Alcohol impairs hippocampal function: From NMDA receptor synaptic transmission to mitochondrial function

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Many studies have reported that alcohol produces harmful effects on several brain structures, including the hippocampus, in both rodents and humans. The hippocampus is one of the most studied areas of the brain due to its function in learning and memory, and a lot of evidence suggests that hippocampal failure is responsible for the cognitive loss present in individuals with recurrent alcohol consumption. Mitochondria are organelles that generate the energy needed for the brain to maintain neuronal communication, and their functional failure is considered a mediator of the synaptic dysfunction induced by alcohol. In this review, we discuss the mechanisms of how alcohol exposure affects neuronal communication through the impairment of glutamate receptor (NMDAR) activity, neuroinflammatory events and oxidative damage observed after alcohol exposure, all processes under the umbrella of mitochondrial function. Finally, we discuss the direct role of mitochondrial dysfunction mediating cognitive and memory decline produced by alcohol exposure and their consequences associated with neurodegeneration.

Alcohol

Glutamate

Mitochondria

Neurotoxicity

Oxidative stress

Synapses

alcohol

immunoglobulin enhancer binding protein

n methyl dextro aspartic acid receptor

alcohol

n methyl dextro aspartic acid receptor

bioenergy

brain function

cell communication

cell function

cognitive defect

cytokine production

disorders of mitochondrial functions

excitotoxicity

exposure

human

mental deterioration

microglia

nerve cell

nerve degeneration

nervous system inflammation

nonhuman

oxidative stress

priority journal

protein localization

Review

signal transduction

synaptic transmission

animal

drug effect

hippocampus

metabolism

mitochondrion

synaptic transmission

Animals

Ethanol

Hippocampus

Humans

Mitochondria

Receptors, N-Methyl-D-Aspartate

Synaptic Transmission