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Presentation Abstract

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Abstract: The structural disposition of astrocytes around synapses places them at the core of information transmission. These glial cells communicate through type 2 IP3 receptor (IP3R2)-dependent calcium oscillations, which are thought to drive the release of neuroactive substances, such as glutamate, ATP or D-serine. When compared to wild type mice, IP3R2 KO mice exhibit impaired hippocampal LTP following theta-burst stimulation of the alveus, presumptively due to a reduction in astrocyte-derived glutamate levels (Navarrette et al., 2012). Our present goal was to investigate if the absence of IP3R2 in vivo led to alterations upon cognitive behaviors, particularly those requiring activation of the hippocampal formation. We found that IP3R2 KO mice presented significant deficiencies in several memory domains as compared to their wild type littermates, including abnormal spatial memory and impaired capacity to recognize novelty. We further found evidence that said deficiencies were specific to behavioral tasks that depended upon learning and memory employment. These data are in support of an important

role of IP3R2-mediated astrocyte calcium signaling for information storage and its manipulation in the brain.

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