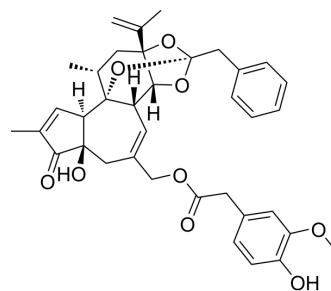


## Resiniferatoxin

**Cat. No.:** HY-N2333  
**CAS No.:** 57444-62-9  
**Molecular Formula:** C<sub>37</sub>H<sub>40</sub>O<sub>9</sub>  
**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	<div>Solvent Concentration</div>	Mass	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 <sup>[1][2]</sup> .
In Vitro	Resiniferatoxin causes extremely prolonged channel opening and calcium influx, which results in cytotoxicity to the TRPV1-positive pain fibers or cell bodies <sup>[1]</sup> . 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

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prolongation of action potential duration (APD) and APD alternans, reduces the inducibilities of ventricular arrhythmias<sup>[2]</sup>. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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## REFERENCES

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[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.

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

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