Title: Acute Traumatic Stress Enhances Hippocampal Theta Activity and Prolongs Sharp-Wave Ripples

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Post-traumatic stress disorder (PTSD) imposes a substantial burden on affected individuals, with current treatments mostly relying on antidepressants and cognitive-behavioral therapy (CBT), providing only partial and variable symptom relief. This has driven growing interest in identifying neurobiological mechanisms that could be targeted for early intervention or prevention after trauma exposure. However, most animal models of PTSD rely on chronic stress paradigms, in which behavioral alterations develop slowly and lack clear temporal anchoring, limiting mechanistic insight. To address this, we used a highly-temporally precise model of traumatic stress, such as immobilization on board, in which animals' limbs are attached to a metal frame for 2 hours in an open space. This model has shown to induce robust anxiety responses and impairments in fear extinction memory. We combined this stress model with chronic high-density silicon probe recordings to characterize the acute and long-term physiological responses to a single traumatic stress) in hippocampal CA1, a region that consistently shows reduced volume and excitability in humans with PTSD. We observed that during this prolonged immobilization, animals exhibited an increase in hippocampal firing rates accompanied by sustained theta-band oscillations (4–8 Hz), a rhythm typically associated with exploratory and attentive behaviors and known to gate synaptic plasticity in the hippocampus. Remarkably, excitability in the pyramidal layer remained significantly elevated for days after immobilization, indicating long-term neurophysiological adaptations following acute traumatic stress. Pyramidal cell activity also increased during hippocampal sharp-wave ripples (SPW-Rs), a key biomarker of episodic memory consolidation, whereas interneurons showed reduced activity relative to pre-stress baseline levels. This shift in SPW-R single neuron dynamics was associated with fewer short ripples and more long ripples, consistent with enhanced memory consolidation and generalization following traumatic stress. In summary, here we describe a mechanism to bridge acute physiological responses with chronic vulnerability markers, providing circuit-level insights into PTSD pathogenesis and prevention. Future experiments will address the contributions of specific inhibitory cell populations to these effects, to better understand circuit mechanisms and inform targeted therapeutic strategies.