

MOLECULAR MECHANISM OF HEPATO-RENAL PROTECTION OF CAMEL MILK AGAINST OXIDATIVE STRESS-PERTURBATIONS

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ABSTRACT

Possible molecular mechanism of camel milk protection of liver and kidney against oxidative stress generated by CCl₄ injection was investigated. Rats injected with carbon tetrachloride (CCl₄) showed upregulation of the mRNA expression of hepatic IL-6 and renal IL-1 β , TGF- β 1, SREBP-1c and caspase-6 and down-regulation of anti-oxidative enzymes SOD, GST and CAT in addition to hepatocellular vacuolation, mononuclear cell infiltration and sinusoidal dilatation and renal glomerular atrophy, capsular space expansion, and adhesion between visceral and parietal layers of Bowman's capsule. Camel milk supplementation prior and with CCl₄ injection to rats attenuated CCl₄-induced hepatic and renal inflammatory cytokines (IL-6, IL-1 β , TGF- β 1 SREBP-1c and caspase-6), upregulated CCl₄-suppressed anti-oxidative markers (SOD, GST and CAT) and induced protective and regenerative mechanism (EPO and IL-10). Additionally camel milk protected the liver and kidney from CCl₄-induced histopathological changes. These results showed the mechanism of camel milk protection of liver and kidney against CCl₄-generated oxidative stress and injuries. These findings may support the beneficial use of camel milk as therapeutic adjuvant with drugs that always associated with production of oxidative stress that injured liver and kidneys as anti-tumor drugs as Cisplatin.

Key words: Camel, hepato-renal protection, milk, oxidative stress

List of abbreviations: (SOD) Superoxide dismutase, (GST) Glutathione S-transferases, (EPO) Erythropoietin, (TG) Triglycerol , (SREBP-1) sterol regulatory element-binding protein-1c, (TGF- β 1) Transforming growth factor beta (TGF β 1), (HCV-infected) Hepatitis C virus infected, (ROS) Reactive Oxygen species, (IL-6) Interleukin-6, (IL-10) Interleukin-10, (IL-1 β) Interleukin-1 beta, (i.p) Intraperitoneal injection, (CAT) Glutathione-S-transferase, (RT PCR) reverse transcription polymerase chain reaction.