Deferiprone

Cat. No.: HY-B0568

CAS No.: 30652-11-0

Molecular Formula: $C_7H_9NO_2$

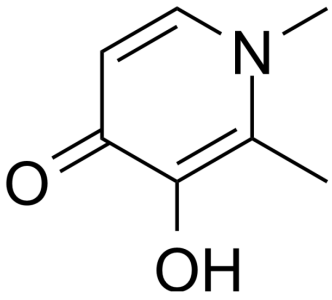
Molecular Weight: 139.15

Target: HCV; Ferroptosis; Apoptosis; COX

Pathway: Anti-infection; Apoptosis; Immunology/Inflammation

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 7.14 mg/mL (51.31 mM; Need ultrasonic)

H_2O : 3.33 mg/mL (23.93 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	7.1865 mL	35.9324 mL	71.8649 mL
5 mM	1.4373 mL	7.1865 mL	14.3730 mL
10 mM	0.7186 mL	3.5932 mL	7.1865 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 10 mg/mL (71.86 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 0.71 mg/mL (5.10 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 0.71 mg/mL (5.10 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.71 mg/mL (5.10 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Deferiprone is a potent, orally active, brain-penetrant, cell-penetrant, skin-permeable, free iron chelating agent. Deferiprone inhibits the proliferation and migration, and stimulates apoptosis in tumor cell. Deferiprone has antianemic, neuroprotective, anti-inflammatory, antioxidant, and antidotal activity. Deferiprone can be used in cancer, cardiovascular disease, infection, inflammation, and neurological disease study[1][2][3][4][5][6][7][8].

In Vitro

Deferiprone (66-660 μM, 48-96 h) has a significant inhibitory effect on proliferation in TRAMP-C2, Myc-CaP, and 22rv1 cells[1].
Deferiprone (100 μM, up to 192 h) inhibits cell migration in TRAMP-C2, Myc-CaP, and 22rv1 cells[1].
Deferiprone (100 μM, 24 h) reduces the expression and activity of m-Acon in TRAMP-C2, Myc-CaP, and 22rv1 cells[1].
Deferiprone (up to 1μM, 0.5-24 h) decreases the free iron in thalassemic red blood cells[2].
Deferiprone (10 mins) inhibits human platelet aggregation stimulated by AA and ADP and epinephrine and collagen, with the IC_{50} values of 0.24, 0.25, 3.36 and 3.73 mM, respectively[3].
Deferiprone (0.1-3.2 μM, 5 mins) inhibits COX-1 activity with the IC_{50} value of 0.33 μM[3].
Deferiprone (4 mM, 5 mins) preventes ADP-induced formation of cAMP[3].
Deferiprone (156.25 μg/mL, 24 h) enhances survival rate and reduces LDH Levels and displays normal cell morphology in aged Fibroblasts[4].
Deferiprone (25μM, 6 h) amplifies the antibacterial activity of conventional antibiotics against S. epidermidis[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	TRAMP-C2, Myc-CaP, and 22rv1 cells
Concentration:	0, 16, 30, 66, 100, 160, 300, 660 μM
Incubation Time:	48 h, 72 h
Result:	Showed a cytostatic effect in three cell lines with an IC_{50} and IC_{90} values of about 50 and 100 μM, respectively.

Cell Migration Assay [1]

Cell Line:	TRAMP-C2, Myc-CaP, and 22rv1 cells
Concentration:	100 μM
Incubation Time:	0 to 30 h for TRAMP-C2, and Myc-CaP; 0 to 192 h for 22rv1
Result:	Inhibited cell migration starting at different time points for each cell line, ranging from 12 h in TRAMP-C2 cell to 48 h in 22rv1 cells, and 30 h in Myc-CaP cells.

Western Blot Analysis[1]

Cell Line:	TRAMP-C2, Myc-CaP, and 22rv1 cells
Concentration:	100 μM
Incubation Time:	24 h
Result:	Reduced the expression of m-Acon, by 2-fold in Myc-CaP and 22 rv1 cells and decreased by 79% in TRAMP-C2 cells.

In Vivo

Deferiprone (100 mg/kg/daily for i.g., 4 weeks) has a neuroprotective effect in the rTg(tauP301L)4510 mouse model of tauopathy[6].
Deferiprone (50-200 mg/kg/daily for p.o., 5-10 day) reduces the nephrotoxicity in Cisplatin (HY-17394)-induced rat acute renal failure[7].
Deferiprone (13.82, 27.64 mg/kg/d for i.g., 4 weeks) exhibits anti- apoptosis and neuroprotective activity in rat Alzheimer's disease model[8].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	The rTg(tauP301L)4510 mouse model of tauopathy[6].
Dosage:	100 mg/kg/daily, 4 weeks

Administration:	Intragastric administration (i.g.)
Result:	Improved Y-maze and open field performance, and decreased 28% iron levels in brain, and reduced AT8-labeled p-tau within the hippocampus in transgenic tau mice.

Animal Model:	Cisplatin(HY-17394)-induced rat acute renal failure model [7]
Dosage:	50, 100, 200 mg/kg, 5-10 day
Administration:	Oral administration
Result:	Reduced the creatinine, BUN, malondialdehyde, iron concentrations, and the amounts of TfR, and indreased the levels of HIF-1a and related anti-apoptotic genes expression in Cisplatin (HY-17394)-injected animals.

Animal Model:	Aluminium-linked apoptosis in rat hippocampus model (Alzheimer's disease model) [8]
Dosage:	13.82, 27.64 mg/kg/d, 4 week
Administration:	Intragastric administration lasting 6 days with 1 day interval per week
Result:	Decreased the apoptosis and the expression of Caspase-3 and Bax, and increased the expression of Bcl-2 in Aluminium-linked apoptosis in rat hippocampus.

CUSTOMER VALIDATION

- Nat Nanotechnol. 2021 Oct;16(10):1150-1160.
- Biomaterials. 2022: 121936.
- Sci Adv. 2023 Nov 15;9(46):eadf4345.
- J Hazard Mater. 2021 May 15;410:124566.
- Autophagy. 2022 Apr 26:1-17.

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[2]. Oded Shalev. et al. Deferiprone (L1) Chelates Pathologic Iron Deposits From Membranes of Intact Thalassemic and Sickle Red Blood Cells Both In Vitro and In Vivo.

[3]. Ngan Thi Tran, et al. Antiplatelet activity of deferiprone through cyclooxygenase-1 inhibition. Platelets 2020 May 18;31(4):505-512.

[4]. Andrea Pagani, MD, et al. Deferiprone Stimulates Aged Dermal Fibroblasts via HIF-1α Modulation.Pathog Dis. 2018 Jul 1;76(5).

[5]. Débora C Coraça-Huber, et al. Iron chelation destabilizes bacterial biofilms and potentiates the antimicrobial activity of antibiotics against coagulase-negative Staphylococci. Pathogens and Disease, Volume 76, Issue 5, July 2018, fty052

[6]. Shalini S. Rao, et al. Deferiprone Treatment in Aged Transgenic Tau Mice Improves Y-Maze Performance and Alters Tau Pathology. Neurotherapeutics. 2021 Apr;18(2):1081-1094.

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[9]. Kontoghiorghes GJ, et al. Benefits and risks of deferiprone in iron overload in Thalassaemia and other conditions: comparison of epidemiological and therapeutic aspects with deferoxamine. Drug Saf. 2003;26(8):553-584.

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