

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 μ M. 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

Caution: Product has not been fully validated for medical applications. For research use only.

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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 IC₅₀ of $0.021 \pm 0.005 \mu\text{M}$, and shows no significant inhibition of HDAC1, HDAC6, HDAC7, or HDAC8 at concentrations up to $10 \mu\text{M}$ [1]. HDAC11-IN-5 (96 h) potently inhibits proliferation of U937, OCI-AML2, THP-1, and MOLM13 AML cell lines with IC₅₀ values of $6.3 \mu\text{M}$, $5.7 \mu\text{M}$, $5.4 \mu\text{M}$, and $6.7 \mu\text{M}$, respectively[1]. HDAC11-IN-5 ($10\text{--}20 \mu\text{M}$; 6 h) selectively inhibits intracellular HDAC11 in U937 AML cells, as shown by increased SHMT2 fatty acylation at $10 \mu\text{M}$ for 6 h and no significant changes in histone H3 or α -tubulin acetylation at $20 \mu\text{M}$ for 6 h[1]. HDAC11-IN-5 ($8 \mu\text{M}$; 48 h) strongly induces apoptosis in U937 and OCI-AML2 AML cells[1]. HDAC11-IN-5 ($8 \mu\text{M}$; 48 h) induces G1-phase cell cycle arrest in U937 AML cells[1]. HDAC11-IN-5 ($8 \mu\text{M}$; 48 h) promotes myeloid differentiation of U937 AML cells, as shown by increased CD14 and CD11b expression[1]. HDAC11-IN-5 ($8 \mu\text{M}$; 48 h) induces ferroptosis in U937 AML cells, as shown by increased lipid peroxidation, cellular ROS, and intracellular Fe²⁺ levels[1]. HDAC11-IN-5 ($5\text{--}20 \mu\text{M}$; 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.

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