Trichostatin A

Cat. No.: HY-15144

CAS No.: 58880-19-6

Molecular Formula: $C_{17}H_{22}N_2O_3$

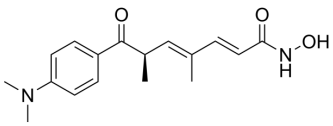
Molecular Weight: 302.37

Target: HDAC; Organoid

Pathway: Cell Cycle/DNA Damage; Epigenetics; Stem Cell/Wnt

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (165.36 mM; Need ultrasonic)
Methanol : 2 mg/mL (6.61 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.3072 mL	16.5360 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

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (8.27 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.27 mM); Clear solution
3. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: 2.5 mg/mL (8.27 mM); Suspended solution; Need ultrasonic
4. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.27 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Trichostatin A (TSA) is a potent and specific inhibitor of HDAC class I/II, with an IC_{50} value of 1.8 nM for HDAC[1].

IC_{50} & Target

HDAC
1.8 nM (IC_{50})

In Vitro	Trichostatin A is a potent and specific inhibitor of HDAC class I/II, with an IC_{50} value of 1.8 nM for HDAC. Trichostatin A (TSA) inhibits proliferation of eight breast carcinoma cell lines with mean±SD IC_{50} of 124.4±120.4 nM (range, 26.4-308.1 nM). HDAC inhibitory activity of Trichostatin A is similar in all cell lines with mean IC_{50} of 2.4±0.5 nM (range, 1.5-2.9 nM)[1]. Trichostatin A (330 nM) increases Gαs protein expression in human myometrial cells, but does not increase Gαs mRNA levels[2]. Trichostatin A (20-75 nM) induces minimal cytotoxicity to adipose-derived stem cells (ADSCs), and enhances the osteogenic differentiation capacity of ADSCs[3]. In addition, Trichostatin A (0, 10, 100, 500 nM) dose-dependently decreases HDAC class I/II activity[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Trichostatin A (500 μg/kg, s.c.) pronounces antitumor activity without causing any measurable toxicity in doses of up to 5 mg/kg by s.c. injection, in randomized controlled efficacy studies using the N-methyl-N-nitrosourea carcinogen-induced rat mammary carcinoma model[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	Cells are cultured in a 96-well plate at 1×10^3 cells per well with 100 μL complete DMEM in the presence or absence of a HDAC inhibitor Trichostatin A for 72 h. Cytotoxicity is measured by performing WST-8 assay using a CCK-8 cell proliferation kit. The 450 nm absorbance is measured with a microplate reader. All experiments are carried out in triplicate and 3 independent experiments are performed[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Rats[1] Twelve rats are randomized to receive 500 μg/kg Trichostatin A in 50 μL DMSO, or 50 μL DMSO as vehicle control, by s.c. injection twice weekly for 4 weeks. In subsequent studies, 30 rats are randomized to receive Trichostatin A 500 μg/kg in 50 μL DMSO, or 50 μL DMSO as vehicle control, by s.c. injection daily for 4 weeks. Weekly tumor measurements, estimated tumor volumes, and body mass are recorded for each animal. Animals are sacrificed at the end of the 4-week study period; palpable tumors are resected and immediately snap-frozen in liquid nitrogen. Animals with tumors <2 cm in diameter or ulcerating tumors are withdrawn from study[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Immunol. 2023 Jan;24(1):162-173.
- Cell Metab. 2021 May 4;33(5):988-1000.e7.
- Nat Biomed Eng. 2018 Aug;2(8):578-588.
- Mil Med Res. 2022 Aug 23;9(1):46.
- Circ Res. 2022 Aug 19;131(5):456-472.

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REFERENCES

[1]. Vigushin DM 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, et al. Expression of the GTP-Binding Protein Gαs in Human Myometrial Cells is Regulated by Ubiquitination and Protein Degradation: Involvement of Proteasomal Inhibition by Trichostatin A.,Reprod Sci. 2012 Aug 8.

[3]. Hu X, 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. 2012 Aug 8.

[4]. Azechi 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.

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