# Data Sheet (Cat.No.T1583)



# Vorinostat

# **Chemical Properties**

CAS No.: 149647-78-9

Formula: C14H20N2O3

Molecular Weight: 264.32

Appearance: no data available

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

# **Biological Description** Description Vorinostat (SAHA) is a pan-histone deacetylase (HDAC) inhibitor (IC50=10 nM) with inhibitory activity against HDAC1/2/3/6/7/11. Vorinostat has antitumor activity, induces cell differentiation, blocks the cell cycle and induces apoptosis. Targets(IC50) Apoptosis, Mitophagy, HDAC, Autophagy, Virus Protease In vitro METHODS: Synovial sarcoma cells SW-982 and chondrosarcoma cells SW-1353 were treated with Vorinostat (0.5-15 $\mu$ M) for 48 h, and cell viability was measured by MST assay. RESULTS: Vorinostat inhibited the proliferation of SW-982 and SW-1353 cells in a dosedependent manner with IC50s of 8.6 $\mu$ M and 2.0 $\mu$ M, respectively. [1] METHODS: Uterine sarcoma cells MES-SA were treated with Vorinostat (3 µM) for 24-72 h. The expression levels of target proteins were detected using Western Blot. **RESULTS**: There was no difference in the expression of HDAC1 throughout the treatment period, and HDAC2, 3 and 7 showed significant inhibition of expression by Vorinostat. [2] In vivo **METHODS**: To detect anti-tumor activity in vivo, Vorinostat (50 mg/kg in HOP-β-CD) was administered intraperitoneally to Nude-Foxn1nu/nu mice bearing uterine sarcoma MES-SA five times per week for twenty-one days. **RESULTS**: A reduction in tumor growth of more than 50% was observed in the Vorinostat treatment group compared to the placebo group. [1] METHODS: To study the effects in an animal model of true erythrocytosis (PV), Vorinostat (200 mg/kg in 50% PEG-400) was injected intraperitoneally into MxCre; Jak2V617F/+ mice five times per week for two weeks. **RESULTS**: Vorinostat treatment normalized peripheral blood counts and significantly reduced splenomegaly in Jak2V617F knockout mice. Vorinostat may have therapeutic potential for PV and other JAK2V617F-associated myeloproliferative tumors. [3] Cell Research Cells were plated onto 100-mm tissue culture plates at a density of $2 \times 10^6$ for 48 h and then treated with SAHA or equal concentrations of the vehicle. For longer drug exposure times, medium with drug or vehicle were exchanged every 48 h. For wash-out experiments, cells were treated with SAHA daily for 60-72 h (drug and medium were exchanged at 48 h), then SAHA was washed out and replaced with 10% FCS containing DMEM [3]. Animal Research Athymic Nude-Foxn1nu/nu mice were used in the present study. They were housed at

22°C at a constant light-dark cycle (12-h light, 12-h dark) and had free access to water and rodent chow (4-5% fat, 21% protein). Twelve weeks old male mice (n = 14) were

anesthetized with Isofluran and  $5 \times 10^6$  MES-SA cells were injected subcutaneously into the right flank of the animal. Mice from a control group received placebo containing 300 µl of empty HOP- $\beta$ -CD (2-hydroxypropyl- $\beta$ -cyclodextrin) vesicles. Another group of mice received vorinostat dissolved in HOP- $\beta$ -CD at a concentration of 50 mg/kg/day. Both, empty vesicles and vorinostat were administered intraperitoneally, starting on the day 4 after the injection of MES-SA tumor cells. Mice body weight and tumor size (w2 × l × 0.52; measured by caliper) were estimated twice a week. All mice were treated for 21 days and afterward sacrificed by cervical dislocation. Each tumor was isolated as a whole and different tumor parameters (weight, volume, size, and macroscopic appearance) were determined. Finally, tumor slices were cryopreserved and formalin fixed (4%) for further analyses [1].

# **Solubility Information**

Solubility 10% DMSO+40% PEG300+5% Tween 80+45% Saline: 12.5 mg/mL (47.29 mM), Suspension. Ethanol: 2 mg/mL (7.57 mM), Sonication is recommended.

DMSO: 125 mg/mL (472.90 mM), Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

# **Preparing Stock Solutions**

	1mg	5mg	10mg	
1 mM	3.7833 mL	18.9165 mL	37.8329 mL	
5 mM	0.7567 mL	3.7833 mL	7.5666 mL	
10 mM	0.3783 mL	1.8916 mL	3.7833 mL	
50 mM	0.0757 mL	0.3783 mL	0.7567 mL	

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Page 2 of 3 www.targetmol.com

### Reference

Bernhart E, et al. Histone deacetylase inhibitors vorinostat and panobinostat induce G1 cell cycle arrest and apoptosis in multidrug resistant sarcoma cell lines. Oncotarget. 2017 Aug 24;8(44):77254-77267.

Wellinger L C, Hogg S J, Newman D M, et al. BET Inhibition Enhances TNF Mediated Anti-Tumor Immunity. Cancer Immunology Research. 2022, 10(1): 87-107.

Du T, Hu X, Hou Z, et al.Re-expression of epigenetically silenced PTPRR by histone acetylation sensitizes RAS-mutant lung adenocarcinoma to SHP2 inhibition. Cellular and Molecular Life Sciences. 2024, 81(1): 1-14. Hrzenjak A, et al. Histone deacetylase inhibitor vorinostat suppresses the growth of uterine sarcomas in vitro and

in vivo. Mol Cancer. 2010 Mar 4;9:49.

Akada H, et al. Efficacy of vorinostat in a murine model of polycythemia vera. Blood. 2012 Apr 19;119(16):3779-89. Yuting Meng,Xixi Qian,Li Zhao,Nan Li,Shengjie Wu,Baoan Chen,Tong Sun,Xuerong Wang Trichostatin A downregulates bromodomain and extra-terminal proteins to suppress osimertinib resistant non-small cell lung carcinoma. Cancer Cell International. 2021, 21(1): 1-12.

Hockly E, et al. Suberoylanilide hydroxamic acid, a histone deacetylase inhibitor, ameliorates motor deficits in a mouse model of Huntington's disease. Proc Natl Acad Sci U S A. 2003 Feb 18;100(4):2041-6.

Wang C, Huang M, Lin Y, et al.ENO2-derived phosphoenolpyruvate functions as an endogenous inhibitor of HDAC1 and confers resistance to antiangiogenic therapy. Nature Metabolism. 2023: 1-22.

Liang X L, Ouyang L, Yu N N, et al. Histone deacetylase inhibitor pracinostat suppresses colorectal cancer by inducing CDK5-Drp1 signaling-mediated peripheral mitofission. Journal of Pharmaceutical Analysis. 2023 Wang L, Wu J, Sramek M, et al. Heterogeneous enhancer states orchestrate β cell responses to metabolic stress. Nature Communications. 2024, 15(1): 9361.

Wang J, et al. Snail determines the therapeutic response to mTOR kinase inhibitors by transcriptional repression of 4E-BP1. Nat Commun. 2017 Dec 20;8(1):2207.

Bian R, Shang Y, Xu N, et al. HDAC inhibitor enhances ferroptosis susceptibility of AML cells by stimulating iron metabolism. Cellular Signalling. 2025: 111583.

Wellinger L C, Hogg S J, Newman D M, et al. BET Inhibition Enhances TNF Mediated Anti-Tumor Immunity[J]. bioRxiv. 2021

In vivo vulnerabilities to GPX4 and HDAC inhibitors in drug-persistent versus drug-resistant BRAFV600E lung adenocarcinoma

 $\textbf{Inhibitor} \cdot \textbf{Natural Compounds} \cdot \textbf{Compound Libraries} \cdot \textbf{Recombinant Proteins}$ 

This product is for Research Use Only. Not for Human or Veterinary or Therapeutic Use

Tel:781-999-4286 E\_mail:info@targetmol.com Address:36 Washington Street, Wellesley Hills, MA 02481

Page 3 of 3 www.targetmol.com