Clopidogrel hydrogen sulfate

Cat. No.: HY-17459

CAS No.: 120202-66-6

Molecular Formula: $C_{16}H_{18}ClNO_6S_2$

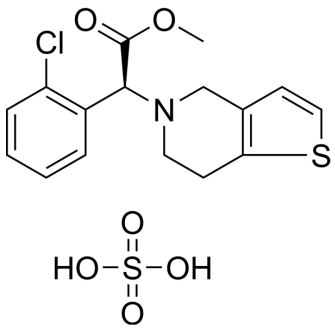
Molecular Weight: 420

Target: P2Y Receptor; Cytochrome P450

Pathway: GPCR/G Protein; Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 46.7 mg/mL (111.19 mM)

H_2O : 16.67 mg/mL (39.69 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3810 mL	11.9048 mL	23.8095 mL
5 mM	0.4762 mL	2.3810 mL	4.7619 mL
10 mM	0.2381 mL	1.1905 mL	2.3810 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 50 mg/mL (119.05 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.95 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Clopidogrel hydrogen sulfate is an antiplatelet agent to prevent blood clots. Clopidogrel hydrogen sulfate inhibits CYP2B6 and CYP2C19 with IC_{50}s of 18.2 nM and 524 nM, respectively[1]. Clopidogrel hydrogen sulfate is a potent antithrombotic agent that inhibits ADP-induced platelet aggregation[2]. Clopidogrel hydrogen sulfate also is an orally active P2Y(12) inhibitor[5].

IC_{50} & Target			
	CYP2B6 18.2 nM (IC_{50})	P2Y12 Receptor	CYP2C19 524 nM (IC_{50})

In Vitro

Clopidogrel (1.5 mM;12 and 24 hours) up-regulates the expression of TRIB3 and CHOP in a concentration- and time-dependent manner, as measured by real-time PCR and Western blot analysis[3].
Clopidogrel (1.5 mM; 24 hours) significantly induces human gastric epithelial cell (GES-1) apoptosis[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[3]

Cell Line:	GES-1 cells
Concentration:	1.5 mM
Incubation Time:	12 and 24 hours
Result:	The mRNA expression levels of both CHOP and TRIB3 were up-regulated in a concentration- and time-dependent manner. The protein expression levels of both CHOP and TRIB3 were up-regulated in a concentration- and time-dependent manner.

Apoptosis Analysis[3]

Cell Line:	Gastric epithelial cell (GES-1) cells
Concentration:	1.5 mM
Incubation Time:	24 hours
Result:	Induced apoptosis of gastric epithelial cells

In Vivo

Clopidogrel (5 mg/kg) significantly inhibits thrombus formation compared with vehicle. Acetylsalicylic acid (0.15 mg/kg) can add to the antithrombotic effect of Clopidogrel in mice; Acetylsalicylic acid (0.6 mg/kg) blunts the antithrombotic effect of Clopidogrel[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Age-matched C57BL/6J male mice weighing at least 25 g were used at 8 to 12 weeks of age [4]
Dosage:	5 mg/kg
Administration:	Gavage treatment
Result:	Clopidogrel significantly inhibited thrombus formation compared with vehicle. When Clopidogrel was given in combination with 0.6 mg/kg ASA, thrombi were significantly larger than those measured with Clopidogrel alone. In contrast, when Clopidogrel was given in combination with 0.15 mg/kg ASA , thrombi were significantly smaller than those in mice treated with Clopidogrel and 0.6 mg/kg ASA, and smaller than those in mice given Clopidogrel alone.

CUSTOMER VALIDATION

- ACS Nano. 2023 Mar 27.
- Theranostics. 2023; 13(6):2040-2056.
- Int J Biol Sci. 2019 Jan 1;15(1):239-252.
- Thromb Res. 2023 May 8.

- Front Pharmacol. 2022 Jan 10;12:792263.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Katsunobu Hagihara, et al. Comparison of human cytochrome P450 inhibition by the thienopyridines prasugrel, Clopidogrel, and ticlopidine. Drug Metab Pharmacokinet. 2008;23(6):412-20.

[2]. Jean-Marc Herbert, et al. P2Y12, a new platelet ADP receptor, target of Clopidogrel.Semin Vasc Med. 2003 May;3(2):113-22.

[3]. Hai-Lu Wu, et al. Increased endoplasmic reticulum stress response is involved in Clopidogrel-induced apoptosis of gastric epithelial cells. PLoS One. 2013 Sep 13;8(9):e74381.

[4]. Ran Ni, et al. Effect of Different Doses of Acetylsalicylic Acid on the Antithrombotic Activity of Clopidogrel in a Mouse Arterial Thrombosis Model.Arterioscler Thromb Vasc Biol. 2018 Oct;38(10):2338-2344.

[5]. Kuszynski DS, et al. Clopidogrel treatment inhibits P2Y2-Mediated constriction in the rabbit middle cerebral artery [published online ahead of print, 2021 Oct 1]. Eur J Pharmacol. 2021;174545.

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