Prasugrel

Cat. No.: HY-15284

CAS No.: 150322-43-3

Molecular Formula: $C_{20}H_{20}FNO_3S$

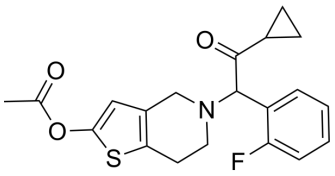
Molecular Weight: 373.44

Target: P2Y Receptor

Pathway: GPCR/G Protein

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 : 100 mg/mL (267.78 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6778 mL	13.3890 mL	26.7781 mL
5 mM	0.5356 mL	2.6778 mL	5.3556 mL
10 mM	0.2678 mL	1.3389 mL	2.6778 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 (6.69 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.69 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.69 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Prasugrel (PCR 4099), a thienopyridine and proagent, inhibits platelet function. Prasugrel is an orally active and potent P2Y12 receptor antagonist, and inhibits ADP-induced platelet aggregation[1].

IC_{50} & Target

P2Y12 Receptor

In Vivo

In rat platelets, Prasugrel active metabolite inhibits in vitro platelet aggregation induced by adenosine ADP (10μM) with an

IC_{50} value of 1.8 μM[2].

Prasugrel acts faster and is significantly more potent than Clopidogrel in vivo. Prasugrel is an inactive prodrug that requires metabolic processing in vivo to generate the active antiplatelet metabolite. Prasugrel is rapidly absorbed from the gut. After oral administration of standard-loading doses of 60 mg, maximum plasma levels of the active metabolite are achieved within 1 h, effective, maximum inhibition of platelet aggregation at 1-2 h[1].

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

REFERENCES

[1]. Wijeyeratne YD, et al. Anti-platelet therapy: ADP receptor antagonists. Br J Clin Pharmacol. 2011 Oct;72(4):647-57.

[2]. Sugidachi A, et al. The greater in vivo antiplatelet effects of prasugrel as compared to clopidogrel reflect more efficient generation of its active metabolite with similar antiplatelet activity to that of clopidogrel's active metabolite. J Thromb Haemost. 2007 Jul;5(7):1545-51.

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