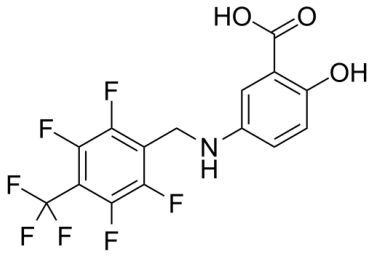
Data Sheet

Product Name:	Nelonemdaz
Cat. No.:	CS-0025740
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
Solubility:	DMSO : ≥ 112.5 mg/mL (293.57 mM)



BIOLOGICAL ACTIVITY:

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]. IC50 & 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]. **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].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [1]Whole-cell voltage-clamp recordings are performed on primary cultured cortical neurons (11 to 14 DIV) at room temperature (18°C to 23°C). Whole-cell currents are recorded and analyzed using an amplifier. The 2 to 3-MΩ-resistance recording pipettes are filled with an internal solution containing 135 mM CsCl, 10 mM N-2-hydroxyethylpiperazine-N′-2-ethanesulfonic acid, 1.2 mM $MgCl_2$, 4 mM ATP-Na_2, 0.5 mM $CaCl_2$, and 11 mM ethyleneglycol tetraacetate (pH adjusted to 7.3 with CsOH). The external solution is composed of 140 mM NaCl, 2 mM KCl, 2 mM $CaCl_2$, 10 mM N-2-hydroxyethylpiperazine-N′-2-ethanesulfonic acid, and 0.01 mM glycine (pH adjusted to 7.3 to 7.4 with NaOH). N-methyl-D-aspartate and Neu2000 solutions are prepared by dissolving these chemicals in the external solutions, and they are applied to target neurons using a gravity-driven superfusion system with a linear array of 8 to 10 barrels. Graphing of data and dose-response analysis are performed and all data are presented as mean±s.e[1]. **Animal Administration:** [2]The pharmacokinetic profile of Neu2000 in normal rats is examined using intraperitoneal (ip) route of administration. Rats are anesthetized by ether inhalation and the femoral artery cannulated. Rats are placed into cages for 1 h and

then receive injections (10, 25, or 50 mg/kg ip, n=6 per dose) of Neu2000 dissolved in sterile saline. Blood is collected from the femoral artery at 15, 30, 60, 120, 240, 480, and 1440 min following injection. Plasma aliquots of 50 µL are spiked with 100 µL of internal standard solution (Neu2000IS, 10 µg/mL in acetonitrile). After vortex mixing for 1 min, the samples are centrifuged at 12,000 g for 5 min. A total of 50 µL of the supernatant phase is separated and diluted with 50 µL of distilled water. Plasma concentrations relative to time are obtained using liquid chromatography-mass spectrometry[2].

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.

CAIndexNames:

Benzoic acid, 2-hydroxy-5-[[[2,3,5,6-tetrafluoro-4-(trifluoromethyl)phenyl]methyl]amino]-

SMILES:

OC(C1=C(O)C=CC(NCC2=C(C(F)=C(C(F)(F)F)C(F)=C2F)F)=C1)=O

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