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Studied the protective effect of taurine on oxidative processes in alloxan diabetes

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Abstract

In this work we tried to find out some of the mechanisms of taurine action on the main metabolic processes that characterize this disease in the general homogenate and mitochondrial fraction of the liver of alloxandiabetic rats.

The purpose of this work is to assess the ability of the sulfur-containing amino acid, taurine, to exhibit anti-glycating and antioxidant activity and correct metabolic disorders in experimental alloxan diabetes.Analysis of the levels of the studied parameters that alloxan diabetes is characterized by the activation of free radical oxidation processes, as well as a decrease in glutathione content and a decrease in the activity of glutathione-containing enzymes. The introduction of taurine resulted in a decrease in the content of lipid peroxidation products, partial normalization of oxidative modification of proteins and Schiff bases in the rat liver homogenate alloxan diabetes.

The protective effect of taurine is associated with its effect on glutathione content, due to the presence in the structure of taurine SH-group, which has pronounced reducing properties and the ability to take electrons from various reactive oxygen forms, inactivate free radicals and maintain the level of endogenous glutathione.

Introduction: Oxidative stress is associated with various diabetic complications and taurine plays an important role in ameliorating those difficulties. In the present study we, therefore, investigated whether taurine plays any beneficial role against diabetes induced liver dysfunction and if it does, what cellular mechanism it follows during protective action. Induction of diabetes by alloxan (ALX) (at a dose of 120 mg/kg body weight, i. p., once) reduced body weight and plasma insulin level, enhanced blood glucose and serum markers related to hepatic injury, accelerated ROS production, disturbed the intra-cellular antioxidant machineries and disintegrated hepatic cells near central vein. This pathophysiology leads to apoptotic cell death as evidenced from DNA fragmentation and TUNEL aasay. Studies on the mechanism of apoptosis showed that ALX accelerated the markers of mitochondrial dependent apoptotic pathway (enhanced cytochrome C release in cytosol from mitochondria, altered the expression of Bax, Bcl-2, Apaf-1, Caspase-9, caspase-3). Treatment with taurine

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(1% w/v for three weeks) post-hyperglycemia, however, could restore all the alteration caused by ALX. Moreover, taurine activates hepatic PI3Kinase, Akt, hexokinase and augments the translocation of GLUT 2 to hepatic membrane in diabetic rats. Combining all, as a potential therapeutic, taurine may normalize the complications of diabetic liver injury.

Conclusions: Thus, we can conclude that the administration of taurine to rats with alloxan diabetes leads to the normalization of oxidative processes.

Biography

Zanginyan Hasmik, scientific worker of the Laboratory of Molecular Membraneology of the Institute of Molecular Biology of NAS RA. In 2013, she defended her thesis and received her PhD in Biological Sciences.He is the author of more than 25 works published in various journals and conferences.

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