KN-93 hydrochloride

Cat. No.: HY-15465A

CAS No.: 1956426-56-4

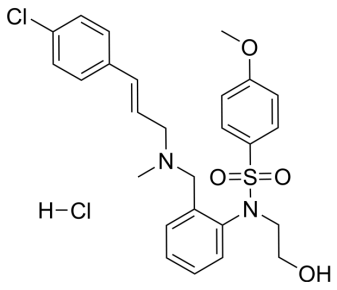
Molecular Formula: $C_{26}H_{30}Cl_2N_2O_4S$

Molecular Weight: 537.5

Target: CaMK; Autophagy

Pathway: Neuronal Signaling; Autophagy

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 : ≥ 31 mg/mL (57.67 mM)

H_2O : 0.45 mg/mL (0.84 mM; Need ultrasonic and warming)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Concentration / Solvent / Mass	1 mg	5 mg	10 mg
	1 mM	1.8605 mL	9.3023 mL	18.6047 mL
	5 mM	0.3721 mL	1.8605 mL	3.7209 mL
	10 mM	0.1860 mL	0.9302 mL	1.8605 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 (4.65 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.65 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.65 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

KN-93 hydrochloride is a cell-permeable, reversible and competitive inhibitor calmodulin-dependent kinase type II (CaMKII) with a K_i of 370 nM.

IC_{50} & Target

CaMK II

In Vitro

After 2 days of KN-93 treatment, 95% of cells are arrested in G1. G1 arrest is reversible; 1 day after KN-93 release, a peak of cells had progressed into S and G2-M. KN-93 also blocks cell growth stimulated by basic fibroblast growth factor, platelet-

derived growth factor-BB, and epidermal growth factor in NIH 3T3 fibroblasts[1]. KN-93 inhibits the H^+, K^+-ATPase activity but strongly dissipates the proton gradient formed in the gastric membrane vesicles and reduces the volume of luminal space[2]. KN-93 (0.5 μM) prevents increased LV developed pressure during action potential prolongation and early afterdepolarizations. Ca^{2+}-independent CaM kinase activity is increased during early afterdepolarizations and this increase is prevented by KN-93[3].

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

CUSTOMER VALIDATION

- Cell. 2022 Jun 23;185(13):2354-2369.e17.
- Nat Commun. 2022 Jul 22;13(1):4255.
- Redox Biol. October 2021, 102115.
- EMBO Mol Med. 2022 Dec 13;e16373.
- Sci Total Environ. 2020 Feb 10;703:134702.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Tombes RM, et al. G1 cell cycle arrest and apoptosis are induced in NIH 3T3 cells by KN-93, an inhibitor of CaMK-II (the multifunctional Ca2+/CaM kinase). Cell Growth Differ. 1995 Sep;6(9):1063-70.

[2]. Mamiya N, et al. Inhibition of acid secretion in gastric parietal cells by the Ca2+/calmodulin-dependent protein kinase II inhibitorKN-93. Biochem Biophys Res Commun. 1993 Sep 15;195(2):608-15.

[3]. Anderson ME, et al. KN-93, an inhibitor of multifunctional Ca++/calmodulin-dependent protein kinase, decreases early afterdepolarizations in rabbit heart. J Pharmacol Exp Ther. 1998 Dec;287(3):996-1006.

[4]. Li J, et al. Curcumin Attenuates Retinal Vascular Leakage by Inhibiting Calcium/Calmodulin-Dependent Protein Kinase II Activity in Streptozotocin-Induced Diabetes. Cell Physiol Biochem. 2016;39(3):1196-208.

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