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Dendritic spine morphology and excitatory neurotransmission in prefrontal cortex is altered after early life sleep disruption

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Abstract

Sleep deprivation studies indicate an essential role for sleep in the pathophysiology of many conditions, with sleep at a lifetime maximum during early life. Previous research in our lab using an early life sleep disruption (ELSD) paradigm has shown long lasting deficits in complex social and cognitive behaviors in adult prairie voles. We hypothesized that increased time spent awake during ELSD may cause a transient increase in excitatory neurotransmission, leading to morphological changes in dendritic spines and long lasting changes in excitatory neurotransmission. Here, we measured spine density and morphology on pyramidal neurons in layers 2/3 of the prelimbic cortex, a region of the brain involved in the behavioral changes observed in this model. Our approach combined light microscopy of Golgi-Cox stained brain tissue and ultrastructural examination of spines using electron microscopy. We found that ELSD resulted in an increase in thin spines, consistent with spines that are more immature. We also found a decrease in the area of nerve terminals expressing the vesicular glutamate transporter 1 (VGLUT1), a protein that is essential for quantal release of glutamate in presynaptic terminals, and a decrease in the size of the postsynaptic spine contacted by these VGLUT1 labeled presynaptic terminals, all within layer II of the prefrontal cortex. Our results suggest that early life sleep is important in development of excitatory connections and may play a causative role in the pathophysiology of neurodevelopmental disorders featuring impaired social and cognitive behaviors.