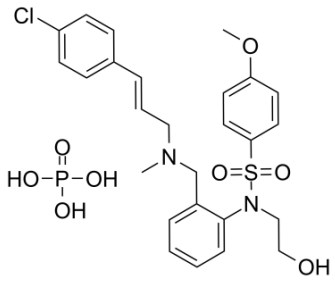
KN-93 phosphate

Cat. No.:	HY-15465B
CAS No.:	1913269-12-1
Molecular Formula:	$C_{26}H_{32}ClN_2O_8PS$
Molecular Weight:	599.03
Target:	CaMK; Autophagy
Pathway:	Neuronal Signaling; Autophagy
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 : 100 mg/mL (166.94 mM; Need ultrasonic)
H_2O : 50 mg/mL (83.47 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.6694 mL	8.3468 mL	16.6937 mL
5 mM	0.3339 mL	1.6694 mL	3.3387 mL
10 mM	0.1669 mL	0.8347 mL	1.6694 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: ≥ 10 mg/mL (16.69 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 10 mg/mL (16.69 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 10 mg/mL (16.69 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	KN-93 phosphate is a novel membrane-permeant synthetic inhibitor of purified neuronal CaMK-II, with K_i of 370 nM.
IC_{50} & Target	Ki: 370 nM (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 Syst. 2018 Apr 25;6(4):424-443.e7.
- Diabetes. 2018 Sep;67(9):1748-1760.
- Sci Total Environ. 2020 Feb 10;703:134702.
- Oxid Med Cell Longev. 2019 Dec 7;2019:2193019.
- Oxid Med Cell Longev. 2019 Dec 7;2019:2193019.

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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.

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