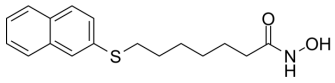
HNHA

Cat. No.:	HY-118672
CAS No.:	926908-04-5
Molecular Formula:	$C_{17}H_{21}NO_2S$
Molecular Weight:	303.42
Target:	HDAC; MMP; HIF/HIF Prolyl-Hydroxylase
Pathway:	Cell Cycle/DNA Damage; Epigenetics; Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	HNHA is a potent histone deacetylase (HDAC) inhibitor. HNHA arrests the cell cycle at the G1/S phase via p21 induction. HNHA inhibits tumor growth and tumor neovascularization. HNHA may be a potent anti-cancer agent against breast cancer [1].

IC_{50} & Target	MMP-2	MMP-9

In Vitro

HNHA (0-100 μM, 96 h) shows strong inhibition at lower concentrations on cancer cell lines, especially on breast cancer cells, mouse FM3A and human MCF-7[1].

HNHA (15 μM, 24 h) arrests cancer cells at the G1/S phase of the cell cycle, activates p21and rescues strongly protein acetylation[1].

HNHA (15 μM, 12 h) inhibits angiogenic proteins in breast cancer cells, effectively inactivates MMP-2, MMP-9, VEGF and HIF-1 α[1].

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

Cell Proliferation Assay

Cell Line:	FM3A, C1300, LA-N-1, LA-N-2, LA-N-5, NB16, NB19, NB69, SK-N-SH, MCF-7 and HT-29[1]
Concentration:	0-100 μM
Incubation Time:	96 h
Result:	Showed strong inhibition at lower concentrations on all cancer cell lines (FM3A, C1300, LA-N-1, LA-N-2, LA-N-5, NB16, NB19, NB69, SK-N-SH, MCF-7 and HT-29), with IC_{50} values of 15.70, 55.63, 22.78, 23.18, 26.70, 19.64, 21.26, 22.31, 65.09, 14.33, and 16.98 μM, respectively.

Cell Viability Assay

Cell Line:	FM3A and MCF-7[1]
Concentration:	0, 0.1, 1, 5, 10, 15, 20, 25,30 μM
Incubation Time:	48 h
Result:	Showed dose-dependent inhibition of viability in mouse and human breast cancer cells.

Cell Cycle Analysis

Cell Line:	FM3A and MCF-7 cells[1]
Concentration:	15 μM
Incubation Time:	24 h
Result:	Arrested FM3A and MCF-7 cells in the G1/S phase.

Western Blot Analysis

Cell Line:	FM3A and MCF-7 cells[1]
Concentration:	0, 0.1, 1, 10, and 20 μM (24 h)
Incubation Time:	1, 6, 24, 48, and 72 h (15 μM)
Result:	Activated a cell proliferation arrestor p21, increased histone and non-histone protein acetylation and inhibited FM3A and MCF-7 proliferation in vitro, and was very effective in increasing the acetylation level of histone H3 protein in FM3A and MCF-7. The most effective dose point for acetylation of histone H3 was 10-20 μM. Histone H3 acetylation peaked after 1 h of exposure to the drugs and remained stable for 1-6 h.

Western Blot Analysis

Cell Line:	FM3A and MCF-7 cells[1]
Concentration:	15 μM
Incubation Time:	12 h
Result:	Showed a strong induction of TIMP-1 and TIMP-2, and effectively inactivated MMP-2, MMP-9, VEGF and HIF-1α.

In Vivo

HNHA (20 μM/mouse, IP, once every 2 days for a total of six injections) reduces tumor burden and extends the survival rate, activates TIMP-1, TIMP-2 and p21 and inhibits MMP-2, MMP-9, HIF-1α and VEGF protein expression[1].

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

Animal Model:	C3H/HeJ-FasL mice (FM3A breast cancer cell tumor xenograft, 6 weeks, n = 25/group)[1]
Dosage:	20 μM/mouse
Administration:	IP, once every 2 days for a total of six injections
Result:	Reduced tumor burden and extended the survival rate. Effectively inhibited cancer development and angiogenesis in vivo. Increased TIMP-1, TIMP-2 and p21, decreased MMP-2, MMP-9, HIF-1α and VEGF protein expression, and reduced the distribution of CD34, HIF-1α and VEGF.

REFERENCES

[1]. Park KC, et al. Potential anti-cancer activity of N-hydroxy-7-(2-naphthylthio) heptanomide (HNHA), a histone deacetylase inhibitor, against breast cancer both in vitro and in vivo. Cancer Sci. 2011 Feb;102(2):343-50.

Caution: Product has not been fully validated for medical applications. For research use only.

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