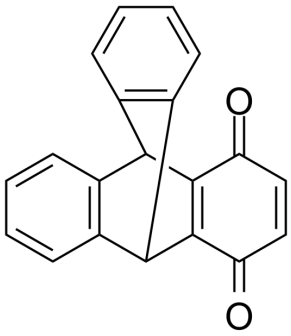
INCA-6

Cat. No.:	HY-108544
CAS No.:	3519-82-2
Molecular Formula:	$C_{20}H_{12}O_2$
Molecular Weight:	284.31
Target:	Others
Pathway:	Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

INCA-6 (Triptycene-1,4-quinone) is a cell-permeable NFAT inhibitor. INCA-6 specifically blocks targeting of NFAT(P) substrate to the calcineurin (CN) phosphatase site and is an effective inhibitor of CN-NFAT signaling[1][2][3].

In Vitro

INCA-6 (5 μM; for 24-hour) prevents transient outward K+ current (Ito) downregulation in 3-Hz cells[1].
Pre-treatment of BV-2 cells with INCA-6 (10 μM) significantly inhibits ATP-induced CXCL2 expression in BV-2 cells. INCA-6 also inhibits ATP-induced CXCL2 expression in rat primary microglia[2].
INCA-6 (5 μM) reduces SERCA2 transcript levels as well as protein expression, in the absence or in the presence of thapsigargin (TG)[3].
INCA-6 (1.0 and 2.5 μM; 24 hours) treatment significantly decreases both VEGF and serum-induced human retinal microvascular endothelial cells (HRMEC) proliferation, but does not affect baseline proliferation[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[4]

Cell Line:	Human retinal microvascular endothelial cells
Concentration:	0.5, 1.0, or 2.5 μM
Incubation Time:	24 hours
Result:	Significantly inhibited VEGF-induced proliferation at 1.0 and 2.5 μM concentrations.

In Vivo

INCA-6 (5.0, or 25.0 μM) treatment significantly reduces pathologic neovascularization in oxygen-induced retinopathy (OIR)[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Rats bearing OIR model[4]
Dosage:	2.5, 5.0, or 25.0 μM
Administration:	Intravitreal injection on days 14(0) and 14(3)
Result:	Decreased the severity of OIR in a dose dependent manner. Significant inhibition was seen at 5.0 and 25.0 μM concentrations.

REFERENCES

[1]. Ling Xiao, et al. Mechanisms underlying rate-dependent remodeling of transient outward potassium current in canine ventricular myocytes. Circ Res. 2008 Sep 26;103(7):733-42.

[2]. Miho Shiratori,et al. P2X7 receptor activation induces CXCL2 production in microglia through NFAT and PKC/MAPK pathways. J Neurochem. 2010 Aug;114(3):810-9.

[3]. Anand Mohan Prasad, et al. Silencing calcineurin A subunit reduces SERCA2 expression in cardiac myocytes. Am J Physiol Heart Circ Physiol. 2011 Jan;300(1):H173-80.

[4]. Colin A Bretz, et al. The role of the NFAT signaling pathway in retinal neovascularization. Invest Ophthalmol Vis Sci. 2013 Oct 25;54(10):7020-7.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA