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Poster Presentation

Probing mechanisms of gamma rhythmogenesis with cell type-specific optical neural control

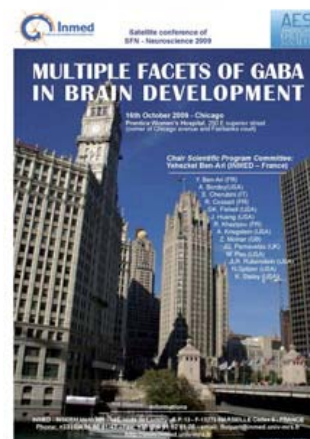
[Edward Boyden](#), [Giovanni T Franzesi](#), [Xiaofeng Qian](#), [Mingjie Li](#), [Xue Han](#), [Christoph Borgers](#), [Nancy J Kopell](#), [Fiona Le Beau](#) and [Miles A Whittington](#)

Rhythms within the gamma frequency range, 30-80Hz, are involved in the brain's strategy for neural coding and information processing. They are generated by networks of neurons in mammalian cortex but the precise nature of the neurons that constitute these networks is far from certain. Many electrophysiological and computational studies have implicated the interplay between inhibitory interneurons and excitatory principal cells as critical for gamma rhythmogenesis. However, lack of neuron subtype-specific tools to probe network function has hampered further investigation. For example, it is still unclear precisely which of the many subtypes of cortical interneuron are critical for generating and modulating gamma rhythms; nor is it clear just how such network activity interacts with principal cells to temporally modulate network outputs. This information is vital if we are to understand the processes underlying disruption of gamma rhythms associated with neurological illnesses such as schizophrenia and many neurodegenerative conditions.

Here we demonstrate neuron subtype-specific targeting of the light-activated cation channel channelrhodopsin-2 (ChR2) and the light-activated chloride pump halorhodopsin (Halo/NpHR) to either excitatory pyramidal neurons or parvalbumin (PV)-positive interneurons in the mouse hippocampus and neocortex. We use these techniques in the context of in vitro pharmacological models of persistent gamma rhythms, showing that bi-directional modulation of the activity levels of these different cell types results in predictable and repeatable changes in amplitude of the gamma oscillation.

These preliminary data demonstrate that each neuronal subtype makes a critical contribution to the emergent network dynamics, and suggest that, far from being a simple 'clock-like' rhythm, the gamma oscillation is an exquisitely labile network phenomenon whose manifestation is controlled by the pattern of excitation of both principal neurons and local circuit interneurons. The specificity of these manipulations allows the acquisition of data ideally suited to computational modeling of network dynamics. These methods can be used, not only for the gamma rhythm, but for the mechanistic exploration of other EEG rhythms that can be produced in vitro and in vivo. Thus, they provide new ways to probe the pathophysiology of neurological illnesses in which disruption of brain dynamics are a core feature.

Conference: Computational and systems neuroscience. Salt Lake City, UT, USA, February 26 - March 03, 2009.



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Citation: Boyden E, Franzesi GT, Qian X, Li M, Han X, Borgers C, Kopell NJ, Le Beau F and Whittington MA (2009). Probing mechanisms of gamma rhythmogenesis with cell type-specific optical neural control. *Frontiers in Systems Neuroscience. Conference Abstract: Computational and systems neuroscience*. doi: 10.3389/conf.neuro.06.2009.03.299

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