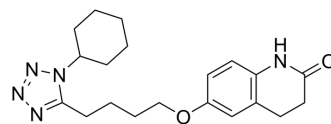


Cilostazol

Cat. No.:	HY-17464		
CAS No.:	73963-72-1		
Molecular Formula:	C ₂₀ H ₂₇ N ₅ O 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	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro	DMSO : 50 mg/mL (135.33 mM; Need ultrasonic)					
		Solvent Concentration	Mass	1 mg	5 mg	10 mg
	Preparing Stock Solutions	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	<ol style="list-style-type: none"> 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 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	IC ₅₀ : 0.2 μM (PDE 3A) ^[1]
In Vitro	<p>Cilostazol selectively inhibits cGMP-inhibited phosphodiesterase (PDE 3) and is a potent inhibitor of platelet aggregation induced by various agonists^[2].</p> <p>?Cilostazol inhibits stress-induced human platelet aggregation (SIPA) dose-dependently,? with an IC₅₀ of 15 μM for SIPA, and with a similar IC₅₀ of 12.5 μM for ADP-induced platelet aggregation^[2].</p> <p>?Cilostazol directly and effectively inhibits the activation of HSC but not of Kupffer cells^[3].</p>

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 effective effects for the late injury.

CUSTOMER VALIDATION

- Cephalalgia. 2021 Aug 18;3331024211038884.
- Cardiovasc Eng Technol. 2019 Dec;10(4):638-647.
- Patent. US20230111925A1.

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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.

Caution: Product has not been fully validated for medical applications. For research use only.

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