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Cannabidiol, a safe and non-psychotropic ingredient of the marijuana plant Cannabis sativa, is protective in a murine model of colitis.

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Author information

Abstract

Inflammatory bowel disease affects millions of individuals; nevertheless, pharmacological treatment is disappointingly unsatisfactory. **Cannabidiol**, a **safe** and **non-psychotropic ingredient** of **marijuana**, exerts pharmacological effects (e.g., antioxidant) and mechanisms (e.g., inhibition of endocannabinoids enzymatic degradation) potentially beneficial for the inflamed gut. Thus, we investigated the effect of **cannabidiol** in a **murine model** of **colitis**. **Colitis** was induced in mice by intracolonic administration of dinitrobenzene sulfonic acid. Inflammation was assessed both macroscopically and histologically. In the inflamed colon, cyclooxygenase-2 and inducible nitric oxide synthase (iNOS) were evaluated by Western blot, interleukin-1beta and interleukin-10 by ELISA, and endocannabinoids by isotope dilution liquid chromatography-mass spectrometry. Human colon adenocarcinoma (Caco-2) cells were used to evaluate the effect of **cannabidiol** on oxidative stress. **Cannabidiol** reduced colon injury, inducible iNOS (but not cyclooxygenase-2) expression, and interleukin-1beta, interleukin-10, and endocannabinoid changes associated with 2,4,6-dinitrobenzene sulfonic acid administration. In Caco-2 cells, **cannabidiol** reduced reactive oxygen species production and lipid peroxidation. In conclusion, **cannabidiol**, a likely **safe** compound, prevents experimental **colitis** in mice.

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