Paroxetine hydrochloride

Cat. No.: HY-B0492

CAS No.: 78246-49-8

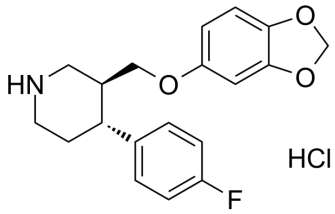
Molecular Formula: $C_{19}H_{21}ClFNO_3$

Molecular Weight: 365.83

Target: Serotonin Transporter; Autophagy

Pathway: Neuronal Signaling; Autophagy

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 1 year; -20°C, 6 months (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (273.35 mM; Need ultrasonic)
H_2O : 5 mg/mL (13.67 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7335 mL	13.6676 mL	27.3351 mL
5 mM	0.5467 mL	2.7335 mL	5.4670 mL
10 mM	0.2734 mL	1.3668 mL	2.7335 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 (6.83 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.83 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.83 mM); Clear solution
4. Add each solvent one by one: PBS
   Solubility: 2.03 mg/mL (5.55 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Paroxetine hydrochloride is a potent selective serotonin-reuptake inhibitor, commonly prescribed as an GRK2 inhibitor with IC_{50} of 14 μM. Paroxetine hydrochloride can be used for the research of depressive disorder[1][2][3].

IC_{50} & Target

IC50: 14 μM (GRK2)[3]

In Vitro

Paroxetine (1 μM and 10 μM) distinctly restrains T cell migration induced by CX3CL1 through inhibiting GRK2. Paroxetine

	inhibits GRK2 induced activation of ERK[1]. Paroxetine (10 μM) reduces pro-inflammatory cytokines in LPS-stimulated BV2 cells. Paroxetine (0-5 μM) leads to a dose-dependent inhibition on LPS-induced production of TNF-α and IL-1β in BV2 cells. Paroxetine also inhibits lipopolysaccharide (LPS)-induced nitric oxide (NO) production and inducible nitric oxide synthase (iNOS) expression in BV2 cells. Paroxetine (5 μM) blocks LPS-induced JNK activation and attenuates baseline ERK1/2 activity in BV2 cells. Paroxetine relieves microglia-mediated neurotoxicity, and suppresses LPS-stimulated pro-inflammatory cytokines and NO in primary microglial cells[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Paroxetine treatment obviously attenuates the symptoms of CIA rats. Paroxetine treatment clearly prevents the histological damage of joints and alleviates T cells infiltration into synovial tissue. Paroxetine hydrochloride reveals a strong effect on inhibiting CX3CL1 production in synovial tissues[1]. Paroxetine hydrochloride (20 mg/kg/day) reduces the myocyte cross-sectional area in rat and ROS formation in the remote myocardium. Paroxetine reduces the susceptibility to ventricular tachycardia. Paroxetine treatment following MI decreases LV remodeling and susceptibility to arrhythmias, probably by reducing ROS formation[2]. In CCI paroxetine-treated group, paroxetine (10 mg/kg, i.p.) produces hyperalgesia at days 7 and 10 ($P<0.01$), but a decrease in pain behavior is seen at day 14. Moreover, paroxetine (10 mg/kg) significantly attenuates tactile hypersensitivity when compared to CCI vehicle-treated group[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [4]	Cell viability is determined by the tetrazolium salt 3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide (MTT) assay. BV2 and primary microglial cells are initially seeded into 96-well plates at a density of 1×10^4 cells/well and 5×10^4 cells/well, respectively. Following treatment, MTT (5 mg/mL in PBS) is added to each well and incubated at 37°C for four hours. The resulting formazan crystals are dissolved in dimethylsulfoxide (DMSO). The optical density is measured at 570 nm, and results are expressed as a percentage of surviving cells compared with the control. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [5]	Animals are divided into two main groups: 1) pre-emptive and 2) post-injury group. Each main group is divided into three different subgroups: I) CCI vehicle-treated group, II) sham group, and III) CCI paroxetine-treated group. Vehicle is injected i.p. to CCI and sham-operated animals. In the pre-emptive study, paroxetine (10 mg/kg) is injected 1 h before surgery and continued daily until day 14 post surgery. In the post-injury group, paroxetine (10 mg/kg) is administered at day 7 post injury and continued daily until day 14. All behavioral tests are recorded on day 0 (control day) before surgery and on days 1, 3, 5, 7, 10, and 14 post-nerve injury. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell. 2021 Apr 15;184(8):2167-2182.e22.
- Front Pharmacol. 22 June 2022.
- J Chem Inf Model. 2021 Jul 21.
- Brain Res. 2019 Oct 1;1720:146296.

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REFERENCES

[1]. Wang Q, et al. Paroxetine alleviates T lymphocyte activation and infiltration to joints of collagen-induced arthritis. Sci Rep. 2017 Mar 28;7:45364.

[2]. Lassen TR, et al. Effect of paroxetine on left ventricular remodeling in an in vivo rat model of myocardial infarction. Basic Res Cardiol. 2017 May;112(3):26.

[3]. Waldschmidt HV, et al. Structure-Based Design of Highly Selective and Potent G Protein-Coupled Receptor Kinase 2 Inhibitors Based on Paroxetine. J Med Chem. 2017 Apr 13;60(7):3052-3069.

[4]. Liu RP, et al. Paroxetine ameliorates lipopolysaccharide-induced microglia activation via differential regulation of MAPK signaling. J Neuroinflammation. 2014 Mar 12;11:47.

[5]. Zarei M, et al. Paroxetine attenuates the development and existing pain in a rat model of neurophatic pain. Iran Biomed J. 2014;18(2):94-100.

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