Lumiracoxib

Cat. No.: HY-13507

CAS No.: 220991-20-8

Molecular Formula: $C_{15}H_{13}ClFNO_2$

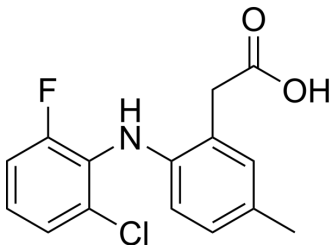
Molecular Weight: 293.72

Target: COX

Pathway: Immunology/Inflammation

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 : 125 mg/mL (425.58 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4046 mL	17.0230 mL	34.0460 mL
	5 mM	0.6809 mL	3.4046 mL	6.8092 mL
	10 mM	0.3405 mL	1.7023 mL	3.4046 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: ≥ 2.08 mg/mL (7.08 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (7.08 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (7.08 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Lumiracoxib is a potent,selective and orally active COX-2 inhibitor with a K_i value of 0.06 μM[1]. Lumiracoxib acts as a nonselective NSAID with anti-inflammatory, analgesic and antipyretic activities. Lumiracoxib can be used for osteoarthritis and bone cancer research[1][2].

IC_{50} & Target

COX-2	COX-1
0.06 μM (Ki)	3 μM (Ki)

In Vitro	Lumiracoxib inhibits purified COX-1 and COX-2 with K_i values of 3 μM and 0.06 μM, respectively. In cellular assays, Lumiracoxib has an IC_{50} of 0.14 μM in COX-2-expressing dermal fibroblasts, but causesno inhibition of COX-1 at concentrations up to 30 μM in HEK293 cells transfected with human COX-1[1]. In a human whole blood assay, IC_{50} values for Lumiracoxib are 0.13 μM for COX-2 and 67 μM for COX-1[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Lumiracoxib (oral administration; 10 and 30 mg/kg; single dose) significantly reverses the established hyperalgesia with a maximal 58% reversal observed 3 h following administration in rat model[1]. Lumiracoxib (oral administration; 10 and 30 mg/kg; twice daily; from day 10 to day 20 following MRMT-1 cell injection) significantly attenuates the weight-bearing difference observed on days 14, 17 and 20. The repeated administration significantly reverses static allodynia measured 90 min following the final administration.It significantly reduces the radiologically observed structural changes 20 days after inoculation of MRMT-1 cells in rat[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Rat model of bone cancer pain with injection of MRMT-1 tumour cells into one tibia[1]
Dosage:	10 and 30 mg/kg
Administration:	Oral administration; 10 and 30 mg/kg; twice daily; from day 10 to day 20 following MRMT-1 cell injection
Result:	Had an effect on mechanical hyperalgesia in a model of bone cancer pain.

REFERENCES

[1]. Ronald Esser, et al. Preclinical pharmacology of lumiracoxib: a novel selective inhibitor of cyclooxygenase-2. Br J Pharmacol. 2005 Feb;144(4):538-50.

[2]. Alyson Fox, et al. Anti-hyperalgesic activity of the cox-2 inhibitor lumiracoxib in a model of bone cancer pain in the rat. Pain

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA