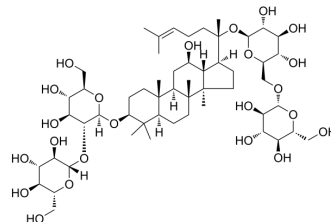


Ginsenoside Rb1

Cat. No.:	HY-N0039		
CAS No.:	41753-43-9		
Molecular Formula:	C ₅₄ H ₉₂ O ₂₃		
Molecular Weight:	1109.29		
Target:	Na ⁺ /K ⁺ ATPase; IRAK; NF-κB; Autophagy; Mitophagy; HSV		
Pathway:	Membrane Transporter/Ion Channel; Immunology/Inflammation; NF-κB; Autophagy; Anti-infection		
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 (90.15 mM; Need ultrasonic)
 H₂O : ≥ 33.33 mg/mL (30.05 mM)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	0.9015 mL	4.5074 mL	9.0148 mL
	5 mM	0.1803 mL	0.9015 mL	1.8030 mL
	10 mM	0.0901 mL	0.4507 mL	0.9015 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (2.25 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (2.25 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (2.25 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ginsenoside Rb1, a main constituent of the root of Panax ginseng, inhibits Na⁺, K⁺-ATPase activity with an IC₅₀ of 6.3±1.0 μM. Ginsenoside also inhibits IRAK-1 activation and phosphorylation of NF-κB p65.

IC₅₀ & Target

Na ⁺ , K ⁺ -ATPase	IRAK-1	p65	Autophagy
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	6.3 μ M (IC ₅₀)		
	Mitophagy	HSV-1	
In Vitro	<p>Rat brain microsomal Na⁺, K⁺-ATPase activity is inhibited significantly and rapidly by Ginsenoside Rb1. The IC₅₀ of Ginsenoside Rb1 for Na⁺,K⁺-ATPase is 6.3±1.0 μM. The inhibition is enhanced with increasing the concentration of Ginsenoside Rb1 or decreasing that of Na⁺ and K⁺. Kinetic analysis reveals that Ginsenoside is an uncompetitive inhibitor with respect to ATP^[1]. Ginsenoside Rb1 significantly inhibits the activation of interleukin-1 receptor-associated kinase-1 (IRAK-1), IKK-β, NF-κB, and MAP kinases (ERK, JNK, and p-38); however, interaction between LPS and Toll-like receptor-4, IRAK-4 activation and IRAK-2 activation are unaffected^[2]. Ginsenoside Rb1 is an ingredient of a Chinese medicine Panax ginseng. Ginsenoside Rb1 is a major bioactive compound in the regulating pregnane X receptor (PXR)/NF-κB signaling. Ginsenoside Rb1 is the compound with potent anti-inflammatory activity in ginseng saponin extract (GSE). The concentration for Ginsenoside Rb1 (10 μM) is optimized from a preliminary study to ensure sufficient anti-inflammatory activity and without apparent cytotoxicity. Ginsenoside Rb1 significantly reduces TNF-α-induced upregulation of IL-1β and iNOS mRNA levels, and restores the mRNA levels of PXR and CYP3A4 in LS174T cells. TNF-α causes a significant reduction in PXR protein levels and increase in the ratio of phosphorylated to total NF-κB p65, both of which are significantly abrogated by Ginsenoside Rb1^[3].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>		
In Vivo	<p>Ginsenoside Rb1 at the both doses of 30 mg/kg and 60 mg/kg significantly attenuates the histological lung injury. Ginsenoside Rb1 at the dose of 30 mg/kg and 60 mg/kg both significantly attenuates the histological intestine injury^[4]. Ginsenoside Rb1 (Rb1), an ingredient of a Chinese medicine Panax ginseng, has beneficial effects on mesentery microvascular hyperpermeability induced by Lipopolysaccharide (LPS) and the underlying mechanisms. In some rats, Ginsenoside Rb1 (5 mg/kg per hour) is administrated through the left jugular vein 30 min after LPS infusion. Ginsenoside Rb1 decreases caveolae number in endothelial cells of microvessels. Ginsenoside Rb1 ameliorates microvascular hyperpermeability after the onset of endotoxemia and improves intestinal edema through inhibiting caveolae formation and junction disruption, which is correlated to suppression of NF-κB and Src activation^[5].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>		

PROTOCOL

Cell Assay ^[3]

LS174T cells are seeded in cell imaging dish. After overnight incubation, cells are treated with GSE (100 μ g/mL), Ginsenoside Rb1 (10 μ M), or CK (10 μ M) for 3 hours, followed by an additional incubation with or without TNF- α (20 ng/ml) for 6 hours. At the end of the incubation, cells are harvested and fixed with 4% paraformaldehyde solution at 20°C for 20 minutes. After washing in PBS, cells are permeabilized with 0.2% Triton X-100 in PBS at room temperature for 5 minutes. After incubation in blocking buffer containing 0.1% Triton X-100 and 5% bovine serum albumin, cells are incubated with rabbit NF- κ B p65 antibody at 4°C overnight and then with Alexa Fluor 488-conjugated anti-rabbit IgG antibody at room temperature for 30 minutes in 1% bovine serum albumin in PBS. Fluorescence photographs are obtained using a Zeiss 710 confocal microscope ^[3].

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Animal Administration ^{[4][5]}

Mice^[4]
 Male C57BL/6 mice (9-12 weeks old; 17-22 g) are randomly allocated into eight groups (n=8 in each group): (1) Sham surgical preparation including isolation of the superior mesenteric artery (SMA) without occlusion is performed (Sham); (2) mice are subjected to II/R without treatment (II/R); (3) mice are subjected to II/R with treatment of normal saline 10 minutes before reperfusion (II/R+NS); (4), (5) mice are treated with 30 mg/kg (II/R+Rb1-30) or 60 mg/kg (II/R+Rb1-60) Ginsenoside Rb1, in which surgery is performed as in the II/R group with administration of the Ginsenoside Rb1 intraperitoneally 10 minutes before reperfusion; (6) mice are subjected to Sham surgery and treated with ATRA (ATRA+Sham), which is the inhibitor of Nrf2/ARE signaling pathway; (7) mice are subjected to II/R and treated with ATRA (ATRA+II/R); (8) mice are subjected to II/R and treated with ATRA and 60 mg/kg Ginsenoside Rb1 as group 5 (ATRA+II/R+Rb1-60). During the last two weeks before the operation, the mice in the group 6, 7, 8 receive ATRA i.p. daily at 10 mg/kg and fed on a vitamin A-deficient diet, and the mice in the other groups receive the equivalent volume of corn oil and fed on a control normal diet.

Rats^[5]

Male Wistar rats weighing 200-250 g are used. The rats are randomly divided into four groups, 26 animals in each. After being anesthetized with urethane (2 g/kg body wt im), the left femoral vein and left jugular vein of the rat are cannulated. In the LPS group, LPS solution in saline is infused (5 mg/kg per hour) for 90 min via the left femoral vein. The vehicle, instead of LPS solution, is administered in Sham and Ginsenoside Rb1 alone (Rb1, 5 mg/kg) groups. In the Rb1 posttreatment (LPS+Rb1) group, Ginsenoside Rb1 solution is infused continuously through the left jugular vein 30 min after LPS administration at the dose of 5 mg/kg. Ginsenoside Rb1 solution and the same volume of saline are infused in Ginsenoside Rb1 and Sham groups, respectively, without subsequent LPS administration. In a separate set of experiments, the rats are anesthetized with 2% penobarbital sodium (60 mg/kg body wt ip), and saline, LPS, and Ginsenoside Rb1. After recovery from anesthesia, the animals are allowed access to water and rodent chow, and survival rate is recorded over time until 4 days after LPS stimulation.

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

CUSTOMER VALIDATION

- Cell Death Dis. 2022 Sep 26;13(9):824.
- Phytomedicine. 2023 Dec, 121, 155083.
- Front Pharmacol. 2021 Apr 27;12:643188.
- Research Square Print. September 1st, 2022.
- Research Square Preprint. 2021 Feb.

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- [2]. Joh EH, et al. Ginsenoside Rb1 and its metabolite compound K inhibit IRAK-1 activation--the key step of inflammation. Biochem Pharmacol. 2011 Aug 1;82(3):278-86.
- [3]. Zhang J, et al. Ginsenosides Regulate PXR/NF-κB Signaling and Attenuate Dextran Sulfate Sodium-Induced Colitis. Drug Metab Dispos. 2015 Aug;43(8):1181-9.
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- [5]. Zhang Y, et al. Ginsenoside Rb1 ameliorates lipopolysaccharide-induced albumin leakage from rat mesenteric venules by intervening in both trans- and paracellular pathway. Am J Physiol Gastrointest Liver Physiol. 2014 Feb 15;306(4):G289-300.

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