



Cytoprotective effects of taurine against the toxicity induced by isoniazid and hydrazine in isolated rat hepatocytes

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Background and Aims: The cytoprotective effects of taurine against isoniazid and its suspected toxic metabolite, hydrazine in isolated rat hepatocytes were evaluated.

Methods: Isolated hepatocytes were prepared by collagenase enzyme perfusion method. Reactive oxygen species (ROS) formation, lipid peroxidation, mitochondrial depolarization and cellular reduced and oxidized glutathione were assessed to evaluate the toxicity induced by isoniazid and its metabolite.

Results: Isoniazid caused no significant ROS formation in intact hepatocytes but the amount of ROS formed in glutathione depleted cells was considerable. Hydrazine caused ROS formation and lipid peroxidation in both intact and glutathione depleted cells. Taurine (200 μ M) and N-acetylcysteine (200 μ M) effectively prevented isoniazid and hydrazine cytotoxicity as revealed by decreasing ROS formation and lipid peroxidation and preventing mitochondrial damage. Taurine prevented the reduction in cellular GSH and reduced the level of oxidized glutathione (GSSG) produced in hydrazine treated cells.

Conclusions: This study suggests that taurine protective role is attributed to its effect on attenuating oxidative stress and protecting subcellular components such as mitochondria from adverse effects of isoniazid and its metabolite.

Keywords: Isoniazid; Hydrazine; Taurine; Mitochondria; Oxidative stress; Lipid peroxidation