

The Role of Alcohol in Hippocampal Calcium Channel (Ca_v1.2) expression

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Background and Hypothesis:

Voltage-gated L-type calcium channels (Ca_v1.2 and Ca_v1.3) in the hippocampus play important roles in glutamatergic neurotransmission underlying memory and learning. Their overexpression has been implicated in neuroexcitatory cell death and disease states including chronic alcoholism. While increases in Ca_v1.2 gene expression have been reported in the hippocampus after chronic ethanol exposure in rats, the regional distribution of Ca_v1.2 protein after voluntary ethanol (EtOH) drinking has not been reported. We hypothesize that the expression of Ca_v1.2 channels within the hippocampus is increased by EtOH drinking in a region-specific manner.

Methods:

Male Sprague Dawley rats were allowed 28 days of intermittent access to a 10% EtOH solution. At 24 hours after the last exposure to EtOH, brains were collected and processed for immunohistochemistry. Ca_v1.2 associated immunofluorescent signal from subregions of the hippocampus was quantified using ImageJ analysis software.

Results:

Immunohistochemical results indicate that Ca_v1.2 immunoreactivity in the hippocampal stratum granulosum layer within the Dentate Gyrus and the stratum pyramidale layer within CA1 and CA3 regions was increased in response to EtOH treatment. There was no significant change in Ca_v1.2 immunoreactivity for the CA2 region.

Conclusion:

This study suggests that calcium signaling in subregions of the hippocampus is differentially affected by EtOH consumption that may contribute to eventual calcium-mediated apoptosis.

Impact and Implications:

Understanding the process of EtOH-induced hippocampal calcium signaling presents opportunities for understanding the consequences of chronic alcohol exposure related to hippocampal function including memory and learning, and possible interventional therapies for alcohol damage.