Emergent oscillations in neocortex: a simulation study

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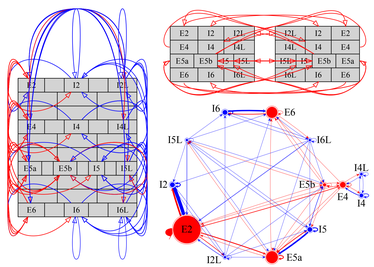
Introduction

Coordination of neocortical oscillations has been hypothesized to underlie cognitive "binding". The mechanisms that generate neocortical oscillations in physiological frequency bands remain unknown. We simulated a neocortical network wired according to known anatomical data. A physiological frequency spectrum appeared as an emergent property. We monitored spectral changes while gently perturbing network dynamics by gradually introducing hubs into individual layers one at a time.

Methods

Network structure and wiring.

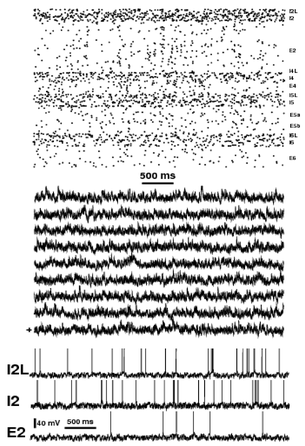


Red for excitatory and blue for inhibitory connections. Cell types are E (excitatory) or I (inhibitory), followed by layer number (2 represents 2/3) and an additional letter for cell subsets: L,low-threshold spiking cells; 2 cell subpopulations in layer 5: 5a,5b. Wiring among the 9 columns was all-to-all.

On the graph-theoretic representation of intracolumnar wiring, circle size represents the number of cells in the population (E red, I blue). Line thickness represents connection strength (divergence multiplied by average synaptic weight).

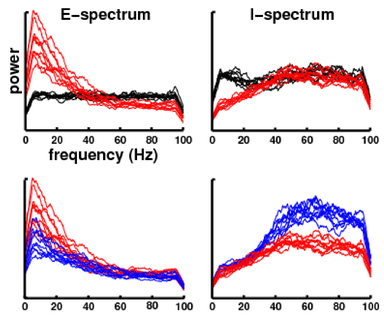
Results

Basic activity of network



LFPs from 9 columns are shown. '+' (lowest trace) indicates LFP corresponding to raster. Also shown are single cell voltage traces from low-threshold spiking interneuron(I2L), fast-spiking interneuron (I2), and pyramidal neuron (E2) in layers 2/3.

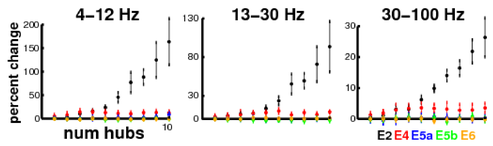
Spectral peaks emerge from cell-cell interactions in network.



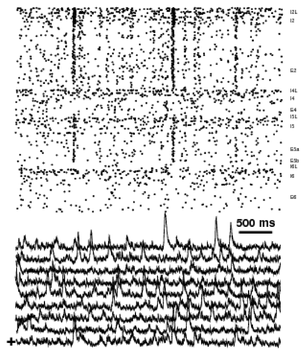
Connecting the cells into the individual columns(red) produces a large theta peak in E-cells and a broad gamma peak in I-cells.

In connected columns, lateral inhibition (blue) is seen as augmentation of gamma activation in the I-cells which leads to broad dampening of the spectrum for the E-cells.

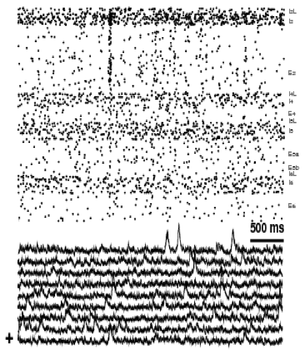
Addition of hubs to E2 produces substantial power increases in unconnected columns.



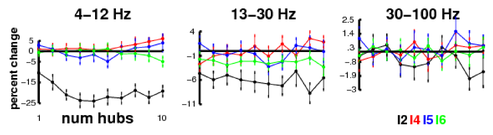
Population spikes are prominent in unconnected columns of 10 E2 hub network.



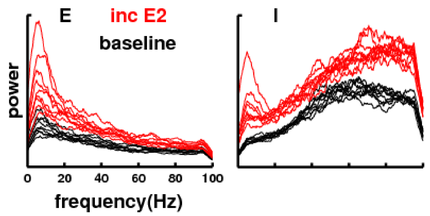
Lateral inhibition across columns reduces population spikes.



I2 hubs sculpt out additional high gamma.

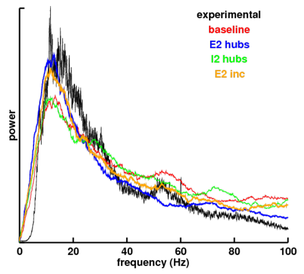


Frequency homeostasis



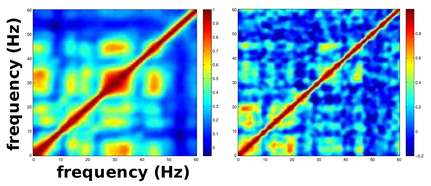
No appreciable shift in E MUA peak with increased external inputs to E2 cells.

Comparison with experimental data



Comparison of power spectra from LFP recorded in left medial prefrontal cortex of awake rat to normalized MUA power spectra from the different simulation types

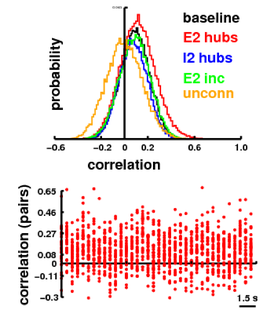
The frequencies that show coupling, and those that don't, are similar in both experiment and simulation.



Power-fluctuation correlation matrices from LFP recorded in left medial prefrontal cortex of awake rat (left) to non-normalized E MUA from baseline simulation(right).

Our simulations suggest that this pattern would be expected to emerge from the correlation of E2 generation of theta with I2 sculpting of gamma.

Intercolumnar correlations



There was a substantial additional increase in correlations in the 10 E2 hub models. Added excitation spread first within, and then across columns, as a result of the hub activity.

Correlation structure demonstrated periodicity across time. Recurrence of correlation strength may indicate recurrence of correlation structure.

Conclusions

Layer 2/3, excitatory cells were the primary drivers (for E cells) or sculptor (for I cells) of overall spectral patterns.

Lateral inhibition between columns reduced activity, but did not change the form of the spectrum.

The network possessed internal homeostatic mechanisms.

The network produced a complex pattern of intercolumnar correlation relationships that could provide the basis for the neocortical phase relations putatively used in cognitive binding.

Acknowledgments

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