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Presentation Abstract

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Title:	Dynamical properties of gamma-frequency cell assemblies in the hippocampus probed with optical neural control and computational modeling
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Abstract: Neurons in many brain circuits fire in assemblies at a gamma frequency (30-80 Hz) rhythm, presumably coordinated by several classes of interneurons. However, how these cells work together to create and preserve cell assemblies is unclear. Here we use an in vitro model of the gamma oscillation, in which mouse hippocampal slices (400 um) are bathed in 400 nM kainate to drive oscillations at 25-40 Hz, a model that recapitulates many of the properties of gamma oscillations in vivo. Using mice expressing channelrhodopsin-2 (ChR2) under the Thy1 promoter as well as mice in which viruses carrying the channelrhodopsin-2 variant ChIEF are injected into hippocampal area CA3, we studied the effect of driving sets of pyramidal cells with different powers of light (0.5-40 mW/mm2) using field recording. We found that brief (10 ms) moderate-intensity light pulses resulted in abrupt phase resets of the gamma oscillator. Stronger pulses resulted in stereotyped silent periods (150 + 50 ms long) after light cessation, reminiscent of those we previously found in awake monkey and mouse cortex when optically driving excitatory cells. With longer (100 ms) light pulses, novel phenomena appeared during light exposure: weak light quieted gamma, whereas strong light pushed the network into high-frequency gamma (40-70Hz).
In order to understand the properties of gamma frequency cell assemblies in hippocampus, we created a computational model that includes pyramidal cells,

parvalbumin (PV)+ basket cells, and a second class of inhibitory interneurons. The second class of interneurons receive weak input from pyramidal cells, and release GABA asynchronously; cholecystokinin (CCK)+ basket cells have both properties. We reproduced the responses to light activation of pyramidal cells observed in vitro. A brief light pulse generates a spike volley in pyramidal cells; the PV+ basket cells respond promptly, resetting the gamma oscillation. A stronger brief input pulse activates the second class of interneurons, producing the ~100 ms silent period observed in slice. A longer input pulse to a small subpopulation of pyramidal cells can desynchronize the network, leading to the experimentally-observed decrease in gamma power. A strong longer input pulse generates fast oscillations in the model and also leads to activation of the second class of interneurons, generating the 100 ms silencing following pulse offset.
Through such efforts we aim to understand how multiple interneuron classes contribute to the creation and modulation of cell assemblies, and to derive principles of how to control neural circuits to yield generalizable strategies for remedying pathological neural dynamics.

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