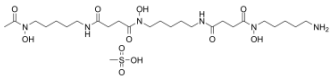
Deferoxamine mesylate

Cat. No.: HY-B0988

CAS No.: 138-14-7

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

Molecular Weight: 656.79



Target: Autophagy; Amyloid-β; Mitophagy; Ferroptosis

Pathway: Autophagy; Neuronal Signaling; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month

SOLVENT & SOLUBILITY

In Vitro

H_2O : 125 mg/mL (190.32 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.5226 mL	7.6128 mL	15.2256 mL
5 mM	0.3045 mL	1.5226 mL	3.0451 mL
10 mM	0.1523 mL	0.7613 mL	1.5226 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 33.33 mg/mL (50.75 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Deferoxamine mesylate is an iron chelator that binds free iron in a stable complex, preventing it from engaging in chemical reactions.

In Vitro

Deferoxamine treatment significantly increases HIF-1α binding under all culture conditions, including hypoxic and high-glucose. The mechanism of deferoxamine is through improving HIF-1α biological function through scavenging oxygen free radicals[1]. Deferoxamine (5 μM) has significant effect on the tumor-associated stromal cells cellular multiplication, and cells die at day 7 after exposure to 50 μM and 100 μM deferoxamine. Deferoxamine (5 μM-100 μM) inhibits the proliferation of BMMSCs, and induces apoptosis of MSCs in a dose-dependent manner. Deferoxamine influences the expression of adhesion proteins on MSCs[3]. Deferoxamine (30, 60, 120 μM) shows lower expression of HIF-1α in a concentration dependent way in AdMSCs[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	Deferoxamine (100 mg/kg, i.p.) lowers the mortality rate of subarachnoid hemorrhage (SAH) rat. Deferoxamine (100 mg/kg, i.p.) attenuates Evan's blue extravasation in cortex, ameliorates the tight junction detachment and preserves the integrity of the base membrane examined in electron microscope at day 3 after SAH. Deferoxamine attenuates degradation of BBB proteins after SAH and significantly reduces ferritin expression at day 3 in the cortex, and improves neurologic behavior and cognitive deficits after experimental[1]. Ten μL of 1 mM deferoxamine-treated wounds display significantly accelerated healing from day 7 onward and heal significantly faster than control-treated wounds in diabetic mice. Deferoxamine-treated wounds and dimethyloxalylglycine-treated wounds heal significantly faster than control-treated wounds in aged mice[2]. In deferoxamine (10 mg/mL)-treated TG mice, there is a decrease in both soluble and insoluble Aβ40 and Aβ42. Both pGSK3β and β-catenin are significantly increased by approximately 50% in the deferoxamine-treated mice[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	Harvested murine tumor and bone marrow-derived MSCs are exposed to varying doses of deferoxamine. Cell viability is assessed by trypan blue exclusion assay. The viable cells are more than 98% before enrolled for experiments. A total of 1.5×10^5 TAMSCs/well or 3×10^5 BMMSCs/well are seeded in 6-well plates. Then MSCs are exposed to 5, 10, 25, 50, and 100 μM deferoxamine on the following day. After 7 days, the number of TAMSCs is counted. To assess the cytotoxicity of deferoxamine to primary bone marrow MSCs, 2×10^6 bone marrow cells/well are seeded in 24-well plates. After 9 days, the number of survival cells is counted. To assess the cell cycle, TAMSCs are stained with propidium iodide, and cell cycle distribution is analyzed by flow cytometry. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [5]	Mice are divided into three treatment groups of 17 each: (1) TG mice given IN Deferoxamine (TG-DFO), (2) TG mice given IN phosphate buffered saline (TG-PBS), and (3) WT mice given IN PBS (WT-PBS). At 30 weeks of age, mice are acclimated to handling and then treated intranasally every monday, wednesday, and friday, starting at 36 weeks of age. Mice are dosed for 18 weeks, until behavior tests at 54 weeks. Dosing continues during the 4 weeks of behavior to measure both chronic and acute effects. After behavior mice are dosed a final time, and 24 h later euthanized and tissues collected for biochemical analyses. These include soluble and insoluble amyloid as measured by ELISA and IHC, quantification of proteins with Western blot and oxidative markers. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Res. 2018 Dec;28(12):1171-1185.
- Signal Transduct Target Ther. 2020 May 8;5(1):51.
- Redox Biol. 2020 Jan;29:101402.
- ACS Appl Mater Interfaces. 2018 Feb 21;10(7):6180-6189.
- Theranostics. 2020 Apr 6;10(11):5107-5119.

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REFERENCES

[1]. Li Y, et al. Effects of deferoxamine on blood-brain barrier disruption after subarachnoid hemorrhage. PLoS One. 2017 Mar 1;12(3):e0172784

[2]. Duscher D, et al. Comparison of the Hydroxylase Inhibitor Dimethyloxalylglycine and the Iron Chelator Deferoxamine in Diabetic and Aged Wound Healing. Plast Reconstr Surg. 2017 Mar;139(3):695e-706e

[3]. Wang G, et al. In vitro assessment of deferoxamine on mesenchymal stromal cells from tumor and bone marrow. Environ Toxicol Pharmacol. 2017 Jan;49:58-64

[4]. Wahl EA, et al. VEGF released by deferoxamine preconditioned mesenchymal stem cells seeded on collagen-GAG substrates enhances neovascularization. Sci Rep. 2016 Nov 10;6:36879

[5]. Fine JM, et al. Intranasal deferoxamine engages multiple pathways to decrease memory loss in the APP/PS1 model of amyloid accumulation. Neurosci Lett. 2015 Jan 1;584:362-7

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