Clopidogrel

Cat. No.: HY-15283

CAS No.: 113665-84-2

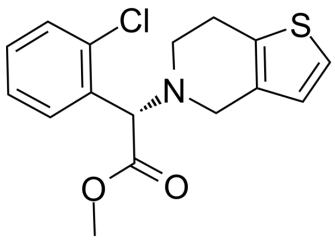
Molecular Formula: $C_{16}H_{16}ClNO_2S$

Molecular Weight: 322

Target: P2Y Receptor

Pathway: GPCR/G Protein

Storage: -20°C, stored under nitrogen, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (155.28 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1056 mL	15.5280 mL	31.0559 mL
	5 mM	0.6211 mL	3.1056 mL	6.2112 mL
	10 mM	0.3106 mL	1.5528 mL	3.1056 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 (7.76 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.76 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (7.76 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Clopidogrel is an orally active platelet inhibitor that targets P2Y12 receptor. Clopidogrel is used to inhibit blood clots in coronary artery disease, peripheral vascular disease, and cerebrovascular disease.

IC_{50} & Target

P2Y12 Receptor

In Vivo

Clopidogrel, administered during the last three months, significantly decreases blood glucose, collagen and fibronectin expression compared to vehicle-treated diabetic mice. Clopidogrel markedly ameliorates hyperglycemia-induced renal fibrosis[1]. The combination therapy of clopidogrel and aspirin (dual-antiplatelet therapy) has been shown to be significantly

beneficial compared to aspirin monotherapy and has also shown to decrease sub-acute stent thrombosis as well as recurrent ischemic events following ACS[2].

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

PROTOCOL

Animal Administration [1]

Mice[1]

13-week-old C57BL/6J male mice are used throughout the study. After 1 week of acclimation, 15 mice are injected I.P. with streptozotocin (STZ) at a dosage of 55 mg/kg body weight daily for five consecutive days. Additional 15 mice as controls (Ctrl) are injected with a vehicle solution (0.1 mol/L citrate acid buffer, pH 4.3-4.5). Seven days after the last STZ administration, hyperglycemic mice (3-hour fasting blood glucose ≥250 mg/dL) are considered T1D (DM). This time point is defined as a baseline. Three months after diabetes induction, five diabetic and five control mice are sacrificed and blood and kidneys harvested. The remaining animals are divided in four groups: Normal control with vehicle (Ctrl), Normal control with Clopidogrel (Ctrl+ Clo), T1D (DM) with vehicle, and DM with Clopidogrel treatment (DM+Clo) and are treated with 20 mg/kg b.w./day Clopidogrel or with vehicle administered in their drinking water for three additional months. At the end of experiment, mice are intraperitoneally anesthetized with Avertin (tribromoethanol, 350 mg/kg) and sacrificed to collect blood and kidneys for mRNA, protein, and histological analyses[1].

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

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.

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REFERENCES

[1]. Zongyu Zheng, et al. Clopidogrel Reduces Fibronectin Accumulation and Improves Diabetes-Induced Renal Fibrosis. Int J Biol Sci. 2019 Jan.

[2]. An insight into the interaction between clopidogrel and proton pump inhibitors By Shah, Bhavik S.; Parmar, Sanjay A.; Mahajan, Shailaja; Mehta, Anita A. From Current Drug Metabolism (2012), 13(2),225-235.

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