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Berberine attenuates 1,2-dimethylhydrazine dihydrochloride-(DMH-) induced colorectal cancer in rat using network pharmacology approach and downregulation of specific proteins (interleukin-6, Tumor Necrosis Factor, and high-mobility group box 1)

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Introduction: Colorectal cancer (CRC) is the commonly and most occurring type of colon or rectal tumour and accounts for approximately 10% of all types of cancer and about 718,000 untimely deaths per year. Berberine shows various therapeutic effects and found effective against colorectal cancer, but it does remain unclear that how it works. We have therefore scrutinized the possible underlying mode of action of Berberine on colorectal cancer. Various researches have revealed that it shows anticancer property and used as an adjuvant therapy in the colorectal cancer.

Methods: A network of pharmacology approaches have been used to predict berberine-related proteins and colorectal cancer-related genes and therapeutic objectives for berberine in Colorectal cancer. The contemplation for mechanism for berberine was associated with five targets, a. the proteasome (26S), b. interleukin 6 (IL-6), c. p53, d. the transforming growth factor beta 1 (TGF- β 1), e. the Tumor Necrosis Factor (TNF- α) and corresponding high-mobility group box 1 (HMGB1) signaling and T helper cell differentiation. The expression of forecast serum objectives has been checked in 18-weeks of interapertitoneal treatment of berberine in 1,2-dimethylhydrazine dihydrochloride-induced Colorectal cancer in rats.

Results: As per Ingenuity Knowledge Database, it was observed that five target protein and signaling of HMGB1 were highly linked molecule associated with the underlying mechanism in the treatment of CRC and potential target protein for berberine. Results reveal that high dose of berberine repressed the growth of colorectal cancer, tumour number, tumour.

Conclusions: Pharmacological efficacy of berberin on colorectal cancer may have taken place through HMGB1 inhibition.

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