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Extrinsic control and intrinsic computation in the hippocampal CA1 network

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A key issue in understanding circuit operations is the extent to which neuronal spiking reflects local computation or responses to upstream inputs. Several studies have lesioned or silenced inputs to area CA1 of the hippocampus - either area CA3 or the entorhinal cortex and examined the effect on CA1 pyramidal cells. However, the types of the reported physiological impairments vary widely, primarily because simultaneous manipulations of these redundant inputs have never been performed. In this study, I combined optogenetic silencing of unilateral and bilateral mEC, of the local CA1 region, and performed bilateral pharmacogenetic silencing of CA3. I combined this with high spatial resolution extracellular recordings along the CA1-dentate axis. Silencing the medial entorhinal largely abolished extracellular theta and gamma currents in CA1, without affecting firing rates. In contrast, CA3 and local CA1 silencing strongly decreased firing of CA1 neurons without affecting theta currents. Each perturbation reconfigured the CA1 spatial map. Yet, the ability of the CA1 circuit to support place field activity persisted, maintaining the same fraction of spatially tuned place fields. In contrast to these results, unilateral mEC manipulations that were ineffective in impacting place cells during awake behavior were found to alter sharp-wave ripple sequences activated during sleep. Thus, intrinsic excitatory-inhibitory circuits within CA1 can generate neuronal assemblies in the absence of external inputs, although external synaptic inputs are critical to reconfigure (remap) neuronal assemblies in a brain-state dependent manner.

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