HXR9 hydrochloride

Cat. No.: HY-P3245A

Molecular Formula: $C_{119}H_{194}ClN_{53}O_{20}S$

Molecular Weight: 2754.67

Sequence Shortening: WYPWMKKHHRRRRRRRRR

WYPWMKKHHRRRRRRRRR (HCl salt)

Target: Apoptosis

Pathway: Apoptosis

Storage: Sealed storage, away from moisture and light, under nitrogen

Powder -80°C 2 years

-20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light, under nitrogen)

SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (18.15 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.3630 mL	1.8151 mL	3.6302 mL
	5 mM	0.0726 mL	0.3630 mL	0.7260 mL
	10 mM	0.0363 mL	0.1815 mL	0.3630 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (36.30 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

HXR9 hydrochloride is a cell-permeable peptide and a competitive antagonist of HOX/PBX interaction. HXR9 hydrochloride antagonizes the interaction between HOX and a second transcrip-tion factor (PBX), which binds to HOX proteins in paralogue groups1 to 8. HXR9 hydrochloride selectively decreases cell proliferation and promotes apoptosis in cells with a high level of expression of the HOXA/PBX3 genes, such as MLL-rearranged leukemic cells[1][2][3].

In Vitro

HXR9 hydrochloride (60μM; 4 hours) blocks the interaction between PBX and HOX[1].
HXR9 hydrochloride (60μM; 2 hours) triggers apoptosis in B16 and primary melanoma cells[1].
HXR9 hydrochloride (60μM; 2 hours) causes specific transcriptional changes[1].
HXR9 hydrochloride (B16 cells) shows antiproliferative activity with an IC_{50} of 20μM[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis

Cell Line:	murine B16melanoma cells
Concentration:	60 μM
Incubation Time:	4 hours
Result:	Blocked the binding of HOXD9 to PBX.

Apoptosis Analysis

Cell Line:	B16 cells
Concentration:	60 μM
Incubation Time:	2 hours
Result:	A significant proportion of cells were in late phases of apoptosis.

RT-PCR

Cell Line:	B16F10cells
Concentration:	60 μM
Incubation Time:	2 hours
Result:	Fos, Jun, Dusp1, and Atf1 were allsignificantly up-regulate.

In Vivo

HXR9 hydrochloride (10 mg/kg; i.v. via the tail vein; twice weekly) blocks tumor growth[1].
HXR9 hydrochloride (Initial dose of 100 mg/kg (subsequent dosing of 10 mg/kg twice weekly);Intraperitoneal; twice weekly for 18 days) blocks A549 tumour growth in vivo[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57black/6 mice (bearing B16 cells)
Dosage:	10 mg/kg
Administration:	I.v. via the tail vein; twice weekly (~30 days)
Result:	Tumors showed a significant degree of growth retardation.

Animal Model:	Athymic nude mice (bearing A549 cells)
Dosage:	Initial dose of 100 mg/kg (subsequent dosing of 10 mg/kg twice weekly)
Administration:	Intraperitoneal; twice weekly for 18 days
Result:	The tumours of HXR9-treated mice were considerably smaller than those of the control groups.

REFERENCES

[1]. Morgan R, et al. Antagonism of HOX/PBX dimer formation blocks the in vivo proliferation of melanoma. Cancer Res. 2007;67(12):5806-5813.

[2]. Li Z, et al. PBX3 is an important cofactor of HOXA9 in leukemogenesis. Blood. 2013;121(8):1422-1431.

[3]. Plowright L, et al. HOX transcription factors are potential therapeutic targets in non-small-cell lung cancer (targeting HOX genes in lung cancer). Br J Cancer. 2009;100(3):470-475.

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