Alterations of in vivo CA1 network activity in Dp(16)1Yey Down syndrome model mice

Laboratory for Neurogenetics, RIKEN Brain Science Institute, Wako-shi , Japan
Laboratory for Circuit and Behavioral Physiology, RIKEN Brain Science Institute, Wako-shi , Japan

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Abstract

Down syndrome, the leading genetic cause of intellectual disability, results from an extra-copy of chromosome 21. Mice engineered to model this aneuploidy exhibit Down syndrome-like memory deficits in spatial and contextual tasks. While abnormal neuronal function has been identified in these models, most studies have relied on *in vitro* measures. Here, using *in vivo* recording in the Dp(16)1Yey model, we find alterations in the organization of spiking of hippocampal CA1 pyramidal neurons, including deficits in the generation of complex spikes. These changes lead to poorer spatial coding during exploration and less coordinated activity during sharp-wave ripples, events involved in memory consolidation. Further, the density of CA1 inhibitory neurons expressing neuropeptide Y, a population key for the generation of pyramidal cell bursts, were significantly increased in Dp(16)1Yey mice. Our data refine the 'over-suppression' theory of Down syndrome pathophysiology and suggest specific neuronal subtypes involved in hippocampal dysfunction in these model mice.

Additional information

Competing interest

The authors declare that no competing interests exist.

Ethics

Animal experimentation: All handling and experiments were conducted in accordance with the protocols approved by the RIKEN Animal Care and Use Committee (#H29-2-218(2) , # H29-2-224(3)).

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