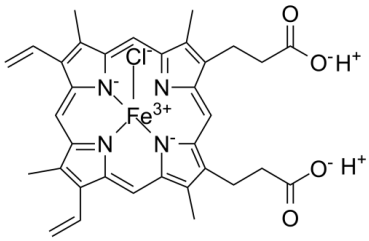
Data Sheet

Product Name:	Hemin
Cat. No.:	CS-5882
CAS No.:	16009-13-5
Molecular Formula:	C34H32ClFeN4O4
Molecular Weight:	651.94
Target:	Autophagy; Ferroptosis; Mitophagy
Pathway:	Apoptosis; Autophagy
Solubility:	DMSO : 17.33 mg/mL (26.58 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

Hemin is an iron-containing porphyrin. Hemin is an **Heme oxygenase (HO)-1** inducer. IC50 & 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]. **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].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: Hemin is dissolved with DMSO and diluted with appropriate media[1].[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]. **Animal Administration:** Hemin is prepared in PBS (Mice)[2].[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.

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.

CAIndexNames:

Ferrate(2-), chloro[7,12-diethenyl-3,8,13,17-tetramethyl-21H,23H-porphine-2,18-dipropanoato(4-)-κN21,κN22,κN23,κN24]-, hydrogen (1:2), (SP-5-13)-

SMILES:

[Cl-][Fe+3]123[N-]4C5=C(CCC([O-])=O)C(C)=C4C=C(C(C=C)=C6C)[N]1=C6C=C(C(C=C)=C7C)[N-]2C7=CC(C(C)=C8CCC([O-])=O)=[N]3C8=C5.[H+].[H+]

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