Impact of intrinsic neuronal properties in cortical network-dynamics

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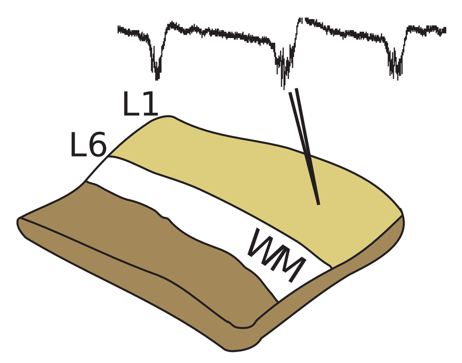
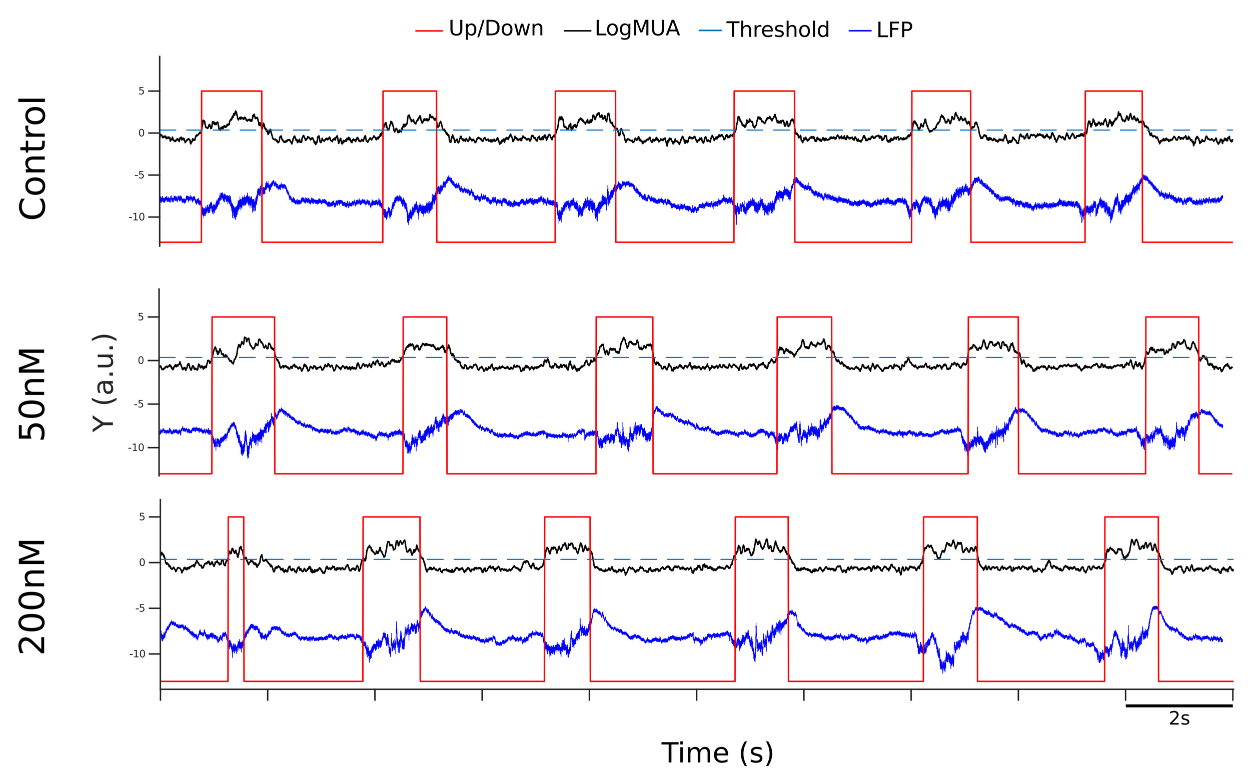
1 INTRODUCTION

During slow wave activity, neuronal circuits can spontaneously switch between periods of sustained firing (Up states) and quiescence (Down states) [1]. The mechanisms controlling this phenomenon has been explored, but the focus has remained largely on synaptic properties and network connectivity. Our objective is to investigate up to what extent the network is sensitive to ionic currents. With this aim we (i) analyzed *in vitro* extracellular multiunit recordings from cortical slices under physiological conditions following bath application of apamin, a K_{Ca} channel blocker and (ii) used a computational data-based model to validate the experimental findings and further explore the effects of K_{Na} ion channels.

2 METHODS

- Local field potentials were obtained from *in vitro* cortical slices with 2-4 MΩ tungsten electrodes;
- Bath application of K_{Ca} blocker (Apamin: 50 and 200nM);

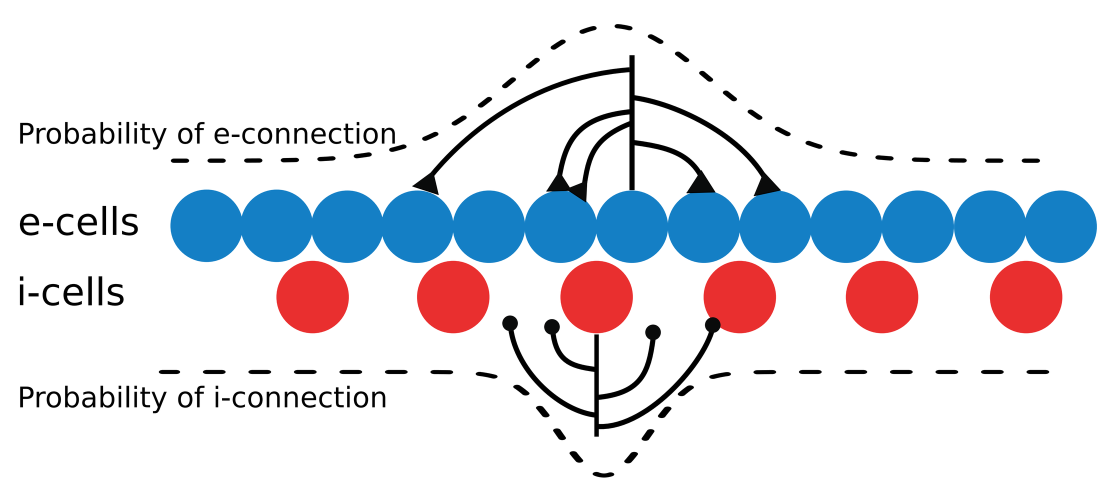




- Multiunit activity (MUA) was estimated as the power change in the Fourier components at high frequencies (>200 Hz) of the recorded signal. The normalized MUA spectrum can be interpreted as the population firing rate, as shown by theoretical studies [3].

- State transition times (Up-to-Down, Down-to-Up) were detected applying a threhsold on the MUA signal.

A data-based model for Slow-Oscillations

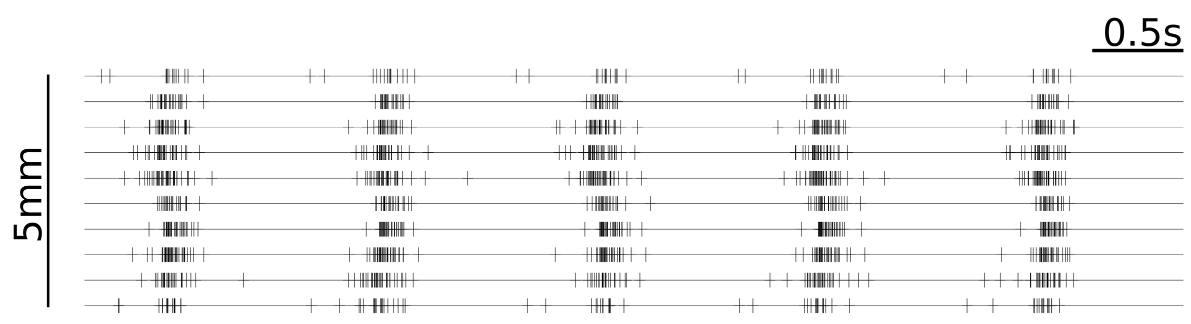


two-compartment model

soma $$C_m A_s \frac{dV_s}{dt} = -A_s(I_L + I_{Na} + I_K + I_A + I_{KS} + I_{KNa}) - I_{syn,s} - g_{sd}(V_s - V_d)$$

dend. $$C_m A_d \frac{dV_d}{dt} = -A_d(I_{Ca} + I_{KCa} + I_{NaP} + I_{AR}) - I_{syn,d} - g_{sd}(V_d - V_s)$$

The model [2] is able to reproduce the slow oscillatory activity observed *in vitro*.



References:

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2. Compte A., et al., *J. Neurophys.*, 89(5), 2003.
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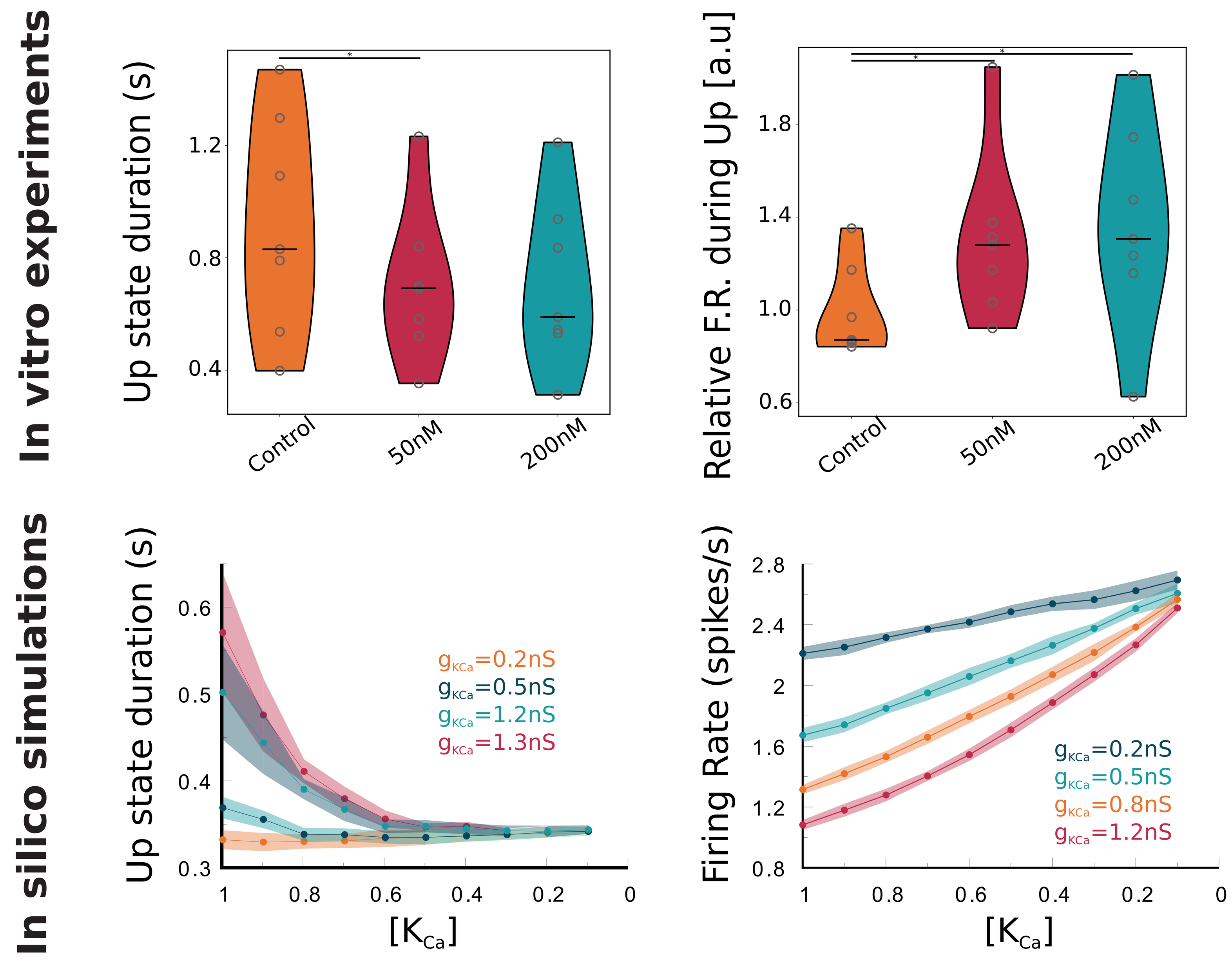
3 RESULTS

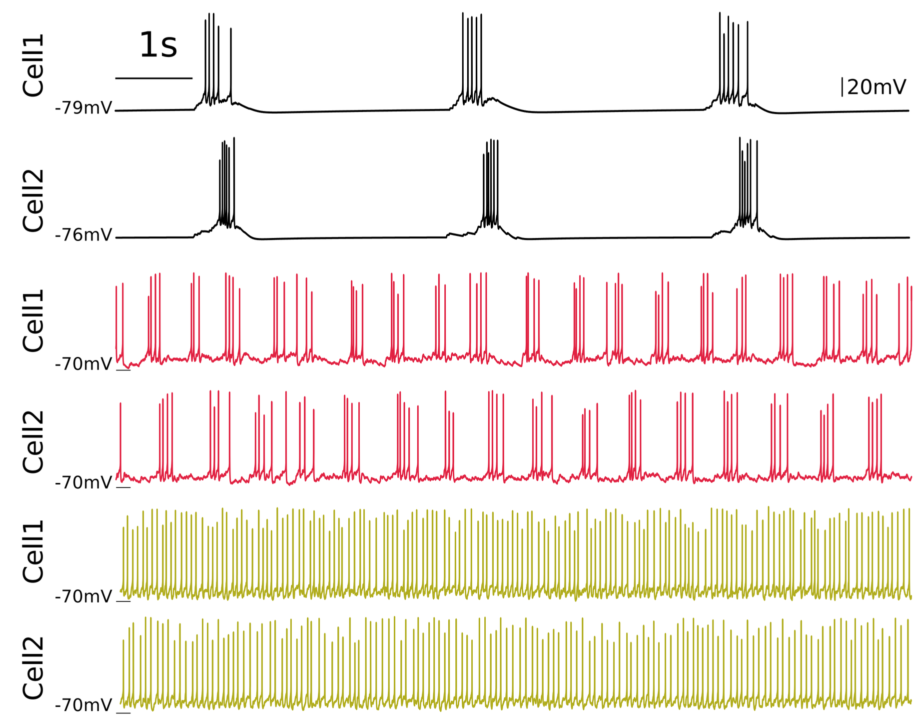
Ca^{2+}-dependent K^+-current

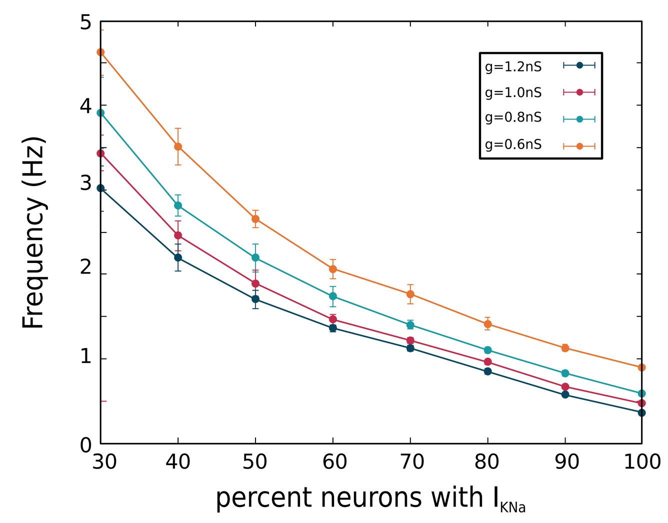
In vitro, application of the K_{Ca} channel blocker decreases the duration and increases the firing rate during Up states. This phenomenon is also observed *in silico* when we parametrically reduce the percentage of neurons containing K_{Ca} channel.

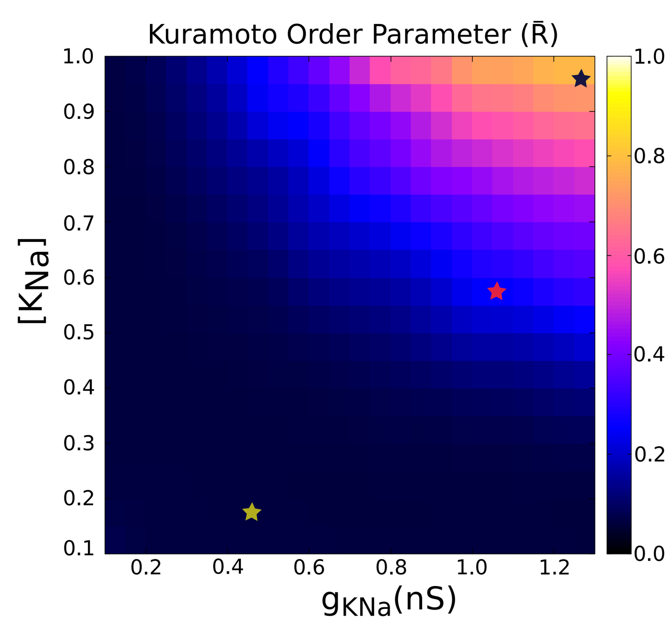


Na^+-dependent K^+ -current

We find that *in silico* K_{Na} current modulates the network's dynamical state. The reduction of such channel brings network from synchronous to asynchronous regime, showed here by the Kuramoto Order Parameter.







4 CONCLUSIONS

1. The modulation of apamine-sensitive K_{Ca} channel blocker is able to regulate Up states, shortening its duration and increasing its firing rate, therefore, increasing the synchronization of the network. Although it keeps the network under Slow Oscillation regime.

2. Simulations *in silico* reproduce the K_{Ca} blockade effects observed *in vitro* and show a dependence of the activity on the conductance strength.

3. The slow oscillatory regime is modulated through the presence of K_{Na} ionic current. Therefore the activation of a single ionic channel, K_{Na} channel, is enough to switch the cortical state.

4. From the extracellular and intracellular spontaneous activity, it is not possible to distinguish neurons without K_{Ca} or K_{Na} currents from the remaining ones. Therefore, the properties of the ionic channels become a network property.

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