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Presentation Abstract

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Title:	The role of the cortex in sleep spindles termination
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Abstract:	Spindle waves are a hallmark of non rapid-eye-movement (NREM) sleep stage II of mammals. In humans EEG sleep spindles are 11-15 Hz bursts of spikes grouped in short 1-3 s periods organized within a waxing-and-waning envelope that recur periodically every 5-15 s. There is a growing body of evidence linking spindles to fundamental physiological functions, such as memory and recovery, and pathological spindles occur in some forms of sleep disorders. Therefore, much effort has been devoted to unraveling the mechanisms underlying sleep spindles. Despite the growing number of physiological, anatomical, and computational studies, some essential questions remain unanswered. Spindles are generated in the thalamus and are normally sustained through interactions between thalamic reticular neurons and thalamocortical relay neurons that involve two ionic currents - the cation non-specific hyperpolarization-activated current, I_h, and the low-threshold Ca^{2+} current, I_T, found in thalamocortical cells. In addition, it has been proposed that spindles are terminated by Ca^{2+}-induced up-regulation of I_h, without cortical involvement. Here, using Hodgkin-Huxley based modeling studies and *in vivo* electrophysiological experiments, we demonstrate a direct contribution of cortical cells to the termination of spindles. We investigated the impact of corticothalamic and thalamocortical synaptic connections on spindle modulation under conditions

when the up-regulation of the H-type current was not sufficient to terminate spindles. Increasing the strength of corticothalamic projections transformed continuous spindle-like activity into well separated spindles. Correlation analysis revealed that cortical cells have a higher propensity to fire bursts of action potentials at spindle offset. This increase of cortical activity onto thalamic neurons caused a decreased input resistance of thalamic cells, and concomitantly reduced the rate of deinactivation of channels mediating T-type current, preventing the low threshold spikes from being generated. We concluded that cortical activity caused desynchronization of the thalamocortical network leading ultimately to the spindle termination.
Thus, consistent with the initial suggestion of Andersen & Anderson (1968), we propose that spindle termination is mediated by several complimentary mechanisms, one being the intrinsic thalamic mechanism via H-type current, and the other by synaptic modulation from the cortex. Our results shed new light onto a fundamental cortical mechanism responsible for a central sleep oscillation.

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