Prasugrel hydrochloride

Cat. No.: HY-15284A

CAS No.: 389574-19-0

Molecular Formula: $C_{20}H_{21}ClFNO_3S$

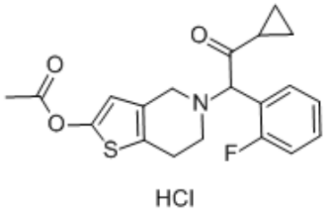
Molecular Weight: 409.9

Target: P2Y Receptor

Pathway: GPCR/G Protein

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 41.67 mg/mL (101.66 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4396 mL	12.1981 mL	24.3962 mL
	5 mM	0.4879 mL	2.4396 mL	4.8792 mL
	10 mM	0.2440 mL	1.2198 mL	2.4396 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: ≥ 2.08 mg/mL (5.07 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.07 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.07 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

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

IC_{50} & Target

P2Y12 receptor[1]

In Vivo

In rat platelets, Prasugrel hydrochloride active metabolite inhibits in vitro platelet aggregation induced by adenosine ADP (10μM) with an IC_{50} value of 1.8 μM[2].
Prasugrel hydrochloride acts faster and is significantly more potent than Clopidogrel in vivo. Prasugrel hydrochloride is an inactive prodrug that requires metabolic processing in vivo to generate the active antiplatelet metabolite. Prasugrel

hydrochloride 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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