Adolescence binge alcohol consumption induces hippocampal mitochondrial impairment that persists during the adulthood

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Binge alcohol drinking is a well characterized consumption pattern related with drinking five or more alcoholic beverages during a short period of time followed by a non-drinking period. Several studies showed that this pattern of alcohol intake is becoming very popular among adolescents. However, little is known about the cellular mechanisms involved in ethanol toxicity under these conditions and if these negative changes could be extending to the adulthood. We previously reported that adolescent binge-ethanol consumption impairs brain function acutely. More importantly, we found that animals exposed to this alcohol treatment showed a decrease in the ATP production that remain over time. Therefore, in the present study, we will evaluate the mitochondrial and oxidative alterations that could occur in the adult hippocampus of rats exposed to a unique binge-drinking episode during the adolescence. Our results indicate that adult hippocampus after one adolescent binge-drinking episode presents an increase in the reactive oxygen species production accompanied of mitochondrial dysfunction. Adolescent binge-like ethanol exposure reduced the expression of the mitochondrial respiration complexes, induced mitochondrial depolarization, increased mitochondrial calcium levels, and reduced ATP production in the adult hippocampus. Altogether, our results indicate that adolescence binge alcohol drinking affects the electron transport chain components expression resulting in mitochondrial failure and loss of calcium buffering in the adult hippocampus. Therefore, we reported for first time that adolescent binge-alcohol consumption has severe repercussions on mitochondrial bioenergetics during the adulthood; and that this is not a transitory change until the state of drunkenness disappears as previously believed.

adolescence

alcohol

binge-drinking

hippocampus

mitochondria

adenosine triphosphate

alcohol

calcium

reactive oxygen metabolite

reactive oxygen metabolite

adolescent

adult

adulthood

alcohol consumption

animal experiment

animal tissue

Article

binge drinking

brain function

calcium cell level

cognitive defect

controlled study

depolarization

disorders of mitochondrial functions

electron transport

exposure

mitochondrial respiration

nonhuman

priority journal

rat

adverse event

age

animal

binge drinking

complication

drinking behavior

drug effect

hippocampus

male

metabolism

mitochondrion

organ culture technique

pathology

Sprague Dawley rat

Age Factors

Alcohol Drinking

Animals

Binge Drinking

Hippocampus

Male

Mitochondria

Organ Culture Techniques

Rats

Rats, Sprague-Dawley

Reactive Oxygen Species