Web: www.taiclone.com
Tel: +886-2-2735-9682
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# 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].