

Product Data Sheet

Product Name: Fatostatin A hydrobromide

Cat. No.: GC18121

Chemical Properties

Cas. No. 298197-04-3

Chemical Name 2-(2-propylpyridin-4-yl)-4-(p-tolyl)thiazole hydrobromide

SMILES CCCC1=NC=CC(C2=NC(C3=CC=C(C=C3)C)=CS2)=C1.Br

Formula $C_{18}H_{18}N_2S.HBr$ M.Wt 375.33

Solubility ≥ 19.76 mg/mL in DMSO, ≥ 5.02 mg/mL in EtOH Storage Store at $-20^{\circ}C$

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

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

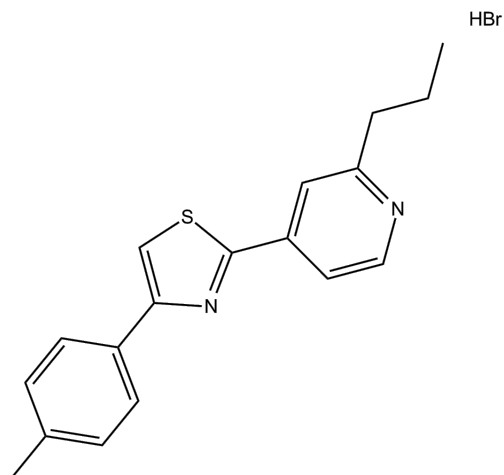
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:

Cell lines HeLa cells (human cervical carcinoma cell line), U87 and T98G (glioblastoma cell lines), MDA-MB-453 (breast cancer cell line), Jurkat T-cells (human T-cell leukemia line)

Preparation Method Cells were maintained in DMEM/F12, Iscove's modified Dulbecco's medium, or RPMI 1640 supplemented with 10% fetal bovine serum (FBS) at 37°C, 5% CO₂. Cells were treated with Fatostatin A hydrobromide at concentrations of 2.11–5μM for 20–48 hours.

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Reaction
Conditions 2.11-5μM; 20-48h

Applications Fatostatin A hydrobromide induced G₂/M phase arrest in cancer cells, disrupted mitotic spindle assembly, and triggered multipolar spindle formation. Fatostatin A hydrobromide inhibited tubulin polymerization, activated the spindle assembly checkpoint, and promoted caspase-3/7-dependent mitotic catastrophe, leading to reduced cell viability. Fatostatin also suppressed SREBP target gene expression (FASN, HMGCS1) independent of its antimitotic effects.

Animal experiment [2]:

Animal models BALB/c nude mice (female, 4-5 weeks old)

Preparation Method Mice were subcutaneously inoculated with IshikawaMR (IshMR) endometrial carcinoma cells (1×10^7 cells/mouse). When tumor diameter reached 5mm, mice were intraperitoneally administered Fatostatin A hydrobromide (25mg/kg) three times per week, either alone or in combination with Medroxyprogesterone acetate (MPA; 100mg/kg every two days) for 4 weeks. Tumor volume and body weight were monitored every two days.

Dosage form 25mg/kg; i.p.; three times per week for 4 weeks

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Applications

Fatostatin A hydrobromide significantly suppressed tumor growth in IshMR xenograft models and enhanced the sensitivity of endometrial cancer to progesterone therapy. Combined treatment with Fatostatin A hydrobromide and MPA synergistically reduced tumor volume, downregulated SREBP1 and Ki-67 expression, and increased cleaved-caspase-3 levels, indicating reversal of progesterone resistance and induction of apoptosis.

References:

- [1] Gholkar AA, Cheung K, Williams KJ, et al. Fatostatin Inhibits Cancer Cell Proliferation by Affecting Mitotic Microtubule Spindle Assembly and Cell Division. *J Biol Chem.* 2016 Aug 12;291(33):17001-8.
- [2] Cai J, Ye Z, Hu Y, et al. Fatostatin induces ferroptosis through inhibition of the AKT/mTORC1/GPX4 signaling pathway in glioblastoma. *Cell Death Dis.* 2023 Mar 25;14(3):211.

Background

Fatostatin A hydrobromide is a compound that specifically inhibits the activity of sterol regulatory element-binding proteins (SREBPs), effectively suppressing the activation of SREBP-1 and SREBP-2^[1]. By binding to the SREBP cleavage-activating protein (SCAP), Fatostatin A hydrobromide inhibits the transport of SREBPs from the endoplasmic reticulum to the Golgi apparatus, ultimately reducing the transcriptional level of intracellular lipogenesis-related genes^[2]. Fatostatin A hydrobromide also regulates glucose and lipid metabolism and suppresses cancer progression^[3-4].

In vitro, treatment of cancer cells (such as HeLa, U87, and SH-SY5Y) with Fatostatin A hydrobromide (2.11–5 μ M) for 24–48 hours inhibits SREBP maturation and induces G₂/M phase arrest. Concurrently, Fatostatin A hydrobromide disrupts mitotic spindle assembly by inhibiting tubulin polymerization, activates the spindle assembly checkpoint, and ultimately leads to multipolar division and caspase 3/7-dependent cell death^[5]. Pretreatment of pancreatic cancer cells (BxPC-3, MIAPaCa-2) with Fatostatin A hydrobromide (10–30 μ M) for 24–72 hours significantly suppresses cell proliferation and clonogenic ability while reducing intracellular lipid synthesis. Furthermore, Fatostatin A hydrobromide induces iron accumulation, increases reactive oxygen species (ROS) and malondialdehyde (MDA) levels, and downregulates GPX4 expression, triggering ferroptosis^[6].

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In vivo, Fatostatin A hydrobromide (25mg/kg) administered via tail vein injection in a 6-week-old BALB/c nude mouse model of intracranial glioblastoma (three times per week for 4 weeks) significantly inhibits tumor growth and prolongs survival^[7]. Fatostatin A hydrobromide (25mg/kg; injected three times a week for 4 weeks) was administered intraperitoneally to 4-week-old BALB/c nude mice to establish a xenograft model of endometrial cancer. Fatostatin A hydrobromide significantly inhibited tumor growth and enhanced sensitivity to progesterone therapy^[8].

References:

- [1] Xue L, Qi H, Zhang H, et al. Targeting SREBP-2-Regulated Mevalonate Metabolism for Cancer Therapy. *Front Oncol*. 2020 Aug 21;10:1510.
- [2] Soyal SM, Nofziger C, Dossena S, et al. Targeting SREBPs for treatment of the metabolic syndrome. *Trends Pharmacol Sci*. 2015 Jun;36(6):406-16.
- [3] Li M, Lu Q, Zhu Y, et al. Fatostatin inhibits SREBP2-mediated cholesterol uptake via LDLR against selective estrogen receptor α modulator-induced hepatic lipid accumulation. *Chem Biol Interact*. 2022 Sep 25;365:110091.
- [4] Yao L, Chen S, Li W. Fatostatin inhibits the development of endometrial carcinoma in endometrial carcinoma cells and a xenograft model by targeting lipid metabolism. *Arch Biochem Biophys*. 2020 May 15;684:108327.
- [5] Gholkar AA, Cheung K, Williams KJ, et al. Fatostatin Inhibits Cancer Cell Proliferation by Affecting Mitotic Microtubule Spindle Assembly and Cell Division. *J Biol Chem*. 2016 Aug 12;291(33):17001-8.
- [6] Cao R, Feng Z, Mo J, et al. Pharmacological inhibition of SREBP1 suppresses pancreatic cancer growth via inducing GPX4-mediated ferroptosis. *Cell Signal*. 2024 Dec;124:111381.
- [7] Cai J, Ye Z, Hu Y, et al. Fatostatin induces ferroptosis through inhibition of the AKT/mTORC1/GPX4 signaling pathway in glioblastoma. *Cell Death Dis*. 2023 Mar 25;14(3):211.
- [8] Ma X, Zhao T, Yan H, et al. Fatostatin reverses progesterone resistance by inhibiting the SREBP1-NF- κ B pathway in endometrial carcinoma. *Cell Death Dis*. 2021 May 26;12(6):544.

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