Product Data Sheet

Product Name: HDAC11-IN-5
Cat. No.: GC81758

Chemical Properties

Cas. No.

Formula	$C_{24}H_{25}Cl_2F_3N_4O_2$	M.Wt	529.38
Solubility	DMSO: 100 mg/mL (188.90 mM; Need ultrasonic)	Storage	Store at 4°C, sealed storage, away from moisture
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure

Background

HDAC11-IN-5 is a selective, potent and orally active HDAC11 inhibitor with an IC_{50} of 0.021 μM. HDAC11-IN-5 increases fatty acylation levels of substrate SHMT2 in AML cells. HDAC11-IN-5 induces apoptosis, G1 phase cell cycle arrest, ferroptosis, ROS production and terminal myeloid differentiation in AML cells. HDAC11-IN-5 demonstrates anti-tumor potency in an MLL-AF9-induced mouse AML model. HDAC11-IN-5 can be used for the research of cancer, such as acute myeloid

leukemia [1].

In Vivo, HDAC11-IN-5 (Compound 25) (50 mg/kg; i.g.; once daily; 14 days) reduces peripheral blood leukemic cell burden and significantly extends survival in an MLL-AF9-induced AML mouse model, with enhanced efficacy when combined with Ivosidenib [1].

In Vitro, HDAC11-IN-5 (Compound 25) potently inhibits purified HDAC11 protein with an IC_{50} of 0.021 ± 0.005 μM, and shows no significant inhibition of HDAC1, HDAC6, HDAC7, or HDAC8 at concentrations up to 10 μM[1]. HDAC11-IN-5 (96 h) potently inhibits proliferation of U937, OCI-AML2, THP-1, and MOLM13 AML cell lines with IC_{50} values of 6.3 μM, 5.7 μM, 5.4 μM, and 6.7 μM, respectively[1]. HDAC11-IN-5 (10-20 μM; 6 h) selectively inhibits intracellular HDAC11 in U937 AML cells, as shown by increased SHMT2 fatty acylation at 10 μM for 6 h and no significant changes in histone H3 or α-tubulin acetylation at 20 μM for 6 h[1]. HDAC11-IN-5 (8 μM; 48 h) strongly induces apoptosis in U937 and OCI-AML2 AML cells[1]. HDAC11-IN-5 (8 μM; 48 h) induces G1-phase cell cycle arrest in U937 AML cells[1]. HDAC11-IN-5 (8 μM; 48 h) promotes myeloid differentiation of U937 AML cells, as shown by increased CD14 and CD11b expression[1]. HDAC11-IN-5 (8 μM; 48 h) induces ferroptosis in U937 AML cells, as shown by increased lipid peroxidation, cellular ROS, and intracellular Fe2+ levels[1]. HDAC11-IN-5 (5-20 μM; 96 h) exhibits strong synergistic antiproliferative activity with ivosidenib in U937, OCI-AML2, and MLL-AF9 mouse AML cells[1].

References:
[1]. Chai Q, et al. Discovery of a Hydroxamic Acid-Based HDAC11 Isoform-Selective Inhibitor with Oral Anti-AML Potency. J Med Chem. 2026;69(6):6816-6834.