Cannabinoid receptors in GtoPdb v.2023.1

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Abstract

Cannabinoid receptors (nomenclature as agreed by the NC-IUPHAR Subcommittee on Cannabinoid Receptors [119]) are activated by endogenous ligands that include N-arachidonoylethanolamine (anandamide), N-homo-γ-linolenoylethanolamine, N-docosatetra-7,10,13,16-enoylethanolamine and 2-arachidonoylglycerol. Potency determinations of endogenous agonists at these receptors are complicated by the possibility of differential susceptibility of endogenous ligands to enzymatic conversion [5].

There are currently three licenced cannabinoid medicines each of which contains a compound that can activate CB₁ and CB₂ receptors [111]. Two of these medicines were developed to suppress nausea and vomiting produced by chemotherapy. These are nabilone (Cesamet®), a synthetic CB₁/CB₂ receptor agonist, and synthetic Δ⁹-tetrahydrocannabinol (Marinol®; dronabinol), which can also be used as an appetite stimulant. The third medicine, Sativex®, contains mainly Δ⁹-tetrahydrocannabinol and cannabidiol, both extracted from cannabis, and is used to treat multiple sclerosis and cancer pain.

Contents

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are given here under database links.

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Please note that the database version for the citations given in GtoPdb are to the most recent preceding version in which the family or its subfamilies and targets were substantially changed. The links below are to the current version. If you need to consult the cited version, rather than the most recent version, please contact the GtoPdb curators.

**Database links**

**Cannabinoid receptors**

https://www.guidetopharmacology.org/GRAC/FamilyDisplayForward?familyId=13

**Introduction to Cannabinoid receptors**

https://www.guidetopharmacology.org/GRAC/FamilyIntroductionForward?familyId=13

Receptors

- **CB₁ receptor**
  https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=56

- **CB₂ receptor**
  https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=57

**References**


155. Song ZH and Bonner TI. (1996) A lysine residue of the cannabinoid receptor is critical for receptor recognition by several agonists but not WIN55212-2. *Mol Pharmacol* **49**: 891-6 [PMID:8622639]


