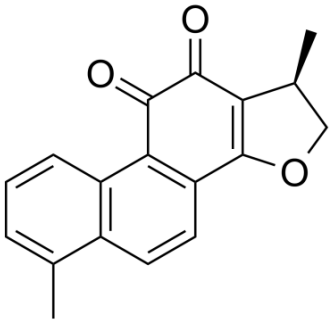
Dihydrotanshinone I

Cat. No.:	HY-N0360
CAS No.:	87205-99-0
Molecular Formula:	$C_{18}H_{14}O_3$
Molecular Weight:	278.3
Target:	SARS-CoV
Pathway:	Anti-infection
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 : 4.76 mg/mL (17.10 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.5932 mL	17.9662 mL	35.9324 mL
	5 mM	0.7186 mL	3.5932 mL	7.1865 mL
	10 mM	0.3593 mL	1.7966 mL	3.5932 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: 0.48 mg/mL (1.72 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 0.48 mg/mL (1.72 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.48 mg/mL (1.72 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dihydrotanshinone I is a natural compound extracted from Salvia miltiorrhiza Bunge which has been widely used for treating cardiovascular diseases. Dihydrotanshinone I exhibits entry-blocking effect for MERS-CoV.

In Vitro

In lipopolysaccharide (LPS)-stimulated human umbilical vein endothelial cells (HUVECs), DHT (10 nM) decreases lectin-like ox-LDL receptor-1 (LOX-1) and NADPH oxidase 4 (NOX4) expression, reactive oxygen species (ROS) production, NF-κB nuclear translocation, ox-LDL endocytosis and monocytes adhesion[1]. Dihydrotanshinone I induces caspase dependent apoptosis induced in HCT116 cells. Dihydrotanshinone I induces concentration and ROS dependent caspase activation.

	Apoptosis induced by Dihydrotanshinone I is completely prevented by Z-VAD-fmk. Apoptosis induced by Dihydrotanshinone I is significantly inhibited by pretreatment of Z-LEHD-fmk but only is partially inhibited by Z-IETD-fmk. Apoptosis induced by Dihydrotanshinone I is significantly increased by caspase-2 knockdown[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	DHT (10 and 25 mg/kg) significantly attenuates atherosclerotic plaque formation, alteres serum lipid profile, decreases oxidative stress and shrinks necrotic core areas in ApoE$^{-/-}$ mice. DHT dramatically inhibits the enhanced expression of LOX-1, NOX4, and NF-κB in aorta[1]. Dihydrotanshinone I (1, 2, 4 mg/kg) treatment can improve cardiac function, reduce infarct size, ameliorate the variations in myocardial zymogram and histopathological disorders, decrease 20-HETE generation, and regulate apoptosis-related protein in myocardial ischemia-reperfusion rats[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [3]	Cells are treated with various concentrations of Dihydrotanshinone I (3.13-20 μM) for 48 h. For the activity assay, Ac-DEVD-AMC (1 μg/μL), Ac-IETD-AMC (1 μg/μL) or Ac-LEDH-AMC (1 μg/μL) and cell lysate are added into Protease Assay Buffer in 96-well plate. Reaction mixtures with lysis buffer are used as negative controls. Cells treated with DMSO (0.1%) are treated as vehicle control. The reaction mixtures are incubated for 1 h at 37°C. The AMC liberated from the substrates is measured using spectrofluorometer of Victor 2 plate reader with an excitation wavelength of 380 nm and an emission wavelength of 430 nm. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Male ApoE$^{-/-}$ mice (6-8 weeks old) on C57BL/6J background and age-matched wild-type C57BL/6J controls housed in SPF-grade animal facilities with a 12 h light/dark cycle, at 23°C (±2°C). Starting from 6 weeks, the mice are fed with a HCD (54.35% raw grain, 20% lard, 0.15% cholesterol, 15% sucrose, 0.5% Sodium Cholate, 10% yolk powder) for 12 weeks. All ApoE$^{-/-}$ mice are dosed daily via intragastric gavage with 10 and 25 mg/kg Dihydrotanshinone I dissolved in 0.5% CMC-Na or administered 0.5% CMC-Na alone (vehicle control) (n=8 per group). MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nucleic Acids Res. 2020 Nov 9;gkaa969.
- Cell Mol Life Sci. 2020 Aug 13.

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REFERENCES

[1]. Zhao W, et al. Dihydrotanshinone I Attenuates Atherosclerosis in ApoE-Deficient Mice: Role of NOX4/NF-κB Mediated Lectin-Like Oxidized LDL Receptor-1 (LOX-1) of the Endothelium. Front Pharmacol. 2016 Nov 8;7:418. eCollection 2016.

[2]. Wei Y, et al. The cardioprotection of dihydrotanshinone I against myocardial ischemia-reperfusion injury via inhibition of arachidonic acid ω-hydroxylase. Can J Physiol Pharmacol. 2016 Dec;94(12):1267-1275. Epub 2016 Jun 24.

[3]. Wang L, et al. Dihydrotanshinone I induced apoptosis and autophagy through caspase dependent pathway in colon cancer. Phytomedicine. 2015 Nov 15;22(12):1079-87

[4]. Ji Yeun Kim, et al. Safe, High-Throughput Screening of Natural Compounds of MERS-CoV Entry Inhibitors Using a Pseudovirus Expressing MERS-CoV Spike Protein. Int J Antimicrob Agents. 2018 Nov;52(5):730-732.

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

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