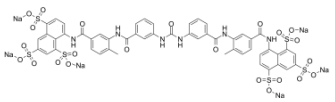
Suramin sodium salt

Cat. No.:	HY-B0879A
CAS No.:	129-46-4
Molecular Formula:	$C_{51}H_{34}N_6Na_6O_{23}S_6$
Molecular Weight:	1429.17
Target:	Phosphatase; Sirtuin; Reverse Transcriptase; Topoisomerase; SARS-CoV; Parasite; Apoptosis
Pathway:	Metabolic Enzyme/Protease; Cell Cycle/DNA Damage; Epigenetics; Anti-infection; Apoptosis
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 200 mg/mL (139.94 mM)
DMSO : 5.6 mg/mL (3.92 mM; Need warming)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.6997 mL	3.4985 mL	6.9971 mL
5 mM	0.1399 mL	0.6997 mL	1.3994 mL
10 mM	0.0700 mL	0.3499 mL	0.6997 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 (69.97 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Suramin sodium salt (Suramin hexasodium salt) is a reversible and competitive protein-tyrosine phosphatases (PTPases) inhibitor[1]. Suramin sodium salt is a potent inhibitor of sirtuins: SirT1 (IC_{50}=297 nM), SirT2 (IC_{50}=1.15 μM), and SirT5 (IC_{50}=22 μM)[2]. Suramin sodium salt is a competitive inhibitor of reverse transcriptase (DNA topoisomerase II: IC_{50}=5 μM)[3][4]. Suramin sodium salt is a potent SARS-CoV-2 RNA-dependent RNA polymerase (RdRp) inhibitor[5]. Suramin sodium salt efficiently inhibits IP5K and is an antiparasitic, anti-neoplastic and anti-angiogenic agent[6][7][8].

IC_{50} & Target

SIRT1	SIRT2	SIRT5
297 nM (IC_{50})	1.15 μM (IC_{50})	22 μM (IC_{50})

In Vitro

Suramin sodium salt (Suramin hexasodium salt; 50-600 μg/mL; for 24-96 hours) inhibits cells proliferation in a dose-

dependent and time-dependent manner and decreases viability in cancer cells[7].
Suramin sodium salt (300 μg/mL; for 48 hours) induces cells apoptosis and down-regulates mRNA expression in HeLa cells[7].

Suramin sodium salt (1 mg/mL; 1 hour) significantly suppresses the phosphorylated ERK1/2[8].
The IC_{50} values of HO-8910 PM and HeLa are 319 μg/mL, 476 μg/mL, respectively[7].
Suramin blocks viral replication in Vero E6 cells[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[6]

Cell Line:	HO-8910 PM ovarian and Hela cervical cancer cells
Concentration:	50, 100, 200, 300, 400, 500 and 600 μg/mL
Incubation Time:	For 24, 48, 72 and 96 hours
Result:	Inhibited cells proliferation in a dose-dependent and time-dependent manner.

Apoptosis Analysis[6]

Cell Line:	HeLa cells
Concentration:	300 μg/mL
Incubation Time:	For 48 hours
Result:	Induced cells apoptosis.

Western Blot Analysis[7]

Cell Line:	PA-SMCs cells
Concentration:	1 mg/mL
Incubation Time:	For 1 hour
Result:	Significantly suppressed the phosphorylated ERK1/2.

In Vivo

Suramin sodium salt (Suramin hexasodium salt; 10 mg/kg; IV; twice weekly for 3 weeks) reverses established pulmonary hypertension (PH), thereby normalizing the pulmonary artery pressure values and vessel structure[8].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult male Wistar rats (200-225 g)[7]
Dosage:	10 mg/kg
Administration:	IV; twice weekly for 3 weeks
Result:	Reversed established PH, thereby normalizing the pulmonary artery pressure values and vessel structure.

CUSTOMER VALIDATION

- Nat Struct Mol Biol. 2021 Mar;28(3):319-325.
- J Biol Chem. 2020 Jul 24;295(30):10281-10292.

- Biomicrofluidics. 2019 Nov 21;13(6):064117.
- Research Square Preprint. 2020 Nov.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Jindal HK, et al. Suramin affects DNA synthesis in HeLa cells by inhibition of DNA polymerases. Cancer Res. 1990 Dec 15;50(24):7754-7.

[2]. Izikki M, et al. The beneficial effect of suramin on monocrotaline-induced pulmonary hypertension in rats. PLoS One. 2013 Oct 15;8(10):e77073.

[3]. Zhang YL, et al. Suramin is an active site-directed, reversible, and tight-binding inhibitor of protein-tyrosine phosphatases. J Biol Chem. 1998 May 15;273(20):12281-7.

[4]. Trapp J, et al. Structure-activity studies on suramin analogues as inhibitors of NAD+-dependent histone deacetylases (sirtuins). ChemMedChem. 2007 Oct;2(10):1419-31.

[5]. Schuetz A, et al. Structural basis of inhibition of the human NAD^+-dependent deacetylase SIRT5 by suramin. Structure. 2007 Mar;15(3):377-89.

[6]. De Clercq E, et al. Suramin: a potent inhibitor of the reverse transcriptase of RNA tumor viruses. Cancer Lett. 1979 Nov;8(1):9-22.

[7]. Novaes RD, et al. Purinergic Antagonist Suramin Aggravates Myocarditis and Increases Mortality by EnhancingParasitism, Inflammation, and Reactive Tissue Damage in Trypanosoma cruzi-Infected Mice. Oxid Med Cell Longev. 2018 Sep 30;2018:7385639.

[8]. Xiaozhe Zhang, et al. Suramin and NF449 Are IP5K Inhibitors That Disrupt IP6-mediated Regulation of Cullin RING Ligase and Sensitize Cancer Cells to MLN4924/pevonedistat. J Biol Chem. 2020 Jun 3;jbc.RA120.014375.

[9]. Xiaozhe Zhang, et al. Suramin and NF449 Are IP5K Inhibitors That Disrupt IP6-mediated Regulation of Cullin RING Ligase and Sensitize Cancer Cells to MLN4924/pevonedistat. J Biol Chem. 2020 Jun 3;jbc.RA120.014375.

[10]. Wanchao Yin, et al. Structural basis for inhibition of the SARS-CoV-2 RNA polymerase by suramin. Nat Struct Mol Biol. 2021 Mar;28(3):319-325.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA