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Presentation Abstract

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Title: Experience-dependent reactivation of the calcium signal transduction pathway in the rat hippocampus during sleep

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Abstract: Sleep-dependent plastic changes play a key role in the consolidation of newly acquired memories. Two distinct and successive phases of sleep, slow wave sleep (SWS), and rapid eye movement (REM) sleep can be recognized in mammals. Both phases have been implicated in the sensorimotor processing of daytime events, but the molecular mechanisms involved remain poorly understood. Brain expression of the plasticity-associated immediate-early gene (IEG) zif-268 is upregulated during REM sleep in the cerebral cortex and hippocampus of animals exposed to rich sensorimotor experience in the preceding waking period (Learn Mem. 6; 500, 1999). Zif-268 integrates a major calcium signal transduction pathway which includes Ca(2+)/calmodulin-dependent protein kinase II (CaMKII) and mitogen activated protein kinase (MAPK). CaMKII autophosphorylation of T286 is of special importance because it makes the enzyme active in the absence of Ca(2+), providing a biochemical memory that is critical for plasticity. MAPK, an integral component of cellular signaling during mitotic cell differentiation, has been implicated in hippocampal long-term potentiation (LTP) and learning and memory in behaving animals.
Our goal here is to investigate the phosphorylation levels of CaMKII and MAPK during sleep in rats exposed to a new rich environment in the preceding waking period. Intracranial local field potentials (LFPs) recorded in the cortex and hippocampus were used to characterize the wake-sleep cycle (J. Neurosci. 24; 11137, 2004). The phosphorylation levels of CaMKII and MAPK were assessed using specific antibodies for western blots and immunohistochemistry. Our preliminary data (WK n=3, SWS n=5 and REM n=3)) indicate that the MAPK pathway was reactivated in the hippocampus after a few minutes of SWS. Interestingly, for reasons still unknown, MAPK phosphorylation decreased to WK level after a single episode of REM sleep. Our results also showed CaMKII

reactivation during REM sleep in the hippocampus of rats previously exposed to novel objects in the preceding WK period. Controls unexposed to novel experience did not show kinase reactivation during sleep. Our results support the notion that sleep harbors experience-dependent mechanisms of synaptic upscaling (Learn Mem. 6; 500, 1999, J. Neurosci. 22; 10914, 2002, J. Neurochem. 95; 418, 2005, Science, 313; 1775, 2006, FINS 1; 43, 2007, Neuron 61; 454, 2009).

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