HDAC-IN-53

Cat. No.: HY-149208

CAS No.: 2921948-27-6

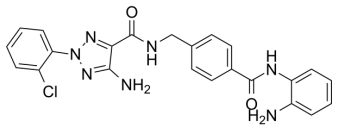
Molecular Formula: $C_{23}H_{20}ClN_7O_2$

Molecular Weight: 461.9

Target: HDAC; Apoptosis

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-53 is an orally active, and selective HDAC1-3 inhibitor with IC_{50} values of 47 nM, 125 nM, and 450 nM, respectively. HDAC-IN-53 does not inhibit class II HDACs (HDAC4, 5, 6, 7, 9; IC_{50}>10 μM). HDAC-IN-53 induces caspase-dependent apoptosis . HDAC-IN-53 significantly inhibits the growth of human tumor xenografts in nude mice and murine tumor growth in immune-competent mice bearing MC38 colon cancer[1].

IC_{50} & Target

HDAC1 47 nM (IC_{50})	HDAC2 125 nM (IC_{50})	HDAC3 450 nM (IC_{50})	HDAC4 >10 μM (IC_{50})
HDAC5 >10 μM (IC_{50})	HDAC6 >10 μM (IC_{50})	HDAC7 >10 μM (IC_{50})	HDAC8 >10 μM (IC_{50})
HDAC9 >10 μM (IC_{50})			

In Vitro

HDAC-IN-53 (compound 19h) has good antiproliferative activity against a panel of cancer cell lines, for example MC38 (IC_{50} =0.66 μM), HCT116 cell (IC_{50}=0.56 μM) [1].

HDAC-IN-53 (0.1-1 μM; 24 h) causes G0/G1 cell cycle arrest in MC38 cells and induces G2/M cell cycle arrest in HCT116 cells[1].

HDAC-IN-53 (0.1-1 μM; 24 h) upregulates the expressions of cleaved caspase-3 and cleaved PARP in a dose-dependent manner[1].

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

Cell Cycle Analysis[1]

Cell Line:	MC38 and HCT116 cells
Concentration:	0.1, 0.3, 1 μM
Incubation Time:	24 h
Result:	Caused G0/G1 cell cycle arrest in MC38 cells and induced G2/M cell cycle arrest in HCT116 cells. Significantly decreased the proportion of S phase cells in MC38 cells.

Western Blot Analysis[1]

Cell Line:	MC38 and HCT116 cells
Concentration:	0.1, 0.3, 1 μM
Incubation Time:	24 h
Result:	Upregulated the expressions of cleaved caspase-3 and cleaved PARP in a dose-dependent manner.

In Vivo

HDAC-IN-53 (60 or 120 mg/kg; PO; daily for 15 days) exerts antitumor activities by both direct tumor growth inhibition and indirect immune cell-mediated antitumor effect[1].

Pharmacokinetic Parameters of HDAC-IN-53 in Mice[1].

	IV (5 mg/kg)	PO (20 mg/kg)
T_{max} (h)		0.42
C_{max} (ng/mL)	8129	9558
AUC_{0-t} (ng/mL·h)	5864	15278
$t_{1/2}$ (h)	0.85	2.49
F (%)		65.1%

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

Animal Model:	C57BL/6 Mice or athymic nude mice (female, 6-8 weeks old) with MC38 cells[1]
Dosage:	60 or 120 mg/kg
Administration:	PO; daily for 15 days
Result:	Yielded TGI values of 60.3 and 87.6%, respectively. Increased the percentage of CD4+ T cells.

REFERENCES

[1]. Nan Sun, et al. Design and Synthesis of Triazole-Containing HDAC Inhibitors That Induce Antitumor Effects and Immune Response. J Med Chem. 2023 Apr 13;66(7):4802-4826.

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