increasing further the depression a bifurcation is observed consisting of the saddle-focus point becoming stable; at the network level, it means that both populations reach a constant firing rate, mostly due to the tonic activity and asynchronous activity of the cells.

The critical value of the depression plays an important role in the changes of the conductances. As the depression level decreases we observe that the excitatory and inhibitory conductances exponentially increase (the goodness of fit presents an $R^2$ coefficient greater than 0.8) for all levels of depression that are not close to the critical value. These two different behaviors could be explained by observing that for low values of facilitation the AMPA conductances are almost zero.

On the other hand, the network shows a different dynamics under the presence of short-term facilitation. In this case, we observed that the transition time of the saddle point at the origin is increasing when decreasing the level of facilitation. In these cases, the network presents UP and DOWN states. However, at the some
critical value of the facilitation level, the saddle point becomes stable causing a permanent DOWN state.

This is a joint work with Paolo Massobrio, Antoni Guillamon.

**REFERENCES**


