**Frequency homeostasis in a computer model of neocortex**

SA Neymotin, GL Chadderdon, CC Kerr, JT Francis, WW Lytton

**Introduction**

Oscillations are ubiquitous in both central and peripheral nervous systems, though their significance remains uncertain. We hypothesize that these oscillations, particularly those centered in the alpha band (8-12 Hz), are critical to cognitive and motor performance. We have begun by looking at the source, genesis, and robustness of alpha oscillations. We have shown that frequencies of this range could emerge in the network via a process of spike-timing-dependent plasticity (STDP). We note that this learning generally tends to reinforce, rather than shift, alpha, a phenomenon we term frequency homeostasis. This central resonant tendency would allow a neuroprosthesis to readily restore an oscillation that has been shifted or attenuated after brain damage. We have begun to explore the relevance of this oscillation for the generation of the physiological tremor that underlies movement as a specific application of oscillatory assistance.

**Methods**

- **Connectivity:** Color represents the normalized probability that a neuron from a given column projects to a neuron from a given row.
- **Frequency Homeostasis I:** Network stability after external perturbation. Increased input -> higher amplitude, same peak frequency.
- **Frequency Homeostasis II:** Network stability after balanced learning. Balanced STDP with subcortical, 8 Hz signal to layer 4 excitatory (E4) cells enhances alpha (and low beta) oscillations.
- **Frequency Homeostasis III:** Target-directed STDP parameter modulation (max weights, increments) during runtime allows network (red dots) to tune to desired frequency (blue).
- **Frequency Homeostasis IV:** Stable, neocortical alpha as functional signal.

**Results**

- Network demonstrates physiological oscillations: but can oscillation peak be easily modulated?
- Cell voltage traces
- Connectivity: Color represents the normalized probability that a neuron from a given column projects to a neuron from a given row.

**Activity Homeostasis:**

Learning to avoid epilepsy and silence.

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.

**Conclusions**

1. Learning at excitatory to excitatory synapses must be modulated by learning at excitatory to inhibitory synapses to avoid a transition to epileptic dynamics.
2. Alpha oscillations could develop via STDP learning coupled with training signals projected from sub-cortical areas.
3. Training signals provided to the cortex could be used as a neuroprosthetic to assist in recovery of normal neurodynamics in a neocortex damaged by trauma or stroke.
4. Neocortical oscillations demonstrate homeostasis to external perturbations and to changes in the network induced by plasticity.
5. The balance between E/I strength sets the oscillatory peak frequency by controlling temporal precision of spiking activity and can be exploited to tune a network's oscillations.

**Supported by DARPA grant N6001-10-C-0808**

Acknowledgments: Larry Eberle (SUNY Downstate)