Thiomyristoyl

Cat. No.: HY-101278

CAS No.: 1429749-41-6

Molecular Formula: $C_{34}H_{51}N_3O_3S$

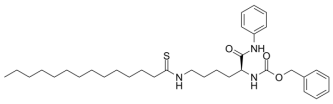
Molecular Weight: 581.85

Target: Sirtuin

Pathway: Cell Cycle/DNA Damage; Epigenetics

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 : ≥ 32 mg/mL (55.00 mM)

Ethanol : 15.29 mg/mL (26.28 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.7187 mL	8.5933 mL	17.1866 mL
5 mM	0.3437 mL	1.7187 mL	3.4373 mL
10 mM	0.1719 mL	0.8593 mL	1.7187 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 (4.30 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Thiomyristoyl is a potent and specific SIRT2 inhibitor with an IC_{50} of 28 nM.

IC_{50} & Target

SIRT2	SIRT1
28 nM (IC_{50})	98 μM (IC_{50})

In Vitro

Thiomyristoyl (TM) is a potent SIRT2-specific inhibitor with broad anticancer activity but little effect on non-cancerous cells. SIRT2-inhibition promotes c-Myc ubiquitination and degradation, suggesting the therapeutic potential of TM to target certain c-Myc-driven cancers. TM could inhibit SIRT2 with an IC_{50} of 28 nM, but inhibits SIRT1 with an IC_{50} value of 98 μM and

	does not inhibit SIRT3 even at 200 μM. TM inhibits three human breast cancer cell lines, MCF-7, MDA-MB-468, and MDA-MB-231[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	TM inhibits tumor growth in mouse models of breast cancer. TM does not cause significant toxicity in mice and no significant weight loss is observed in TM-treated mice. S5H, the acetyl-a-tubulin level is moderately but statistically significantly increased in tumors from TM-treated mice compared with those from vehicle-treated mice, suggesting that TM indeed inhibits SIRT2 in vivo[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Cells are seeded into 96-well plates at 3,000–4,000 cells per well. After 24 hr, test compounds (Thiomyristoyl) are added to cells to final concentrations ranging from 1 to 50 μM. Cells are then incubated for 72 hr and cell viability is measured using the CellTiter-Blue viability assay. Relative cell viability in the presence of test compounds is normalized to the vehicle-treated controls after background subtraction. GraphPad Prism software is used to determine the IC_{50} values. Knockdown of SIRT1-7 in various cell lines is achieved by lentiviral infection[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Pharmacol Sin. 2021 Apr 13.
- Int Immunopharmacol. 2021 Jan;90:107212.
- Neoplasia. 2019 Mar 29;21(5):429-441.
- Prostate. 2022 Sep 8.

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REFERENCES

[1]. Jing H, et al. A SIRT2-Selective Inhibitor Promotes c-Myc Oncoprotein Degradation and Exhibits Broad Anticancer Activity. Cancer Cell. 2016 Mar 14;29(3):297-310.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA