Short-Term Plasticity modulates UP and DOWN cortical dynamics

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Abstract:

Neuronal dynamics are profoundly influenced by short-term plasticity (STP), which modulates synaptic efficacy on timescales ranging from milliseconds to seconds. Depending on the brain region, STP can be dominated by short-term depression (STD), short-term facilitation (STF), or a combination of both. These mechanisms shape electrophysiological activity patterns, particularly the alternation between UP and DOWN states. The UP-DOWN pattern is characterized by periods of tonic firing (UP states) followed by periods of silence (DOWN states), occurring sequentially during synchronized brain activity. Different levels of plasticity could alter these UP-DOWN dynamics, causing the network to stop exhibiting this neuronal behavior.

In this study, we investigate the impact of short-term plasticity on simulated cortical activity patterns. To achieve this, we adapt a bio-inspired network model from [1] designed to simulate the visual cortex, incorporating both STD and STF. The model consists of a population of 320 multi-compartment neurons, with 80% excitatory and 20% inhibitory neurons, and includes various membrane channels modeled using the Hodgkin–Huxley formalism. The neurons are spatially organized to replicate the connectivity observed in the visual cortex.

Our findings primarily show that STD and STF jointly govern transitions between distinct activity regimes: from alternating UP and DOWN states (with low STD and high STF) to asynchronous firing (with high STD and low STF). We identify the critical depression and facilitation levels that define these regimes and demonstrate how they influence the network's mean synaptic conductance growth rate. This relationship provides crucial insights into the balance of excitation and inhibition underlying different brain activity patterns. Finally, by analyzing the trajectories of excitatory and inhibitory firing rates near these critical boundaries, we reveal dynamic signatures that suggest the types of bifurcations that a rate model for this complex network should capture.

Bibliography

[1] Compte A, Sánchez-Vives MV, McCormick DA, Wang X-J. Cellular and network mechanisms of slow oscillatory activity. J Neurophysiol 2003;89(5):2707–25

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