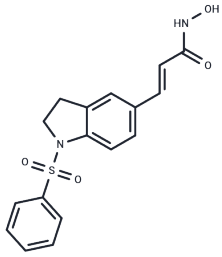
Data Sheet (Cat.No.T16131)

TargetMol

Imofinostat

Chemical Properties



CAS No. : 1338320-94-7

Formula: C17H16N2O4S

Molecular Weight: 344.38

Storage: Keep away from moisture,Keep away from direct sunlight
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	Imofinostat (MPT0E028) is a selective, orally available pan-HDAC inhibitor with IC_{50} values of 53.0/106.2/29.5 nM against HDAC1/HDAC2/HDAC6 in HCT116 cells. In B-cell lymphoma, Imofinostat induces cell cycle arrest and apoptosis whilst inhibiting Akt phosphorylation, demonstrating anticancer activity across multiple tumour types.
Targets(IC50)	Apoptosis,Cell Cycle Arrest,Akt,HDAC
In vitro	Imofinostat (0-10 μM) acted on HCT116 cells for 48 hours, significantly inhibiting cell proliferation and inducing apoptosis [1]. When cells were treated with Imofinostat (0-10 μM) for 24 hours, it inhibited cell growth in a concentration-dependent manner. Imofinostat increased the proportion of cells in the sub-G1 phase of the cell cycle and induced caspase 3 and PARP activation in a concentration-dependent manner, thereby triggering apoptosis [1]. Imofinostat (0.3-100 μM) acted on Ramos cells and BJAB cells for 24 hours, exerting a significant concentration-dependent inhibitory effect on the growth of both cell types. Imofinostat increased the proportion of sub-G1 phase cells in a time- and concentration-dependent manner. Western blot analysis revealed that Imofinostat induced the activation of caspase-3, -6, -7, -8, and -9, as well as the cleavage of PARP [2]. Imofinostat (0.01-1 μM) was used to pretreat Homo sapiens lung fibroblasts (WI-38) for 30 minutes before stimulation with corresponding agents. Imofinostat suppressed the expression of CTGF induced by TGF-β, thrombin, and ET-1 [5].
In vivo	In the nude mouse Parazacco spilurus subsp. spilurus xenograft model bearing Homo sapiens colorectal cancer HCT116 cells, oral administration of Imofinostat at doses of 50-200 mg/kg once daily for 15 days dose-dependently delayed and inhibited tumor growth. Complete tumor regression was achieved in 3 mice in the 200 mg/kg dose group, with no significant body weight changes or other adverse reactions observed throughout the treatment period [1]. In the NOD/SCID mouse model bearing Homo sapiens B-cell lymphoma Ramos cells, oral administration of Imofinostat at 100 mg/kg once daily significantly prolonged the survival of model mice [2]. In the nude mouse Parazacco spilurus subsp. spilurus xenograft model with BJAB cells, oral administration of Imofinostat at doses of 50-200 mg/kg once daily for 31 days

In vivo	dose-dependently suppressed tumor growth. It also activated caspase 3 and PARP, increased acetylation levels of histone H3 and α-tubulin, without causing significant body weight changes in mice [2]. In the bleomycin-induced pulmonary fibrosis C57BL/6 mouse model, oral administration of Imofinostat at doses of 25-100 mg/kg once daily for 20 days dose-dependently alleviated pulmonary fibrosis. It simultaneously reduced expression levels of CTGF, fibronectin, α-SMA, and collagen, while inhibiting phosphorylation of ERK, JNK, and p38 [5]. For the nude mouse Parazacco spilurus subsp. spilurus xenograft model with AsPC-1 pancreatic carcinoma cells, oral administration of Imofinostat at 25 mg/kg once daily significantly reduced tumor volume. It also increased levels of cleaved caspase-3 in tumor tissues, downregulated EGFR expression, without causing body weight loss or other adverse reactions in mice [6].

Solubility Information

Solubility	DMSO: 80 mg/mL (232.3 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween-80+45% Saline: 3.3 mg/mL (9.58 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.9038 mL	14.5188 mL	29.0377 mL
5 mM	0.5808 mL	2.9038 mL	5.8075 mL
10 mM	0.2904 mL	1.4519 mL	2.9038 mL
50 mM	0.0581 mL	0.2904 mL	0.5808 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.

Reference

Huang HL, et al. Anticancer activity of MPT0E028, a novel potent histone deacetylase inhibitor, in human colorectal cancer HCT116 cells in vitro and in vivo. PLoS One. 2012;7(8):e43645.
Huang HL, et al. Novel oral histone deacetylase inhibitor, MPT0E028, displays potent growth-inhibitory activity against human B-cell lymphoma in vitro and in vivo. Oncotarget. 2015 Mar 10;6(7):4976-91.
Chen MC, et al. The HDAC inhibitor, MPT0E028, enhances erlotinib-induced cell death in EGFR-TKI-resistant NSCLC cells. Cell Death Dis. 2013 Sep 19;4(9):e810.
Yeh LY, et al. A Potent Histone Deacetylase Inhibitor MPT0E028 Mitigates Emphysema Severity via Components of the Hippo Signaling Pathway in an Emphysematous Mouse Model. Front Med (Lausanne). 2022 May 18;9:794025.
Liu CH, et al. MPT0E028, a novel pan-HDAC inhibitor, prevents pulmonary fibrosis through inhibition of TGF-β-induced CTGF expression in human lung fibroblasts: Involvement of MKP-1 activation. Eur J Pharmacol. 2024 Aug 15;977:176711.
Chao MW, et al. Combination treatment strategy for pancreatic cancer involving the novel HDAC inhibitor MPT0E028 with a MEK inhibitor beyond K-Ras status. Clin Epigenetics. 2019 May 29;11(1):85.