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 does the body respond 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.

*Corresponding author: Nabil Eid, Department of Anatomy and Cell Biology, Division of Life Sciences, Osaka Medical College, 2-7 Daigaku-machi, Takatsuki, Osaka 569-8686, Japan

Received May 17, 2013; Accepted June 11, 2013; Published June 13, 2013


Copyright: © 2013 Eid N. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.