ON THE ORIGINS OF OSCILLATION IN CELLS OF THE CAT STRIATE CORTEX. A.B. Bonds[1], R.K. Snider[1], J.F. Kabara[1], P. Bush[2] and T.J. Sejnowski[2], Dept. of Electrical Engineering[1], Vanderbilt University., Nashville, TN and CNL[2], The Salk Institute, La Jolla, CA.

Problem. The origin and significance of oscillation near 40 Hz in cortical spike sequences is unknown. Method. We recorded responses to drifting sinewave gratings in Area 17 of anesthetized (N_2O) cats and constructed a network model. Results. Patches of oscillation are found in about 25% of striate cortical cells. Oscillation is not confined to a given cell type or cortical layer, but is predominantly found in cells that burst. Bursts are brief (2-5 spikes at 1-3 msec intervals) and are followed by an absolute refractory period of 10-15 msec. When cells are strongly driven, oscillation could result from simple abutment of bursts. We found a graded dependence of oscillation frequency on contrast that is consistent with this hypothesis. The relationship between oscillation and bursts was tested with a compartmental network model. Eight excitatory cells (driven by in vivo recorded spike trains) and two inhibitory cells (driven by the excitatory cells) were fully interconnected. The model yielded ISI histograms and autocorrelations similar to those found in actual cells. Oscillation could be produced in the network using as input spike trains that themselves showed no oscillation. Burst and oscillatory behavior in the model was disrupted severely by reducing either inhibitory feedback or a potassium current dependent on intracellular calcium concentration. Conclusion. We suggest that cortical oscillation can be a natural consequence of intrinsic cellular mechanisms combined with simple inhibitory feedback.

NIH: EY03778 and core grant EY08126, Howard Hughes Med. Inst. and ONR.