Balakrishnan Aristatile, J Mol Biomark Diagn 2011, 2:4
doi: http://dx.doi.org/10.4172/2155-9929.S1.10

OMICS Group Conferences
Accelerating Scientific Discovery

2nd World Congress on
Biomarkers & Clinical Research

12-14 September 2011 Baltimore, USA

Effect of carvacrol on D-galactosamine-induced mitochondrial enzymes and DNA damage by single-cell gel Electrophoresis

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In the present study, we investigate the effect of carvacrol on the activities of serum hepatic marker enzymes, mitochondrial enzymes and the DNA damage in D-galactosamine (D-GalN)-induced hepatotoxic rats. The hepatic marker enzymes such as aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP) and γ-glutamyl transpeptidase (GGT) activities are elevated in (D-GalN)-induced rats. In addition, the activities of hepatic mitochondrial enzymes (such as isocitrate dehydrogenase, ά-ketoglutarate dehydrogenase, succinate dehydrogenase, malate dehydrogenase, NADPH dehydrogenase and cytochrome c-oxidase) are significantly decreased in D-GalN- hepatotoxic rats. Oral administration of carvacrol brought these hepatic markers and mitochondrial enzymes activities to near normal levels. In D-GalN-induced hepatotoxic rats, the hepatic mitochondrial thiobarbituric acid reactive substances (TBARS) significantly increased and the activities of enzymatic antioxidants such as superoxide dismutase (SOD), glutathione peroxidase (GPx) and non-enzymatic antioxidants such as vitamin C, vitamin E and reduced glutathione (GSH) decreased significantly in the liver and mitochondria. Administration of carvacrol restores the enzymatic activities and non-enzymatic antioxidants levels towards normal. DNA damage was observed in D-GalN-hepatotoxic rats and treatment with carvacrol significantly decreased the DNA damage. These results suggest that carvacrol is having hepato-protective and antioxidant properties and can also protect the liver mitochondrial damage in D-GalN-induced rats.