

Product Data Sheet

Product Name: Trichostatin A (TSA)

Cat. No.: GC15526

Chemical Properties

Cas. No. 58880-19-6

Chemical Name (2E,4E,6R)-7-[4-(dimethylamino)phenyl]-N-hydroxy-4,6-dimethyl-7-oxohepta-2,4-dienamide

SMILES CC(C=C(C)C=CC(=O)NO)C(=O)C1=CC=C(C=C1)N(C)C

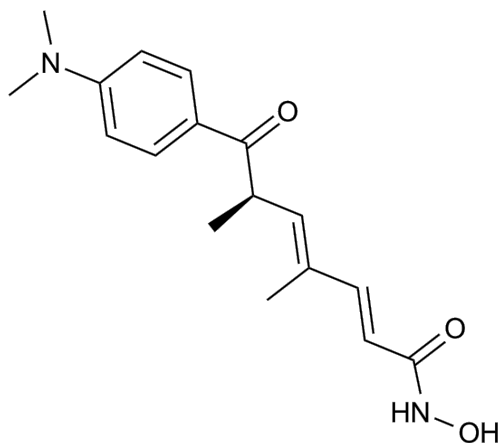
Formula $C_{17}H_{22}N_2O_3$ M.Wt 302.37

Solubility $\geq 15.12\text{mg/mL}$ in DMSO, ≥ 16.56 mg/mL in EtOH with ultrasonic Storage Desiccate at -20°C

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

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

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Cell experiment [1]:

Cell lines Human small cell lung cancer DMS53 cells

DMS53 cells were maintained in Waymouth's MB752/1 medium supplemented Preparation Method with 10% fetal bovine serum at 37°C, 5% CO₂. Cells were plated at 50-60% confluence and treated with Trichostatin A (25-400nM) for 2 days.

Reaction
Conditions 25-400nM; 2 days

Applications Trichostatin A dose-dependently increased acetylated histone H4 levels, induced morphological changes (flattened, rounded, spindle shapes with distinct cellular borders and neuronal-like projections), reduced cell numbers, inhibited cell proliferation (complete growth suppression at 100, 200, and 400nM), upregulated p21 and p27 protein levels, downregulated Bcl-2 expression, and increased cleaved PARP levels.

Animal experiment [2]:

Animal models Female C57BL/6 mice (8 weeks old)

Mice were exposed to cigarette smoke twice daily for 30 days using a whole-body mainstream smoke exposure system (6 cigarettes per session, mixed with room air and delivered into the chamber over 120min), with a 3-day Preparation Method acclimatization period before the experiment (exposed to cigarette smoke from 1 cigarette for 20min twice daily). Trichostatin A (0.6mg/kg) was intraperitoneally injected into cigarette smoke-exposed mice on alternate days for 30 days. Vaginal exfoliative cells were examined to confirm the estrous cycle at the end of exposure, and mice in the estrous phase were euthanized for uterine collection.

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Dosage form 0.6mg/kg; i.p.; every other day for 30 days

Applications Trichostatin A attenuated cigarette smoke-induced loss of body weight and uterine wet weight, restored the thickness of the myometrium and the number of glandular and interstitial cells in the uterus. Trichostatin A inhibited cigarette smoke-induced HDAC1/2 activation in uterine smooth muscle, glandular endothelial and luminal endothelial cells, restored macro-autophagy markers (elevated LC3-II and Beclin-1 levels, reduced p62 accumulation), limited phosphorylated mTOR and IRS activation, and inhibited cigarette smoke-induced FOXO1, FOXO3 and caspase-9 activation in the uterus.

References:

- [1] Scarpellini C, Klejborowska G, Lanthier C, et al. Beyond ferrostatin-1: a comprehensive review of ferroptosis inhibitors. Trends Pharmacol Sci. 2023 Dec;44(12):902-916.
- [2] Miotto G, Rossetto M, Di Paolo ML, et al. Insight into the mechanism of ferroptosis inhibition by ferrostatin-1. Redox Biol. 2020 Jan;28:101328.

Background

Trichostatin A (TSA) is an antifungal antibiotic with histone deacetylase (HDAC) inhibitory activity ($IC_{50}=1.8nM$). Trichostatin A induces hyperacetylation of histone H4 through inhibition of HDAC activity, thereby regulating gene transcription, inducing cell cycle arrest and apoptosis. Trichostatin A exerts neuroprotective effects through antioxidant, anti-inflammatory and epigenetic regulation. Trichostatin A can be used in research related to cancer and Alzheimer's disease^[1-4].

In vitro, Trichostatin A (25-400nM) was applied to DMS53 cells for 2 days. Trichostatin A dose-dependently increased acetylated histone H4 levels and inhibited HDAC activity in DMS53 cells. Trichostatin A induced morphological changes including round, spindle and flat shapes, and suppressed cell proliferation^[5]. Trichostatin A (1 μ M) combined with cisplatin (8 μ M) was applied to A549 cells for 48h. Trichostatin A enhanced cisplatin-induced late apoptosis and G2/M phase arrest.

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Trichostatin A synergized with cisplatin to activate INF signaling, reduce cell migration ability, and promote ferroptosis^[6].

In vivo, Trichostatin A (0.6mg/kg; every other day injection) was intraperitoneally injected into female C57BL/6 mice exposed to cigarette smoke for 30 days. Trichostatin A inhibited HDAC1/2 activation in mouse uterine tissue, alleviated cigarette smoke-induced decreases in body weight and uterine wet weight, and restored myometrial thickness and the numbers of glandular and interstitial cells. Trichostatin A restored macroautophagy markers LC3-II and Beclin-1 expression, and inhibited phosphorylation of mTOR and IRS, as well as activation of FOXO1, FOXO3 and caspase-9^[7]. One hour before mechanical ventilation, Trichostatin A (2mg/kg) was intraperitoneally injected into bleomycin-pretreated C57BL/6 mice and Akt-deficient C57BL/6 mice, followed by low tidal volume (6mL/kg) or high tidal volume (30mL/kg) mechanical ventilation with room air for 5h. Trichostatin A attenuated high tidal volume ventilation-induced alveolar capillary microvascular leakage and increased lung water in mice. Trichostatin A improved lung structure destruction and narrowed the alveolar-arterial oxygen gradient^[8].

References:

- [1] Vigushin DM, Ali S, Pace PE, et al. Trichostatin A is a histone deacetylase inhibitor with potent antitumor activity against breast cancer in vivo. Clin Cancer Res. 2001 Apr;7(4):971-6.
- [2] Karolczak-Bayatti M, Abu-Amara TM, Europe-Finner GN. Expression of the GTP-binding protein G α s in human myometrial cells is regulated by ubiquitination and protein degradation: involvement of proteasomal inhibition by trichostatin A. Reprod Sci. 2012 Dec;19(12):1323-31.
- [3] Hu X, Zhang X, Dai L, et al. Histone deacetylase inhibitor trichostatin A promotes the osteogenic differentiation of rat adipose-derived stem cells by altering the epigenetic modifications on Runx2 promoter in a BMP signaling-dependent manner. Stem Cells Dev. 2013 Jan 15;22(2):248-55.
- [4] Azechi T, Kanehira D, Kobayashi T, et al. Trichostatin A, an HDAC class I/II inhibitor, promotes Pi-induced vascular calcification via up-regulation of the expression of alkaline phosphatase. J Atheroscler Thromb. 2013;20(6):538-47.
- [5] Platta CS, Greenblatt DY, Kunnimalaiyaan M, et al. The HDAC inhibitor trichostatin A inhibits growth of small cell lung cancer cells. J Surg Res. 2007 Oct;142(2):219-226.

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[6] Ding J, Liu B, Han P, et al. Trichostatin A inhibits uterine histomorphology alterations induced by cigarette smoke exposure in mice. Life Sci. 2019 Jul;228:112-120.

[7] Zhang L, Dong Z, Jiang F, et al. Ferrostatin-1 ameliorates Cis-dichlorodiammineplatinum(II)-induced ovarian toxicity by inhibiting ferroptosis. Molecular Medicine. 2024 Sep;30:150.

[8] Li L-F, Lee C-S, Lin C-W, et al. Trichostatin A attenuates ventilation-augmented epithelial-mesenchymal transition in mice with bleomycin-induced acute lung injury by suppressing the Akt pathway. PLoS ONE. 2017 Feb;12(2):e0172571.

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