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Modeling ripple oscillations in the hippocampus

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Sharp wave-ripples (SWRs) are highly synchronous network events displayed by the mammalian hippocampus during slow-wave sleep and immobile resting periods. A SWR event (~100 ms duration) is characterized by fast network oscillations (~200 Hz "ripples") superimposed by a sharp wave, which is a large-amplitude, low-frequency (<10 Hz) signature in the local field potential of the hippocampal CA1 region. Such events are involved in memory consolidation. Understanding the mechanisms that give rise to SWRs can help us to gain insights on the computations being implemented during memory consolidation.

Despite a large amount of electrophysiological data on SWRs *in vivo* (e.g. [1]) and *in vitro* (e.g. [2-4]) and many computational models (e.g. [5] and [6]), the basic mechanisms behind SWR generation remain elusive. Regarding the origin of the high-frequency ripple component during SWRs, two (not mutually exclusive) generative mechanisms have been proposed: First, a rhythmic output of a network of principal cells coupled by gap junctions, presumably between axons of pyramidal cells [5]. Second, an interneuron network coupled by chemical synapses that modulates the firing of pyramidal cells [6]. Modeling studies showed that both mechanisms can give rise to a prominent ripple component in the 200 Hz range. These two mechanisms could not only coexist but also be differentially expressed during SWR generation.

Here we explore the oscillatory behavior of an *in-silico* model of a CA1 interneuron network to mimic *in-vitro* recordings from CA1 slices in which excitatory synaptic transmission is blocked and SWRs are induced by application of brief potassium puffs, which transiently excite neurons [3]. In such a preparation, the two proposed ripple generating mechanisms are decoupled and can be studied in isolation. Focusing on a model of a random interneuron network, we found that the power of ripple oscillations is stable for a wide range of the strength of the recurrent inhibitory coupling. A reduction of inhibitory conductance leads to a slight increase in network frequency, which is in line with [4]. Furthermore, larger reductions of inhibitory coupling leads to an abrupt decay of ripple power (see also [3]). Thus, some of the phenomenology of SWRs *in vitro* can be described in terms of chemical synaptic inhibition.

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