kb NB 142-70

Cat. No.:	HY-15528		
CAS No.:	1233533-04-4		
Molecular Formula:	C ₁₁ H ₉ NO ₂ S ₂		
Molecular Weight:	251.32		
Target:	PKD		
Pathway:	Apoptosis		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year

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SOLVENT & SOLUBILITY

Preparing Stock Solutions		Mass Solvent Concentration	1 mg	5 mg	10 mg		
	1 mM	3.9790 mL	19.8950 mL	39.7899 mL			
		5 mM	0.7958 mL	3.9790 mL	7.9580 mL		
		10 mM	0.3979 mL	1.9895 mL	3.9790 mL		
	Please refer to the so	Please refer to the solubility information to select the appropriate solvent.					
n Vivo	1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 3.5 mg/mL (13.93 mM); Clear solution						
		2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 3.5 mg/mL (13.93 mM); Clear solution					

BIOLOGICAL ACTIVITY			
Description	kb NB 142-70 is a potent PKD inhibitor, with IC ₅₀ s of 28.3, 58.7 and 53.2 nM for PKD1, PKD2, and PKD3, respectively. kb NB 142-70 also has antitumor activity.		
IC₅₀ & Target	PKD1 28.3 nM (IC ₅₀)	PKD3 53.2 nM (IC ₅₀)	PKD2 58.7 nM (IC ₅₀)
In Vitro	kb NB 142-70 is a potent PKD inhibitor, with IC ₅₀ s of 28.3, 58.7 and 53.2 nM for PKD1, PKD2, and PKD3, respectively. kb NB 142-70 also inhibits Ser ⁹¹⁶ phosphorylation of PKD1 (IC ₅₀ , 2.2 ± 0.6 μ M) in LNCaP cells. Moreover, kb NB 142-70 is cytotoxic against PC3 cells with an EC ₅₀ of 8.025 μ M ^[1] . kb NB 142-70 (0-5 μ M) concentration-dependently prevents ANG II-induced phosphorylation of HDAC4 at Ser ²⁴⁶ and Ser ⁶³² , HDAC5 at Ser ²⁵⁹ and Ser ⁴⁹⁸ , and HDAC7 at Ser ¹⁵⁵ in IEC-18 cells. In addition,		

Product Data Sheet

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()) 0 kb NB 142-70 (3.5 μM) also suppresses HDAC4, HDAC5, and HDAC7 phosphorylation in IEC-18 cells stimulated with ANG II for 0-240 min or with vasopressin, lysophosphatidic acid (LPA), or phorbol 12,13-dibutyrate (PDBu)^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL	
Cell Assay ^[1]	Briefly, PC3 cells are treated with kb NB 142-70 at 10 μM concentration for 48 h, and then fixed in 70% ice-cold ethanol overnight and labeled with propidium iodide. The labeled cells are analyzed using a FACScan Benchtop Cytometer ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Lavalle CR, et al. Novel protein kinase D inhibitors cause potent arrest in prostate cancer cell growth and motility. BMC Chem Biol. 2010 May 5;10:5.

[2]. James Sinnett-Smith, et al. Protein kinase D1 mediates class IIa histone deacetylase phosphorylation and nuclear extrusion in intestinal epithelial cells: role in mitogenic signaling. Am J Physiol Cell Physiol. 2014 May 15; 306(10): C961-C971.

Caution: Product has not been fully validated for medical applications. For research use only.