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MODEL FOR WAVE PROPAGATION IN THE THALAMIC RETICULAR NUCLEUS IN VIVO. M. Bazhenov[1]*, I. Timofeev[2], M. Steriade[2] and T.J. Sejnowski[1]. [1]The Salk Institute, PO Box 85800, San Diego, CA 92186 and [2]Lab. of Neurophysiology, School of Medicine, Laval University, Quebec, Canada G1K 7P4.

Recent intracellular recordings from reticular thalamic (RE) neurons at resting and hyperpolarized membrane potentials *in vivo* suggest that the reversed inhibitory postsynaptic potentials (IPSPs) between RE cells can directly trigger a low-threshold (LT) spike. The oscillatory mechanisms underlying IPSP-triggered LT spikes crowned by spike-bursts within the RE nucleus were investigated in one- and two-dimensional models of isolated RE networks, which included voltage and Ca^{2+}-dependent currents described by the Hodgkin-Huxley type of kinetics. *(a)* In a one-dimensional network model the external stimulation resulted in waves of excitation propagating with constant velocity 25-150 cells/sec controlled by the $GABA_A$ conductance and radius of synaptic interconnections in the network. The mechanism of propagation depended on the level of membrane potential in RE cells. For membrane potentials below Cl^- reversed potential, bursts of spikes in presynaptic RE cells led to the reversal of $GABA_A$ IPSP followed by a LT Ca^{2+} spike and a burst of Na^+ spikes in neighboring RE cells. The temporal inactivation of the LT Ca^{2+} current in an RE cell after a burst discharge prevented oscillations from persisting in the cell. *(b)* In a large-scale two-dimensional model of the reticular nucleus, the network displayed transient or self-sustained oscillations controlled by the maximum conductance of the LT Ca^{2+} current. Computer simulation of a model including RE and thalamocortical (TC) cells have revealed that the wave patterns in the RE network trigger oscillations involving both RE and TC cells. This model predicts that the isolated reticular nucleus may initiate sequences of spindle oscillations in thalamocortical networks *in vivo*. Supported by the Howard Hughes Medical Institute, Sloan Foundation, MRC of Canada, Human Frontier Science Program and Savoy Foundation.