Tubacin

Cat. No.: HY-13428

CAS No.: 537049-40-4

Molecular Formula: $C_{41}H_{43}N_3O_7S$

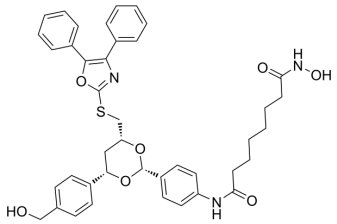
Molecular Weight: 721.86

Target: HDAC; Virus Protease; Beta-lactamase

Pathway: Cell Cycle/DNA Damage; Epigenetics; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (138.53 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.3853 mL	6.9266 mL	13.8531 mL
5 mM	0.2771 mL	1.3853 mL	2.7706 mL
10 mM	0.1385 mL	0.6927 mL	1.3853 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 (3.46 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.46 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tubacin is a potent and selective inhibitor of HDAC6, with an IC_{50} value of 4 nM and approximately 350-fold selectivity over HDAC1. Tubacin also inhibits metallo-β-lactamase domain-containing protein 2 (MBLAC2).

IC_{50} & Target

HDAC6 4 nM (IC_{50})	HDAC3 1.27 μM (IC_{50})	HDAC8 1.27 μM (IC_{50})	HDAC1 1.40 μM (IC_{50})
HDAC5 3.35 μM (IC_{50})	HDAC10 3.71 μM (IC_{50})	HDAC11 3.79 μM (IC_{50})	HDAC9 4.31 μM (IC_{50})
HDAC2	HDAC7	HDAC4	

	6.27 μM (IC_{50})	9.70 μM (IC_{50})	17.30 μM (IC_{50})

In Vitro

Tubacin preferentially induces α-tubulin hyperacetylation at a concentration of 2.5 μM, and induces α-tubulin acetylation at 5 μM and protects prostate cancer (LNCaP) cells from hydrogen peroxide-induced death at 8 μM via peroxiredoxin acetylation[1]. Tubacin (2.5 and 5 μM) specifically induces acetylation of α-tubulin in MM cells. Tubacin significantly inhibits both drug-sensitive and drug-resistant MM cell growth, with IC_{50} 5-20 μM at 72 h. Tubacin also induces apoptosis by activation of caspases. Moreover, Tubacin inhibits binding of HDAC6 with dynein, and it induces significant accumulation of polyubiquitinated proteins, when combined with bortezomib. Tubacin and bortezomib induce synergistic antitumor activity in MM cell lines, and inhibits paracrine MM Cell Growth. Tubacin (5 μM) synergistically enhances bortezomib-induced cytotoxicity in patient MM cells without cytotoxicity to PBMCs[2]. Tubacin can concentration-dependently inhibits JEV-induced cytopathic effect and apoptosis, as well as reduces virus yield in human cerebellar medulloblastoma cells. The IC_{50} of virus yield is 0.26 μM for Tubacin. Tubacin also meaningfully blocks the production of intracellular infectious virus particles, with an IC_{50} of 1.52 μM. Tubacin induces the hyperacetylation of a HDAC6 substrate Hsp90 and reduces the interaction of Hsp90 with JEV NS5 protein[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]

HDAC inhibitors TSA, VPA, tubacin, and TBSA are used in the assay. Cytotoxicity of HDACi to TE671 and BHK-21 cells is evaluated by MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) assay. 5×10^4 cells per well are seeded in 96-well plates and then treated with the indicated concentration of each HDACi. After 48-h of treatment, 25 μL of MTT solution (5 mg/mL) is added to each well and incubated at 37 °C with 5% CO2 for 3 h. After three washings with phosphate buffer saline (PBS), 100 μL DMSO is added into each well for dissolving formazan crystals. OD570−630 is measured by micro-ELISA reader and survival rate are calculated to indicate suppressive effects of each HDACi on the survival of TE671 and BHK-21 cells. Survival rate (%) = ((Acontrol − Aexperiment)/Acontrol) × 100%. 50% cytotoxic concentration (CC_{50}) values are calculated by computer program[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2021 Jan 28;12(1):662.
- Genes Dev. 2020 Feb 1;34(3-4):194-208.
- JCI Insight. 2021 Dec 7;e153948.
- Biomed Pharmacother. 2019 Jun;114:108805.
- Apoptosis. 2020 Oct;25(9-10):697-714.

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REFERENCES

[1]. Severin Lechner, et al. Target deconvolution of HDAC pharmacopoeia reveals MBLAC2 as common off-target. Nat Chem Biol. 2022 Apr 28.

[2]. Butler KV, et al. Rational design and simple chemistry yield a superior, neuroprotective HDAC6 inhibitor, tubastatin A. J Am Chem Soc. 2010 Aug 11;132(31):10842-6.

[3]. Hideshima T, et al. Small-molecule inhibition of proteasome and aggresome function induces synergistic antitumor activity in multiple myeloma. Proc Natl Acad Sci U S A. 2005 Jun 14;102(24):8567-72. Epub 2005 Jun 3.

[4]. Lu CY, et al. Tubacin, an HDAC6 Selective Inhibitor, Reduces the Replication of the Japanese Encephalitis Virus via the Decrease of Viral RNA Synthesis. Int J Mol Sci.

2017 May 1;18(5).

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