

Product Data Sheet

Product Name: Deferoxamine mesylate

Cat. No.: GC13554

Chemical Properties

Cas. No. 138-14-7

Chemical Name (Z)-4-((5-aminopentyl)(hydroxyamino)-N-(5-((Z)-N,4-dihydroxy-4-((5-(N-hydroxyacetamido)pentyl)imino)butanamido)pentyl)-4-oxobutanimidic acid compound with methanesulfonic acid (1:1)

SMILES CC(N(O)CCCCC/N=C(O)/CCC(N(O)CCCCC/N=C(O)/CCC(N(O)CCCCCN)=O)=O)=O.CS(=O)(=O)=O

Formula $C_{26}H_{52}N_6O_{11}S$

M.Wt 656.79

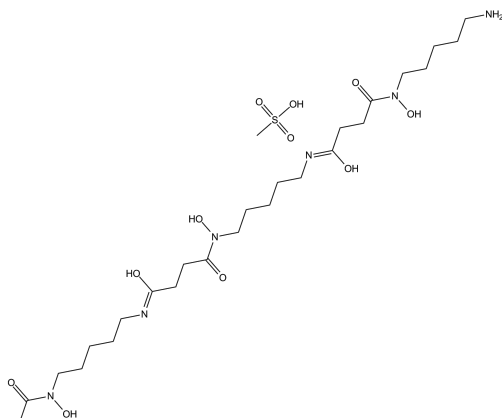
Solubility $\geq 65.7\text{mg/mL}$ in Water

Storage Store at -20°C , protect from light

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	HEI-OC1 cells (mouse cochlear hair cell-like cell line)
Preparation Method	HEI-OC1 cells were maintained in medium at 33°C, 10% CO ₂ . HEI-OC1 cells were pretreated with 200µM Deferoxamine mesylate for 2h followed by 100µM tert-butyl hydroperoxide for 3h. After treatment, CCK-8 assay, Calcein/PI staining, TUNEL assay, transmission electron microscopy, GSH/GSSG quantification, and western blot for Nrf2, FTH1, GPX4, LC3B, BAX, P53, ACSL4, SOD2, GCLC, GSS were performed.
Reaction Conditions	200µM; 2h pretreatment
Applications	Deferoxamine mesylate maintained HEI-OC1 cell viability and adherence, reduced TUNEL-positive and PI-positive dead cell counts, reversed mitochondrial shrinkage and cristae loss, alleviated GSH depletion and GSH/GSSG ratio drop, upregulated Nrf2 and FTH1 protein expression, increased LC3B-II protein level, but did not restore GPX4, BAX, P53, ACSL4, SOD2, GCLC or GSS expression changes induced by tert-butyl hydroperoxide.
Animal experiment [2]:	
Animal models	Male Sprague-Dawley rats (180-200g)

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Preparation Method	Rats were intraperitoneally injected with 200mg/kg Deferoxamine mesylate daily for 2 weeks; control rats received 0.9% saline injections. After treatment, rats were fasted overnight and subjected to intraperitoneal glucose tolerance test (2g/kg), then sacrificed under anesthesia by exsanguination for collection of liver, calf muscle, and peritoneal adipose tissue. Serum iron, blood hematocrit, hepatic iron concentration, serum insulin, hepatic qRT-PCR and western blot (normalized to β -actin) were performed. A separate group of high-calorie high-fat diet-fed rats received the same Deferoxamine mesylate regimen for 2 weeks with intraperitoneal GTT assessment.
Dosage form	200mg/kg; i.p.; daily for 2 weeks
Applications	Deferoxamine mesylate decreased serum iron, blood hematocrit, and hepatic iron concentration, improved intraperitoneal glucose tolerance test glucose clearance, increased hepatic HIF-1 α , insulin receptor, Akt/PKB and phosphorylated Akt/PKB protein levels, upregulated hepatic Glut1 mRNA, and in high-fat-diet rats improved glucose clearance during GTT without altering body weight or nonfasting glucose.
References: [1] Lu PH, Ma PW, Wang WL, et al. Deferoxamine protects cochlear hair cells and hair cell-like HEI-OC1 cells against tert-butyl hydroperoxide-induced ototoxicity. BBA - Molecular Basis of Disease. 2024;1870:167024. [2] Dongiovanni P, Valenti L, Fracanzani AL, et al. Iron Depletion by Deferoxamine Up-Regulates Glucose Uptake and Insulin Signaling in Hepatoma Cells and in Rat Liver. American Journal of Pathology. 2008 Mar;172(3):738-747.	

Background

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Deferoxamine mesylate is an iron chelator that binds Fe(III) and many other metal cations. Deferoxamine mesylate binds Fe(III) to form water-soluble ferrioxamine chelates, thereby preventing iron-mediated Fenton reactions and reactive oxygen species generation. Deferoxamine mesylate upregulates HIF-1 α levels and inhibits tumor cell proliferation, induces apoptosis and autophagy in cancer cells. Deferoxamine mesylate can be used in research related to cancer, spinal cord injury, neurodegenerative diseases, diabetes, and ferroptosis^[1-4].

In vitro, treatment of SH-SY5Y cells with 100 μ M Deferoxamine mesylate and 200 μ M 6-hydroxydopamine for 24 hours restored cell viability, inhibited intracellular reactive oxygen species generation, and reduced the numbers of early and late apoptotic cells^[5]. Co-treatment of PC3 cells with 200 μ M Deferoxamine mesylate and 200 μ g/mL nickel-iron layered double hydroxide for 24 hours disrupted mitochondrial membrane potential, destroyed lysosomal integrity, upregulated IGFBP3, inhibited the PI3K/AKT/mTOR pathway, and induced apoptosis^[6]. Pretreatment of HEI-OC1 cells with 200 μ M Deferoxamine mesylate for 2 hours followed by 100 μ M tert-butyl hydroperoxide for 3 hours maintained cell morphology and adherence, reduced the proportion of dead cells, alleviated mitochondrial shrinkage, mitochondrial cristae reduction or loss, and mitochondrial vacuolization, and mitigated GSH depletion^[7].

In vivo, subcutaneous injection of 100mg/kg Deferoxamine mesylate into the distal flap of streptozotocin-induced diabetic C57BL/6 mice for 3 days increased flap survival area, enhanced distal flap blood perfusion, and increased CD31-positive capillary density^[8]. Daily topical application of 10 μ L of 1mM Deferoxamine mesylate solution to full-thickness excisional wounds on the backs of type II diabetic C57BL/6J mice for 15 days accelerated wound healing, shortened healing time, and increased CD31-positive microvessel density in healed tissues^[9]. Daily intraperitoneal injection of 200mg/kg Deferoxamine mesylate into Sprague-Dawley rats for 2 consecutive weeks decreased serum iron and blood hematocrit, reduced liver iron concentration, and improved glucose clearance in the intraperitoneal glucose tolerance test^[10].

References:

[1] Seligman PA, Schleicher RB. Deferoxamine mesylate (Desferal mesylate). A specific iron-chelating agent for treating acute iron intoxication. Clinical Pharmacology and Therapeutics. 1969 Jul-Aug;10(4):595-596.

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- [2] Wang F, Elliott RL, Head JF. Inhibitory effect of deferoxamine mesylate and low iron diet on the 13762NF rat mammary adenocarcinoma. *Anticancer Research*. 1999 Jan-Feb;19(1A):333-339.
- [3] Vogelaar CF, König B, Krafft S, et al. Pharmacological suppression of CNS scarring by deferoxamine reduces lesion volume and increases regeneration in an in vitro model for astroglial-fibrotic scarring and in rat spinal cord injury in vivo. *PLoS ONE*. 2015 Jul 29;10(7):e0134371.
- [4] Gholami M, Khodagholi F, Hafizi M, et al. The role of the iron-chelating agent deferoxamine in spinal cord injury: a systematic review and meta-analysis of preclinical studies. *Journal of Neurosurgery: Spine*. 2026;44(2):123-134.
- [5] Rakshit J, Priyam A, Gowrishetty KK, et al. Iron chelator Deferoxamine protects human neuroblastoma cell line SH-SY5Y from 6-Hydroxydopamine-induced apoptosis and autophagy dysfunction. *Journal of Trace Elements in Medicine and Biology*. 2020;59:126406.
- [6] Wang Z, Wang H, Yang L, et al. Controllable nickel ions release from deferoxamine mesylate-triggered nickel-iron layered double hydroxide for eliciting apoptotic cell death in prostate cancer. *Journal of Nanobiotechnology*. 2025 May 30;23:399.
- [7] Lu PH, Ma PW, Wang WL, et al. Deferoxamine protects cochlear hair cells and hair cell-like HEI-OC1 cells against tert-butyl hydroperoxide-induced ototoxicity. *BBA - Molecular Basis of Disease*. 2024;1870:167024.
- [8] Wang C, Cai Y, Zhang Y, et al. Local Injection of Deferoxamine Improves Neovascularization in Ischemic Diabetic Random Flap by Increasing HIF-1 α and VEGF Expression. *PLoS ONE*. 2014 Jun 25;9(6):e100818.
- [9] Duscher D, Januszyk M, Maan ZN, et al. Comparison of the Hydroxylase Inhibitor DMOG and the Iron Chelator Deferoxamine in Diabetic and Aged Wound Healing. *Plastic and Reconstructive Surgery*. 2017 Mar;139(3):695e-706e.
- [10] Dongiovanni P, Valenti L, Fracanzani AL, et al. Iron Depletion by Deferoxamine Up-Regulates Glucose Uptake and Insulin Signaling in Hepatoma Cells and in Rat Liver. *The American Journal of Pathology*. 2008 Mar;172(3):738-747.

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