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Autophagy: A Sensor of Ethanol Toxicity in Various Body Organs

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Alcohol abuse is a major public health challenge worldwide and a leading risk factor for death. Excessive alcohol consumption results in various pathologies in almost all body systems. These pathological changes include apoptosis, necrosis, fibrosis, altered fat metabolism and carcinogenesis. The most affected organs by ethanol toxicity are the liver, gut, testis, breast and brain. Toxic effects of alcohol on organs and tissues result from its metabolism to acetaldehyde, oxidative stress, DNA damage and mitochondrial injury. So, how the body responds to ethanol toxicity? Is there any mechanism sensing and subsequently ameliorating ethanol toxicity?

Autophagy is a survival catabolic pathway for clearance of cellular components and damaged organelles within autophagic vacuoles following multiple forms of cellular stress. Enhanced autophagic sequestration of damaged mitochondria in hepatocytes and Sertoli cells of ethanol-treated rats has been recently reported. This elevated autophagic response may reflect ethanol toxicity and could represent a survival mechanism through the autophagic clearance of pro-apoptotic damaged mitochondria. Elevated autophagy has been also observed in ethanol-treated neuronal cells in vivo and in vitro and suggested to be a neuroprotective response to alleviate ethanol toxicity.

Understanding the molecular mechanisms of autophagy may help in diagnosis and treatment of ethanol-induced damage in various body organs.

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