

Product Data Sheet

Product Name: Ferroprotoporphyrin

Cat. No.: GC49230

Chemical Properties

Cas. No. 14875-96-8

SMILES [O-]C(CCC(C1=CC(C(CCC([O-])=O)=C2C)=[N](C2=C3)[Fe+2]4([N-]5C3=C6C)[N-]1C7=CC(C(C=C)=C8C)=[N]4C8=CC5=C6C=C)=C7C)=O.[H+].[H+]

Formula C₃₄H₃₀FeN₄O₄·2H M.Wt 616.5

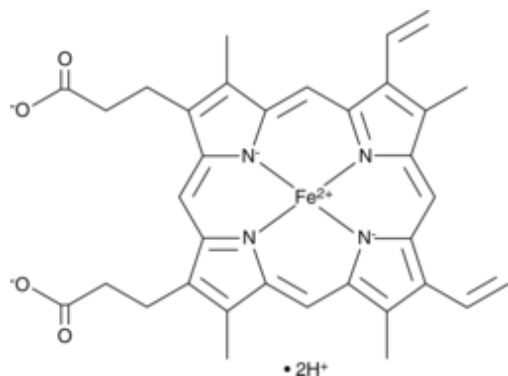
Solubility 10 mg/mL in Water (ultrasonic and adjust pH to 10 with 1M NaOH); < 1 mg/mL in DMSO (insoluble or slightly soluble)

Storage Store at -20°C, protect from light

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines THP-1 cells and BMDMs

Caution: Product has not been fully validated for medical applications. For research use only.

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Preparation Method

Bone marrow-derived macrophages (BMDMs) were isolated from the femurs and tibias of C57BL/6 mice aged 3-5 months. For macrophage differentiation, bone marrow cells were cultured in RPMI-1640 medium supplemented with 20ng/mL recombinant mouse macrophage colony-stimulating factor at 37°C in a humidified atmosphere with 5% CO₂. On day 3, half the medium was replaced with fresh M-CSF-containing medium, and cultures were maintained for a total of 7 days to generate macrophages for downstream applications. THP-1 human monocyte-derived macrophages were cultured in RPMI-1640 medium supplemented with 2mM L-glutamine, 1% penicillin-streptomycin, and 10% FBS. Differentiation into macrophages was induced by treating cells with 100nM phorbol 12-myristate 13-acetate (PMA) for three days. Cells were washed twice with pre-warmed PBS to remove residual PMA and rested for 24 hours before experimental use. Bone marrow-derived macrophages (BMDMs) and THP-1 macrophages were treated with the following stimuli for 6-24h, alone or in combination, as indicated: Ferroprotoporphyrin (10μM), heat-killed *E. coli* (MOI: 20). Cells were treated for the indicated times to assess mitochondrial ROS production using MitoSOX Red, and mitochondrial membrane potential using JC-1 staining. Cell death was evaluated using propidium iodide (PI) staining. Mitochondrial ROS and membrane potential assessments, along with PI staining, were analyzed by flow cytometry, confocal imaging, and a fluorescence plate reader. Cell senescence was determined via β-galactosidase (SA-β-Gal) activity using the Senescence β-Galactosidase Staining Kit. Additionally, the expression of senescence markers was analyzed by Western blot and immunostaining.

Reaction Conditions

10μM; 6-24h

Applications

Treatment of THP-1 cells and BMDMs with Ferroprotoporphyrin induces PANoptosis, triggers mitochondrial dysfunction, upregulates senescence markers (p21, p16, acetylated p53), and causes cell death when combined with heat-killed *E. coli*.

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Animal experiment [2]:

Animal models Male and female Townes' knocked-in transgenic sickle mice

Preparation Method Male and female Townes' knocked-in transgenic sickle mice (SS) and strain controls expressing normal human Hb (AA mice) were used. Mouse genotypes were confirmed by PCR. Freshly prepared Ferroprotoporphyrin solution was protected from light and injected into 12-16 week old mice. A range of doses and times were tested and 3h after injection produced consistent survival with no adverse effects on all strains of mice in this study. The mice were injected in the tail vein with a Ferroprotoporphyrin dose of 50µmoles/kg body weight for SS and AA mice. Control mice received sterile vehicle containing 0.25M NaOH adjusted to pH 7.5 with HCl used in preparation of Ferroprotoporphyrin. Whole organs were harvested from mice 3h after Ferroprotoporphyrin injection. Freshly isolated organs (300mg) were snap-frozen and kept at -80°C for further RT-PCR analysis. Heart IL-6 concentration was measured using the mouse IL-6 ELISA kit.

Dosage form 50µmoles/kg body weight; intravenous injection

Applications Ferroprotoporphyrin significantly increased cardiac IL-6 mRNA expression and induced cardiac hypertrophy in the Townes sickle cell disease (SS) mice model.

References:

[1] Li T, Adams J, Zhu P, et al. The role of heme in sepsis induced Kupffer cell PANoptosis and senescence. *Cell Death Dis.* 2025;16(1):284.

[2] Gbotosho OT, Kapetanaki MG, Ghosh S, Villanueva FS, Ofori-Acquah SF, Kato GJ. Heme Induces IL-6 and Cardiac Hypertrophy Genes Transcripts in Sickle Cell Mice. *Front Immunol.* 2020;11:1910.

Background

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Ferroprotoporphyrin is the ferrous form of heme in hemoglobin [1]. Ferroprotoporphyrin is involved in several biological processes such as the transport or storage of oxygen by hemoglobin or myoglobin, respectively, electron transfer by cytochrome b5, and oxidation of xenobiotics or endogenous substrates by cytochrome P450s (CYPs) [2]. Ferroprotoporphyrin used in experiments is typically prepared by dissolving Hemin in an alkaline solution (such as NaOH) and reducing it to functional Heme [3]. Ferroprotoporphyrin is usually used in studies of iron-overload disorders and ferroptosis-related pathologies [4][5].

In vitro, treatment of THP-1 cells and BMDMs with Ferroprotoporphyrin (10µM; 6-24h) induces PANoptosis, triggers mitochondrial dysfunction, upregulates senescence markers (p21, p16, acetylated p53), and causes cell death when combined with heat-killed *E. coli* [6].

In vivo, Ferroprotoporphyrin (50µmoles/kg body weight; intravenous injection) significantly increased cardiac IL-6 mRNA expression and induced cardiac hypertrophy in the Townes sickle cell disease (SS) mice model [7].

References:

- [1] Pauling L, Coryell CD. The Magnetic Properties and Structure of Hemoglobin, Oxyhemoglobin and Carbonmonoxyhemoglobin. *Proc Natl Acad Sci U S A*. 1936;22(4):210-216.
- [2] Shimizu T, Lengalova A, Martínek V, Martínková M. Heme: emergent roles of heme in signal transduction, functional regulation and as catalytic centres. *Chem Soc Rev*. 2019;48(24):5624-5657.
- [3] Kapetanaki MG, Gbotosho OT, Sharma D, Weidert F, Ofori-Acquah SF, Kato GJ. Free heme regulates placenta growth factor through NRF2-antioxidant response signaling. *Free Radic Biol Med*. 2019;143:300-308.
- [4] Navaneethabalakrishnan S, An X, Vinchi F. Heme- and iron-activated macrophages in sickle cell disease: an updated perspective. *Curr Opin Hematol*. 2024;31(6):275-284.
- [5] Derry PJ, Vo ATT, Gnanansekaran A, et al. The Chemical Basis of Intracerebral Hemorrhage and Cell Toxicity With Contributions From Eryptosis and Ferroptosis. *Front Cell Neurosci*. 2020;14:603043.
- [6] Li T, Adams J, Zhu P, et al. The role of heme in sepsis induced Kupffer cell PANoptosis and senescence. *Cell Death Dis*. 2025;16(1):284.
- [7] Gbotosho OT, Kapetanaki MG, Ghosh S, Villanueva FS, Ofori-Acquah SF, Kato GJ. Heme Induces IL-6 and Cardiac Hypertrophy Genes Transcripts in Sickle Cell Mice. *Front Immunol*.

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