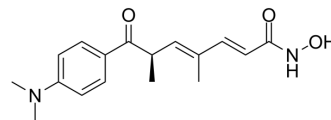


Trichostatin A

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		
Pathway:	Cell Cycle/DNA Damage; Epigenetics		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro	DMSO : 25 mg/mL (82.68 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	<ol style="list-style-type: none"> 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 IC ₅₀ value of 1.8 nM for HDAC ^[1] .
IC₅₀ & Target	HDAC 1.8 nM (IC ₅₀)

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 Biomed Eng. 2018 Aug;2(8):578-588.
- Nat Commun. 2021 Jul 9;12(1):4227.
- Nat Commun. 2021 Feb 23;12(1):1237.
- Nucleic Acids Res. 2020 May 21;48(9):4858-4876.
- Cancer Res. 2021 Jan 25;canres.2808.2020.

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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.

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