

Abstract.

Long term arsenic exposure finally results in cancer in different parts of the body. Some synthetic therapeutic agents show insufficient potency with severe side effects against arsenic toxicity. The flesh of *Bellamya bengalensis*, an edible snail has long been used by rural people. This organism has been used as traditional medicine in several health-anomalies/ arthritis, conjunctivitis, impaired immune system, blood impurities and liver-disorders. In the study, the possible protective and therapeutic effect against arsenic induced rat tissue damage are attributed by antioxidative mechanism and attenuation of pro-inflammatory cytokine. This potent organism might be a choice against arsenic and toxicities. Currently to investigate the possible protective and therapeutic effect against arsenic induced hepatotoxicity, the extract of *Bellamya bengalensis* was tested in arsenic intoxicated rat model. The time- and dose-dependent effect of arsenic toxicity was also experimented against *Bellamya bengalensis*. Sodium-meta-arsenite NaAsO_2 (0.6 ppm/100g bw/day for 28 days, as earlier reported) was treated alone or in combination with the *Bellamya bengalensis* water extract (BBE, 100mg/100g bw) to rat and compared with vehicle treated control. In a separate experiment, the *Bellamya bengalensis* was exposed to high concentration of NaAsO_2 contaminated water (5 to 20 ppm for 1 to 9 days) in laboratory condition and their DNA quality was evaluated in relation to its possible oxidative threat. Data show that arsenic was incapable to initiate a significant DNA damage in *B. bengalensis*. Lipid peroxidation was increased in arsenic exposed *Bellamya* after longer time of exposure. Increase in reduced antioxidant like non-protein-soluble thiol (NPSH) is paralleled with the decrease in lipid peroxidation and DNA stability in this organism. In rat experiment, the BBE supplementation strongly prevented arsenic-induced oxidative, necrotic and apoptotic damages to liver tissue/DNA by

strengthening antioxidant systems. And these have been demonstrated in hepatic DNA-fragmentation, mitochondrial membrane stability, comet-assay, histo-architecture (hematoxylin/eosin), study increase in pro-inflammatory cytokine TNF- α was reverted terminating an acute phase reaction. But arsenic exposure decreased hepatic superoxide-dismutase (SOD) *in-vivo* and *in-vitro enzymatic* activities and the level of NPSH with a simultaneous increase in malondialdehyde. This protective result in the restoration liver DNA and tissue histoarchitecture. The present investigation offers strong evidence on the hepatoprotective and medicinal efficiencies of BBE against oxidative stress induced by arsenic.