Cilostazol

Cat. No.: HY-17464

CAS No.: 73963-72-1

Molecular Formula: $C_{20}H_{27}N_5O_2$

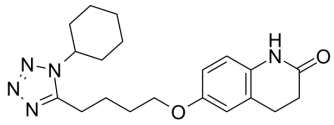
Molecular Weight: 369.46

Target: Phosphodiesterase (PDE); Autophagy

Pathway: Metabolic Enzyme/Protease; 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 (135.33 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7067 mL	13.5333 mL	27.0665 mL
5 mM	0.5413 mL	2.7067 mL	5.4133 mL
10 mM	0.2707 mL	1.3533 mL	2.7067 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 mg/mL (5.41 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2 mg/mL (5.41 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cilostazol (OPC 13013) is a potent and selective inhibitor of phosphodiesterase (PDE) 3A, the isoform of PDE 3 in the cardiovascular system, with an IC_{50} of 0.2 μM[1][2].

IC_{50} & Target

IC50: 0.2 μM (PDE 3A)[1]

In Vitro

Cilostazol selectively inhibits cGMP-inhibited phosphodiesterase (PDE 3) and is a potent inhibitor of platelet aggregation induced by various agonists[2].

Cilostazol inhibits stress-induced human platelet aggregation (SIPA) dose-dependently, with an IC_{50} of 15 μM for SIPA, and with a similar IC_{50} of 12.5 μM for ADP-induced platelet aggregation[2].

Cilostazol directly and effectively inhibits the activation of HSC but not of Kupffer cells[3].

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

In Vivo

Cilostazol (clinically used doses; p.o.; for 2 weeks) could alleviate CCl4 -induced hepatic fibrogenesis in vivo, presumably due to its direct effect to suppress HSC activation[3].

Cilostazol (intraperitoneal injection; 10 mg/kg; 7 consecutive days after ischemia) attenuates neurological dysfunctions, brain atrophy and infarct volume, and inhibits astrocyte proliferation/glial scar formation and accelerated the angiogenesis in the ischemic boundary zone 7 and 28 days after ischemia[4].

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

Animal Model:	Male C57BL/6J mice[3]
Dosage:	0.1% w/w, 0.3% w/w
Administration:	Oral administration; fed a normal diet for 2 weeks
Result:	Exhibited a lesser fibrotic area than control groups.

Animal Model:	Male ICR mice[4]
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection; 7 consecutive days after ischemia
Result:	Had an effectve effects for the late injury.

CUSTOMER VALIDATION

- Cephalalgia. 2021 Aug 18;3331024211038884.
- Cardiovasc Eng Technol. 2019 Dec;10(4):638-647.

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REFERENCES

[1]. Schr?r K. The pharmacology of cilostazol. Diabetes Obes Metab. 2002 Mar;4 Suppl 2:S14-9.

[2]. Minami N, et al. Inhibition of shear stress-induced platelet aggregation by cilostazol, a specific inhibitor of cGMP-inhibited phosphodiesterase, in vitro and ex vivo. Life Sci. 1997;61(25):PL 383-9.

[3]. Saito S, et al. Cilostazol attenuates hepatic stellate cell activation and protects mice against carbon tetrachloride-induced liver fibrosis. Hepatol Res. 2013 Apr 19.

[4]. Ye YL,et al. Cilostazol, a phosphodiesterase 3 inhibitor, protects mice against acute and late ischemic brain injuries.Eur J Pharmacol. 2007 Feb 14;557(1):23-31. Epub 2006 Nov 10.

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