Resiniferatoxin

Cat. No.: HY-N2333

CAS No.: 57444-62-9

Molecular Formula: $C_{37}H_{40}O_9$

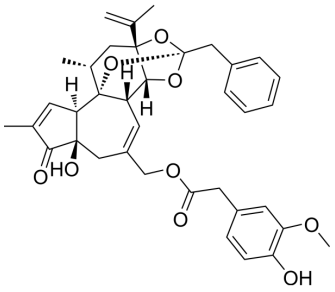
Molecular Weight: 628.71

Target: TRP Channel

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: -20°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (79.53 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.5906 mL	7.9528 mL	15.9056 mL
	5 mM	0.3181 mL	1.5906 mL	3.1811 mL
	10 mM	0.1591 mL	0.7953 mL	1.5906 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: ≥ 1.25 mg/mL (1.99 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.25 mg/mL (1.99 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Resiniferatoxin ((+)-Resiniferatoxin), is a selective agonist of transient receptor potential vanilloid 1 (TRPV1) receptor agonist. Resiniferatoxin can be isolated from the Euphorbia resinifera plant. Resiniferatoxin eliminates TRPV1+ primary sensory afferents and blunt cardiac sympathetic afferent reflex for a relatively long period[1][2].

In Vitro

Resiniferatoxin causes extremely prolonged channel opening and calcium influx, which results in cytotoxicity to the TRPV1-positive pain fibers or cell bodies[1].

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

In Vivo

Resiniferatoxin (2 μg/10 μl; injected intrathecally into the T2/T3 interspace; four weeks after coronary artery occlusion to induce heart failure in rats) significantly and selectively abolishes the afferent markers expression (TRPV1 and calcitonin gene-related peptide) in dorsal horn and reduced overactivated CSNA. Resiniferatoxin significantly reverses the

prolongation of action potential duration (APD) and APD alternan, reduces the inducibilities of ventricular arrhythmias[2].

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

REFERENCES

[1]. Brown DC. Resiniferatoxin: The Evolution of the "Molecular Scalpel" for Chronic Pain Relief. Pharmaceuticals (Basel). 2016;9(3):47. Published 2016 Aug 11.

[2]. Wu Y, et al. Resiniferatoxin reduces ventricular arrhythmias in heart failure via selectively blunting cardiac sympathetic afferent projection into spinal cord in rats. Eur J Pharmacol. 2020;867:172836.

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