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Presentation Abstract

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Title:	Thalamocortical feedback controls the properties of sleep spindles in vivo
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Abstract:	Sleep spindles are 7-14 Hz waxing-and-waning oscillations that last for few seconds and repeat every 5-15 seconds. Spindles are commonly observed during stage II of non-rapid eye movement (NREM) sleep and may be involved in memory consolidation. In slice experiments, spindle initiation usually requires external stimulation and spindle termination is mediated by the hyperpolarization-activated current, I_h, which de-inactivates the low threshold T-current in thalamocortical neurons with calcium accumulation. *In vivo* data suggests, however, that the cortex may control spindle properties, but specific mechanisms remain unknown. We constructed a computational model of thalamocortical circuits in which spindle initiation was triggered by spontaneous activity in cortical layers and the termination of spindle oscillations was mediated by corticothalamic feedback. The primary mechanism of spindle initiation was cortical input to thalamic reticular neurons. The primary mechanism of termination was a desynchronization of firing between thalamic and cortical neurons leading to cortical spiking after rebound bursts in the thalamus. This prevented the T-channel deinactivation and promoted spindle termination. In burst-triggered averages, the cortical population activity became more desynchronized and out-of-phase from the thalamic relay neurons during the waning spindle. The time lag between the occurrence of a thalamic

burst and the onset of a cortical spike increased during the waning part of the spindle, leading to decreased synchronization. Increasing the cortical inhibition led to longer spindle epochs in the thalamus (up to 42% longer when the cortical inhibition doubled). Finally, thalamic populations were more synchronized at spindle offset than at the onset when the cortical feedback was strong. *In vivo* experiments revealed progressive decrease of synchronization between cortex and thalamus during spindle, therefore, confirming the predictions of the computational model. Furthermore, under ketamine-xylazine anesthesia, spindle duration was significantly increased after decortications in agreement with the proposed mechanism of cortical involvement to spindle termination. Through regulation of spindle duration, the neocortex could affect cognitive and pathological processes, including memory consolidation.

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