Nelonemdaz

Cat. No.: HY-106408

CAS No.: 640290-67-1

Molecular Formula: $C_{15}H_8F_7NO_3$

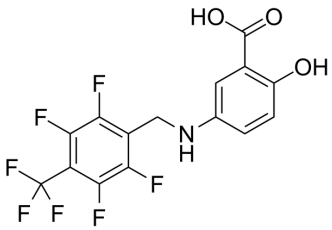
Molecular Weight: 383.22

Target: iGluR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 112.5 mg/mL (293.57 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6095 mL	13.0473 mL	26.0947 mL
5 mM	0.5219 mL	2.6095 mL	5.2189 mL
10 mM	0.2609 mL	1.3047 mL	2.6095 mL

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

BIOLOGICAL ACTIVITY

Description

Nelonemdaz (Salfaprodil free base) is an NR2B-selective and uncompetitive antagonist of N-methyl-D-aspartate (NMDA). Nelonemdaz is also a free radical scavenger. Nelonemdaz has excellent neuroprotection against NMDA- and free radical-induced cell death[1][2].

IC_{50} & Target

NMDA receptor[1]

In Vitro

Nelonemdaz (10-300 μM) shows apparent neuroprotection against 300 μM N-methyl-d-aspartate (NMDA) at doses as low as 30 μM[1].

Nelonemdazl (10-500 μM) inhibits the electrophysiologic response of cultured cortical neurons to 300 μM NMDA in a concentration-dependent manner[1].

Nelonemdaz (0.1-1 μM) produces a marked reduction of Fe^{2+}-induced neurotoxicity, even at doses of 0.1 to 0.3 μM[1].

Nelonemdaz (0.1-1 μM) blocks the degeneration of neurons and glia in cortical cell cultures[1].

Nelonemdaz (0-350 μM) effectively scavenges superoxide radicals (IC_{50}=63.07±1.44 μM), nitric oxide (IC_{50}=155.8±4.88 μM), and hydroxyl radicals (IC_{50}=58.45±1.74 μM)[3].

Nelonemdaz (0.78-12.5 μM) decreases the amount of antimycin A-induced ROS/RNS formation in a dose-dependent manner, with an IC_{50} of 2.21±0.11 μM[3].
Nelonemdaz (0.19-12.5 μM) inhibits malondialdehyde (MDA) formation with an IC_{50} of 2.72±0.26 μM[3].
Nelonemdaz (0-125 μM) effectively reduces iron-ascorbate-induced lipid peroxidation (IC_{50}=24.56±0.07 μM)[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Nelonemdaz (0.5-20 mg/kg; i.v.) reduces cerebral infarct evolving 24 h after 60-mins occlusion of the middle cerebral artery occlusion (MCAO) substantially and dose dependently[1].
Nelonemdaz (5 mg/kg; i.v.) protects white matter such as axons and myelin as well as gray matter from ischemic brain injury [1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (260 to 300 g) (clip occlusion model)[1]
Dosage:	0.5-20 mg/kg
Administration:	I.v. administration 5 mins after reperfusion
Result:	Produced a large neuroprotective effect, with a maximal reduction in infarct volume of 66% at doses of 2.5 to 5 mg/kg. Not observed neuronal damage in the most vulnerable cortical area after administration of 5 mg/kg.

Animal Model:	Male Sprague-Dawley rats (260 to 300 g) (intraluminal thread occlusion model)[1]
Dosage:	5 mg/kg
Administration:	I.v. administration 30 mins after reperfusion
Result:	Did not change physiologic variables such as arterial pH, PCO_2, PO_2, and hematocrit. Reduced infarct volume evolving in the cortex and the striatum substantially. Reduced white matter damage in the striatum and external capsule markedly.

REFERENCES

[1]. Gwag BJ, et al. Marked prevention of ischemic brain injury by Neu2000, an NMDA antagonist and antioxidant derived from aspirin and sulfasalazine. J Cereb Blood Flow Metab. 2007 Jun;27(6):1142-51.

[2]. Sung IC, et, al. Neu2000, an NR2B-selective, Moderate NMDA Receptor Antagonist and Potent Spin Trapping Molecule for Stroke. Drug News Perspect. 2010 Nov; 23(9): 549-56.

[3]. Nishant PV, et, al. Antioxidant Properties of Neu2000 on Mitochondrial Free Radicals and Oxidative Damage. Toxicol In Vitro. 2013 Mar; 27(2): 788-97.

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