Spike-timing-dependent plasticity and subcortical waves enhance alpha oscillations in a computer model of neocortex

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Introduction

A prominent neocortical alpha oscillation emerges over the first several years of life. Slow oscillations are generated during development in retina and other tissues and contribute to wiring synaptic circuitry. We hypothesized that these waves projected upwards from subcortical centers could also train neocortex to produce alpha oscillations. We here show that frequencies of this range could emerge in the network via a process of spike-timing-dependent plasticity (STDP).

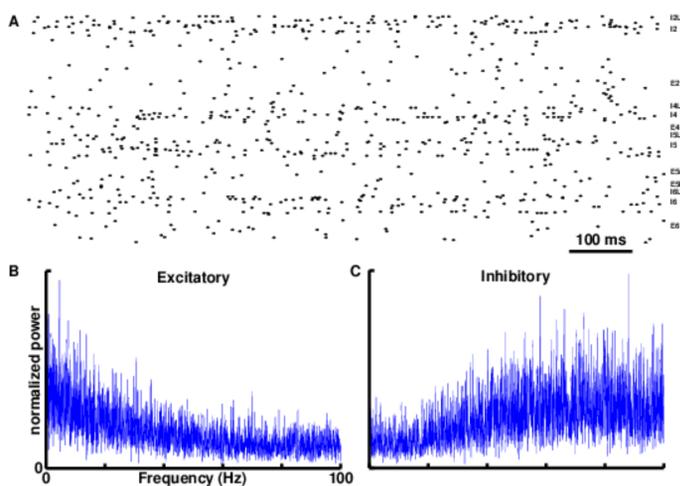## Methods

Layered neocortical column
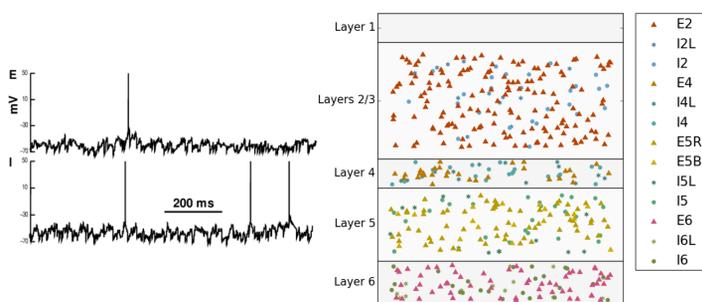
Connectivity: Color represents the normalized probability that a neuron from a given column projects to a neuron from a given row.
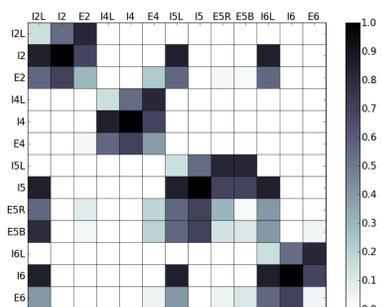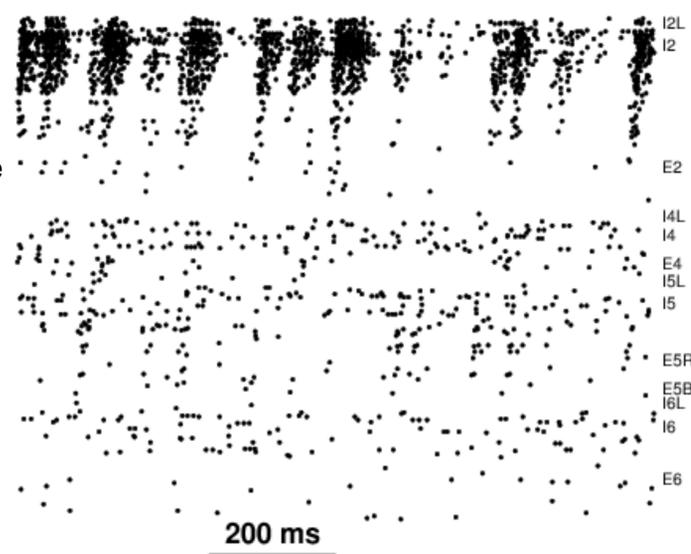
Results

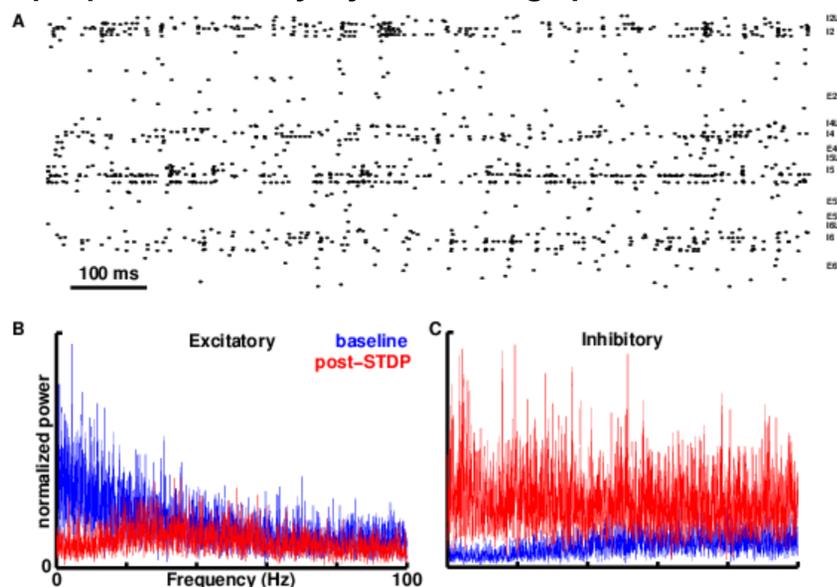**Baseline network activity demonstrates weak oscillations.**

Baseline activity of network: A) raster. B) Excitatory (E) and C) Inhibitory (I) multiunit activity (MUA) power spectra.
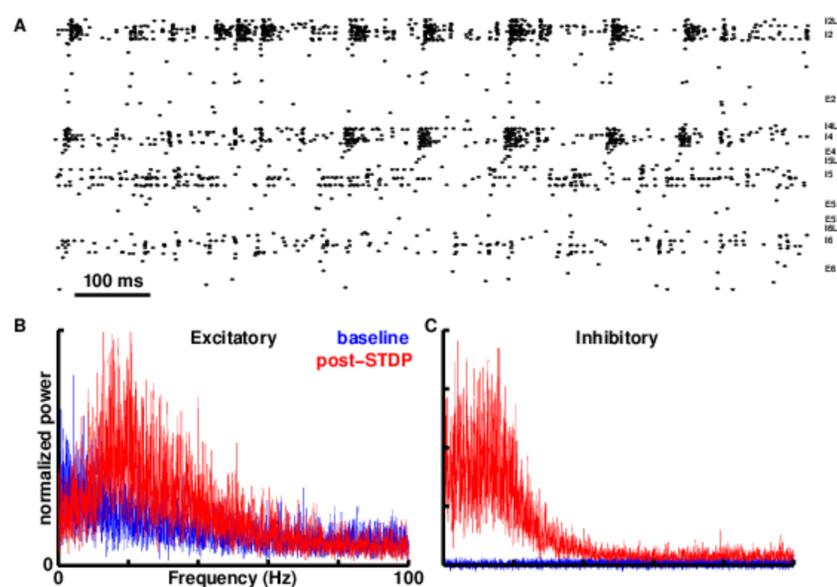
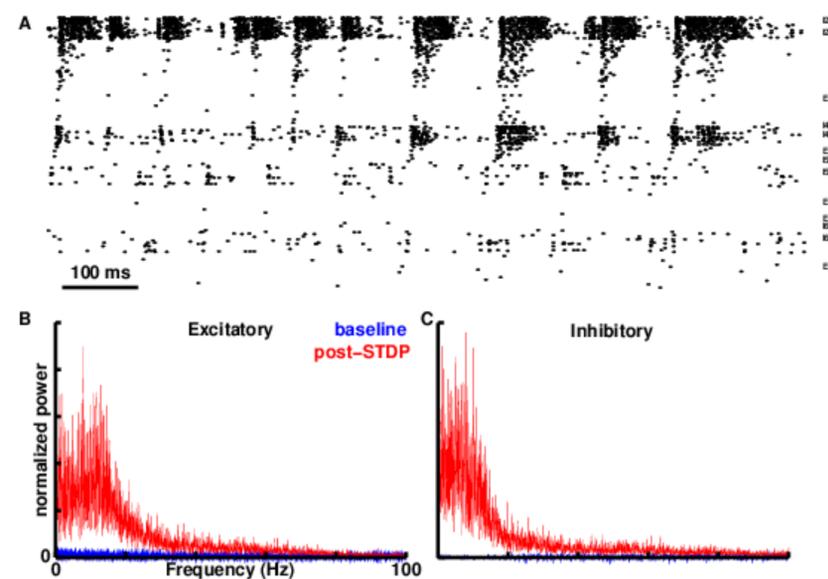**STDP at E->E synapses may promote epileptiform activity.**

Balanced STDP from E->E and E->I synapses prevents epileptiform activity, by attenuating spread of excitation.
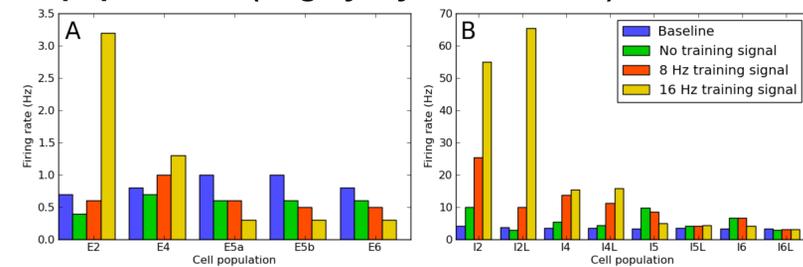
Balanced STDP with subcortical, 8 Hz signal to E4 enhances alpha (and low beta) oscillations.
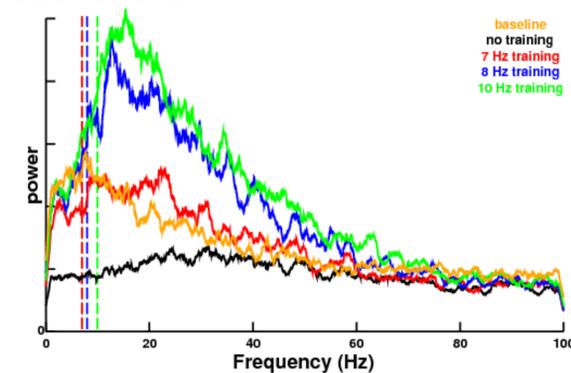
Overlearning: STDP and subcortical, 16 Hz signal to E4 induces epileptiform oscillations

Learning reflected in changes of firing rates of specific subpopulations (largely layers 2/3 and 4).

Summary: subcortical training signals may enhance effectiveness of balanced STDP learning by shifting peak and amplitude of oscillations.

Conclusions

Learning at excitatory to excitatory synapses must be modulated by learning at excitatory to inhibitory synapses to avoid a transition to epileptic dynamics.

Alpha oscillations could develop via STDP learning coupled with training signals projected from sub-cortical areas.

Training signals provided to cortex could be used as a neuroprosthetic to assist in recovery of normal neurodynamics in a neocortex damaged by trauma or stroke.