Hemin

Cat. No.: HY-19424

CAS No.: 16009-13-5

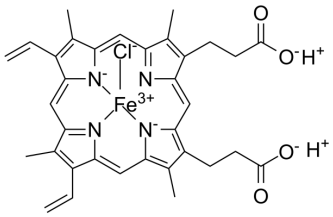
Molecular Formula: $C_{34}H_{32}ClFeN_4O_4$

Molecular Weight: 651.94

Target: Autophagy; Mitophagy; Ferroptosis

Pathway: Autophagy; Apoptosis

Storage: 4°C, protect from light, stored under nitrogen
* In solvent : -80°C, 2 years; -20°C, 1 year (protect from light, stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

1M NaOH : 6.67 mg/mL (10.23 mM; ultrasonic and adjust pH to 12 with NaOH)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.5339 mL	7.6694 mL	15.3388 mL
	5 mM	0.3068 mL	1.5339 mL	3.0678 mL
	10 mM	0.1534 mL	0.7669 mL	1.5339 mL

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

BIOLOGICAL ACTIVITY

Description

Hemin is an iron-containing porphyrin. Hemin is an Heme oxygenase (HO)-1 inducer.

IC_{50} & Target

Heme oxygenase[1]

In Vitro

Hemin and PGJ2, used as positive controls, strongly increase both expression and activity of HMOX after 4 and 12 h, respectively. Indeed, a significant effect is found of 30 μM Hemin on cell proliferation in all used cell lines after 48 h, which is dose-dependent. Hemin treatment decreases cell proliferation to 62±5 %, 51±3 %, and 38±8 % in PA-TU-8902, BxPC-3 and MiaPaCa-2 cancer cells, respectively, with p<0.0001 for all comparisons. Furthermore, enhancement of anti-proliferative effects of statins is observed by Hemin, documented as decreased cell proliferation after 48 h of co-treatment[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Following the i.p. administration of Hemin (100 μmol/kg), the HO-1 level in the renal cortex begins to increase gradually. The HO-1 level reaches its peak 24 h after Hemin preconditioning. HO-1 is expressed mainly in the renal tubules. The HO-2 level in the kidney does not change following Hemin preconditioning[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	For the cell proliferation assay, cells are seeded into 96 well (5-12.5×10^4 cells per mL according to the cell line) and kept at 37°C and 5 % CO_2. After 24 h, cells are treated with statins or/and Hemin, followed by the MTT test as a general cell proliferation assay[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Eight- to ten-week-old male BABL/c mice are used for the ischemia-reperfusion (I/R) experiments. The animals are divided into five groups as follows: (1) the sham group undergo isolation of the bilateral renal arteries without clamping; (2) the vehicle group receive an intraperitoneal (i.p.) injection of 4 mL/kg PBS as a vehicle control (with IRI); (3) the Hemin-preconditioned group receive Hemin, a potent inducer of HO-1, at 100 μmol/kg i.p.; (4) the Hemin plus ZnPP group receive zinc protoporphyrin IX, an inhibitor of HO-1 activity, at 5 mg/kg i.p. 6 h after receiving 100 μmol/kg Hemin i.p.; and (5) the Hemin plus PD98059 group receive PD98059, an inhibitor of ERK1/2 activity, at 10 mg/kg i.p. 6 h after receiving 100 μmol/kg Hemin i.p. Both inhibitors are administered i.p. 2 h before I/R, whereas Hemin was administered 8 h before I/R. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Mol Immunol. 2022 Jan 5.
- Adv Sci (Weinh). 2023 Jun 17;e2206798.
- Genome Biol. 2022 Dec 15;23(1):259.
- Cancer Lett. 2022 Jul 19;215831.
- Int J Nanomedicine. 2021 Feb 26;16:1565-1573.

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REFERENCES

[1]. Vanova K, et al. Heme oxygenase is not involved in the anti-proliferative effects of statins on pancreatic cancer cells. BMC Cancer. 2016 May 12;16:309.

[2]. Chen HH, et al. Heme oxygenase-1 ameliorates kidney ischemia-reperfusion injury in mice through extracellular signal-regulated kinase 1/2-enhanced tubular epithelium proliferation. Biochim Biophys Acta. 2015 Oct;1852(10 Pt A):2195-201.

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