Trichostatin A

Cat. No.: HY-15144
CAS No.: 58880-19-6
Molecular Formula: C₁₇H₂₂N₂O₃
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)

Preparing Stock Solutions

<table>
<thead>
<tr>
<th>Concentration</th>
<th>Mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>3.3072 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.6614 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.3307 mL</td>
</tr>
</tbody>
</table>

1 mg      5 mg      10 mg

<table>
<thead>
<tr>
<th>Concentration</th>
<th>Mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>16.5360 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>3.3072 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>1.6536 mL</td>
</tr>
</tbody>
</table>

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

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 HDAC¹.

IC₅₀ & Target
HDAC
1.8 nM (IC₅₀)

In Vitro
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 Gαs protein expression in human myometrial cells, but does not

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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\(^4\).

**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\(^1\).

**PROTOCOL**

**Cell Assay**\(^3\)

Cells are cultured in a 96-well plate at \(1 \times 10^3\) 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**

- Proc Natl Acad Sci U S A. 2016 Sep 6;113(36):9967-76.
- Theranostics. 2019 Apr 13;9(9):2424-2438.

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**REFERENCES**


[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