GluN2B-NMDAR antagonist-1

Cat. No.: HY-149967

Molecular Formula: $C_{26}H_{23}BrN_2O_2$

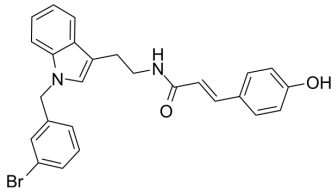
Molecular Weight: 475.38

Target: iGluR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (210.36 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1036 mL	10.5179 mL	21.0358 mL
	5 mM	0.4207 mL	2.1036 mL	4.2072 mL
	10 mM	0.2104 mL	1.0518 mL	2.1036 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 (5.26 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (5.26 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

GluN2B-NMDAR antagonist-1 is an orally active GluN2B-NMDAR antagonist. GluN2B-NMDAR antagonist-1 has neuroprotective activity. GluN2B-NMDAR antagonist-1 can be used for research of ischemic injury[1].

In Vitro

GluN2B-NMDAR antagonist-1 (Compound Z25) (0.05 μM, 0.5 μM, 5 μM) shows neuroprotection percentage of 35.7%, 48.8%, 55.8% against NMDA-induced cell injury in SH-SY5Y cells[1].

GluN2B-NMDAR antagonist-1 (5 μM) reduces Ca^{2+} influx in SH-SY5Y cells induced by NMDA (500μM)[1].

GluN2B-NMDAR antagonist-1 (0.05-5 μM, 6 h) increases NMDA-induced down-regulation of p-ERK1/2 expression in SH-SY5Y cells[1].

GluN2B-NMDAR antagonist-1 shows favorable plasma stability, with a half-life value greater than 289.1 min[1].

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

Western Blot Analysis[1]

Cell Line:	SH-SY5Y cells
Concentration:	0.05, 0.5, 5 μM
Incubation Time:	6 h
Result:	Increased NMDA-induced down-regulation of p-ERK1/2 expression, and reached the same level as Ifenprodil at 0.5 μM.

In Vivo

GluN2B-NMDAR antagonist-1 (Compound Z25) (20-80 mg/kg, intragastric administration) improves cognitive ability in ICV-ET1-induced vascular dementia mice model[1].

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

Animal Model:	ICV-ET1-induced vascular dementia mice model[1]
Dosage:	20, 40, and 80 mg/kg
Administration:	Intragastric administration, daily.
Result:	Decreased escape latency and swimming distance.

Animal Model:	Mouse (PK Assay)[1]
Dosage:	i.v. (1 mg/kg) and p.o. (10 mg/kg)
Administration:	i.v., p.o.
Result:	Pharmacokinetic profile of Nemvaleukin alfa.

dose (mg/kg)	$T_{1/2}$ (h)	C_{max} (ng/mL)	Cl (mL/min/kg)	F%
p.o. (10 mg/kg)	1.11	181.7		3.12
i.v. (1 mg/kg)	0.67	1913	20.45	

REFERENCES

[1]. Quan J,et al. Discovery of novel tryptamine derivatives as GluN2B subunit-containing NMDA receptor antagonists via pharmacophore-merging strategy with orally available therapeutic effect of cerebral ischemia. Eur J Med Chem. 2023 May 5;253:115318.

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