Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

KN-93

Catalog No: tcsc1095

Available Sizes

Size: 1mg

Size: 5mg

Size: 10mg

Size: 25mg

Size: 50mg

Specifications

CAS No:
139298-40-1

Formula:
$C_{26}H_{29}ClN_2O_4S$

Pathway:
Neuronal Signaling

Target:
CaMK

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 50 mg/mL (99.79 mM)

Observed Molecular Weight:
501.04

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Product Description

KN-93 is a cell-permeable, reversible and competitive inhibitor calmodulin-dependent kinase type II (**CaMKII**) with a $\mathbf{K_i}$ of 370 nM.

IC50 & Target: Ki: 370 nM (CaMK)

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, epidermal growth factor, and insulin-like growth factor-1 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]. KN-93 (10 μM)significantly inhibits the activation of CaMKII/NF-κB signaling induced by elevated glucose, and subsequently decreases the expression of VEGF, iNOS and ICAM-1 in Müller cells[4].

In Vivo: KN-93 (1 mg/kg/day, i.p.) inhibits retinal vascular leakage induced by diabetes, and suppresses phosphorylation of CaMKII and NF-κB in diabetic retina[4].