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Br J Pharmacol. 2011 Aug;163(7):1507-19. doi: 10.1111/j.1476-5381.2011.0

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Cannabidiol inhibits pathogenic T cells, decreases spinal microglial activation and ameliorates multiple sclerosis-like disease in C57BL/6 mice.

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Author information

Abstract

BACKGROUND AND PURPOSE: Cannabis extracts and several cannabinoids have been shown to exert broad anti-inflammatory activities in experimental models of inflammatory CNS degenerative diseases. Clinical use of many cannabinoids is limited by their psychotropic effects. However, phytocannabinoids like cannabidiol (CBD), devoid of psychoactive activity, are, potentially, safe and effective alternatives for alleviating neuroinflammation and neurodegeneration.

EXPERIMENTAL APPROACH: We used experimental autoimmune encephalomyelitis (EAE) induced by myelin oligodendrocyte glycoprotein (MOG) in C57BL/6 mice, as a model of multiple sclerosis. Using immunocytochemistry and cell proliferation assays we evaluated the effects of CBD on microglial activation in MOG-immunized animals and on MOG-specific T-cell proliferation.

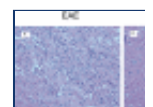
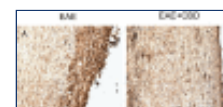
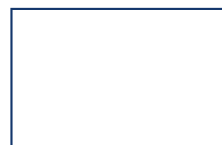
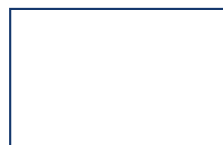
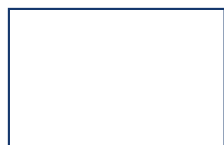
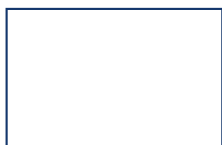
KEY RESULTS: Treatment with CBD during disease onset ameliorated the severity of the clinical signs of EAE. This effect of CBD was accompanied by diminished axonal damage and inflammation as well as microglial activation and T-cell recruitment in the spinal cord of MOG-injected mice. Moreover, CBD inhibited MOG-induced T-cell proliferation in vitro at both low and high concentrations of the myelin antigen. This effect was not mediated via the known cannabinoid CB(1) and CB(2) receptors.

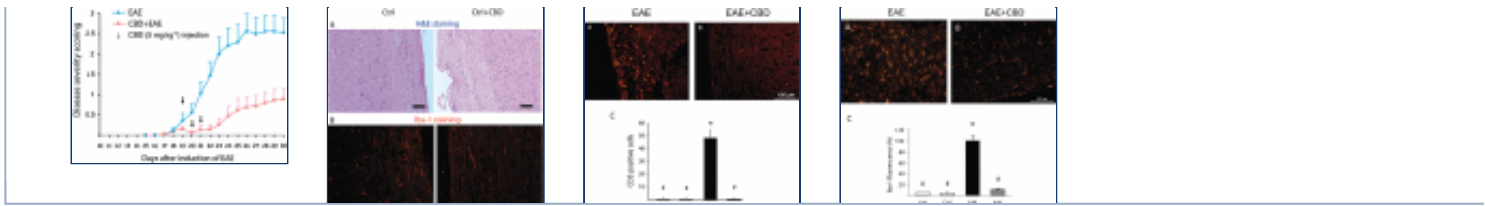
CONCLUSIONS AND IMPLICATIONS: CBD, a non-psychoactive cannabinoid, ameliorates clinical signs of EAE in mice, immunized against MOG. Suppression of microglial activity and T-cell proliferation by CBD appeared to contribute to these beneficial effects.

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