MGH-CP1

Cat. No.: HY-139330

CAS No.: 896657-58-2

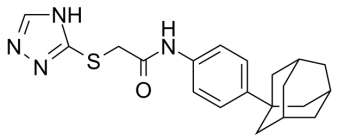
Molecular Formula: $C_{20}H_{24}N_4OS$

Molecular Weight: 368.5

Target: Apoptosis

Pathway: Apoptosis

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (271.37 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7137 mL	13.5685 mL	27.1370 mL
	5 mM	0.5427 mL	2.7137 mL	5.4274 mL
	10 mM	0.2714 mL	1.3569 mL	2.7137 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 (6.78 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.78 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.78 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

MGH-CP1 is a potent and orally active TEAD2 and TEAD4 auto-palmitoylation inhibitor with IC_{50}s of 710 nM and 672 nM, respectively. MGH-CP1 can decrease the palmitoylation levels of endogenous or ectopically expressed TEAD proteins in cells. MGH-CP1 can suppress Myc expression, inhibit epithelial over-proliferation, and induce apoptosis when together with Lats1/2 deletion[1].

IC_{50} & Target

IC_{50}: 710 nM (TEAD2), 672 nM (TEAD4) [1]

In Vitro

MGH-CP1 (0-100 μM) inhibits auto-palmitoylation of recombinant TEAD2 and TEAD4 in a dose-dependent manner[1].

MGH-CP1 (0-2 μM) inhibits TEAD-binding sites (TBS)-Luc reporter activity in a dose-dependent manner in YAP-expressing HEK293 cells[1].
MGH-CP1 does not affect YAP nuclear localization or protein levels but potently inhibits TEAD-mediated transcription in a dose-dependent manner and effectively blocks cell over-proliferation[1].
MGH-CP1 can suppress Myc expression, inhibit epithelial over-proliferation, and induce apoptosis when together with Lats1/2 deletion[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

MGH-CP1 (75mg/kg; PO; daily, for 2 weeks) inhibits the palmitoylation of TEAD proteins in the intestinal epithelium in wild-type mice, but inhibits upregulation of the TEAD target genes, CTGF and ANKRD1, in Lats1/2 KO mice intestine[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Mice (induced high-dose Cre recombination by intraperitoneal injection of 120mg/kg Tamoxifen for two consecutive days)[1]
Dosage:	75 mg/kg
Administration:	PO; daily, for 2 weeks
Result:	Effectively inhibited the palmitoylation of TEAD proteins in the intestinal epithelium in wild-type mice, but effectively inhibited upregulation of the TEAD target genes, CTGF and ANKRD1, in Lats1/2 KO mice intestine.

REFERENCES

[1]. Li Q, Sun Y, Jarugumilli GK, et al. Lats1/2 Sustain Intestinal Stem Cells and Wnt Activation through TEAD-Dependent and Independent Transcription. Cell Stem Cell. 2020;26(5):675-692.e8. doi:10.1016/j.stem.2020.03.002

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