Effect of β-selenoamines in Thioacetamide-induced Acute Hepatotoxicity in Mice

Hartmann, D.D. [1]; Stefanello, S.T. [1]; da Silva, T.C. [1]; Leite, M.T.B. 1; Amaral, G.P. [1]; Souza, M.B. [1];Soares, F.A.A. [1]

[1]Dep. de Bioquímica e Biologia Molecular, Centro de Ciências Naturais e Exatas, UFSM, RS, Brazil.

Introduction: Thioacetamide (TAA) administration is an elegant model that can produce liver injury in mice. This model is useful for evaluate the therapeutic potential of new drugs. Our group already shown that organoselenium compounds (diselenides) were able to protect the liver function and diminish the oxidative stress generated by TAA induction. **Objective:** Thus, the objective of this study was to analyze the effect β-selenoamines or monoselenides (1-phenyl-3-(p-tolylselanyl)propan-2-amine (C1) and 1-(2-methoxyphenylselanyl)-3-phenylpropan-2-amine (C2) on the TAA-induced acute hepatotoxicity. **Materials and Methods:** The animals received a TAA dose of 200 mg/kg intraperitoneally and then one hour later, they received 15.6 mg/kg intraperitoneally of organoselenium compounds. After twenty three hours, the animals were killed and blood and liver samples were collected for analysis (serum alanine and aspartate aminotransferase levels, methyl-tetrazolium reduction (MTT), lipid peroxidation and reactive oxygen species (ROS) production). **Results and Discussion:** The TAA group presented high levels of transaminases and the treatment with β-selenoamines did not reduce these levels. In addition, β-selenoamines treatment did not protect against the TAA ROS production. However, the MTT assay showed that TAA caused severe cellular injury and we observed that the treatment with organoselenium compounds protect partially this damage, demonstrating that the treatment had little beneficial effect against TAA-induced cell damage. Additionalyl, the administration of C1 reduce significantly the lipid peroxidation when compared with the TAA group. **Conclusions:** Our results suggest that β-selenoamines was able to decrease the lipid peroxidation and maintain the liver cell viability; however, we observed that the treatment did not protect against the TAA-induced oxidative damage because ROS, alanine and aspartate aminotransferase levels remain elevated.

Keywords: Thioacetamide, Hepatotoxicity, β-selenoamines.

Supported by CAPES, FAPERGS and CNPq.