XMD8-92

Cat. No.: HY-14443

CAS No.: 1234480-50-2

Molecular Formula: $C_{26}H_{30}N_6O_3$

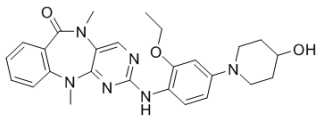
Molecular Weight: 474.55

Target: ERK; Epigenetic Reader Domain

Pathway: MAPK/ERK Pathway; Stem Cell/Wnt; Epigenetics

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 (105.36 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1073 mL	10.5363 mL	21.0726 mL
5 mM	0.4215 mL	2.1073 mL	4.2145 mL
10 mM	0.2107 mL	1.0536 mL	2.1073 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 (5.27 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.27 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

XMD8-92 is a potent ERK5 (BMK1)/BRD4 inhibitor with K_ds of 80 and 190 nM, respectively. XMD8-92 inhibits DCAMKL2, PLK4 and TNK1 with K_ds of 190, 600 and 890 nM, respectively. Anti-cancer activity[1][2].

IC_{50} & Target

BMK1	BRD4
80 nM (Kd)	190 nM (Kd)

In Vitro

XMD8-92 (0-5 Mm; 48 hours) inhibits the proliferation of HMEC and cancer cells[1].
XMD8-92 effectively inhibits BMK1 activation as well as induces PML's (promyelocytic leukemia protein) downstream effector, p21. XMD8-92 significantly inhibits basic fibroblast growth factor (bFGF) induced angiogenesis in Matrigel plugs. XMD8-92 significantly induces p21 expression in HeLa and A549 cells[1].

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

Cell Viability Assay[1]

Cell Line:	HMEC, A549 and HeLa cells
Concentration:	0.16, 0.32, 0.63, 1.25, 2.5 or 5 μM
Incubation Time:	48 hours
Result:	Inhibited the proliferation of HMEC and cancer cells.

In Vivo

XMD8-92 (i.p.; twice a day for 28 days) significantly inhibits the growth of the xenografted human tumors[1].

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

Animal Model:	HeLa Xenograft Model (6-week-old Nod/Scid mice)[1]
Dosage:	50 mg/kg
Administration:	I.p.; twice a day for 28 days
Result:	Significantly inhibited the growth of the xenografted human tumors.

CUSTOMER VALIDATION

- Cancer Cell. 2018 Nov 12;34(5):807-822.
- Sci Signal. 2014 Oct 28;7(349):ra102.
- Stem Cell Reports. 2018 Oct 9;11(4):929-943.
- Target Oncol. 2020 Oct;15(5):659-671.
- PLoS One. 2015 Apr 17;10(4):e0125054.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Yang Q, et al. Pharmacological inhibition of BMK1 suppresses tumor growth through promyelocytic leukemia protein.Cancer Cell. 2010 Sep 14;18(3):258-67.

[2]. Yang Q, et al. Targeting the BMK1 MAP kinase pathway in cancer therapy. Clin Cancer Res. 2011 Jun 1;17(11):3527-32.

[3]. Umapathy G, et al. The kinase ALK stimulates the kinase ERK5 to promote the expression of the oncogene MYCN in neuroblastoma. Sci Signal. 2014 Oct 28;7(349):ra102.

[4]. Lin EC, et al. ERK5 kinase activity is dispensable for cellular immune response and proliferation. Proc Natl Acad Sci U S A. 2016;113(42):11865-11870.

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