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Partial response to supra-threshold excitation desynchronizes spiking neurons

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Desynchronization of neuronal dynamics is highly relevant, occurring for example in pathological synchronized neuronal activity involved in Parkinson tremor or in epileptic seizures [1-3]. Several mechanisms for desynchronization have been proposed and are based on heterogeneity, noise, or delayed feedback [1-6]. Here we reveal a different desynchronization mechanism based on the neuron's *partial response* to supra-threshold inputs [7-9]. In medical applications, desynchronization may be controlled by appropriate low-level drugs that change a neuron's intrinsic response properties.

Typically, a spike generated at the soma affects the dendritic part only indirectly due to intra-neuronal interactions. Thus excitatory input charges may partly remain on the dendrite and contribute to the membrane potential integration after the reset at the soma [10-12]. Several multi-compartment models have been proposed to characterize this effect. For instance, in a two-compartment model [13] of coupled dendrite and soma, the membrane potential at the soma is reset after spike emission, while the dendritic dynamics is affected only by the resistive coupling to the soma.

Here we propose an idealized neural network model that captures the partial response to residual input charges after spike emission by a partial reset. We analytically study the effect of the partial reset onto the collective network dynamics and uncover a *desynchronization mechanism* that causes a sequential desynchronization transition: In globally coupled neurons an increase in the strength of the partial response induces a sequence of bifurcations from states with large clusters of synchronously firing neurons, through states with smaller clusters to completely asynchronous spiking. The mechanism is robust against structural perturbations in the network and neuron properties.

We link our simple model to biophysically more detailed ones by comparing *spike time response curves* (STRCs). STRCs encode the shortening of the inter-spike intervals (ISI) following an excitatory input at different phases of the neural oscillation. An excitatory stimulus that causes the neuron to spike will maximally shorten the ISI in which the stimulus is applied. Additionally the following ISI is typically affected as well. This effect can be characterized by an appropriately chosen partial reset in our simple system.

We find a similar desynchronization transition in networks of two-compartment conductance based neurons when varying the resistive coupling between soma and dendrite. By linking the two-compartment neuron model via STRCs to our simple model we observe a change in the partial reset strength and identify it as the underlying desynchronization mechanism.

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