



PRINCIPE FELIPE

CENTRO DE INVESTIGACION

Seminario CIPF

Neuroinflammation alters neurotransmission in hippocampus: molecular mechanisms and therapeutic implications

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Neurobiology Lab

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Abstract: Hepatic encephalopathy (HE) is a complex neuropsychiatric syndrome which appears in patients with liver diseases leading to mild cognitive impairment, attention deficits, psychomotor slowing, altered visuo-motor coordination, and may progress to coma and death. Animal models of HE reproduce these neurological alterations, including impaired motor coordination and spatial memory and learning. Hyperammonemia leads to neuroinflammation, which is the main cause of the neurological alterations in animal models of HE. Neuroinflammation alters neurotransmission leading to the cognitive and motor alterations. Previous results have shown that reducing neuroinflammation restores the hippocampus-dependent memory and learning in rats with hyperammonemia and HE. The main aim of this work is to unveil the molecular mechanisms by which neuroinflammation leads to alterations in neurotransmission in hippocampus of hyperammonemic rats. Using hippocampal slices *ex vivo* as experimental model, we found that IL-1 β , a pro-inflammatory cytokine which is increased in hyperammonemia, alters membrane expression of NMDA and AMPA receptors by altering several intracellular signalling pathways. These alterations are reversed by blocking the IL-1 receptor in hippocampal slices. The alteration of these pathways would be the molecular basis of the impairment of spatial memory and learning in hyperammonemic rats. These studies may allow the identification of therapeutic targets to improve cognitive and motor function and quality of life in patients with hepatic encephalopathy.

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