CBG studies

CBG Clinical Trials:

New formulation is 120 mg CBD/90 mg CBG per drop.

Potential benefits: cancer, cancer appetite, IBD, neural inflammation, skin

Colon carcinogenesis is inhibited by the TRPM8 antagonist cannabigerol, a Cannabis-derived non-psychotropic cannabinoid

Francesca Borrelli [1], Ester Pagano [1], Barbara Romano [1], Stefania Panzera [1], Francesco Maiello [2], Diana Coppola [2], Luciano De Petrocellis [3], Lorena Buono [3], Pierangelo Orlando [4], Angelo A Izzo [5]

Affiliations + expand

PMID: 25269802 DOI: 10.1093/carcin/bgu205

Abstract

Cannabigerol (CBG) is a safe non-psychotropic Cannabis-derived cannabinoid (CB), which interacts with specific targets involved in carcinogenesis. Specifically, CBG potently blocks transient receptor potential (TRP) M8 (TRPM8), activates TRPA1, TRPV1 and TRPV2 channels, blocks 5-hydroxytryptamine receptor 1A (5-HT1A) receptors and inhibits the reuptake of endocannabinoids. Here, we investigated whether CBG protects against colon tumourigenesis. Cell growth was evaluated in colorectal cancer (CRC) cells using the 3-(4,5-dimethylthiazole-2-yl)-2,5-diphenyl tetrazolium bromide and 3-amino-7-dimethylamino-2-methylphenazine hydrochloride assays; apoptosis was examined by histology and by assessing caspase 3/7 activity; reactive oxygen species (ROS) production by a fluorescent probe; CB receptors, TRP and CCAAT/enhancer-binding protein homologous protein (CHOP) messenger RNA (mRNA) expression were quantified by reverse transcription-polymerase chain reaction; small hairpin RNA-vector silencing of TRPM8 was performed by electroporation. The in vivo antineoplastic effect of CBG was assessed using mouse models of colon cancer. CRC cells expressed TRPM8, CB1, CB2, 5-HT1A receptors, TRPA1, TRPV1 and TRPV2 mRNA. CBG promoted apoptosis, stimulated ROS production, upregulated CHOP mRNA and reduced cell growth in CRC cells. CBG effect on cell growth was independent from TRPA1, TRPV1 and TRPV2 channels activation, was further increased by a CB2 receptor antagonist, and mimicked by other TRPM8 channel blockers but not by a 5-HT1A antagonist. Furthermore, the effect of CBG on cell growth and on CHOP mRNA expression was reduced in TRPM8 silenced cells. In vivo, CBG inhibited the growth of xenograft tumours as well as chemically induced colon carcinogenesis. CBG hampers colon cancer progression in vivo and selectively inhibits the growth of CRC cells, an effect shared by other TRPM8 antagonists. CBG should be considered translationally in CRC prevention and cure.

CBG studies

> Biochem Pharmacol. 2013 May 1;85(9):1306-16. doi: 10.1016/j.bcp.2013.01.017.
Epub 2013 Feb 12.

Beneficial effect of the non-psychotropic plant cannabinoid cannabigerol on experimental inflammatory bowel disease

Francesca Borrelli [1], Ines Fasolino, Barbara Romano, Raffaele Capasso, Francesco Maiello, Diana Coppola, Pierangelo Orlando, Giovanni Battista, Ester Pagano, Vincenzo Di Marzo, Angelo A Izzo

Affiliations + expand

PMID: 23415610 DOI: 10.1016/j.bcp.2013.01.017

Abstract

Inflammatory bowel disease (IBD) is an incurable disease which affects millions of people in industrialized countries. Anecdotal and scientific evidence suggests that Cannabis use may have a positive impact in IBD patients. Here, we investigated the effect of cannabigerol (CBG), a non-psychotropic Cannabis-derived cannabinoid, in a murine model of colitis. Colitis was induced in mice by intracolonic administration of dinitrobenzene sulphonic acid (DNBS). Inflammation was assessed by evaluating inflammatory markers/parameters (colon weight/colon length ratio and myeloperoxidase activity), by histological analysis and immunohistochemistry; interleukin-1β, interleukin-10 and interferon-γ levels by ELISA, inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2) by western blot and RT-PCR; CuZn-superoxide dismutase (SOD) activity by a colorimetric assay. Murine macrophages and intestinal epithelial cells were used to evaluate the effect of CBG on nitric oxide production and oxidative stress, respectively. CBG reduced colon weight/colon length ratio, myeloperoxidase activity, and iNOS expression, increased SOD activity and normalized interleukin-1β, interleukin-10 and interferon-γ changes associated to DNBS administration. In macrophages, CBG reduced nitric oxide production and iNOS protein (but not mRNA) expression. Rimonabant (a CB1 receptor antagonist) did not change the effect of CBG on nitric oxide production, while SR144528 (a CB2 receptor antagonist) further increased the inhibitory effect of CBG on nitric oxide production. In conclusion, CBG attenuated murine colitis, reduced nitric oxide production in macrophages (effect being modulated by the CB2 receptor) and reduced ROS formation in intestinal epithelial cells. CBG could be considered for clinical experimentation in IBD patients.