LY2183240

Cat. No.: HY-10865

CAS No.: 874902-19-9

Molecular Formula: $C_{17}H_{17}N_5O$

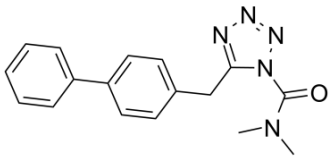
Molecular Weight: 307.35

Target: FAAH; Autophagy

Pathway: Metabolic Enzyme/Protease; Neuronal Signaling; Autophagy

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 : 50 mg/mL (162.68 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2536 mL	16.2681 mL	32.5362 mL
5 mM	0.6507 mL	3.2536 mL	6.5072 mL
10 mM	0.3254 mL	1.6268 mL	3.2536 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.13 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.13 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

LY2183240 is a highly potent blocker of anandamide uptake (IC_{50}= 270 pM; K_i=540 nM). LY2183240 is a potent, covalent inhibitor of the EC-degrading enzyme fatty acid amide hydrolase (FAAH) with an IC_{50} of 12.4 nM. LY2183240 inactivates FAAH by carbamylation of the enzyme's serine nucleophile. LY2183240 also inhibits several other brain serine hydrolases with IC_{50} s of 5.3, 0.09, 8.2 nM for MAG lipase, bh6 and KIAA1363, respectively[1][2] [3].

In Vivo

LY2183240 (3-30mg/kg; i.p.) dose-dependently attenuates formalin-induced paw-licking pain behavior in the formalin model of persistent pain mechanisms[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (Formalin Pain Model)[1]
Dosage:	3, 10, 30 mg/kg
Administration:	I.p.
Result:	Dose-dependently attenuated formalin-induced paw-licking pain behavior in the formalin model of persistent pain mechanisms.

CUSTOMER VALIDATION

- Eur J Pain. 2017 May;21(5):804-814.

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REFERENCES

[1]. Moore SA, et al. Identification of a high-affinity binding site involved in the transport of endocannabinoids. Proc Natl Acad Sci U S A. 2005;102(49):17852-17857.

[2]. Alexander JP, Cravatt BF. The putative endocannabinoid transport blocker LY2183240 is a potent inhibitor of FAAH and several other brain serine hydrolases. J Am Chem Soc. 2006 Aug 2;128(30):9699-704.

[3]. Maione S, et al. Antinociceptive effects of tetrazole inhibitors of endocannabinoid inactivation: cannabinoid and non-cannabinoid receptor-mediated mechanisms. Br J Pharmacol. 2008 Nov;155(5):775-82.

[4]. Pelorosso FG, et al. The endocannabinoid anandamide inhibits kinin B1 receptor sensitization through cannabinoid CB1 receptor stimulation in human umbilical vein. Eur J Pharmacol. 2009 Jan 5;602(1):176-9.

[5]. Powers MS, et al. Effects of the novel endocannabinoid uptake inhibitor, LY2183240, on fear-potentiated startle and alcohol-seeking behaviors in mice selectively bred for high alcohol preference. Psychopharmacology (Berl). 2010 Dec;212(4):571-83.

[6]. Sun L, et al. Endocannabinoid activation of CB1 receptors contributes to long-lasting reversal of neuropathic pain by repetitive spinal cord stimulation. Eur J Pain. 2017 May;21(5):804-814.

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