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Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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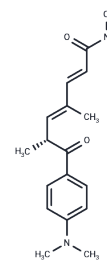
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Trichostatin A

Chemical Properties

CAS No. :	58880-19-6
Formula:	C ₁₇ H ₂₂ N ₂ O ₃
Molecular Weight:	302.37
Appearance:	no data available
Storage:	store at low temperature, store under nitrogen Powder: -20°C for 3 years In solvent: -80°C for 1 year



Biological Description

Description	Trichostatin A (TSA) is a natural derivative of diene isohydroxamic acids. Trichostatin A is a histone deacetylase inhibitor (IC ₅₀ =1.8 nM) that is reversible and specific. Trichostatin A leads to the hyperacetylation of core histones, which regulates chromatin structure.
Targets(IC ₅₀)	HDAC
In vitro	<p>METHODS: Eight breast cancer cells MCF-7, T-47D, ZR-75-1, BT-474, MDA-MB-231, MDA-MB-453, CAL 51 and SK-BR-3 were treated with Trichostatin A (10⁻¹² -10⁻⁵ M) for 96 h. The viability of the cells was determined by SRB. Cell viability was determined by SRB</p> <p>RESULTS: Trichostatin A inhibited the proliferation of eight breast cancer cell lines with a mean IC₅₀=124.4±120.4 nM (range 26.4-308.1 nM). [1]</p> <p>METHODS: Esophageal squamous cell carcinoma cells EC9706 and EC1 were treated with Trichostatin A (0.3-1 μM) for 48 h. Apoptosis was detected using Flow Cytometry.</p> <p>RESULTS: There was no significant increase in the percentage of early apoptosis at 0.3 and 0.5 μM Trichostatin A doses. However, 1.0 μM Trichostatin A treatment significantly induced early apoptosis compared with control. In addition, the percentage of mid- and late-stage apoptosis increased in a concentration-dependent manner. [2]</p> <p>METHODS: Esophageal squamous cell carcinoma cells EC9706 and EC1 were treated with Trichostatin A (0.3-1 μM) for 60 min, and the expression levels of target proteins were detected using Western Blot.</p> <p>RESULTS: Trichostatin A decreased the protein levels of PI3K as well as p-Akt and p-ERK1/2 in a dose-dependent manner. acetylation of histone H4 was increased in a concentration-dependent manner. [2]</p>
In vivo	<p>METHODS: To assay antitumor activity in vivo, Trichostatin A (500 μg/kg) was injected subcutaneously into rats with NMU-induced mammary carcinoma tumors once daily for four weeks.</p> <p>RESULTS: Trichostatin A showed significant antitumor activity in vivo. tumors in Trichostatin A-treated rats had benign phenotypes, fibroadenomas or tubular adenomas, suggesting that the antitumor activity of Trichostatin A may be attributable to induction of differentiation. [1]</p> <p>METHODS: To assay antitumor activity in vivo, Trichostatin A (0.5-1 mg/kg twice weekly) and Quercetin (10 mg/kg three times weekly) were injected intraperitoneally into nude mice bearing human lung adenocarcinoma tumor A549 for thirteen weeks.</p> <p>RESULTS: High-dose Trichostatin A significantly inhibited tumor growth, while low-dose Trichostatin A and Quercetin alone had no effect. However, the combination of low-dose</p>

Trichostatin A and Quercetin significantly inhibited tumor growth. [3]

Kinase Assay	In vitro HDAC activity: Total cellular extracts are prepared from each breast cancer cell line (MCF-7, T-47D, ZR-75-1, BT-474, MDA-MB-231, MDA-MB-453, CAL 51, or SK-BR-3). A 20 μ L crude cell extract ($\sim 2.5 \times 10^5$ cells), in the presence of varying concentrations of Trichostatin A in 0.1% (v/v) ethanol or 0.1% (v/v) ethanol as vehicle control, are incubated for 60 minutes at 25 $^{\circ}$ C with 1 μ L ($\sim 1.5 \times 10^6$ cpm) of [3 H]acetyl-labeled histone H4 peptide substrate (NH ₂ -terminal residues 2-20) that has been acetylated with [3 H]acetic acid, sodium salt (3.7 GBq/mmol) by an in vitro incorporation method. Each 200 μ L reaction is quenched with 50 μ L of 1 M HCl/0.16 M acetic acid and extracted with 600 μ L of ethyl acetate, and released [3 H]acetate is quantified by scintillation counting. IC ₅₀ values are determined graphically using nonlinear regression to fit inhibition data to the appropriate dose-response curve.
Cell Research	Cells are exposed to various concentrations of Trichostatin A for 96 hours. After treatment, cell proliferation is estimated using the sulforhodamine B colorimetric assay. Cell viability is determined by trypan blue exclusion. (Only for Reference)

Solubility Information

Solubility	DMSO: 15.1 mg/mL (50 mM), Ethanol: 3 mg/mL (10 mM), (< 1 mg/ml refers to the product slightly soluble or insoluble)
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Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.3072 mL	16.536 mL	33.0721 mL
5 mM	0.6614 mL	3.3072 mL	6.6144 mL
10 mM	0.3307 mL	1.6536 mL	3.3072 mL
50 mM	0.0661 mL	0.3307 mL	0.6614 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

Wang C, Huang M, Lin Y, et al. ENO2-derived phosphoenolpyruvate functions as an endogenous inhibitor of HDAC1 and confers resistance to antiangiogenic therapy. *Nature Metabolism*. 2023: 1-22. Vigushin DM, et al.

Inhibitor · Natural Compounds · Compound Libraries · Recombinant Proteins

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