Disufenton sodium

Cat. No.: HY-13244

CAS No.: 168021-79-2

Molecular Formula: $C_{11}H_{13}NNa_2O_7S_2$

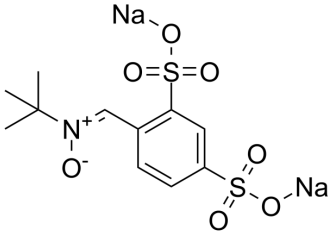
Molecular Weight: 381.33

Target: Reactive Oxygen Species

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (327.80 mM; Need ultrasonic)

H_2O : ≥ 50 mg/mL (131.12 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6224 mL	13.1120 mL	26.2240 mL
5 mM	0.5245 mL	2.6224 mL	5.2448 mL
10 mM	0.2622 mL	1.3112 mL	2.6224 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (262.24 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Disufenton sodium (NXY-059) is the disulfonyl derivative of the neuroprotective spin trap phenylbutynitrone(PBN), both NXY-059, its parent PBN and their hydrolysis/oxidation product MNT are very powerful scavengers of free radicals. IC50 value:Target: Neuroprotectantin vitro: Disufenton sodium is more soluble than the spin trapping agent α-phenyl-N-tert-butyl nitrone (PBN) [1]. In an in vitro blood-brain barrier (BBB) model, 250 mM of Disufenton sodium administered at the onset or up to 4 h after oxygen glucose deprivation (OGD) produces a significant reduction in the increased BBB permeability caused by OGD. Furthermore, OGD produces a huge influx of tissue plasminogen activator across the BBB, which is substantially reduced by Disufenton sodium [2]. in vivo: Disufenton sodium reduces infarct volume in rats subjected to 2 hours of middle cerebral artery occlusion in a dose-dependent manner. At equimolar doses (3.0 mg/kg for Disufenton sodium and 1.4 mg/kg for PBN), Disufenton sodium is more efficacious than PBN. Similar results are obtained when a recovery period of 7 days is allowed. The window of therapeutic opportunity for Disufenton sodium is 3 to 6 hours after the start of recirculation [1]. Disufenton sodium, a free radical-trapping agent, has a substantial protective effect, lessening the disability caused by an experimentally induced stroke in a primate species. Disufenton sodium treatment reduces the

overall amount of brain damage by >50% of saline-treatment values, with similar levels of protection afforded to both white and gray matter [3].

REFERENCES

[1]. Kuroda S, et al. Neuroprotective effects of a novel nitrone, NXY-059, after transient focal cerebral ischemia in the rat. J Cereb Blood Flow Metab, 1999, 19(7), 778-787.

[2]. Marshall JW, et al. NXY-059, a free radical--trapping agent, substantially lessens the functional disability resulting from cerebral ischemia in a primate species. Stroke, 2001, 32(1), 190-198.

[3]. Culot 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, 19(1294), 144-152.

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