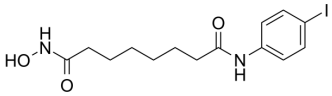
4-Iodo-SAHA

Cat. No.:	HY-124007
CAS No.:	1219807-87-0
Molecular Formula:	$C_{14}H_{19}IN_2O_3$
Molecular Weight:	390.22
Target:	HDAC
Pathway:	Cell Cycle/DNA Damage; Epigenetics
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

4-Iodo-SAHA (1k) is an orally active class I and class II histone deacetylase (HDAC) inhibitor with EC_{50}s of 1.1, 0.95, 0.12, 0.24, 0.85 and 1.3 μM for Skbr3, HT29, U937, JA16 and HL60 cell lines, respectively. 4-Iodo-SAHA (1k) can be used for the research of cancer[1].

In Vitro

4-Iodo-SAHA (0.1-100 μM; 48 h) inhibits Skbr3, HT29, U937, JA16 and HL60 cell lines [1].
4-Iodo-SAHA (2 μM; 6-24 h) affects acetylated H4 and p21 levels in SKBR3 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	SKBR3, HT29, U937, JA16 and HL60 cell lines
Concentration:	0.1-100 μM
Incubation Time:	48 h
Result:	Inhibited SKBR3, HT29, U937, JA16 and HL60 cell lines with EC_{50}s of 1.1, 0.95, 0.12, 0.24, 0.85 and 1.3 μM , respectively. Showed 10-fold potent as an inhibitor of U937 cell line compared to SAHA.

Western Blot Analysis[1]

Cell Line:	SKBR3-breast-derived cell line
Concentration:	2 μM
Incubation Time:	6, 12 and 24 h
Result:	Time-dependently up regulated histone H4 acetylation and p21/WAF1 cell cycle inhibitor accumulation in SKBR3 cells.

In Vivo

4-Iodo-SAHA (1k) (50 mg/kg; p.o. five times a week for two weeks) compares to SAHA-treated and control mice with similar toxicity[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	8-week-old fvb mice[1]
Dosage:	50 mg/kg
Administration:	Oral gavage; 50 mg/kg five times per week; for 2 weeks
Result:	Compared to both SAHA-treated and control mice with similar body weights and hematological counts.

REFERENCES

[1]. Salmi-Smail C, et al. Modified cap group suberoylanilide hydroxamic acid histone deacetylase inhibitor derivatives reveal improved selective antileukemic activity. J Med Chem. 2010 Apr 22;53(8):3038-47.

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