

Data-driven modeling of gamma activity in mouse visual cortex

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The human cortex contains $\sim 10^{10}$ interconnected neurons and underlies all cognitive processes linking sensory perception to behavior. Electrical recordings of cortical activity exhibit power in frequency bands such as delta (0.5-4Hz), alpha (8-12Hz), beta (15-30Hz), or gamma (30-80Hz). Gamma band activity, the fastest of these patterns, is a hallmark of active cortical processing and is impacted in major psychiatric disorders such as schizophrenia and autism. However, despite years of active research, the mechanism and function of gamma band activity are still unclear. The present project aims to develop a comprehensive model of gamma activity in mouse visual cortex based on recent experimental work [1].

The cortical circuit comprises areas specialized in sensory, associative or motor processing. Each area is contacted by the thalamus, a region relaying sensory input or output from distant cortical and motor regions. Thalamic input is integrated through six layers of excitatory neurons regulated in turn by inhibitory interneurons. Earlier models have popularized the idea

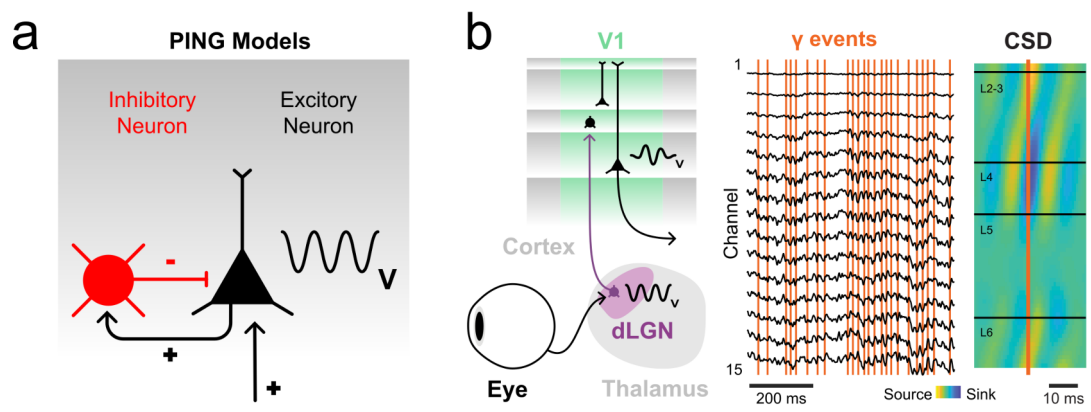


Fig. 1: a) Pyramidal Interneuron Network Gamma (PING) model [2]. Reciprocally connected excitatory pyramidal neurons and inhibitory neurons generate gamma activity when submitted to an excitatory drive. b) Observed gamma activity in the visual cortex (V1). *Left:* input from the retina is relayed by the visual thalamus (dLGN) to V1. The thalamus shows sustained rhythmic gamma activity while gamma activity is transient in the cortex. *Middle:* Electric field through V1 layers. Propagated gamma events are highlighted in orange. *Right:* Current Source Density (CSD) analysis around gamma events. Sinks (i.e. neural activation: blue) propagate through layers 4, 2-3 and 5 following the path of thalamocortical integration.

that gamma activity is generated locally by interactions between connected excitatory and inhibitory neurons submitted to a common excitatory drive (Fig. 1a). This notion was strongly challenged by recent experimental data [1] showing that gamma in the visual cortex is linked to brief events of cascading synchrony between cortical layers triggered by the thalamus (Fig. 1b). Interestingly, while thalamic input maintains a steady rhythmicity in the gamma range, gamma events in the cortex occur flexibly according to context (Fig. 1b). This suggests that gamma activity in the visual cortex is driven by rhythmic thalamic input and that unknown mechanisms help facilitate or attenuate the propagation of this rhythm through cortical layers dynamically. Possible actors of this modulation could be neuromodulators such as noradrenaline or acetylcholine but also background synaptic activity received by cortical neurons which might modulate the conductance of cortical neurons.

The goal of the present project is to explore these possibilities using computational models. While relatively simple rate models may suffice to capture the interplay between thalamic inputs and locally coupled excitatory and inhibitory populations [3], recurrent networks of spiking (e.g. leaky integrate and fire) neurons may be necessary to capture spike-gamma event synchrony. Model predictions will be cross-checked using publicly available datasets of mouse V1 activity and intracellular recordings of V1 excitatory and inhibitory neurons.

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