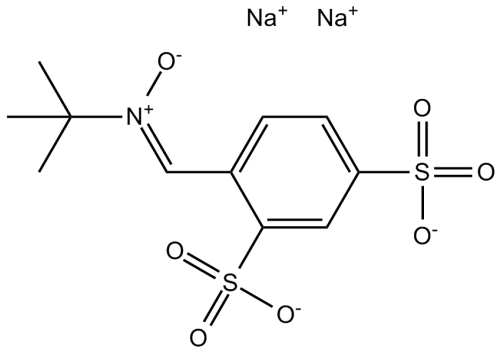
Product Data Sheet

Product Name: NXY-059
Cat. No.: GC13345

Chemical Properties

Cas. No.	168021-79-2		
Chemical Name	disodium;4-[(Z)-[tert-butyl(oxido)azaniumylidene]methyl]benzene-1,3-disulfonate		
SMILES	CC(C)(C)[N+](=CC1=C(C=C(C=C1)S(=O)(=O)[O-])S(=O)(=O)[O-])[O-].[Na+].[Na+]		
Formula	$C_{11}H_{13}NNa_2O_7S_2$	M.Wt	381.33
Solubility	DMF: 20 mg/ml,DMSO: 20 mg/ml,Ethanol: 2 mg/ml,PBS (pH 7.2): 10 mg/ml	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

Cell lines	N1E-115 neuroblastoma cells (mouse neuroblastoma-derived clonal cell line)

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Preparation Method	N1E-115 cells were maintained in Dulbecco's modified Eagle medium:Ham F-12 medium supplemented with 10% heat-inactivated fetal bovine serum at 37°C, 5% CO_2. Cells were pretreated with NXY-059 at a concentration known to be neuroprotective in vivo (300μM) for 30 minutes.
Reaction Conditions	300μM; 24 hours
Applications	NXY-059 showed no cytoprotective effect on SNP-induced cell death in N1E-115 neuroblastoma cells. NXY-059 was also without effect on H_2O_2-mediated cytotoxicity. These results suggest that the neuroprotective effects of NXY-059 observed in vivo are not mediated via a direct cytoprotective action on neuronal-type cells.

Animal experiment [2]:

Animal models	Male Wistar Kyoto rats
Preparation Method	Rats were subjected to either transient (2-hour) or permanent occlusion of the middle cerebral artery (MCA). For the transient model, NXY-059 was administered via continuous intravenous infusion (3, 10, and 30mg/kg/h) for 21.75 hours, starting 2.25 hours after occlusion.
Dosage form	3-30mg/kg/h; i.v. infusion; starting 2.25 hours after occlusion for 21.75 hours.

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Applications	NXY-059 produced a dose-dependent reduction in infarct volume and neurological impairment in both transient and permanent MCA occlusion models. In the transient model, a dose of 10mg/kg/h decreased in infarct volume.

References:
[1] Hainsworth AH, Bhuiyan N, Green AR. The nitrone disodium 2,4-sulphophenyl-N-tert-butylnitrone is without cytoprotective effect on sodium nitroprusside-induced cell death in N1E-115 neuroblastoma cells in vitro. J Cereb Blood Flow Metab. 2008 Jan;28(1):24-8.
[2] Sydserff SG, Borelli AR, Green AR, et al. NXY-059 on infarct volume after transient or permanent middle cerebral artery occlusion in the rat; studies on dose, plasma concentration and therapeutic time window. Br J Pharmacol. 2002 Jan;135(1):103-12.

Background

NXY-059 is a neuroprotective free radical trapping agent that reduces oxidative damage by neutralizing free radicals, while also mitigating neuroinflammation by protecting the integrity of the blood-brain barrier[1-2]. NXY-059 is applicable for research related to acute ischemic stroke [3-4].

In vitro, NXY-059 (250μM) was applied to a co-culture model of bovine brain capillary endothelial cells and glial cells under oxygen-glucose deprivation (OGD) conditions. NXY-059 significantly inhibited the OGD-induced increase in sucrose permeability and reduced the abnormal transcellular transport of tissue-type plasminogen activator (tPA) from the luminal to the brain side [5]. NXY-059 (300μM) pretreated N1E-115 neuroblastoma cells for 30 minutes. NXY-059 failed to inhibit cell death induced by sodium nitroprusside (SNP; 100μM)[6].

In vivo, NXY-059 (3, 10, and 30mg/kg/h) was intravenously infused in a rat model of transient middle cerebral artery occlusion, starting 2.25 hours after occlusion and continuing for 21.75 hours. NXY-059 significantly reduced neurological deficits and histologically measured infarct volume, with the protective effect linearly correlating with plasma concentration[7]. NXY-059 (3 or 10mg/kg) was intravenously administered in a transient focal cerebral ischemia model in Sprague-Dawley rats, with injections at 0 and 30 minutes after occlusion. NXY-059 significantly reduced cortical infarct volume,

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decreased the number of apoptotic cells, and suppressed ischemia-induced cell death[8].

References:

[1] Fong JJ, Rhoney DH. NXY-059: review of neuroprotective potential for acute stroke. Ann Pharmacother. 2006 Mar;40(3):461-71.
[2] Wang CX, Shuaib A. NXY-059: a neuroprotective agent in acute stroke. Int J Clin Pract. 2004 Oct;58(10):964-9.
[3] Maples KR, Green AR, Floyd RA. Nitrone-related therapeutics: potential of NXY-059 for the treatment of acute ischaemic stroke. CNS Drugs. 2004;18(15):1071-84.
[4] Lees KR, Zivin JA, Ashwood T, et al. NXY-059 for acute ischemic stroke. N Engl J Med. 2006 Feb 9;354(6):588-600.
[5] Culot M, Mysiorek C, Renftel M, et al. Cerebrovascular protection as a possible mechanism for the protective effects of NXY-059 in preclinical models: an in vitro study. Brain Res. 2009 Oct 19;1294:144-52.
[6] Hainsworth AH, Bhuiyan N, Green AR. The nitrone disodium 2,4-sulphophenyl-N-tert-butylnitrone is without cytoprotective effect on sodium nitroprusside-induced cell death in N1E-115 neuroblastoma cells in vitro. J Cereb Blood Flow Metab. 2008 Jan;28(1):24-8.
[7] Sydserff SG, Borelli AR, Green AR, et al. NXY-059 on infarct volume after transient or permanent middle cerebral artery occlusion in the rat; studies on dose, plasma concentration and therapeutic time window. Br J Pharmacol. 2002 Jan;135(1):103-12.
[8] Takizawa S, Izuhara Y, Kitao Y, et al. A novel inhibitor of advanced glycation and endoplasmic reticulum stress reduces infarct volume in rat focal cerebral ischemia. Brain Res. 2007 Dec 5;1183:124-37.

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