Lidorestat

Cat. No.: HY-106198

CAS No.: 245116-90-9

Molecular Formula: $C_{18}H_{11}F_3N_2O_2S$

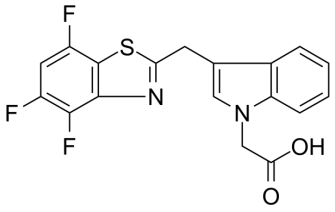
Molecular Weight: 376.35

Target: Aldose Reductase

Pathway: Metabolic Enzyme/Protease

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 (132.86 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6571 mL	13.2855 mL	26.5710 mL
	5 mM	0.5314 mL	2.6571 mL	5.3142 mL
	10 mM	0.2657 mL	1.3286 mL	2.6571 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: ≥ 2.08 mg/mL (5.53 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 0.56 mg/mL (1.49 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.56 mg/mL (1.49 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Lidorestat (IDD-676) is a potent, selective and orally active aldose reductase inhibitor with an IC_{50} of 5 nM. Lidorestat can be used for chronic diabetes complications. Lidorestat also improves nerve conduction and reduces cataract formation[1][2][3].

IC_{50} & Target

IC50: 5 nM (Rldose reductase)[1]

In Vitro

From in vitro experiments, Lidorestat has a reported IC_{50} against recombinant human aldose reductase (/h/-ALR2) of 5 μM. Against recombinant human aldehyde reductase (/h/-ALR1), Lidorestat has a reported IC_{50} of 27,000 μM, yielding a

	selectivity of /h/-ALR1//h/-ALR2 of 5400:1[1][2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Lidorestat (25 mg/kg/day; oral administration; twice daily; for 6 weeks; diabetic mice) treatment decreases fructose and reduces mortality in diabetic hAR-expressing mice. And Lidorestat does not affect weight[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Diabetic low-density lipoprotein (LDL) receptor-deficient [Ldlr(-/-)] mice[1]
Dosage:	25 mg/kg/day
Administration:	Oral administration; twice daily; for 6 weeks
Result:	Diabetic hAR-expressing mice had decreased fructose and reduced mortality.

REFERENCES

[1]. Noh HL, et al. Regulation of plasma fructose and mortality in mice by the aldose reductase inhibitor lidorestat. J Pharmacol Exp Ther. 2009 Feb;328(2):496-503.

[2]. Van Zandt MC, et al. Discovery of 3-[(4,5,7-trifluorobenzothiazol-2-yl)methyl]indole-N-acetic acid (lidorestat) and congeners as highly potent and selective inhibitors of aldose reductase for treatment of chronic diabetic complications. J Med Chem. 2005 May 5;48(9):3141-52.

[3]. Maccari R, et al. Identification of new non-carboxylic acid containing inhibitors of aldose reductase. Bioorg Med Chem. 2010 Jun 1;18(11):4049-55.

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