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Umbelliferon-α-D-glucopyranosyl-(2I→1II)-α-Dglucopyranosideprevents chemically induced renal carcinogenesis by modifying oxidative stress, hyperproliferation and inflammation: role of NF-κB

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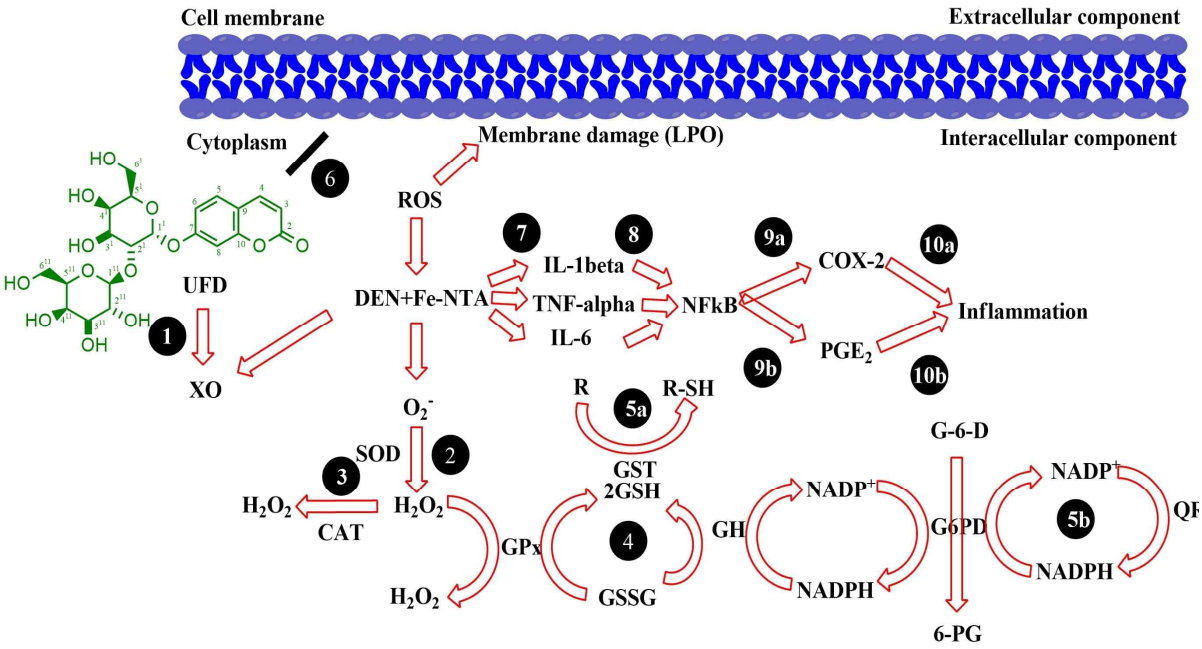
Objectives: Renal cell carcinoma (RCC) is the most serious toxic malignancy in adult kidneys but it is not widely known to account for 3% of all adult malignancies and 90% of kidney neoplasms. An RCC patient's overall survival time is about 4 months and only 10 per cent of patients with RCC live for a year. Umbelliferone (7-hydroxycoumarin) is strongly anti-inflammatory and has free radical scavenging activity. The aim of the current experimental study to scrutinize the renal protective effect of Umbelliferon-α-D-glucopyranosyl-($2^{I}\rightarrow1^{II}$)-α-Dglucopyranoside (UFD) against diethylnitrosamine (DEN) and ferric nitrilotriacetate (Fe-NTA) induced renal cancer and explore the possible mechanism of action.

Methods: DEN (200 mg/kg) and Fe-NTA (9 mg/kg) was used for the induction of renal cancer. To estimate the molecular mechanism involved in UFD renal potential, its effect on the inflammation of the renal tumor was evaluated; the pro-inflammatory cytokines included interleukin-1β (IL-1β), interlukin-6 (IL-6) and tumor necrosis factor (TNF-α); the inflammatory mediator included prostaglandin E2 (PGE2), ornithine decarboxylase (ODC) and the nuclear factor kappa B (NFÿB) were estimated.

Results: UFD showed the 92% and 98% antioxidant activity in the 2,2′-azino-bis (3-ethylbenzothiazoline-6-sulfonic acid) (ABTS) and 2,2-diphenyl-1-picrylhydrazyl (DPPH) models. UFD significantly inhibited the RNS and ROS radical and indicated the antioxidant activity (in vitro). The results showed momentous renal markers such as creatinine, lactate dehydrogenase (LDH), blood urea nitrogen (BUN) and thymidine [3H].and oxidative stress protection such as lipid peroxidation (LPO), endogenous antioxidant enzymes, phase II metabolizing enzymes, and concomitant glutathione (GSH) reduction have been decreased impaired by UFD. UFD also restored the altered inflammatory and proinflammatory cytokines, which further strengthens the renal protection of UFD in DEN + Fe-NTA induced renal carcinogenesis.

Conclusions: UFD is an efficient chemoprotective agent with the ability to thwart induced DEN and that Fe-NTA promoted renal carcinoma through inflammatory pathway.

Mode of Action



Graphical Abstract