Ro 31-8220 mesylate

Cat. No.: HY-13866

CAS No.: 138489-18-6

Molecular Formula: $C_{26}H_{27}N_5O_5S_2$

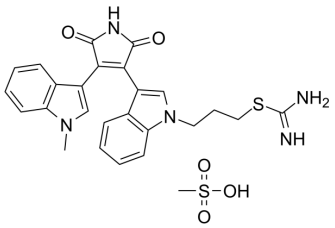
Molecular Weight: 553.65

Target: PKC

Pathway: Epigenetics; TGF-beta/Smad

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 1 years; -20°C, 6 months (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 35.71 mg/mL (64.50 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.8062 mL	9.0310 mL	18.0620 mL
5 mM	0.3612 mL	1.8062 mL	3.6124 mL
10 mM	0.1806 mL	0.9031 mL	1.8062 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (3.76 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.76 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ro 31-8220 mesylate is a potent PKC inhibitor, with IC_{50}s of 5, 24, 14, 27, 24 and 23 nM for PKCα, PKCβI, PKCβII, PKCγ, PKCε and rat brain PKC, respectively. Ro 31-8220 also significantly inhibits MAPKAP-K1b, MSK1, S6K1 and GSK3β (IC_{50}s, 3, 8, 15, and 38 nM, respectively), with no effect on MKK3, MKK4, MKK6 and MKK7.

IC_{50} & Target

PKC-α 5 nM (IC_{50})	PKC-βII 14 nM (IC_{50})	PKC-βI 24 nM (IC_{50})	PKC-ε 24 nM (IC_{50})
PKC-γ 27 nM (IC_{50})	Rat Brain PKC 23 nM (IC_{50})	MAPKAP-K1b 3 nM (IC_{50})	MSK1 8 nM (IC_{50})
S6K1	GSK3β		

	15 nM (IC_{50})	38 nM (IC_{50})
In Vitro	Ro 31-8220 mesylate is a potent PKC inhibitor, with IC_{50}s of 5, 24, 14, 27, 24 and 23 nM for PKCα, PKCβI, PKCβII, PKCγ, PKCε and rat brain PKC, respectively[1]. Ro 31-8220 also significantly inhibits MAPKAP-K1b, MSK1, S6K1 and GSK3β (IC_{50}s, 3, 8, 15, and 38 nM, respectively), with no effect on MKK3, MKK4, MKK6 and MKK7. Moreover, Ro 31-8220 directly suppresses voltage-dependent Na^+ channels[2]. Ro 31-8220 (1 μM) is neuroprotective against paraoxon-induced neuronal cell death in cerebellar granule neurons, blocks paraoxon-induced caspase-3 activity, and reduces the paraoxon-induced increase in phospho-PKC pan levels[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
In Vivo	Ro 31-8220 (6 mg/kg/d, s.c.) is well tolerated, and has half-life of 5.7 hours in mice. Ro 31-8220-treated $MLP^{-/-}$ mice show a dramatic rescue in fractional shortening after treatment for 6 weeks, but the WT mice shows no change[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.	

PROTOCOL

Cell Assay [1]	A neurotoxic concentration of paraoxon (200 μM) is added to the granule cell cultures for the indicated time on day in vitro (DIV) 8. The following drugs are added to the granule cell cultures prior to or after paraoxon exposure on DIV 8: Ro-81-3220 (1 μM) is added 15 min prior to or 3 h after the addition of paraoxon. TPA (0.1 μM) is added 15 min prior to the addition of paraoxon[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4]	Mice[4] The affects of long-term Ro 31-8220 administration over 4 to 6 weeks in $MLP^{-/-}$ heart failure mice are investigated. All mice are assessed for ventricular performance by echocardiography at the beginning of the study and 6 weeks later. Ro 31-8220 (or vehicle) is injected subcutaneously once per day at a dosage of 6 mg/kg/d[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2018 Sep 11;9(1):3688.
- J Clin Invest. 2021 Dec 29;e150101.
- EMBO Mol Med. 2022 Dec 13;e16373.
- Aging Cell. 2022 Feb 23;e13573.
- Aging Cell. 2020 Oct;19(10):e13217.

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REFERENCES

[1]. Wilkinson SE, et al. Isoenzyme specificity of bisindolylmaleimides, selective inhibitors of protein kinase C. Biochem J. 1993 Sep 1;294 (Pt 2):335-7.

[2]. Davies SP, et al. Specificity and mechanism of action of some commonly used protein kinase inhibitors. Biochem J. 2000 Oct 1;351(Pt 1):95-105.

[3]. Tian F, et al. Inhibition of protein kinase C protects against paraoxon-mediated neuronal cell death. Neurotoxicology. 2007 Jul;28(4):843-9. Epub 2007 Apr 20.

[4]. Hambleton M, et al. Pharmacological- and gene therapy-based inhibition of protein kinase Calpha/beta enhances cardiac contractility and attenuates heart failure. Circulation. 2006 Aug 8;114(6):574-82.

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

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