Notoginsenoside R1

Cat. No.: HY-N0615

CAS No.: 80418-24-2

Molecular Formula: $C_{47}H_{80}O_{18}$

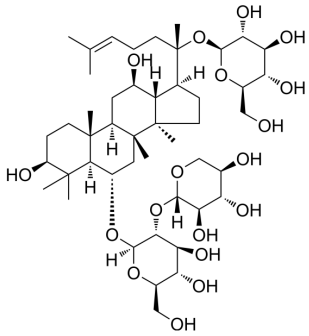
Molecular Weight: 933.13

Target: Amyloid-β; Apoptosis

Pathway: Neuronal Signaling; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (107.17 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.0717 mL	5.3583 mL	10.7166 mL
	5 mM	0.2143 mL	1.0717 mL	2.1433 mL
	10 mM	0.1072 mL	0.5358 mL	1.0717 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: ≥ 2.5 mg/mL (2.68 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (2.68 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (2.68 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Notoginsenoside R1 (Sanchinoside R1), a saponin, is isolated from P. notoginseng. Notoginsenoside R1 exhibits anti-oxidation, anti-inflammatory, anti-angiogenic, and anti-apoptosis activities. Notoginsenoside R1 provides cardioprotection against ischemia/reperfusion (I/R) injury. Notoginsenoside R1 also provides neuroprotection in H_2O_2-induced oxidative damage in PC12 cells[1][2][3].

In Vitro

Notoginsenoside R1 (2.5-80 μM; 24 h) inhibits the hypoxia-reoxygenation (H/R)-induced cell death, intracellular ROS accumulation, and mitochondrial membrane depolarization in H9c2 cardiomyocytes[1].

Notoginsenoside R1 (5-20 μM; 24 h) inhibits the H/R-induced H9c2 cardiomyocytes apoptosis in a concentration-dependent manner[1].
Notoginsenoside R1 (1-100 μM; 24 h) dose-dependently protects PC12 cells and primary neurons from Aβ-induced cell death and apoptosis[2].
Notoginsenoside R1 (10 μM; 24 h) inhibits $A\beta_{25-35}$-induced ROS production, mitochondrial damage and MAPK activation in PC12 cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Notoginsenoside R1 (5 mg/kg/h; infused via the right jugular vein) increases red blood cell velocity, reduces the number of adherent leukocytes and inhibits mast cell degranulation and cytokine elevation in rats[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley (SD) rats (200-250 g)[3]
Dosage:	5 mg/kg/h
Administration:	Infused 20 min before LPS infusion via the right jugular vein
Result:	Ameliorated the LPS-induced reduction in the mesenteric venular shear rate to some extent. Attenuated the LPS-induced adhesion of leukocytes to the venular wall. Inhibited mast cell degranulation and cytokine elevation.

CUSTOMER VALIDATION

- Front Cell Neurosci. 2020 Sep 4;14:280.

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REFERENCES

[1]. Yu Y, et, al. Cardioprotective effects of Notoginsenoside R1 against ischemia/reperfusion injuries by regulating oxidative stress- and endoplasmic reticulum stress-related signaling pathways. Sci Rep. 2016 Feb 18;6:21730.

[2]. Ma B, et, al. Notoginsenoside R1 attenuates amyloid-β-induced damage in neurons by inhibiting reactive oxygen species and modulating MAPK activation. Int Immunopharmacol. 2014 Sep;22(1):151-9.

[3]. Sun K, et, al. Protective effects of ginsenoside Rb1, ginsenoside Rg1, and notoginsenoside R1 on lipopolysaccharide-induced microcirculatory disturbance in rat mesentery. Life Sci. 2007 Jul 19;81(6):509-18.

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