HPOB

Cat. No.: HY-19747

CAS No.: 1429651-50-2

Molecular Formula: $C_{17}H_{18}N_2O_4$

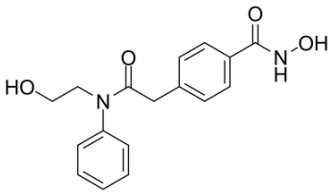
Molecular Weight: 314.34

Target: HDAC; Apoptosis

Pathway: Cell Cycle/DNA Damage; Epigenetics; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (159.06 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1813 mL	15.9063 mL	31.8127 mL
	5 mM	0.6363 mL	3.1813 mL	6.3625 mL
	10 mM	0.3181 mL	1.5906 mL	3.1813 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 (7.95 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.95 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

HPOB is a highly potent and selective inhibitor of HDAC6 with an IC_{50} of 56 nM. HPOB displays >30 fold less potent against other HDACs. HPOB enhances the effectiveness of DNA-damaging anticancer agents in transformed cells but not normal cells. HPOB does not block the ubiquitin-binding activity of HDAC6[1].

IC_{50} & Target

HDAC6 0.056 μM (IC_{50})	HDAC3/NCOR2 1.7 μM (IC_{50})	HDAC8 2.8 μM (IC_{50})	HDAC1 2.9 μM (IC_{50})
HDAC10	HDAC2		

	3.0 μM (IC_{50})	4.4 μM (IC_{50})

In Vitro

HPOB (8, 16, or 32 μM; 72 hours) inhibits growth, however, not viability, of normal or transformed cells[1].
In normal (HFS) and transformed (LNCAP, U87, and A549) cells, HPOB causes accumulation of acetylated α-tubulin and acetylated peroxiredoxin, substrates of HDAC6, but not of acetylated histones. HPOB enhances etoposide-, doxorubicin-, and SAHA-induced transformed cell ((LNCAP, U87, and A549 cells) death but not normal cell death[1].
In LNCaP cells cultured with HPOB and etoposide, there was an increase in cleaved PARP, a marker of apoptosis. Combination of HPOB with etoposide increased the accumulation of DNA damage compared with etoposide alone as evidenced by accumulation of γH2AX in LNCaP cells[1].
HPOB attenuates corticosterone-induced injury in rat adrenal pheochromocytoma PC12 cells by inhibiting mitochondrial GR translocation and the intrinsic apoptosis pathway[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	Normal human foreskin fibroblast (HFS), LNCaP, A549, U87 cells
Concentration:	8, 16, or 32 μM
Incubation Time:	72 hours
Result:	Inhibited cell growth of normal and transformed cells in a concentration-dependent manner but do not induce cell death of normal or transformed cells.

In Vivo

HPOB (300 mg/kg; i.p.; daily for 18 days) and SAHA (50 mg/kg) causes suppression of the growth of established CWR22 tumors[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Nude mice (CWR22 human prostate cancer xenograf)[1]
Dosage:	300 mg/kg
Administration:	I.p.; daily for 18 days
Result:	Combination with SAHA showed significant shrinkage of CWR22 tumors.

CUSTOMER VALIDATION

- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.

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REFERENCES

[1]. Lee JH et al. Development of a histone deacetylase 6 inhibitor and its biological effects. Proc Natl Acad Sci U S A. 2013 Sep 24;110(39):15704-9.

[2]. Li ZY et al. HPOB, an HDAC6 inhibitor, attenuates corticosterone-induced injury in rat adrenal pheochromocytoma PC12 cells by inhibiting mitochondrial GR translocation and the intrinsic apoptosis pathway. Neurochem Int. 2016 Oct;99:239-51.

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

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