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Resveratrol Ameliorates Hepatic Cancer Induced By Diethylnitrosamine Through The Down-regulation Of The NF-kB Pathway

V KUMAR*[1]

[1]*Pharmacy, SHUATS, INDIA*

Background : A leading cause for cancer-related death, hepatocellular carcinoma (HCC) is one of the world's major health problems. The most frequent expansion of HCC due to chronic hepatic inflammation due to alcohol, viral infections, and exposure to dietary and environmental carcinogens. NF-kB pathways are considered to be the unique process that activates the different cellular functions including cell expansion, survival, proliferation, and vesicular transport, and frequently found dysregulated pathways in HCC. Natural product-based inhibitors thus play an important role in the NF-kB pathway and have been widely scrutinized in recent years in targeted cancer. The aim of this research was therefore to scrutinize the role of resveratrol (RT) as hepatic cancer as inhibitors of NF-kB.

Methods : Wistar rats (48) were divided into four groups. DEN (200 mg/kg) dose were used for induction the HCC in rats and treated with the RT for 22 weeks. Macroscopical detection was performed. Body weight, hepatic, biochemical and antioxidant parameters scrutinized, respectively. Pro-inflammatory cytokines including TNF-α, IL-6, IL-1β and NF-κB expression were estimated, respectively. Histopathological observation was also performed.

Results : RT significantly ($p<0.001$) altered the hepatic parameters such as AFP (88.4%), AST (74.5%), ALT (59.3%)alongwith the biochemical and antioxidant parameters. DEN group rats suggest the expansion of hepatic nodules (89%, 68% and 46%), which was reduced by RT at dose dependently. DEN up-regulated the pro-inflammatory cytokines including IL-6 (48.6%), TNF-α (57.8%), IL-1β (45.3%) and NF-κB (45.6%), which suggest the expansion of hepatic inflammation during cancer and down-regulated by RT. RT also reduced the production of COX-2 (84.4%) and PGE2(59.7%), suggest the anti-inflammatory effect.

Conclusions : Collectively, we can conclude that RT down-regulated the HCC via inhibition of NF-kB pathway.

Corresponding Author : V KUMAR (pharmvikaskumar@gmail.com)