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Zuschläge

- Mindermengenzuschlag
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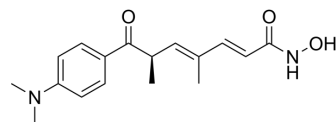
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Trichostatin A

Cat. No.:	HY-15144
CAS No.:	58880-19-6
Molecular Formula:	C ₁₇ H ₂₂ N ₂ O 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)

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	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

- 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
- 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
- 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
- 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 IC₅₀ value of 1.8 nM for HDAC^[1].

IC₅₀ & Target

HDAC
1.8 nM (IC₅₀)

In Vitro	<p>Trichostatin A is a potent and specific inhibitor of HDAC class I/II, with an IC₅₀ value of 1.8 nM for HDAC. Trichostatin A (TSA) inhibits proliferation of eight breast carcinoma cell lines with mean±SD IC₅₀ 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 IC₅₀ of 2.4±0.5 nM (range, 1.5-2.9 nM)^[1]. Trichostatin A (330 nM) increases Gas protein expression in human myometrial cells, but does not increase Gas 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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
In Vivo	<p>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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

PROTOCOL

Cell Assay ^[3]	<p>Cells are cultured in a 96-well plate at 1×10³ 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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
Animal Administration ^[1]	<p>Rats^[1]</p> <p>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].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

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 Gas 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.

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

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