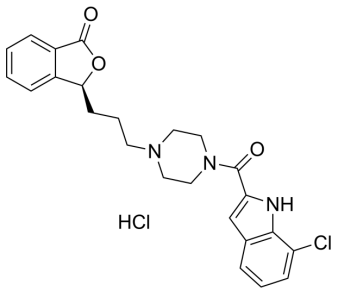
GluN2B-NMDAR antagonist-2

Cat. No.:	HY-157936
Molecular Formula:	$C_{24}H_{25}Cl_2N_3O_3$
Molecular Weight:	474.38
Target:	iGluR
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

GluN2B-NMDAR antagonist-2 (compound S-58) is a potent, selective and cross the blood-brain barrier NMDAR-GluN2B antagonist with an IC_{50} value of 74.01, nM. GluN2B-NMDAR antagonist-2 shows mild cytotoxicity. GluN2B-NMDAR antagonist-2 decreases the cerebral infarction rates and neurologic deficit scores. GluN2B-NMDAR antagonist-2 has the potential for the research of stroke[1].

IC_{50} & Target

GluN2B
74.01 nM (IC_{50})

In Vitro

GluN2B-NMDAR antagonist-2 (compound S-58) (1, 3, 10 μM) shows no significant prolonging effect on the action potential duration (APD90) in hiPSC-CMs[1].
GluN2B-NMDAR antagonist-2 (0-300 μM; 48 h) shows mild cytotoxicity for primary mouse neuron, VERO, L929, HEK293 cells [1].

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

Cell Cytotoxicity Assay[1]

Cell Line:	primary mouse neuron, VERO, L929, HEK293 cells
Concentration:	0, 0.3, 1, 3, 10, 100, 300 μM
Incubation Time:	48 h
Result:	Showed mild cytotoxicity for primary mouse neuron, VERO, L929, HEK293 cells with IC_{50}s of >300, 109.66, 56.34, 22.01 μM, respectively.

In Vivo

GluN2B-NMDAR antagonist-2 (5, 10, 20 mg/kg; i.v.) decreases the cerebral infarction rates and neurologic deficit scores in a dose dependent manner in MCAO rat model[1].
GluN2B-NMDAR antagonist-2 (450, 900 mg/kg; i.v.) shows a good safety profile with maximum tolerated dose (MTD) of 450 mg/kg in ICR mice[1].

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

Animal Model:	Fifty-six Sprague-Dawley rats (MCAO rat model)[1]

Dosage:	5, 10, 20 mg/kg
Administration:	I.v.
Result:	Significantly decreased the cerebral infarction rates and neurologic deficit scores in a dose dependent manner, dramatically improved motor function, decreased cerebral infarct volume, and reduced neurologic scores, significantly reduced the Ca^{2+} concentration in brain tissue.

REFERENCES

[1]. Lin G, et al. Design, Synthesis, and Biological Evaluation of Pierardine Derivatives as Novel Brain-Penetrant and In Vivo Potent NMDAR-GluN2B Antagonists for Ischemic Stroke Treatment. J Med Chem. 2024 Feb 27.

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