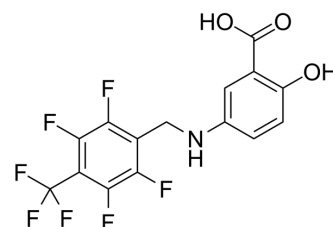


## Nelonemdaz

<b>Cat. No.:</b>	HY-106408		
<b>CAS No.:</b>	640290-67-1		
<b>Molecular Formula:</b>	C <sub>15</sub> H <sub>8</sub> F <sub>7</sub> NO <sub>3</sub>		
<b>Molecular Weight:</b>	383.22		
<b>Target:</b>	iGluR		
<b>Pathway:</b>	Membrane Transporter/Ion Channel; Neuronal Signaling		
<b>Storage:</b>	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		1 mg	5 mg	10 mg
	Concentration	Mass			
	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<sup>[1][2]</sup>.

#### IC<sub>50</sub> & Target

NMDA receptor<sup>[1]</sup>

#### In Vitro

Nelonemdaz (10-300 μM) shows apparent neuroprotection against 300 μM N-methyl-d-aspartate (NMDA) at doses as low as 30 μM<sup>[1]</sup>.  
 Nelonemdaz (10-500 μM) inhibits the electrophysiologic response of cultured cortical neurons to 300 μM NMDA in a concentration-dependent manner<sup>[1]</sup>.  
 Nelonemdaz (0.1-1 μM) produces a marked reduction of Fe<sup>2+</sup>-induced neurotoxicity, even at doses of 0.1 to 0.3 μM<sup>[1]</sup>.  
 Nelonemdaz (0.1-1 μM) blocks the degeneration of neurons and glia in cortical cell cultures<sup>[1]</sup>.  
 Nelonemdaz (0-350 μM) effectively scavenges superoxide radicals (IC<sub>50</sub>=63.07±1.44 μM), nitric oxide (IC<sub>50</sub>=155.8±4.88 μM), and hydroxyl radicals (IC<sub>50</sub>=58.45±1.74 μM)<sup>[3]</sup>.

Nelonemdaz (0.78-12.5  $\mu\text{M}$ ) decreases the amount of antimycin A-induced ROS/RNS formation in a dose-dependent manner, with an  $\text{IC}_{50}$  of  $2.21 \pm 0.11 \mu\text{M}$ <sup>[3]</sup>.  
Nelonemdaz (0.19-12.5  $\mu\text{M}$ ) inhibits malondialdehyde (MDA) formation with an  $\text{IC}_{50}$  of  $2.72 \pm 0.26 \mu\text{M}$ <sup>[3]</sup>.  
Nelonemdaz (0-125  $\mu\text{M}$ ) effectively reduces iron-ascorbate-induced lipid peroxidation ( $\text{IC}_{50} = 24.56 \pm 0.07 \mu\text{M}$ )<sup>[3]</sup>.  
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<sup>[1]</sup>.  
Nelonemdaz (5 mg/kg; i.v.) protects white matter such as axons and myelin as well as gray matter from ischemic brain injury<sup>[1]</sup>.  
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) <sup>[1]</sup>
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) <sup>[1]</sup>
Dosage:	5 mg/kg
Administration:	I.v. administration 30 mins after reperfusion
Result:	Did not change physiologic variables such as arterial pH, $\text{PCO}_2$ , $\text{PO}_2$ , 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.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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