ALCOHOL AND THE HEART MUSCLE

Summary

Alcoholic beverages played an important role throughout the human history. Enriching the cuisine, adding splendor to celebrations, serving as a precious gift and sometimes as means of refuge from the burdens of life, alcoholic drinks intoxicate the recipient in a dose-dependent manner. In individuals drinking chronically 7–8 standard drinks daily over years develop changes in heart structure and performance, which eventually lead to alcoholic cardiomyopathy (ACM). ACM appears clinically as dilated cardiomyopathy (DCM), so that sometimes it may not be diagnosed properly. The course of early stages of the disease is asymptomatic, and then there appear stepwise increase of tiredness and finally signs of severe cardiac insufficiency. The intricate mechanism of myocardial damage involves, among others, changes in sarcoplasmatic protein expression, disturbances of signaling pathways, impairment of mitochondria functions, necrosis and apoptosis. Most of the mechanisms were studied in animal models, where impairments were observed in protein synthesis due to changes in translation, modulation of peptide chain initiation and availability of eukaryotic initiation factor (eIF). Perfusion of myocardium with alcohol resulted in energy-saving adaptations typical for ageing or diseases increasing the sequent load, exemplified by an increase in expression of β-MHC myosine isoforms. Other experiments revealed promotion of apoptosis through fragmentation of DNA and an increase in the content of Bax protein. Among the conditions facilitating cardiomyopathy are also changes in calcium ions flow and oxidative stress. Despite this rather discouraging experimental data, drinking of small quantities of alcohol (21–24 grams/24 hrs) is clinically approved as a form of cardio-protection. Further clinical studies in this field are necessary.