ALK/HDAC-IN-1

Cat. No.: HY-161350

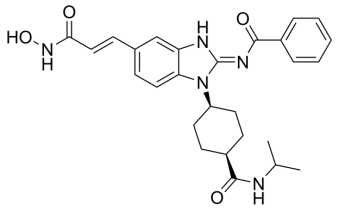
Molecular Formula: $C_{27}H_{31}N_5O_4$

Molecular Weight: 489.57

Target: Anaplastic lymphoma kinase (ALK); HDAC

Pathway: Protein Tyrosine Kinase/RTK; Cell Cycle/DNA Damage; Epigenetics

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

ALK/HDAC-IN-1 is a dual inhibitor for ALK and HADC6, with IC_{50}s of 16 nM and 1.03 μM, respectively. ALK/HDAC-IN-1 exhibits antitumor activity[1].

IC_{50} & Target

HDAC6	HDAC8	HDAC1	HDAC11
1.03 μM (IC_{50})	1.69 μM (IC_{50})	10.8 μM (IC_{50})	16.6 μM (IC_{50})

In Vitro

ALK/HDAC-IN-1 inihibits proliferations of cancer cells A549, HepG2, MCF7, U87MG and H2228, with IC_{50}s of 0.33, 0.59, 0.55, 0.62 and 0.44 μM, respectively[1].

ALK/HDAC-IN-1 inihibits CYP450 enzymes CYP2C9 with IC_{50} of 2.65 μM[1].

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

Cell Proliferation Assay[1]

Cell Line:	A549, HepG2, MCF7, U87MG and H2228
Concentration:	
Incubation Time:	48 h
Result:	Inhibited proliferations with sub-micromolar level IC50 values.

Western Blot Analysis[1]

Cell Line:	H2228
Concentration:	2 μM
Incubation Time:	6 h
Result:	Ptomoted acetylation of α-tubulin and histone H3.

In Vivo

ALK/HDAC-IN-1 (0-20 mg/kg, i.p. for 21 days) inhibits tumor growth without significant toxicity in A549 xenografted BALB/c mice[1].

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Animal Model:	A549 xenografted BALB/c mice[1]
Dosage:	0-20 mg/kg
Administration:	i.p., once daily for 21 days
Result:	Decrease tumor growth with TGIs of 68% and 85%, with dose of 10 and 20 mg/kg, respectively.

REFERENCES

[1]. Wang KL, et al., Discovery of novel anaplastic lymphoma kinase (ALK) and histone deacetylase (HDAC) dual inhibitors exhibiting antiproliferative activity against non-small cell lung cancer. J Enzyme Inhib Med Chem. 2024 Dec;39(1):2318645.

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