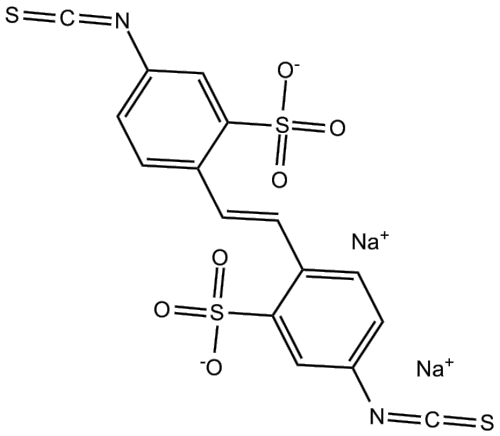
Product Data Sheet

Product Name: DIDS sodium salt
Cat. No.: GC14830

Chemical Properties

Cas. No.	67483-13-0		
Chemical Name	sodium (E)-6,6'-(ethene-1,2-diyl)bis(3-isothiocyanatobenzenesulfonate)		
SMILES	S=C=NC1=CC=C(/C=C/C(C(S([O-])(=O)=O)=C2)=CC=C2N=C=S)C(S([O-])(=O)=O)=C1.[Na+].[Na+]		
Formula	$C_{16}H_8N_2Na_2O_6S_4$	M.Wt	498.48
Solubility	50mg/ml in DMSO (ultrasonic and warming and heat to 60°C); 30mg/ml in Water (Need ultrasonic)	Storage	Store at -20°C, sealed storage, away from moisture
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]:

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Cell lines	DRG(dorsal root ganglion) neurons extracted from the spinal levels of 6- to 8-week-old Sprague-Dawley rats
Preparation method	Soluble in DMSO > 10mM. General tips for obtaining a higher concentration: Please warm the tube at 37°C for 10 minutes and/or shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.
Reacting condition	0.1, 1, 3, 10, 100μm for 2min; simultaneous detection from adding first drop
Applications	In DRG neurons, although DIDS did not induce the activation of TRPV1(Transient receptor potential vanilloid type 1) per se but drastically increased the TRPV1 currents induced by either capsaicin or low pH. DIDS could modify TRPV1 channel function in an agonist-dependent manner.

Animal experiment [2]:

Animal models	4–5 day old newborn healthy Sprague-Dawley rats (both males and females)
Dosage form	5 mg/kg, intraperitoneal injection
Application	DIDS significantly reduced the elevated mRNA levels and protein expression of chloride channel 2 (ClC-2) in neonatal rats induced by ischemia-hypoxia. DIDS application significantly decreased the concentrations of reactive oxygen species (ROS); and the mRNA levels of inducible nitric oxide synthase (iNOS) and tumor necrosis factor-alpha(TNF-α) in neonatal rats with hypoxic-ischemic damage. The elevated number of caspase-3 and neural/glial antigen 2 (NG-2) double-labeled positive cells was attenuated by DIDS after ischemia anoxic injury.

Caution: Product has not been fully validated for medical applications. For research use only.
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Other notes	Please test the solubility of all compounds indoor, and the actual solubility may slightly differ with the theoretical value. This is caused by an experimental system error and it is normal.

References:

[1]. Zhang X1, Du XN, et al, Agonist-dependent potentiation of vanilloid receptor transient receptor potential vanilloid type 1 function by stilbene derivatives. Mol Pharmacol. 2012 May;81(5):689-700. doi: 10.1124/mol.111.076000. Epub 2012 Feb 10.

[2]. Zhao B1, Quan H2, 4,4'-Diisothiocyanostilbene-2,2'-disulfonic Acid (DIDS) Ameliorates Ischemia-Hypoxia-Induced White Matter Damage in Neonatal Rats through Inhibition of the Voltage-Gated Chloride Channel ClC-2. Int J Mol Sci. 2015 May 7;16(5):10457-69. doi: 10.3390/ijms160510457.

Background

DIDS is an anion transport inhibitor, which inhibits the ClC-Ka chloride channel with an IC_{50} of 100 μM and the bacterial ClC-ec1Cl-/H+ exchanger with an IC50 of ~300 μM. [1]

Chloride channels are a superfamily consisting of approximately 13 subgroups and display a variety of functions in physiology. The human genome contains nine CLC proteins, which serve various physiological functions and potentially constitute novel exciting drug targets for the treatment of hypertension, osteoporosis, and gastrointestinal and renal disorders. [1]

DIDS's effect on the calcium-activated chloride current [ICl (ca)] in muscle cells from the rabbit portal vein was studied with the perforated patch technique. Consequently, DIDS reduced the amplitude of spontaneous transient inward currents (STICs) in a concentration-dependent manner with an IC_{50} value of 2.1 x 10-4 M for STICs. Moreover, DIDS was investigated for its action on contraction of cerebral artery smooth muscle cells. DIDS showed a vasodilator effect on pressure-constricted arteries with IC_{50} of 69 ± 14 μM. [2, 3]

In vivo study showed DIDS alone increased the effect of hyperthermia at 42.5 °C or 43.5 °C to suppress tumor growth. The thermosensitization was greater when DIDS was combined with

amiloride. Hyperthermia at 43.5 °C could result in a tumor growth delay for 4 days, while hyperthermia and treatment of 25 mg/kg DIDS prolonged the delay to approximately 6 days. As a proof, in vivo-in vitro excision assays for cell survival illustrated that DIDS enhanced the heat-induced tumor cell death. [4]

References:

[1] Wulff, Heike. "New light on the “Old” chloride channel blocker DIDS." ACS chemical biology 3.7 (2008): 399-401.

[2] Hogg, R. C., Q. Wang, and W. A. Large. "Effects of Cl channel blockers on Ca-activated chloride and potassium currents in smooth muscle cells from rabbit portal vein." British journal of pharmacology 111.4 (1994): 1333-1341.

[3] Nelson, Mark T., et al. "Chloride channel blockers inhibit myogenic tone in rat cerebral arteries." The Journal of Physiology 502.2 (1997): 259-264.

[4] Lyons, John C., Brian D. Ross, and Chang W. Song. "Enhancement of hyperthermia effect in vivo by amiloride and DIDS." International Journal of Radiation Oncology* Biology* Physics 25.1 (1993): 95-103.