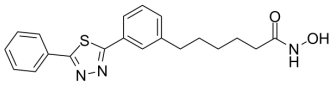
HDAC1-IN-5

Cat. No.:	HY-151153
Molecular Formula:	$C_{20}H_{21}N_3O_2S$
Molecular Weight:	367.46
Target:	HDAC; Microtubule/Tubulin; Caspase; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Cytoskeleton; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

HDAC1-IN-5 is a potent HDAC1 inhibitor with IC_{50} values of 15 nM and 20 nM for HDAC1 and HDAC6, respectively. HDAC1-IN-5 can enhance the acetylation of histone H3 and α-tubulin, as well as promote the activation of caspase 3 in cancer cells, thereby inducing apoptosis. HDAC1-IN-5 induces chromatin damage by binding with DNA. HDAC1-IN-5 has strong inhibitory activity against tumor growth in xenograft mice[1].

IC_{50} & Target

HDAC1	HDAC6
15 nM (IC_{50})	20 nM (IC_{50})

In Vitro

HDAC1-IN-5 (compound 4j) (0-50 μM; 48 h) exhibits strong inhibitory effects on HCT116, HeLa, HepG2, MC38, K562 and HEL[1].

HDAC1-IN-5 (0.16-1.5 μM; 48 h) induces apoptosis in a dose-dependent manner[1].

HDAC1-IN-5 (0.17-1.5 μM; 24 h or 48 h) induces downregulation of SPT16 and SSRP-1, induces the cleavage of caspase-3, and increases the expression of Acetyl-Histone H3 and Acetyl-α-tubulin[1].

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

Cell Proliferation Assay[1]

Cell Line:	HCT116, HeLa, HepG2, MC38, K562 and HEL
Concentration:	0-50 μM
Incubation Time:	48 h
Result:	Showed strong inhibitory effects on HCT116, HeLa, HepG2, MC38, K562 and HEL with IC_{50}s of 0.47 μM, 0.78 μM, 1.4 μM, 0.43 μM, 0.91 μM and 0.28 μM, respectively.

Apoptosis Analysis[1]

Cell Line:	HCT116
Concentration:	0.16 μM, 0.5 μM and 1.5 μM
Incubation Time:	48 h
Result:	Induced apoptosis in a dose-dependent manner, and induced 38.5% at the concentration of 1.5 μM (both early and late apoptotic cells).

Western Blot Analysis[1]

Cell Line:	HCT116 and MC38
Concentration:	0.17 μM, 0.5 μM and 1.5 μM
Incubation Time:	24 h or 48 h
Result:	Induced downregulation of SPT16 and SSRP-1 in HCT116. Induced the cleavage of caspase-3 in HCT116 and MC38. Increased the expression of HDAC1, 2, 6 substrate Acetyl-Histone H3 and Acetyl-α-tubulin with a dose-dependent manner.

In Vivo

HDAC1-IN-5 (50 and 100 mg/kg; IP; every two days; for 16 days) significantly decreases the tumor volume and weight in MC38 xenograft mice[1].

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

Animal Model:	C57BL/6 mice (6-10 weeks; 2×10^6 MC38 cells were injected subcutaneously into the right flank regions)[1]
Dosage:	50 and 100 mg/kg
Administration:	IP; every two days; for 16 days
Result:	Significantly decreased the tumor volume and weight with tumor growth inhibition (TGI) of 66% at 50 mg/kg.

REFERENCES

[1]. Chen C, et al. Discovery of 2,5-diphenyl-1,3,4-thiadiazole derivatives as HDAC inhibitors with DNA binding affinity. Eur J Med Chem. 2022 Jul 31;241:114634.

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