Hacia la utilización de cannabinoides como agentes antitumorales y otras historias de señalización inspiradas por el cannabis.

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Abstract

∆9-Tetrahydrocannabinol (THC) the main active ingredient of Cannabis sativa produces its biological effects in humans by mimicking the effects evoked by a family of endogenous lipid neuromodulators named endocannabinoids. These compounds engage two G protein-coupled cannabinoid receptors named CB1 and CB2. Cannabinoids are currently being investigated as potential therapeutic agents for the management of different diseases, including cancer. Thus, cannabinoids have been shown to exhibit anticancer activity in different animal models of cancer and specifically in gliomas. The mechanism by which THC promotes these anti-tumoral actions has been at least partially unraveled and relies on the stimulation of an ER stress-related signaling pathway that triggers the up-regulation of the protein Tribbles pseudokinase 3 and the subsequent stimulation of autophagy. Likewise, THC induces a modification in the sphingolipid composition of autophagosomes and autolysosomes that plays a crucial role in the regulation of autophagy-mediated cancer cell death. In this talk I will summarize some of these findings that have set the bases for the development of clinical studies in Glioma.

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