Berberine inhibits the proliferation of colon cancer cells by inactivating Wnt/β-catenin signaling.


Source

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Abstract

Colon cancer is one of the most common malignancies, mainly initiated by the abnormal activation of Wnt/β-catenin signaling. In this study, we investigated the proliferation inhibitory effect of berberine on colon cancer cells and the molecular basis underlying this effect. With the viability, apoptosis and cell cycle assay, we demonstrated that berberine can inhibit proliferation, induce apoptosis and cell cycle arrest in colon cancer cells. In in vivo investigation, we demonstrated that berberine can prevent the colon cancer formation initiated by dimethylhydrazine (DMH) and dextran sodium sulfate (DSS) in rats. We employed western blotting, reverse transcription and polymerase chain reaction, special antagonist, overexpression and knockdown techniques to dissect the possible molecular mechanisms mediating the function of berberine. We found that the protein levels of β-catenin in the nucleus and cytoplasm were all reduced after treating the colon cancer cells with berberine, and this may not result from accelerating the degradation of β-catenin in the cytoplasm, but from inhibiting the mRNA expression of β-catenin. Our results indicate that berberine can be a potential chemoprevention and chemotherapy agent for human colon cancer by targeting Wnt/β-catenin signaling.

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