AT-56

Cat. No.: HY-13988

CAS No.: 162640-98-4

Molecular Formula: $C_{25}H_{27}N_5$

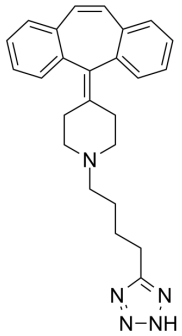
Molecular Weight: 397.52

Target: PGE synthase

Pathway: Immunology/Inflammation

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (251.56 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5156 mL	12.5780 mL	25.1560 mL
	5 mM	0.5031 mL	2.5156 mL	5.0312 mL
	10 mM	0.2516 mL	1.2578 mL	2.5156 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 (6.29 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.29 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.29 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

AT-56 is a potent, selective and orally active inhibitor of lipocalin-type prostaglandin D synthase (L-PGDS), with an IC_{50} of 95 μM and K_i of 75 μM. AT-56 could selectively suppress the drowsiness or pain reaction mediated by L-PGDS-catalyzed PGD_2[1].

IC_{50} & Target

IC50: 95 μM (L-PGDS); Ki: 75 μM (L-PGDS)[1]

In Vitro

AT-56 (1-30 μM; 10 minutes) dose-dependently inhibits the production of PGD_2 in L-PGDS-expressing human medulloblastoma TE-671 cells with an IC_{50} of about 3 μM[1].

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

In Vivo

AT-56 (1-30 mg/kg; p.o.) suppresses the PGD_2 production in the stab-wounded brain[1].
AT-56 (1-10 mg/kg; p.o.) suppresses the L-PGDS-mediated allergic airway inflammation in mice[1].
AT-56 (10 mg/kg; p.o.) exhibits C_{max} (2.15 μg/ml), half-life (1.71 h) and high oral bioavailability (82%)[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	H-PGDS KO mice (14-16weeks, 25-30 g, C57BL/6 strain) with a stab wound brain injury[1]
Dosage:	0, 1, 3, 10, 30 mg/kg
Administration:	P.o. 1 h before the stab wound injury
Result:	Inhibited the L-PGDS reaction in the brain. Decreased the total amount of PGD_2 in the brain to 40% with 30 mg/kg AT-56.

Animal Model:	Human L-PGDS-overexpressing TG mice (males, 14-16 weeks, 25-30 g)[1]
Dosage:	0, 1, 10 mg/kg
Administration:	P.o. 1 h before and 24 h after the antigen exposure
Result:	Prevented the eosinophil infiltration by inhibiting transgened human L-PGDS.

Animal Model:	Male C57BL/6 mice (7 weeks, 22-26 g)[1]
Dosage:	10 mg/kg for p.o. and 2 mg/kg for i.v. (Pharmacokinetic Analysis)
Administration:	P.o. and i.v. administration
Result:	Oral bioavailability (82%); C_{max} (2.15 μg/ml); $T_{1/2}$ (1.71 h, p.o.); $T_{1/2}$ (2.35 h, i.v.).

REFERENCES

[1]. Irikura D, et, al. Biochemical, functional, and pharmacological characterization of AT-56, an orally active and selective inhibitor of lipocalin-type prostaglandin D synthase. J Biol Chem. 2009 Mar 20; 284(12): 7623-30.

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