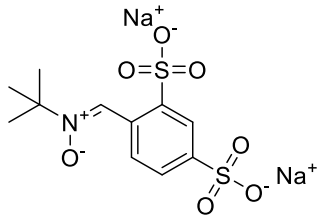
Product data sheet

MedKoo Cat#: 200721 Name: Disufenton sodium (Cerovive) CAS#: 168021-79-2 (sodium) Chemical Formula: $C_{11}H_{13}NNa_2O_7S_2$ Molecular Weight: 381.33	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

Disufenton sodium, also known as Cerovive, OKN007, NXY-059, is a disulfonyl derivative of phenyl-tert-butyl nitrone (PBN), with potential anti-glioma activity. Although the exact mechanism(s) of action of OKN007 are still largely unknown, this agent appears to inhibit cancer cell proliferation and migration. This agent appears to inhibit the activity of sulfatase 2 (SULF2), a highly specific endoglucosamine-6-sulfatase that is overexpressed in the extracellular matrix of cancer cells and catalyzes the removal of sulfate from the 6-O-sulfate esters of heparin.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	44.71	117.25
DMF	20.0	52.45
Ethanol	2.0	5.24
PBS (pH 7.2)	10.0	26.22
Water	54.71	143.47

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	**1 mg**	**5 mg**	**10 mg**
1 mM	2.62 mL	13.11 mL	26.22 mL
5 mM	0.52 mL	2.62 mL	5.24 mL
10 mM	0.26 mL	1.31 mL	2.62 mL
50 mM	0.05 mL	0.26 mL	0.52 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Zheng X, Gai X, Han S, Moser CD, Hu C, Shire AM, Floyd RA, Roberts LR. The human sulfatase 2 inhibitor 2,4-disulfonylphenyl-tert-butylnitrone (OKN-007) has an antitumor effect in hepatocellular carcinoma mediated via suppression of TGFB1/SMAD2 and Hedgehog/GLI1 signaling. Genes Chromosomes Cancer. 2013 Mar;52(3):225-36. doi: 10.1002/gcc.22022. Epub 2012 Oct 29. PMID: 23109092; PMCID: PMC3889201.

2. Mutch NJ, Moore NR, Mattsson C, Jonasson H, Green AR, Booth NA. The use of the Chandler loop to examine the interaction potential of NXY-059 on the thrombolytic properties of rtPA on human thrombi in vitro. Br J Pharmacol. 2008 Jan;153(1):124-31. doi: 10.1038/sj.bjp.0707543. Epub 2007 Nov 5. PMID: 17982476; PMCID: PMC2199381.

In vivo study

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1. Ewert D, Hu N, Du X, Li W, West MB, Choi CH, Floyd R, Kopke RD. HPN-07, a free radical spin trapping agent, protects against functional, cellular and electrophysiological changes in the cochlea induced by acute acoustic trauma. PLoS One. 2017 Aug 23;12(8):e0183089. doi: 10.1371/journal.pone.0183089. PMID: 28832600; PMCID: PMC5568441.
2. Floyd RA, Chandru HK, He T, Towner R. Anti-cancer activity of nitrones and observations on mechanism of action. Anticancer Agents Med Chem. 2011 May 1;11(4):373-9. doi: 10.2174/187152011795677517. PMID: 21651461; PMCID: PMC3246679.

7. Bioactivity

Biological target:

Disufenton sodium (NXY-059) is the disulfonyl derivative of the neuroprotective spin trap phenylbutynitrone (PBN) and is a very powerful scavenger of free radicals.

In vitro activity

To determine the specific molecular mechanisms of the antitumor effect of OKN-007 on HCC cells, this study measured the effect of OKN-007 on the activity of two well-known oncogenic signaling pathways in HCC, including TGFB1/ SMAD2 signaling and Hedgehog/GLI1 signaling. First, luciferase expression was measured in SULF2-positive Huh7 cells transfected with luciferase reporter constructs responsive to activated TGFB1 pathway SMAD transcription factors or to the Hh pathway GLI transcription factors. OKN-007 treatment significantly suppressed SMAD-responsive luciferase activity (Fig. 4A; P = 0.03), as well as GLI-responsive luciferase activity (Fig. 4B; P = 0.03). Immunoblotting confirmed the concomitant suppression of the activated transcription factors pSMAD2 (Fig. 4A) and GLI1 (Fig. 4B). These data support the hypothesis that OKN-007 treatment suppresses the activation of signaling pathways and mediators that we have previously shown to be responsive to SULF2 expression, including TGFB1/SMAD2 and Hedgehog/GLI1.

Reference: Genes Chromosomes Cancer. 2013 Mar;52(3):225-36. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3889201/

In vivo activity

Remarkably, this study observed that OHC (outer hair cell) loss was not only significantly reduced in HPN-07 (Disufenton sodium) treated animals in tonotopic regions corresponding to the initial insult, but its subsequent apical migration was also blocked in the treated animals.In contrast to OHC loss, which plateaued at about 10d post-insult, IHC (inner hair cell) loss continued longitudinally, with most of the loss occurring between 21 and 180d in the untreated animals. In contrast, less than 1% IHC loss was observed at all tonotopic positions in HPN-07-treated animals during the 180d study period. The progressive IHC loss observed in untreated, noise-exposed chinchilla may be attributable to ongoing inflammation or oxidative stress post-injury, both of which have been shown to be attenuated by HPN-07 treatment in other tissue injuries. The observed protection of afferent nerve fibers in the treatment group may, thus, be an indirect effect of HC protection or a direct antioxidant effect on the nerve fibers themselves.

Reference: PLoS One. 2017; 12(8): e0183089. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5568441/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.