Alcohol consumption during adolescence alters the hippocampal response to traumatic brain injury

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Binge drinking is the consumption of large volumes of alcohol in short periods and exerts its effects on the central nervous system, including the hippocampus. We have previously shown that binge drinking alters mitochondrial dynamics and induces neuroinflammation in the hippocampus of adolescent rats. Mild traumatic brain injury (mTBI), is regularly linked to alcohol consumption and share mechanisms of brain damage. In this context, we hypothesized that adolescent binge drinking could prime the development of brain damage generated by mTBI. We found that alcohol binge drinking induced by the ?drinking in the dark? (DID) paradigm increases oxidative damage and astrocyte activation in the hippocampus of adolescent mice. Interestingly, adolescent animals submitted to DID showed decreased levels of mitofusin 2 that controls mitochondrial dynamics. When mTBI was evaluated as a second challenge, hippocampi from animals previously submitted to DID showed a reduction in dendritic spine number and a different spine profile. Mitochondrial performance could be compromised by alterations in mitochondrial fission in DID-mTBI animals. These data suggest that adolescent alcohol consumption can modify the progression of mTBI pathophysiology. We propose that mitochondrial impairment and oxidative damage could act as priming factors, modifying predisposition against mTBI effects.

Alcohol

Dendritic spine

Mitochondria

Oxidative stress

Traumatic brain injury

mitofusin 2

adolescent

alcohol consumption

animal cell

animal experiment

animal model

animal tissue

Article

astrocyte

binge drinking

brain damage

brain level

cell activation

cell structure

controlled study

dendritic spine

disease predisposition

hippocampus

male

mitochondrial dynamics

mouse

nonhuman

oxidation

priority journal

synaptic transmission

traumatic brain injury