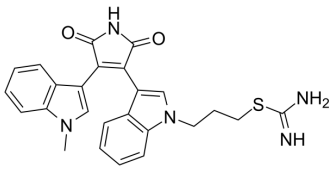
Ro 31-8220

Cat. No.:	HY-13866A
CAS No.:	125314-64-9
Molecular Formula:	$C_{25}H_{23}N_5O_2S$
Molecular Weight:	457.55
Target:	PKC
Pathway:	Epigenetics; TGF-beta/Smad
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Ro 31-8220 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	IC50: 5 nM (PKCα), 24 nM (PKCβI), 14 nM (PKCβII), 27 nM (PKCγ), 24 nM (PKCε), 23 nM (Rat brain PKC)[1], 3 nM (MAPKAP-K1b), 8 nM (MSK1), 15 nM (S6K1), 38 nM (GSK3β)[2]
In Vitro	Ro 31-8220 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.
- Aging Cell. 2022 Feb 23;e13573.
- Aging Cell. 2020 Oct;19(10):e13217.
- Front Immunol. 2021 Feb 2;11:625542.

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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.

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