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Anticancer potential of rhamnocitrin 4′-β-D-galactopyranoside against *N*-diethylnitrosamine-induced hepatocellular carcinoma in rats

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Abstract The hepatoprotective activity of flavonoid rhamnocitrin 4′-β-D-galactopyranoside (RGP) obtained from leaves of *Astragalus hamosus* L. against *N*-diethylnitrosamine (DENA)-induced hepatic cancer in Wistar albino rats was evaluated. Hepatic cancer in rats was induced by single-dose intraperitoneal administration of DENA (200 mg/kg). Induction of hepatic cancer was confirmed after 7 days of DENA administration by measurement of elevated level of serum α-feto protein (AFP). Administration of DENA in a single dose lofted the levels of serum biochemical parameters like alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, total bilirubin, total protein and AFP. Antioxidant enzymes like superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione-*S*-transferase (GST) and lipid per oxidation (LPO) were annealed significantly by administration of RGP in a dose-dependant manner. The histopathological examination of rat liver section was found to reinforce the biochemical observations significantly. It was observed that a substantial and dose-dependent reversal of DENA-diminished activity of antioxidant enzymes like SOD, CAT, GPx, GST and the reduced DENA-elevated level of LPO with a marked change. Any elevation in the levels of serum markers along with suppression of free radical formation by scavenging the hydroxyl radicals is significantly prevented by RGP. It also modulates the levels of LPO and perceptibly increases the endogenous antioxidant enzymes level in DENA-induced hepatocellular carcinogenesis. The findings suggest that RGP prevents hepatocellular carcinoma by suppressing the marked increase in the levels of serum marker enzymes, and suppresses the free radical by scavenging hydroxyl radicals.

Keywords DENA · Hepatocellular carcinoma · Rhamnocitrin 4′-β-D-galactopyranoside · Biochemical parameters · Anticancer

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Introduction

Hepatocellular carcinoma (HCC) is proving to be the plebeian of liver cancer obliterating over 500,000 people around the globe. The incidence is endemically high in Asia and Africa due to the prevalence of hepatitis B and C [1]. HCC is result of the mutation of cellular machinery that increases the rate of replication and results in cell avoiding apoptosis [2]. HCC occurs mainly as a manifestation to