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Toxicol Mech Methods. 2012 May;22(4):277-88. Epub 2012 Jan 9.

Silibinin potentially protects arsenic-induced oxidative hepatic dysfunction in rats.

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Abstract

Arsenic (As) compounds are reported as environmental toxicants and human carcinogens. Exposure to arsenic imposes a big health issue worldwide. Silibinin (SB) is a major flavonolignan compound of silimarin and is found in **milk thistle** of **Silybum marianum**. It has been reported that silibinin has antioxidant efficacy as metal chelators due to the orientation of its functional groups. However, it has not yet been explored in experimental animals. In view of this fact, the purpose of this study was to delineate the ameliorative role of silibinin against arsenic-induced hepatotoxicity in rats. Rats were orally treated with arsenic alone (5 mg/kg body weight (bw)/day) plus silibinin (75 mg/kg bw/day) for 4weeks. Hepatotoxicity was evaluated by the increased activities of serum hepatospecific enzymes namely aspartate transaminase, alanine transaminase, alkaline phosphatase, gamma glutamyl transferase, lactate dehydrogenase and total bilirubin along with increased elevation of lipid peroxidative markers, thiobarbituric acid reactive substances, lipid hydroperoxides, protein carbonyl content and conjugated dienes. The toxic effect of arsenic was also indicated by significantly decreased activities of membrane bound ATPases, enzymatic antioxidants like superoxide dismutase, catalase, glutathione peroxidase, glutathione-S-transferase, glutathione reductase and glucose-6-phosphate dehydrogenase along with nonenzymatic antioxidants like reduced glutathione, total sulfhydryl groups, vitamins C and E. Administration of silibinin exhibited a significant reversal of arsenic-induced toxicity in hepatic tissue. All these changes were supported by reduction of DNA damage in hepatocytes and histopathological observations of the liver. These results suggest that silibinin has a potential protective effect over arsenic-induced hepatotoxicity in rat.

PMID: 22229868 [PubMed - indexed for MEDLINE]

MeSH Terms, Substances

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