MK-2206 dihydrochloride

Cat. No.: HY-10358

CAS No.: 1032350-13-2

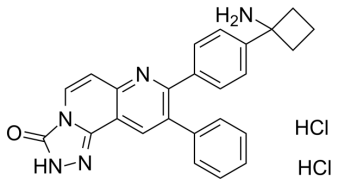
Molecular Formula: $C_{25}H_{23}Cl_2N_5O$

Molecular Weight: 480

Target: Akt; Autophagy; Apoptosis; Organoid

Pathway: PI3K/Akt/mTOR; Autophagy; Apoptosis; Stem Cell/Wnt

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 12.5 mg/mL (26.04 mM; ultrasonic and warming and heat to 60°C)
H_2O : 3.57 mg/mL (7.44 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0833 mL	10.4167 mL	20.8333 mL
	5 mM	0.4167 mL	2.0833 mL	4.1667 mL
	10 mM	0.2083 mL	1.0417 mL	2.0833 mL

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

In Vivo

1. Add each solvent one by one: 20% SBE-β-CD in saline
   Solubility: 25 mg/mL (52.08 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.67 mg/mL (3.48 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.67 mg/mL (3.48 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

MK-2206 dihydrochloride (MK-2206 (2HCl)) is an orally active, BBB-penetrated allosteric AKT inhibitor with IC_{50}s of 5 nM, 12 nM, and 65 nM for AKT1, AKT2, and AKT3, respectively. MK-2206 dihydrochloride induces autophagy[1][2].

IC_{50} & Target

Akt1	Akt2	Akt3
8 nM (IC_{50})	12 nM (IC_{50})	65 nM (IC_{50})

In Vitro

MK-2206 dihydrochloride (MK-2206 (2HCl)) (0-10 μM; 72 and 96 hours) inhibits the nasopharyngeal carcinoma (NPC) cell lines CNE-1, CNE-2, HONE-1, and SUNE-1 proliferation in dose- and time-dependent manner[1].

MK-2206 dihydrochloride (0-10 μM; 24 and 48 hours) results in a dose-dependent increase in the percentage of cells in G0/G1 phase and a concomitant reduction of cell numbers in S phase in CNE-2 and HONE-1 cells[2].
MK-2206 dihydrochloride (0-10 μM; 24 hours) attenuates phosphorylation levels of PRAS40 and S6 in a dose-dependent manner. MK-2206 does not effect phosphorylation of GSKα/β and AKT[2].
MK-2206 dihydrochloride (0-10 μM; 24 hours) increases the appearance of LC3-II in CNE-2 cells dose-dependently. Microtubule-associated protein 1 LC3 is an essential autophagy protein[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	The NPC cell lines CNE-1, CNE-2, HONE-1, and SUNE-1
Concentration:	0.08, 0.16, 0.31, 0.63, 1.25, 2.5, 5, 10 μM
Incubation Time:	72 and 96 hours
Result:	At 72 and 96 hours, the IC_{50} values in CNE-1, CNE-2, and HONE-1 cell lines were 3-5 μM, and in SUNE-1, they were less than 1 μM.

Cell Cycle Analysis[2]

Cell Line:	CNE-2 and HONE-1 cells
Concentration:	0.625, 1.25, 2.5, 5, 10 μM
Incubation Time:	24 or 48 hours
Result:	Induced cell cycle arrest at G1 in a dose-dependent manner.

Western Blot Analysis[2]

Cell Line:	SUNE-1 and CNE-2 cells
Concentration:	0.625, 1.25, 2.5, 5, 10 μM
Incubation Time:	24 hours
Result:	Inhibited phosphorylation of AKT downstream targets.

Cell Autophagy Assay[2]

Cell Line:	CNE-2 cells
Concentration:	0.625, 1.25, 2.5, 5, 10 μM
Incubation Time:	24 hours
Result:	Induced autophagy.

In Vivo

Both MK-2206 dihydrochloride (MK-2206 (2HCl)) doses (oral gavage; 480 mg/kg once a week and 240 mg/kg three times a week; for 2 weeks) can inhibit the growth of human CNE-2 xenografts in nude mice. No other obvious toxicity is observed in mice[2].
MK-2206 dihydrochloride (orally; 120 mg/kg; alternate days; for 3 weeks) significantly inhibits tumor growth in 3-5 week old athymic nude mice with GEO colon carcinoma cells[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Four- to 6-week-old male BALB/c nude mice with CNE-2 xenografts[2]

Dosage:	240 mg/kg and 480 mg/kg
Administration:	Oral gavage; 240 mg/kg for three times a week; 480 mg/kg for once a week; for 2 weeks
Result:	Both doses inhibited the growth of human CNE-2 xenografts in nude mice.

CUSTOMER VALIDATION

- Nature. 2018 Aug;560(7719):499-503.
- Cell. 2014 Feb 13;156(4):771-85.
- Science. 2022 Jul 8;377(6602):eabg9302.
- Cancer Cell. 2018 Jun 11;33(6):1061-1077.e6.
- Signal Transduct Target Ther. 2021 Jun 18;6(1):234.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Zhao YY, et al. Effects of an oral allosteric AKT inhibitor (MK-2206) on human nasopharyngeal cancer in vitro and in vivo. Drug Des Devel Ther. 2014 Oct 10;8:1827-37.

[2]. Agarwal E, et al. Akt inhibitor MK-2206 promotes anti-tumor activity and cell death by modulation of AIF and Ezrin in colorectal cancer. BMC Cancer. 2014 Mar 1;14:145.

[3]. Li Yan, et al. Abstract #DDT01-1: MK-2206: A potent oral allosteric AKT inhibitor. 2009.

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