

Product Data Sheet

Product Name: Erastin
Cat. No.: GC16630

Chemical Properties

Cas. No. 571203-78-6

Chemical Name 2-[1-[4-[2-(4-chlorophenoxy)acetyl]piperazin-1-yl]ethyl]-3-(2-ethoxyphenyl)quinazolin-4-one

SMILES O=C1N(C2=CC=CC=C2OCC)C(C(N3CCN(C(COC4=CC=C(Cl)C=C4)=O)CC3)C)=NC5=C1C=CC=C5

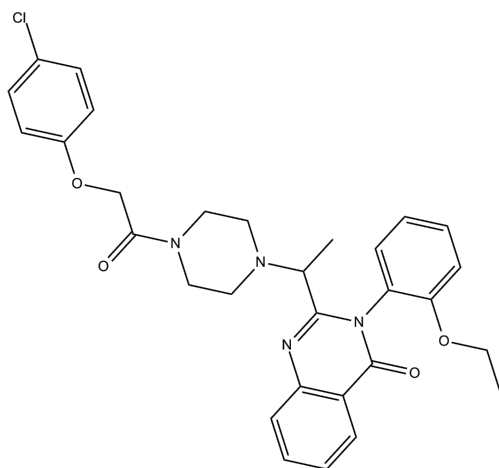
Formula $C_{30}H_{31}ClN_4O_4$ M.Wt 547.04

Solubility $\geq 10.92\text{mg/mL}$ in DMSO with gentle warming ,This product is unstable in solution and it is recommended to prepare and use it immediately. Storage Store at -20°C

General tips This product is unstable in solution and should be prepared immediately before use. We recommend ordering aliquoted pack sizes or aliquoting the product upon receipt.

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

Structure



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

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Protocol

Cell experiment [1]:	
Cell lines	Tu177, FaDu cells (human intrahepatic cholangiocarcinoma cell line)
Preparation Method	Tu177 and FaDu cells were maintained in Dulbecco's modified Eagle's medium at 37°C, 5% CO ₂ . Tu177 and FaDu cells were treated with Erastin (10μM) alone or in combination with BI 2536 (5nM) for 24h, 48h or 72h. After treatment, cell viability was measured by MTS assay, cytotoxicity by LDH release assay, colony formation by clone formation assay, migration by scratch healing assay, invasion by Transwell assay, Fe ²⁺ by iron assay, malondialdehyde by MDA kit, glutathione by GSH kit, mRNA by qRT-PCR, and protein by Western blot for SLC7A11, GPX4, ACSL4.
Reaction Conditions	10μM; 24-72h
Applications	Erastin reduced Tu177 and FaDu cell viability, self-clone formation, invasion and migration abilities. Erastin increased Fe ²⁺ and malondialdehyde content and decreased glutathione content in Tu177 and FaDu cells. Erastin downregulated SLC7A11 and GPX4 mRNA and protein expression and upregulated ACSL4 mRNA and protein expression in Tu177 and FaDu cells.
Animal experiment [2]:	
Animal models	7-8 week old female C57BL/6 mice (20-22g)

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Preparation Method	Endometriosis was induced by autotransplantation of uterine horn fragments onto the peritoneal wall. Postoperative mice received 30µg/kg 17-β-estradiol benzoate intraperitoneally every 3 days for 28 days. At day 14 after surgery, mice were randomized into two groups. The experimental group received 40mg/kg Erastin by intraperitoneal injection once daily for 14 days. The control group received soybean oil instead. At day 28, mice were sacrificed and ectopic lesions were harvested for volume measurement and hematoxylin-eosin staining.
Dosage form	40mg/kg; i.p.; once daily for 14 days
Applications	Erastin administration reduced endometriotic lesion size. Erastin administration did not significantly change mouse body weight. Hematoxylin-eosin staining of lesions showed cystic endometriotic lesions with epithelial and stromal cells.
References: [1] Wu X, Wu J. A polo-like kinase 1 inhibitor enhances erastin sensitivity in head and neck squamous cell carcinoma cells in vitro. Cancer Chemother Pharmacol. 2024;94(2):183-195. [2] Li Y, Zeng X, Lu D, et al. Erastin induces ferroptosis via ferroportin-mediated iron accumulation in endometriosis. Hum Reprod. 2021 Apr;36(4):951-964.	

Background

Erastin is a ferroptosis activator. Erastin inhibits system Xc- to reduce intracellular glutathione levels and weaken GPX4 activity. Erastin binds VDAC2/3 to promote mitochondrial dysfunction and excessive reactive oxygen species production, thereby inducing ferroptosis in tumor cells. Erastin can be used for research on cancer, neurodegenerative diseases and ischemia-reperfusion injury^[1-4].

In vitro, treatment of HeLa and NCI-H1975 cells with 0.1-50µM Erastin for 24 hours dose-dependently reduced cell viability, downregulated GPX4 protein expression, and decreased

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intracellular total glutathione and GSH concentrations^[5]. Treatment of Tu177 cells and FaDu cells with 10 μ M Erastin for 24-72 hours reduced cell viability and colony formation ability, weakened cell invasion and migration ability, and increased Fe²⁺ and malondialdehyde content^[6]. Treatment of HT-29, DLD-1 and Caco-2 cells with 0.1-30 μ M Erastin for 48 hours reduced cell survival rate and colony formation number, increased dead cell proportion, and increased intracellular reactive oxygen species levels^[7].

In vivo, daily intraperitoneal injection of 40mg/kg Erastin for 2 weeks in immunodeficient nude mice subcutaneously implanted with BECN1-overexpressing cells inhibited subcutaneous tumor growth and reduced final tumor volume, increased intratumoral malondialdehyde levels, and decreased intratumoral glutathione levels^[8]. Intraperitoneal injection of 40mg/kg Erastin daily or every other day from day 14 for 14 days in a C57BL/6 mouse model of endometriosis receiving 30 μ g/kg 17- β -estradiol benzoate every 3 days for 28 days reduced ectopic endometriotic lesion volume^[9]. Intraperitoneal injection of 20mg/kg Erastin every other day for 2 weeks in BALB/c-nu mice subcutaneously implanted with SK-OV-3 cells inhibited subcutaneous tumor growth and reduced final tumor volume and weight, decreased intratumoral Ki67-positive cell proportion, and decreased intratumoral GPX4 protein expression^[10].

References:

- [1] Zhao Y, Li Y, Zhang R, et al. The Role of Erastin in Ferroptosis and Its Prospects in Cancer Therapy. *Onco Targets Ther.* 2020 Jun 11;13:5429-5441.
- [2] Koppula P, Zhuang L, Gan B. Cystine transporter SLC7A11/xCT in cancer: ferroptosis, nutrient dependency, and cancer therapy. *Protein Cell.* 2021 Aug;12(8):599-620.
- [3] Gao Z, Du J. Erastin-induced multi-pathway cell death in endometriosis: a mechanistic and translational narrative review. *Front Med (Lausanne).* 2025 Sep 10;12:1594702.
- [4] Gan B. How erastin assassinates cells by ferroptosis revealed. *Protein Cell.* 2023 Mar 16;14(2):84-86.
- [5] Shibata Y, Yasui H, Higashikawa K, et al. Erastin, a ferroptosis-inducing agent, sensitized cancer cells to X-ray irradiation via glutathione starvation in vitro and in vivo. *PLoS ONE.* 2019 Dec 4;14(12):e0225931.

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- [6] Wu X, Wu J. A polo-like kinase 1 inhibitor enhances erastin sensitivity in head and neck squamous cell carcinoma cells in vitro. *Cancer Chemother Pharmacol.* 2024;94:183-195.
- [7] Huo H, Zhou Z, Qin J, et al. Erastin Disrupts Mitochondrial Permeability Transition Pore (mPTP) and Induces Apoptotic Death of Colorectal Cancer Cells. *PLoS ONE.* 2016 May 12;11(5):e0154605.
- [8] Song X, Zhu S, Chen P, et al. AMPK-mediated BECN1 phosphorylation promotes ferroptosis by directly blocking system Xc- activity. *Curr Biol.* 2018 Aug 6;28(15):2388-2399.e5.
- [9] Li Y, Zeng X, Lu D, et al. Erastin induces ferroptosis via ferroportin-mediated iron accumulation in endometriosis. *Hum Reprod.* 2021 Apr;36(4):951-964.
- [10] Li W, Ji T, Ye J, et al. Ferroptosis enhances the therapeutic potential of oncolytic adenoviruses KD01 against cancer. *Cancer Gene Ther.* 2025;32:403-417.

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