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MECHANISMS UNDERLYING OSCILLATORY ACTIVITY IN THE THALAMIC RETICULAR NUCLEUS. A. Destexhe*, D. Contreras§, T.J. Sejnowski and M. Steriade§. The Salk Institute, Computational Neurobiology Laboratory, PO Box 85800, San Diego, CA 92186, USA; § Laboratory of Neurophysiology, Laval University, Quebec, CANADA G1K 7P4.

Single compartment models of thalamic reticular (RE) cells with Hodgkin-Huxley-like kinetics were developed based on in vivo and in vitro recordings. Single RE cells displayed a sequence of rebound bursts following current injection or orthodromic stimuli. The three currents I_T, $I_{K[Ca]}$ and I_{CAN} could generate the rhythmic bursting behavior of RE cells reported in thalamic slices (Bal and McCormick, 1993).

Network models of RE cells interacting with fast and slow inhibition were developed based on kinetic models of $GABA_A$ and $GABA_B$ receptors. Two-dimensional arrays of RE neurons interacting with their neighbors exhibited waxing and waning oscillations at spindle frequency of 6.5-9 Hz, as observed in the isolated RE nucleus in vivo (Steriade et al. 1987). These oscillations were accompanied by wave-like spatiotemporal patterns.

The model predicts that oscillatory activity depends critically on the level of the resting membrane potential relative to the chloride reversal potential. For sufficiently negative resting levels, interconnected RE cells do not sustain oscillatory behavior. This may explain why the isolated RE nucleus in vitro does not exhibit spontaneous oscillations (von Krosigk et al. 1993). We suggest that RE cells in vitro may show spontaneous oscillations if their resting levels were brought to more depolarized values, such as those seen in vivo. We propose that the regulation of the resting level of RE cells by neuromodulatory systems may provide a mechanism for controlling the efficiency of the RE nucleus to synchronize oscillations in the thalamus.