COMPARTMENTAL models of thalamic reticular (RE) neurons were investigated based on current-clamp and voltage-clamp data. Spontaneous oscillations in the model arise from the interaction between inhibitory synaptic currents and the rebound burst of RE cells. These oscillations critically depend on the level of the resting membrane potential. A network of RE neurons can be switched to different oscillation states by neuromodulators.

Modeling the control of reticular thalamic oscillations by neuromodulators.
current, both based on current clamp data.\textsuperscript{5,6} \(I_{\text{Na}}, I_k\) are the fast Na\(^+\) and K\(^+\) currents responsible for the generation of action potentials (taken from Ref. 17). Details of the kinetics of these currents, based on estimates from physiological data, are given in Ref. 10.

The synaptic current \(I_{\text{GABA}}\), represents the intra-RE inhibitory current, mediated by \(\gamma\)-aminobutyric acid (GABA). The receptors on RE cells are of GABA\(_A\) type with a very weak GABA\(_B\) component. Only the GABA\(_A\) synaptic currents were modeled here, using a kinetic scheme for the binding of neurotransmitter to postsynaptic receptors.\textsuperscript{18} The current is described by the first-order equation:

\[ I_{\text{GABA}} = \tilde{g}_{\text{GABA}} m (V - E_C) \]  

\[ \frac{dm}{dt} = \alpha [T] (1 - m) - \beta m \]  

\[ g_{KL} = \tilde{g}_{KL} m \]  

\[ \frac{dm}{dt} = \alpha [S] m - \beta (1 - m) \]  

where \(\tilde{g}_{\text{GABA}}\) is the maximal conductance, \(E_C = -80\) mV is the reversal potential, \(m\) is the fraction of postsynaptic receptors in the open state, \([T]\) is the concentration of neurotransmitter in the cleft and \(\alpha (= 0.53\) ms\(^{-1}\) mM\(^{-1}\)) and \(\beta (= 0.184\) ms\(^{-1}\)) are forward and backward binding rates. The neurotransmitter was released as a pulse (1 ms duration, 1 mM amplitude) when a pre-

where \(\tilde{g}_{KL}\) is the maximal leak conductance for K\(^+\) and \([S]\) represents the concentration of second messenger. For RE neurons, the only experiments available for noradrenergic and serotonergic depolarization are from delivery of agonists \textit{in vitro};\textsuperscript{21} the response lasted up to several minutes. Comparable data are not available for electrical stimulation of noradrenergic and serotonergic receptors. Brief stimulation of peribrachial cholinergic nuclei evoked a short lasting (about 2 s) hyperpolarization in RE neurons\textsuperscript{25} and there are many indications that these muscarinic receptors have the same G protein-based activation mechanisms as noradrenergic and serotonergic receptors.\textsuperscript{22}

Therefore, we chose kinetic parameters to obtain a slow depolarization of 2–3 s following a presynaptic spike (\(\alpha = 0.01\) ms\(^{-1}\) \(\mu\)M\(^{-1}\), \(\beta = 0.001\) ms\(^{-1}\), pulse of [S] of 85 ms duration and 1 \(\mu\)M amplitude). Other receptors may participate in the neuromodulatory control of RE cells, such as the glutamate metabotropic receptor.\textsuperscript{24} In the following, we will use the generic term 'NE/5HT' to refer to the transmitter systems involved in the
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|   a  |   b  |   c  | oscillations. This behavior was extremely robust to changes in parameters such as the reversal potential of GABAergic currents ($E_C$), the values of the synaptic conductances or the amount of leak $K^+$ current affected by NE/5HT synapses. Typically, $E_C$ and the resting level were varied in a range of 5 mV around the present... |
FIG. 2. Dependence of RE oscillatory behavior on the membrane potential. Simulation of a network of 100 RE cells interconnected with their neighbors through GABAergic synapses. The top 10 traces represent the activity of 10 neurons in the network and the bottom trace is the average membrane potential. 20% of NE/SHT synapses were initially activated (as in Fig. 1b). In these conditions, the