Droxinostat

Cat. No.: HY-13267

CAS No.: 99873-43-5

Molecular Formula: $C_{11}H_{14}ClNO_3$

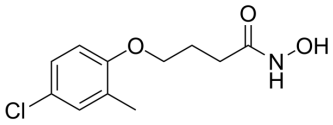
Molecular Weight: 243.69

Target: HDAC; Apoptosis

Pathway: Cell Cycle/DNA Damage; Epigenetics; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 150 mg/mL (615.54 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	4.1036 mL	20.5179 mL	41.0357 mL
5 mM	0.8207 mL	4.1036 mL	8.2071 mL
10 mM	0.4104 mL	2.0518 mL	4.1036 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (10.26 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.26 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Droxinostat (NS 41080) is a histone deacetylase (HDAC) inhibitor. Droxinostat selectively inhibits HDAC3, HDAC6, and HDAC8 with IC_{50} values of 16.9 μM, 2.47 μM, and 1.46 μM, respectively. Droxinostat can be used for the research of hepatocellular carcinoma (HCC)[1][2].

IC_{50} & Target

HDAC8	HDAC6	HDAC3
1.46 μM (IC_{50})	2.47 μM (IC_{50})	16.9 μM (IC_{50})

In Vitro

Droxinostat selectively inhibits HDAC3, HDAC6, and HDAC8 with IC_{50} values of 16.9 μM, 2.47 μM, and 1.46 μM, respectively[1].
Droxinostat (0, 10, 20, or 40 μM; 48 h) suppresses HDAC3 expression and induces acetylation of histones H3 and H4[2].
Droxinostat (0, 10, 20, 40, and 80 μM; 0, 24, 48, 72, 96, and 120 h) inhibits cell proliferation and colony formation in HepG2 and SMMC-7721 cells[2].
Droxinostat (0 to 80 μM; 48 h) induces hepatoma cell apoptosis by activating mitochondrial apoptotic pathways and downregulating FLIP[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	SMMC-7721 and HepG2 (Human liver carcinoma cell lines)
Concentration:	0, 10, 20, or 40 μM
Incubation Time:	48 h
Result:	Significantly decreased the expression of HDAC3 with dose-dependent in HepG2 and SMMC-7721 cell lines. Significantly enhanced the expression of acetyl-H3 (Ac-H3) and acetylH4 (Ac-H4) in HepG2 and SMMC-7721 cells in a dose-dependent manner. Upregulated the levels of phospho-p53 and cleaved caspase 3 protein and downregulated the levels of Bcl-2. Markedly increased the Bax/Bcl-2 ratio in a dose-dependent manner and increased the expression of cleaved PARP protein in HepG2 cells in a dose-dependent manner. Significant reduced the FLIP expression and enhanced caspase 8 activity in both HepG2 and SMMC-7721cell.

Cell Proliferation Assay[2]

Cell Line:	SMMC-7721 and HepG2
Concentration:	0, 10, 20, 40, and 80 μM
Incubation Time:	0, 24, 48, 72, 96, and 120 h
Result:	Decreased the viability with a time-and dose-dependent in both cell lines.

Apoptosis Analysis[2]

Cell Line:	SMMC-7721 and HepG2
Concentration:	0 to 80 μM
Incubation Time:	48 h
Result:	Clearly led to dose-dependent apoptosis, but did not induce hepatoma cell apoptosis at 10 μM and had an apoptotic effect at a starting concentration of 20 μM.

RT-PCR[2]

Cell Line:	SMMC-7721 and HepG2
Concentration:	20 μM and 40 μM
Incubation Time:	48 h
Result:	Significantly increased the mRNA levels of Bax and p53 genes associated with the mitochondrial p53 apoptosis pathway in a dose-dependent manner in HepG2 and SMMC-7721 cells.

	Significantly increased the Bcl-2 mRNA levels in SMMC-7721 cells at a concentration of 40 uM and also increased the Bax/Bcl-2 mRNA ratio.

CUSTOMER VALIDATION

- Int J Mol Sci. 2022 Apr 2;23(7):3980.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.

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REFERENCES

[1]. Liu J, et al. Droxinostat, a Histone Deacetylase Inhibitor, Induces Apoptosis in Hepatocellular Carcinoma Cell Lines via Activation of the Mitochondrial Pathway and Downregulation of FLIP. Transl Oncol. 2016 Feb;9(1):70-8.

[2]. Wood TE et al. Selective inhibition of histone deacetylases sensitizes malignant cells to death receptor ligands. Mol Cancer Ther. 2010 Jan;9(1):246-56.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA