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Inhibition of colon carcinogenesis by a standardized Cannabis sativa extract with high content of cannabidiol.

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Abstract

PURPOSE: Colon cancer is a major public health problem. **Cannabis**-based medicines are useful adjunctive treatments in cancer patients. Here, we have investigated the effect of a **standardized Cannabis sativa extract** with **high content** of **cannabidiol** (CBD), here named CBD BDS, i.e. CBD botanical drug substance, on colorectal cancer cell proliferation and in experimental models of **colon** cancer in vivo.

METHODS: Proliferation was evaluated in colorectal carcinoma (DLD-1 and HCT116) as well as in healthy colonic cells using the MTT assay. CBD BDS binding was evaluated by its ability to displace [(3)H]CP55940 from human cannabinoid CB1 and CB2 receptors. In vivo, the effect of CBD BDS was examined on the preneoplastic lesions (aberrant crypt foci), polyps and tumours induced by the carcinogenic agent azoxymethane (AOM) as well as in a xenograft model of **colon** cancer in mice.

RESULTS: CBD BDS and CBD reduced cell proliferation in tumoral, but not in healthy, cells. The effect of CBD BDS was counteracted by selective CB1 and CB2 receptor antagonists. Pure CBD reduced cell proliferation in a CB1-sensitive antagonist manner only. In binding assays, CBD BDS showed greater affinity than pure CBD for both CB1 and CB2 receptors, with pure CBD having very little affinity. In vivo, CBD BDS reduced AOM-induced preneoplastic lesions and polyps as well as tumour growth in the xenograft model of **colon** cancer.

CONCLUSIONS: CBD BDS attenuates **colon carcinogenesis** and inhibits colorectal cancer cell proliferation via CB1 and CB2 receptor activation. The results may have some clinical relevance for the use of **Cannabis**-based medicines in cancer patients.

KEYWORDS: ?(9)-Tetrahydrocannabinol; Cancer cell growth; **Cannabidiol**; Cannabinoid receptors; Chemoprevention; Colorectal cancer

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