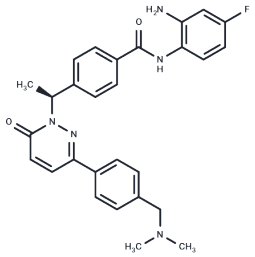
Data Sheet (Cat.No.T86559)

TargetMol

HDAC-IN-56

Chemical Properties



CAS No. : 2814571-89-4

Formula: C28H28FN5O2

Molecular Weight: 485.55

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	HDAC-IN-56 ((S)-17b), an orally active inhibitor of class I histone deacetylase (HDAC), exhibits inhibitory constants (IC 50) of 56.0 ± 6.0 nM for HDAC1, 90.0 ± 5.9 nM for HDAC2, 422.2 ± 105.1 nM for HDAC3, and greater than 10,000 nM for HDAC4-11. This compound displays robust antitumor activity [1], marked by its ability to significantly elevate intracellular acetylhistone H3 and P21 levels, while also inducing G1 cell cycle arrest and apoptosis effectively.
Targets(IC50)	HDAC
In vitro	HDAC-IN-56 exhibits a strong selective inhibitory effect on class I HDACs, specifically 1, 2, and 3, surpassing that of MS-275 [1]. At a concentration of 0.1 μM over 2 hours, HDAC-IN-56 displays significant species-specific metabolic differences across human, monkey, dog, rat, and mouse hepatocytes, yet remains metabolically stable in all five species [1]. When used at 0.01-1 μM for 72 hours, HDAC-IN-56 effectively induces G1 phase cell cycle arrest and apoptosis [1]. Treatment at these concentrations also elevates intracellular levels of acetyl-histone H3 and p21 more effectively than Tucidinostat or MS-275, indicating potent inhibition of class I histone deacetylases [1]. The IC50 of HDAC-IN-56 for SKM-1 is 139.0 ± 8.0 nM [1]. In cell cycle analysis of SKM-1 cells at 0.01, 0.1, and 1 μM for 72 hours, HDAC-IN-56 downregulated c-Myc and CDK4 expression at 0.1 μM more efficiently than MS-275 or Tucidinostat [1]. Apoptosis analysis in SKM-1 cells showed that HDAC-IN-56 triggered strong apoptosis, as evidenced by Annexin V/PI staining, with greater efficacy than MS-275 or Tucidinostat [1]. Western blot analysis demonstrated that HDAC-IN-56 increases the intracellular levels of acetyl-histone H3 and p21 in SKM-1 cells at these concentrations over 72 hours, outperforming Tucidinostat or MS-275 [1].
In vivo	HDAC-IN-56, administered orally at 10-80 mg/kg daily for one month, does not cause significant weight changes even at the highest dose of 80 mg/kg [1]. It exhibits a favorable pharmacokinetic profile, showing oral bioavailability of 47.7% in ICR mice and 39.5% in SD rats when administered to SD rats (10, 20 mg/kg) and ICR mice (20, 40 mg/kg) after a fasting period [1]. HDAC-IN-56 at doses of 20-60 mg/kg effectively inhibits tumor growth of MC38 cells in nude mice and demonstrates enhanced tumor inhibition in immunocompetent C57BL/6 mice, suggesting that the immune system may be involved and activated to enhance the antitumor effect [1]. Animal models used include male SD rats or ICR mice for pharmacokinetic studies and SKM-1 or MC-38 cell xenografts for efficacy evaluation [1].

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.0595 mL	10.2976 mL	20.5952 mL
5 mM	0.4119 mL	2.0595 mL	4.119 mL
10 mM	0.206 mL	1.0298 mL	2.0595 mL
50 mM	0.0412 mL	0.206 mL	0.4119 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.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.