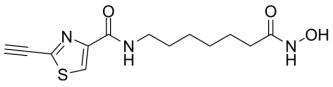
HDAC-IN-48

Cat. No.:	HY-151872
CAS No.:	3031411-05-6
Molecular Formula:	$C_{13}H_{17}N_3O_3S$
Molecular Weight:	295.36
Target:	HDAC; Ferroptosis
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

HDAC-IN-48 is a potent HDAC inhibitor. HDAC-IN-48 is a hybrid molecule with great cytotoxic profile (GI_{50}~20 nM). HDAC-IN-48 consists of harmacophores of SAHA and CETZOLE molecules. HDAC-IN-48 induces ferroptosis and inhibits HDAC proteins [1]. HDAC-IN-48 is a click chemistry reagent, it contains an Alkyne group and can undergo copper-catalyzed azide-alkyne cycloaddition (CuAAc) with molecules containing Azide groups.

In Vitro

HDAC-IN-48 (0-40 μM; 3 d) has superior antiproliferative activity on cancer cells (NCI-H522 and HCT-116) vs the normal cells (WI38 and RPE) with IC_{50}s of 0.5 μM (NCI-H522), 0.61 μM (HCT-116), 8.37 μM (WI38, normal human lung fibroblasts), and 6.13 μM (RPE, retinal pigment epithelial cells), respectively[1].
HDAC-IN-48 (2.5 μM; 24 h) suppresses cell viability by inducing ferroptosis and HDAC inhibition[1].
HDAC-IN-48 (10 μM; 6 h) decreases the lipid peroxide level compared with SAHA[1].
HDAC-IN (0.58 μM, 1.16 μM, and 2.32 μM; 3 d) has no neurotoxic effects and (2.5, 5, and 10 μM; 3 d) leads to hyper acetylation of histones and tubulin[1].
Nondifferentiated PC-12 cells have stem-like properties, but when differentiated by a nerve growth factor, they demonstrate neuronal behavior. HDAC-IN-48 (0.58 μM, 1.16 μM, and 2.32 μM; 24 h) behaves as the HDAC control effect, shows few ferroptosis induction on both differentiated and undifferentiated PC-12 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	NCI-H522 cells
Concentration:	2.5, 5, and 10 μM
Incubation Time:	72 hours; at the corresponding concentrations in the presence of Liproxstatin-1 (0.25 μM)
Result:	Led to hyperacetylation of histones and tubulin in a similar way to SAHA (pan-inhibitor).

Cell Viability Assay[1]

Cell Line:	NCI-H522, WI38, HCT-116, and RPE
Concentration:	0-40 μM
Incubation Time:	72 hours
Result:	Showed selectivity among normal cells over cancer cells.

	Inhibited cell survival with IC50s of 0.5 μM, 8.37 μM, 0.61 μM, and 6.13 μM.

Apoptosis Analysis[1]

Cell Line:	NCI-H522 cells
Concentration:	5 μM
Incubation Time:	24 hours, 48 hours, and 72 hours
Result:	Induced cell ferroptosis.

REFERENCES

[1]. Karaj E, et al. First-in-Class Dual Mechanism Ferroptosis-HDAC Inhibitor Hybrids. J Med Chem. 2022 Nov 10;65(21):14764-14791.

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