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Anti-tumoral action of cannabinoids: involvement of sustained ceramide accumulation and extracellular signal-regulated kinase activation.

Galve-Roperh I¹, Sánchez C, Cortés ML, Gómez del Pulgar T, Izquierdo M, Guzmán M.

Author information

Abstract

Delta9-Tetrahydrocannabinol, the main active component of marijuana, induces apoptosis of transformed neural cells in culture. Here, we show that intratumoral administration of Delta9-tetrahydrocannabinol and the synthetic cannabinoid agonist WIN-55,212-2 induced a considerable regression of malignant gliomas in Wistar rats and in mice deficient in recombination activating gene 2. Cannabinoid treatment did not produce any substantial neurotoxic effect in the conditions used. Experiments with two subclones of C6 glioma cells in culture showed that **cannabinoids** signal apoptosis by a pathway involving cannabinoid receptors, sustained ceramide accumulation and Raf1/extracellular signal-regulated kinase activation. These results may provide the basis for a new therapeutic approach for the treatment of malignant gliomas.

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