



## Presentation Abstract

Program#/Poster#: 765.09/D38

Presentation Title: On the size of cell assemblies and the loss of gamma rhythms

Location: Hall A-C

Presentation time: Wednesday, Nov 16, 2011, 8:00 AM - 9:00 AM

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**Abstract:** Often oscillations in the brain are the result of synaptic interactions between excitatory and inhibitory (E- and I-) cell populations. We examine the boundary of the parameter regime in which gamma frequency (30-80 Hz) rhythms arise in this manner, focusing on the breakup of the rhythms as the number of neurons involved becomes too small, or the synaptic connections become too weak. Our modeling indicates that given synaptic strengths, and given heterogeneities in external drives and synaptic connectivity, there is a minimal possible size of an E/I cell ensemble oscillating at gamma frequency.

Even though the breakdown of the rhythm is linked to network heterogeneity, the point at which the breakdown occurs can be predicted with good accuracy by analyzing highly idealized, homogeneous networks. When the number of E-cells participating in the rhythm is so small that, in a homogeneous network, 1:1 locking of E- and I-cells breaks down, then in a heterogeneous network, the effects of heterogeneity become great, leading to the breakdown of the rhythm. More subtly, when the number of I-cells participating in the rhythm is so small that in a homogeneous network, the gamma rhythm takes several periods to be established, then in a heterogeneous network, the rhythm is not established at all. A tangential, but interesting consequence of the analysis is that gamma oscillations in E/I networks are either established rapidly, within one or two population cycles, or not at all.

In our computational simulations, stimulation of too small a sub-ensemble of E-cells embedded in a larger network fails to establish a gamma oscillation in the stimulated sub-ensemble, but often creates enough asynchronous I-cell activity to disrupt an

ongoing noise-driven weak background gamma oscillation. This offers an explanation of our experimental observation that weak optogenetic stimulation of CA3 pyramidal cells in mouse hippocampal slices can not only fail to create oscillatory activity, but also abolish an ongoing kainite-induced background gamma oscillation.

In a variation on our basic model, we introduce spatial dependence of synaptic connectivity, and find that stimuli with too small a spatial extent fail to elicit gamma oscillations, in agreement with experimental findings of Gieselmann and Thiele (*Eur. J. Neurosci.* 2008). Our model predicts that in these experiments, as in our experiments with optically stimulated hippocampal slices, stimuli that fail to elicit gamma oscillations still generate substantial asynchronous activity in fast-spiking inhibitory interneurons.

Disclosures: **C. Borgers:** None. **G. Talei Franzesi:** None. **E. Boyden:** None. **N.J. Kopell:** None.

Keyword(s): OSCILLATIONS

MODELING

OPTOGENETIC

Support: NIH 1R01 NS067199

NIH 1R01 DA029639, 1RC1 MH088182, DP2OD002002

NSF DMS 0717670

Google

Paul Allen Family Foundation

[Authors]. [Abstract Title]. Program No. XXX.XX. 2011 Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2011. Online.

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