BMS-986122

Cat. No.: HY-120645

CAS No.: 313669-88-4

Molecular Formula: $C_{16}H_{15}BrClNO_3S_2$

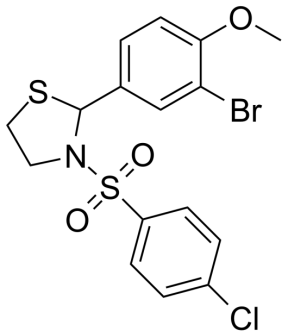
Molecular Weight: 448.78

Target: Opioid Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (222.83 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2283 mL	11.1413 mL	22.2826 mL
5 mM	0.4457 mL	2.2283 mL	4.4565 mL
10 mM	0.2228 mL	1.1141 mL	2.2283 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (5.57 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.57 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BMS-986122 is a selective, potent positive allosteric modulator of the mu-opioid receptor (μ-OR). BMS-986122 shows potentiation of orthosteric agonist-mediated β-arrestin recruitment, adenylyl cyclase inhibition, and G protein activation. BMS-986122 potentiates DAMGO-mediated [^{35}S]GTPγS binding in mouse brain membranes[1][2].

In Vitro

BMS-986122 increases β-arrestin recruitment stimulated by endomorphin 1 (EC_{50}=3 μM) in U2OS-OPRM1 human osteosarcoma cells expressing μ-opioid receptors. BMS-986122 potentiates endomorphin 1-induced inhibition of forskolin-stimulated adenylyl cyclase activity in CHO cells expressing human recombinant μ-opioid receptors (EC_{50}=8.9 μM). BMS-986122 potentiates DAMGO-mediated [35S]GTPγS binding in mouse brain membranes and appears to be, at least in part, a positive affinity modulator of the μ-opioid receptor for DAMGO binding[1].
BMS-986122 enhances the ability of the endogenous opioid Methionine-enkephalin (Met-Enk) to stimulate G protein activity

	in mouse brain homogenates without activity on its own and to enhance G protein activation to a greater extent than β-arrestin recruitment in CHO cells expressing human mu-opioid receptors. BMS-986122 increases the potency of Met-Enk to inhibit GABA release in the periaqueductal gray, an important site for antinociception[2]. BMS-986122 is selective for μ-OR and has no detectable activity at the closely related δ-OR. BMS-986122 is a silent allosteric modulator at δ-OR and κ-OR[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Burford NT, et al. Discovery of positive allosteric modulators and silent allosteric modulators of the μ-opioid receptor. Proc Natl Acad Sci U S A. 2013;110(26):10830-10835.

[2]. Kandasamy R, et al. Positive allosteric modulation of the mu-opioid receptor produces analgesia with reduced side effects. Proc Natl Acad Sci U S A. 2021;118(16):e2000017118.

[3]. Livingston KE, Alt A, Canals M, Traynor JR. Pharmacologic Evidence for a Putative Conserved Allosteric Site on Opioid Receptors. Mol Pharmacol. 2018;93(2):157-167.

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