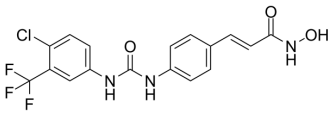
HDAC-IN-35

Cat. No.:	HY-146539
Molecular Formula:	$C_{17}H_{13}ClF_3N_3O_3$
Molecular Weight:	399.75
Target:	HDAC; VEGFR
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Protein Tyrosine Kinase/RTK
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

HDAC-IN-35 (Compound 14) is a potent, selective HDAC and VEGFR-2 inhibitor, with IC_{50} values of 0.166 and 13.2 μM for HDAC6 and VEGFR-2, respectively[1].

IC_{50} & Target

HDAC6	HDAC8	HDAC1	VEGFR2
0.166 μM (IC_{50})	1.99 μM (IC_{50})	7.23 μM (IC_{50})	13.2 μM (IC_{50})

In Vitro

HDAC-IN-35 (Compound 14) (0-10 μM, 48 h) shows anticancer effects in different cancer cells[1].

HDAC-IN-35 (0-10 μM, 48 h) exhibits potent anti-angiogenic activity with a $GI_{>50}$ (50% growth inhibition) value of 1.0 μM on human endothelial progenitor cells (EPCs) through a VEGFR-2-dependent pathway, without obvious systemic toxicity[1].

HDAC-IN-35 exhibits moderate VEGFR-2 inhibitory activities and displays the anticancer effects by inhibiting the enzymatic activity of HDAC[1].

HDAC-IN-35 (0-10 μM, 24 h) concentration-dependently impedes the capillary-like tube formation in human EPCs[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	A549, PC-3, and SK-Hep-1
Concentration:	0-10 μM
Incubation Time:	48 h
Result:	Showed anticancer effects with IC_{50} values of 3.4, 1.9 and 3.2 μM against A549, PC-3, and SK-Hep-1 cells.

Western Blot Analysis[1]

Cell Line:	A549, PC-3, and SK-Hep-1, and human EPCs
Concentration:	0, 5, and 10 μM
Incubation Time:	24 h
Result:	Increased the amount of acetylated α-tubulin and histone H3 in a concentration-dependent manner in cancer cells. Induced mild inhibition of the phosphorylation of VEGFR-2 in human EPCs.

REFERENCES

[1]. Szu Lee, et al. Effect of phenylurea hydroxamic acids on histone deacetylase and VEGFR-2. Bioorg Med Chem. 2021 Nov 15;50:116454.

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