

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

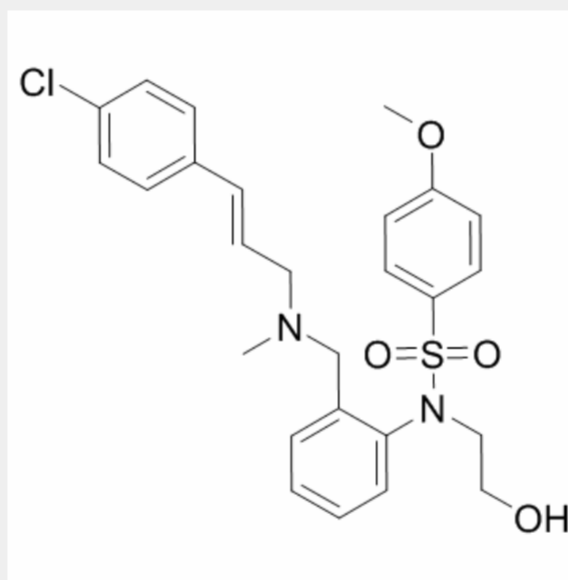
Product Description

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

IC50 & Target: K_i : 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 μ M) 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 μ M) significantly inhibits the activation of CaMKII/NF- κ B 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- κ B in diabetic retina^[4].



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