Loxoprofen sodium

Cat. No.: HY-B0578A

CAS No.: 80382-23-6

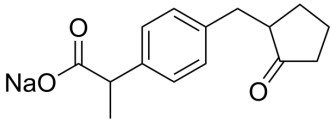
Molecular Formula: $C_{15}H_{17}NaO_3$

Molecular Weight: 268.28

Target: COX

Pathway: Immunology/Inflammation

Storage: 4°C, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (372.74 mM; Need ultrasonic)
DMSO : 19.23 mg/mL (71.68 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7274 mL	18.6372 mL	37.2745 mL
5 mM	0.7455 mL	3.7274 mL	7.4549 mL
10 mM	0.3727 mL	1.8637 mL	3.7274 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: ≥ 1.92 mg/mL (7.16 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.92 mg/mL (7.16 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.92 mg/mL (7.16 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Loxoprofen sodium is a non-steroidal, orally active anti-inflammatory agent with analgesic and anti-pyretic properties. Loxoprofen sodium is a nonselective COX inhibitor with IC_{50}s of 6.5 and 13.5 μM for COX-1 and COX-2, respectively. Loxoprofen sodium can reduce atherosclerosis and shows antitumor activity[1][2][3][4].

IC_{50} & Target

COX-1	COX-2
6.5 μM (IC_{50})	13.5 μM (IC_{50})

In Vitro

Loxoprofen sodium, an anti-inflammatory prodrug (NSAID), is a nonselective COX inhibitor with IC_{50}s of 6.5 and 13.5μM for

COX-1 and COX-2 in human whole blood assays, respectively[1].
Loxoprofen (LOX) sodium is a non-selective cyclooxygenase inhibitor that is widely used for the reasearch of pain and inflammation caused by chronic and transitory conditions. Its alcoholic metabolites are formed by carbonyl reductase (CR) and they consist of trans-LOX, which is active, and cis-LOX, which is inactive. In addition, LOX sodium can also be converted into an inactive hydroxylated metabolite (OH-LOXs) by cytochrome P450 (CYP)[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Loxoprofen sodium (4 mg/kg/day; p.o.; 1 or 8 weeks) reduces atherosclerosis in mice by reducing inflammation[3] .Loxoprofen sodium (60 μg/mL; p.o.; 24 days) suppresses mouse tumor growth by inhibiting VEGF[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	ApoE$^{-/-}$ mice (C57BL/6J-Apoe^{tm1Unc}) with high-fat diet (0.2% cholesterol, 21% saturated fat) from 8 to 16 weeks of age[3]
Dosage:	4 mg/kg/day in drinking water
Administration:	Oral dosing from 8 to 16 weeks of age or from 15 to 16 weeks of age
Result:	Inhibited platelet thromboxane production and platelet aggregation. Reduced extent of atherosclerosis. Suppressed the production of PGE_2, TxB_2 and PGI_2.

Animal Model:	6-week-old male C57BL/6 and BDF1 mice, 100 μL suspensions (2×10^6 cells/mL) of LLC cells and KLN205 cells were injected subcutaneously into C57BL/6 and BDF1 mice, respectively[4].
Dosage:	60 μg/mL
Administration:	Oral dosing in drinking water, every day for 24 days
Result:	Suppressed tumor growth and angiogenesis, suppressed expression of VEGF in mice with LLC tumor, inhibited tubular formation of HUVECs.

REFERENCES

[1]. Hamaguchi M, et al. Loxoprofen Sodium, a Non-Selective NSAID, Reduces Atherosclerosis in Mice by Reducing Inflammation. J Clin Biochem Nutr. 2010 Sep;47(2):138-47.

[2]. Kanda A, et al. Loxoprofen sodium suppresses mouse tumor growth by inhibiting vascular endothelial growth factor. Acta Oncol. 2003;42(1):62-70.

[3]. Riendeau D, et al. Evaluation of loxoprofen and its alcohol metabolites for potency and selectivity of inhibition of cyclooxygenase-2. Bioorg Med Chem Lett. 2004;14(5):1201-1203.

[4]. Paudel S, et al. Assessing Drug Interaction and Pharmacokinetics of Loxoprofen in Mice Treated with CYP3A Modulators. Pharmaceutics. 2019;11(9):479. Published 2019 Sep 16.

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