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Produktinformation

- Forschungsprodukte & Biochemikalien
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- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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AR-42

Cat. No.: HY-13265

CAS No.: 935881-37-1

Molecular Formula: $C_{18}H_{20}N_2O_3$

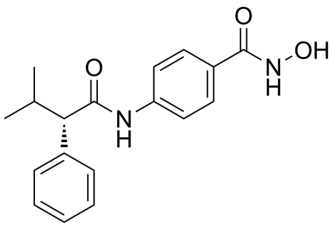
Molecular Weight: 312.36

Target: HDAC; Autophagy; Apoptosis

Pathway: Cell Cycle/DNA Damage; Epigenetics; Autophagy; Apoptosis

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

Ethanol : 50 mg/mL (160.07 mM; Need ultrasonic)
DMSO : 10 mg/mL (32.01 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration / Solvent / Mass	1 mg	5 mg	10 mg
1 mM	3.2014 mL	16.0072 mL	32.0143 mL
5 mM	0.6403 mL	3.2014 mL	6.4029 mL
10 mM	0.3201 mL	1.6007 mL	3.2014 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 5 mg/mL (16.01 mM); Clear solution
2. Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 5 mg/mL (16.01 mM); Clear solution
3. Add each solvent one by one: 10% EtOH >> 90% corn oil
   Solubility: ≥ 5 mg/mL (16.01 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1 mg/mL (3.20 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1 mg/mL (3.20 mM); Clear solution
6. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (3.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	AR-42 (HDAC-42; OSU-HDAC42) is a potent, orally bioavailable pan-HDAC inhibitor (IC_{50}=16 nM). AR-42 induces growth inhibition, cell-cycle arrest, apoptosis, and activation of caspases-3/7. AR-42 promotes hyperacetylation of H3, H4, and alpha-tubulin, and up-regulation of p21. AR-42 shows cytotoxicity against various human cancer cell lines[1][2].
IC_{50} & Target	IC50: 16 nM (HDAC)[2]

In Vitro

AR-42 (0.125-1 μM; 24 hours) inhibits cell proliferation in a dose-dependent manner, and the median IC_{50}s for P815, C2, and BR cells are 0.65, 0.30, and 0.23 μM, respectively[3].
AR-42 (0.5 μM; 24 hours) induces cell-cycle arrest at G1 in the P815 cells and at G1/G2 in the C2 cells[3].
AR-42 (0.13-1 μM; 24 hours) causes a dose-dependent induction of apoptosis P815, C2, BR cells[3].
AR-42 (0.5-3 μM; 24 hours) induces hyperacetylation of histones H3 and H4 and α-tubulin[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[3]

Cell Line:	Mouse (P815) and canine (C2 and BR) malignant mast cells
Concentration:	0.0625, 0.125, 0.25, 0.5, 1 μM
Incubation Time:	24 hours
Result:	Inhibited cell proliferation in a dose-dependent manner, and the median IC_{50}s for P815, C2, and BR cells were 0.65, 0.30, and 0.23 μM, respectively.

Cell Cycle Analysis[3]

Cell Line:	P815,C2 cells
Concentration:	0.5 μM
Incubation Time:	24 hours
Result:	Induced cell-cycle arrest at G1 in the P815 cells and at G1/G2 in the C2 cells.

Apoptosis Analysis[3]

Cell Line:	P815, C2, BR cells
Concentration:	0.13, 0.25, 0.5, 1 μM
Incubation Time:	24 hours
Result:	Caused a dose-dependent induction of apoptosis.

Western Blot Analysis[3]

Cell Line:	P815, C2, BR cell lines
Concentration:	0.5, 1, 3 μM
Incubation Time:	24 hours
Result:	A dose-dependent hyperacetylation of histone H3, histone H4, and α-tubulin.

In Vivo

AR-42 (10 mg/kg; tail vein injection; twice a week for three weeks) significantly inhibites tumor growth[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Nude mice (HepG2 cell tumor xenograft model) [4]
Dosage:	10 mg/kg
Administration:	Tail vein injection; twice a week for three weeks
Result:	Significantly inhibited tumor growth.

CUSTOMER VALIDATION

- J Cell Physiol . 2019 Dec;234(12):22411-22423.
- Patent. US20180263995A1.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Lu YS, Chou CH, Tzen KY, Gao M, ChLu YS, et al. Radiosensitizing effect of a phenylbutyrate-derived histone deacetylase inhibitor in hepatocellular carcinoma. Int J Radiat Oncol Biol Phys. 2012 Jun 1;83(2):e181-9.eng AL, Kulp SK, Cheng JC.Radiosensitizing effect of a phenylbutyrate-derived histone deacetylase inhibitor in hepatocellular carcinoma.Int J Radiat Oncol Biol Phys. 2012 Jun 1;83(2):e181-9. Epub 2012 Feb 28.

[2]. Lu Q, et al. Structure-based optimization of phenylbutyrate-derived histone deacetylase inhibitors. J Med Chem. 2005 Aug 25;48(17):5530-5.

[3]. Lin TY, et al. AR-42, a novel HDAC inhibitor, exhibits biologic activity against malignant mast cell lines via down-regulation of constitutively activated Kit. Blood. 2010 May 27;115(21):4217-25.

[4]. Zhang M, et al. AR-42 induces apoptosis in human hepatocellular carcinoma cells via HDAC5 inhibition. Oncotarget. 2016 Apr 19;7(16):22285-94.

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