Visual alpha generators in a full-density spiking thalamocortical model

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Introduction

One of the most prominent features observed in waking electroencephalograms of a variety of mammals, mainly at eyes-closed rest, is the alpha rhythm (~10 Hz). Although alpha is strongly associated with reduced visual attention, it is also related to other functions such as the regulation of the timing and temporal resolution of perception, and transmission facilitation of predictions to visual cortex [1].

Motivation and goal

Understanding how and where this rhythm is generated can elucidate its functions. In this regard, two potential alpha generators were studied:

1) Pyramidal cortical neurons of layer 5 (L5) producing rhythmic bursts (IB, intrinsically bursting) close to 10 Hz after stimulation by a short current pulse [2].

2) A thalamocortical loop delay around 100 ms previously proposed in mean-field models [3] was evaluated for different combinations of thalamocortical and corticothalamic delays.

Methods

The cortical network is composed of ~200k neurons and the thalamic network of ~1.2k neurons simulated using the AdEx model. Neurons are connected by current-based synapses with instantaneous rise and exponential decay. Network connections are constructed based on the macaque connectivity map given in [4]. All simulations were performed using the neural network simulator NEST.

Neurons (AdEx model)

$$C_m \frac{dV}{dt} = -g_L(V - E_L) + g_L \Delta_T \exp\left(\frac{V - V_T}{\Delta_T}\right) - w + I_{syn} + I_{ext}$$

$$\tau_w \frac{dw}{dt} = a(V - E_L) - w \qquad \text{If } V > V_{cut}\text{, then } \begin{cases} V = V_{reset} \\ w = w + b \end{cases}$$

Thalamocortical network

The thalamic network was composed of 902 thalamocortical (TC) and 301 inhibitory neurons (IN). Cortical neurons in L4 and L6 received thalamocortical connections. In turn, L6 neurons send feedback to thalamus.

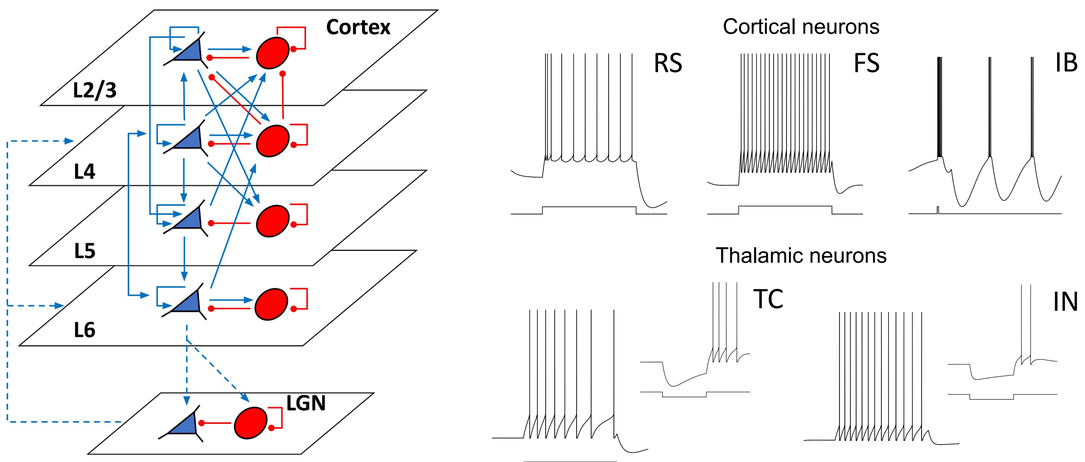

Figure 1. Electrophysiological classes. Cortical neuron patterns on top: regular spiking (RS), fast spiking (FS), and intrinsically bursting (IB). Thalamocortical (TC) and inhibitory (IN) neurons from thalamus on bottom.

Spontaneous activity analysis

- Isolated V1 as the default network for testing the hypotheses
- It mimics resting state with no specific external stimulation
- Non-simulated areas were represented by Poisson spike trains

CSD proxy

- Layer-specific proportions of synapses for each source-target population pair are taken into account
- Excitatory input in a given layer forms a current sink in that layer and a current source in the layer containing the target cell body. Inhibitory input does the opposite

$$CSD_L = \sum_{i \in \mathcal{E}} \sum_{j \in \{\mathcal{E},\mathcal{I}\}} Nsyn_{ij} * \left[-psyn_L^{ij} + \sum_{Lsyn} psyn_{Lsyn}^{ij}\, \delta\left(L_i, L\right) \right] * conv\left(\langle spks_j \rangle \left(t - d^{exc/inh}\right), PSC^{exc/inh} \right)$$

Figure 2. Network activity of the isolated V1 microcircuit. A) raster plot; B) firing rates; C) coefficient of variation of the interspike intervals; D) correlation coefficient; and E) power spectrum calculated from network activity. Each color represents a different population.

Results

L5 rhythmic burst neurons

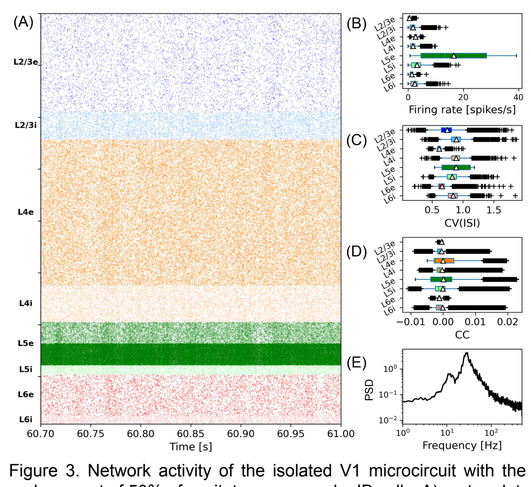

Figure 3. Network activity of the isolated V1 microcircuit with the replacement of 50% of excitatory neurons by IB cells. A) raster plot; B) firing rates; C) coefficient of variation of the interspike intervals; D) correlation coefficient; and E) power spectrum calculated from network activity. Each color represents a different population.

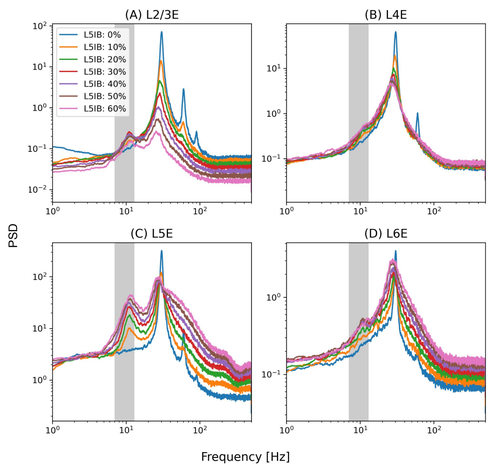

Figure 4. Power spectra estimated from spiking activity by layer for different proportions of IB to RS neurons at L5 (0% to 60% of the total L5 excitatory neurons). The shaded area represents the alpha range.

Thalamocortical loop delay

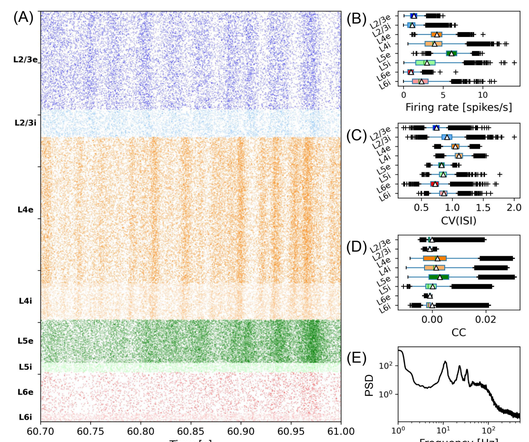

Figure 5. Network activity of the thalamocortical network. Thalamocortical and corticothalamic delays set to 30 and 50 ms respectively. A) raster plot; B) firing rates; C) coefficient of variation of the interspike intervals; D) correlation coefficient; and E) power spectrum calculated from network activity. Each color represents a different population.

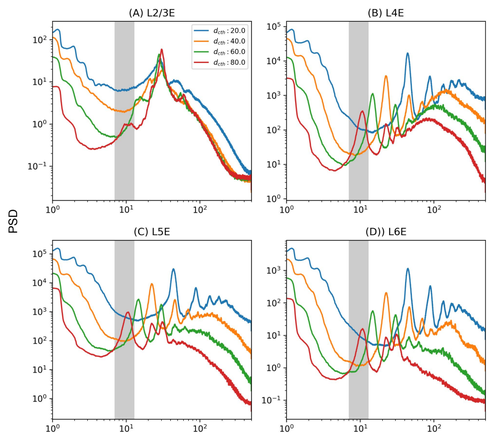

Figure 6. Power spectra estimated from spiking activity by layer for different combinations of corticothalamic synaptic delays. Thalamocortical transmission delays were fixed to 2 ms. The shaded area represents the alpha range.

Alpha rhythm propagation

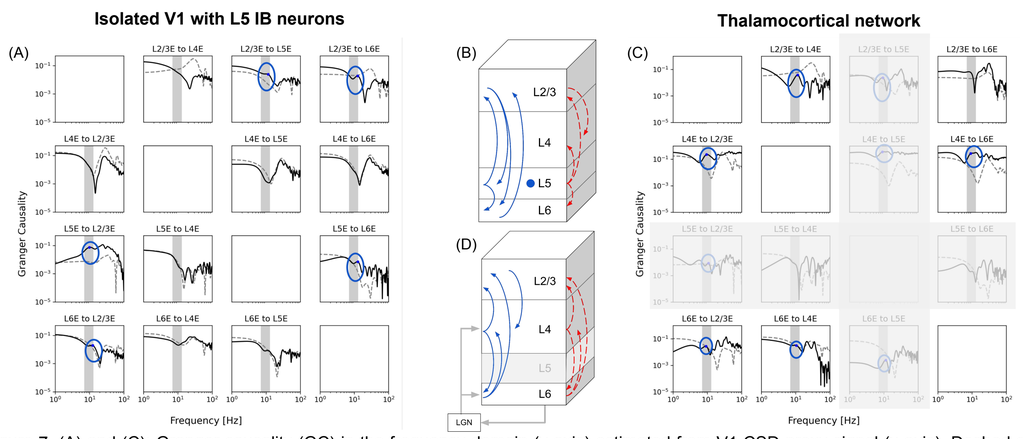

Figure 7. (A) and (C): Granger causality (GC) in the frequency domain (y-axis) estimated from V1 CSD proxy signal (x-axis). Dashed grey lines represent the default network (Fig. 2), while black solid lines represent the networks where both hypotheses were tested. (B) and (D): Schematic diagram illustrating the direction of alpha rhythm propagation through the cortical layers based on GC measures. Blue arrows indicate the circled peaks. Red dashed arrows in (B) and (D) denote experimental results from [5] and [6], respectively.

Summary

- Both mechanisms support alpha oscillations, but with different laminar patterns
- Hypothesis 1 points to Granger causality in the alpha range originating mainly in L5 and L2/3, similar to empirical observations in [5]
- Hypothesis 2 points to L4 and L6 as the main source layers, similar to empirical observations in [6]

References

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