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Produktinformation

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See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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SR33805

Cat. No.: HY-136909

CAS No.: 121345-64-0

Molecular Formula: $C_{32}H_{40}N_2O_5S$

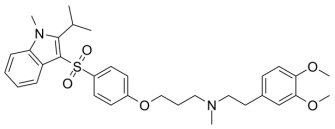
Molecular Weight: 564.74

Target: Calcium Channel

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (177.07 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.7707 mL	8.8536 mL	17.7073 mL
5 mM	0.3541 mL	1.7707 mL	3.5415 mL
10 mM	0.1771 mL	0.8854 mL	1.7707 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 (3.68 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.68 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SR33805 is a potent Ca^{2+} channel antagonist, with EC_{50}s of 4.1 nM and 33 nM in depolarized and polarized conditions, respectively. SR33805 blocks L-type but not T-type Ca^{2+} channels. SR33805 can be used for the research of acute or chronic failing hearts[1][2].

IC_{50} & Target

L-type calcium channel 4.1 nM (EC50, in depolarized conditions)	L-type calcium channel 33 nM (EC50, in polarized conditions)

In Vitro

SR33805 (0.01-10 μM; 3 d) inhibits growth factor-induced proliferation of SMC (0.2050<0.46 μM) in a dose-dependent manner

[3].

SR33805 (10 μM; 10 min) restores the myocardial infarction (MI)-altered cell shortening without affecting the Ca^{2+} transient amplitude[2].

SR33805 (10 μM) decreases the activity of recombinant PKA[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[3]

Cell Line:	Smooth muscle cells (SMC)
Concentration:	0.01, 0.1, 1, 10 μM
Incubation Time:	3 days
Result:	Inhibited in a dose-dependent manner FCS-, bFGF and PDGF-induced proliferation of porcine SMC with IC_{50}s of 0.26±0.08, 0.46±0.1 and 0.20±0.04 μM, respectively.

In Vivo

SR33805 (20 mg/kg; a single i.p.) improves end-systolic strain and fractional shortening of MI hearts in rats[2].

SR33805 (5 mg/kg/day; p.o. for 38 d) significantly reduces intimal hyperplasia in pigs[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar rats (5 weeks) are subjected to coronary artery ligature[2]
Dosage:	0.2, 2, 20 mg/kg
Administration:	A single i.p. injection
Result:	Increased significantly both end-systolic strain (ESS) and fractional shortening (FS) by about +38 and +26%, respectively at the dose of 20 mg/kg. Did not affect other contractile parameters.

REFERENCES

[1]. Romey G, et, al. Effects of two chemically related new Ca2+ channel antagonists, SR33557 (fantofarone) and SR33805, on the L-type cardiac channel. Eur J Pharmacol. 1994 Sep 22; 263(1-2): 101-5.

[2]. Mou YA, et, al. Beneficial effects of SR33805 in failing myocardium. Cardiovasc Res. 2011 Aug 1; 91(3): 412-9.

[3]. Hainaud P, et, al. The calcium inhibitor SR33805 reduces intimal formation following injury of the porcine carotid artery. Atherosclerosis. 2001 Feb 1; 154(2): 301-8.

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