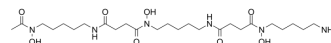


Deferoxamine

Cat. No.:	HY-B1625												
CAS No.:	70-51-9												
Molecular Formula:	C ₂₅ H ₄₈ N ₆ O ₈												
Molecular Weight:	560.68												
Target:	HIF/HIF Prolyl-Hydroxylase; Reactive Oxygen Species; Apoptosis; Akt; Autophagy												
Pathway:	Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB; Apoptosis; PI3K/Akt/mTOR; Autophagy												
Storage:	<table border="0"> <tr> <td>Powder</td> <td>-20°C</td> <td>3 years</td> </tr> <tr> <td></td> <td>4°C</td> <td>2 years</td> </tr> <tr> <td>In solvent</td> <td>-80°C</td> <td>6 months</td> </tr> <tr> <td></td> <td>-20°C</td> <td>1 month</td> </tr> </table>	Powder	-20°C	3 years		4°C	2 years	In solvent	-80°C	6 months		-20°C	1 month
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SOLVENT & SOLUBILITY

In Vitro	DMSO : 10 mg/mL (17.84 mM; ultrasonic and warming and heat to 60°C)				
		Solvent Concentration	Mass		
	Preparing Stock Solutions		1 mg	5 mg	10 mg
		1 mM	1.7835 mL	8.9177 mL	17.8355 mL
		5 mM	0.3567 mL	1.7835 mL	3.5671 mL
10 mM		0.1784 mL	0.8918 mL	1.7835 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	<ol style="list-style-type: none"> Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 1.25 mg/mL (2.23 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 1.25 mg/mL (2.23 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 1.25 mg/mL (2.23 mM); Clear solution 				

BIOLOGICAL ACTIVITY

Description	Deferoxamine (Deferoxamine B) is an iron chelator (binds to Fe(III) and many other metal cations), is widely used to reduce iron accumulation and deposition in tissues. Deferoxamine upregulates HIF-1α levels with good antioxidant activity. Deferoxamine also shows anti-proliferative activity, can induce apoptosis and autophagy in cancer cells. Deferoxamine can be used in studies of diabetes, neurodegenerative diseases as well as anti-cancer and anti-COVID-19 ^{[1][2][3][4][5]} .
In Vitro	Deferoxamine (1 mM; 16 h or 4 weeks) improves HIF-1α function under hypoxic and hyperglycemic conditions and decreases

ROS in MEFs cells^[1].

Deferoxamine (100 μ M; 24 h) increases InsR expression and activity and also induces an increase in p-Akt/total Akt/PKB levels^[2].

Deferoxamine (5, 10, 25, 50, 100 μ M; 7 or 9 days) inhibits the proliferation of tumor-associated MSCs and bone marrow MSCs^[3].

Deferoxamine (5, 10, 25, 50, 100 μ M; 7 days) induces apoptosis of MSCs^[3].

Deferoxamine (10 μ M ; 3 days) influences the expression of adhesion proteins on MSCs^[3].

Deferoxamine (100 μ M; 24 h) induces autophagy mediated by the level of HIF-1 α in SH-SY5Y cells^[4].

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

Western Blot Analysis^[1]

Cell Line:	MEFs cells
Concentration:	1 mM
Incubation Time:	16 h (hypoxia condition); 4 weeks (hyperglycemic conditions)
Result:	Significantly attenuated the hyperglycemia-associated increase in ROS levels under hypoxic high glucose conditions. Notably increased normoxic HIF transactivation in MEFs under both high glucose and normal glucose conditions.

Western Blot Analysis^[2]

Cell Line:	HepG2 cells
Concentration:	100 μ M
Incubation Time:	24 h
Result:	Showed a twofold increase of InsR mRNA levels in cells. Increased by twofold InsR binding activity at the half-maximal concentration of 1.1 nM.

Cell Proliferation Assay^[3]

Cell Line:	TAMSCs and BMMSCs (all isolated from Male C57BL/6J mice (8 week-old; EG-7 induced tumor model))
Concentration:	5, 10, 25, 50, 100 μ M
Incubation Time:	7 days (TAMSCs); 9 days (BMMSCs).
Result:	Inhibited the growth of TAMSCs and BMMSCs, and most cells are died at day 7 or 9 when exposed to 50 and 100 μ M dose.

Apoptosis Analysis^[3]

Cell Line:	TAMSCs, BMMSCs
Concentration:	5, 10, 25, 50, 100 μ M
Incubation Time:	7 days
Result:	Exhibited proapoptotic effect on TAMSCs and BMMSCs cells.

Western Blot Analysis^[3]

Cell Line:	TAMSCs, BMMSCs
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Concentration:	10 μ M
Incubation Time:	3 days
Result:	Remarkably decreased VCAM-1 expression in both TAMSCs and BMMSCs.
Cell Autophagy Assay ^[4]	
Cell Line:	SH-SY5Y cells
Concentration:	100 μ M
Incubation Time:	24 h
Result:	Increased the ratio of LC3-II/I, an indicator of autophagy, which effects were blocked when autophagy-related gene Beclin 1 was suppressed by Beclin 1 siRNA transfection. Caused a time and dose-dependent increase of HIF-1 α , accompanied by the induction of autophagy.

In Vivo	<p>Deferoxamine (560.68 mg/per; drip-on; once daily for 21 days) enhances wound healing and increases neovascularization in aged or diabetic mice^[1].</p> <p>Deferoxamine (200 mg/kg; i.p.; daily for 2 weeks) results in HIF-1α stabilization and increases glucose uptake, hepatic InsR expression, and signaling in vivo^[2].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>															
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CUSTOMER VALIDATION

- Cell Res. 2018 Dec;28(12):1171-1185.
- Signal Transduct Target Ther. 2020 May 8;5(1):51.
- Bioact Mater. 2021 Nov 19;13:23-36.
- Adv Sci (Weinh). 2023 Jun 17;e2206798.
- Adv Sci (Weinh). 2023 Mar 26;e2206007.

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- [2]. Dongiovanni P, et al. Iron depletion by deferoxamine up-regulates glucose uptake and insulin signaling in hepatoma cells and in rat liver. *Am J Pathol*. 2008 Mar;172(3):738-47.
- [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]. Wu Y, et al. Neuroprotection of deferoxamine on rotenone-induced injury via accumulation of HIF-1 alpha and induction of autophagy in SH-SY5Y cells. *Neurochem Int*. 2010 Oct;57(3):198-205.
- [5]. Bellotti D, et al. Deferoxamine B: A Natural, Excellent and Versatile Metal Chelator. *Molecules*. 2021 May 28;26(11):3255.
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