Ginsenoside C-K

Cat. No.: HY-N0904

CAS No.: 39262-14-1

Molecular Formula: $C_{36}H_{62}O_8$

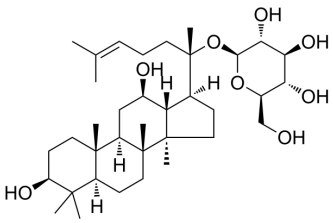
Molecular Weight: 622.87

Target: COX; NO Synthase; Cytochrome P450

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease

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 (160.55 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.6055 mL	8.0274 mL	16.0547 mL
	5 mM	0.3211 mL	1.6055 mL	3.2109 mL
	10 mM	0.1605 mL	0.8027 mL	1.6055 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: ≥ 2.75 mg/mL (4.42 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.75 mg/mL (4.42 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.75 mg/mL (4.42 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ginsenoside C-K, a bacterial metabolite of G-Rb1, exhibits anti-inflammatory effects by reducing iNOS and COX-2. Ginsenoside C-K exhibits an inhibition against the activity of CYP2C9 and CYP2A6 in human liver microsomes with IC_{50}s of 32.0±3.6 μM and 63.6±4.2 μM, respectively.

IC_{50} & Target	COX-2	iNOS	CYP2C9	CYP2A6
			32 μM (IC_{50})	63.6 μM (IC_{50})

In Vitro	Ginsenoside C-K, a bacterial metabolite of G-Rb1, exhibits anti-inflammatory effects mainly by reducing inducible nitric oxide synthase (iNOS), cyclooxygenase (COX)-2, and proinflammatory cytokines. Ginsenoside C-K suppresses the expression of proinflammatory cytokines by downregulating the activities of IRAK-1, MAPKs, IKK-α, and NF-κB in LPS-treated murine peritoneal macrophages. Ginsenoside C-K also suppresses the expression of iNOS and COX-2 by inhibiting NF-κB signaling in LPS-stimulated RAW264.7 cells. In zymosan-treated bone-marrow-derived macrophages (BMDMs) and RAW264.7 cells, Ginsenoside C-K inhibits inflammatory responses by negatively regulating the secretion of proinflammatory cytokines, the activation of MAPKs, and the generation of ROS. In addition, anti-inflammatory activity of Ginsenoside C-K has been observed in LPS-stimulated microglial cells. Ginsenoside C-K hinders inflammatory responses by controlling both the generation of ROS and the activities of MAPKs, NF-κB, and AP-1[1]. Ginsenoside C-K, a major metabolite of ginsenosides in the gastrointestinal tract, inhibits NF-κB signaling in a PXR-dependent manner. Ginsenoside C-K is shown to promote recovery of dextran sulfate sodium (DSS) -induced colitis by suppressing NF-κB activation. Ginsenoside C-K 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[2]. Ginsenoside C-K, one of the intestinal metabolites of 20(S)-protopanaxadiol derivatives, exhibits an inhibition against the activity of CYP2C9 in human liver microsomes with an IC_{50} value of 32.0±3.6 μM, a weak inhibition against the activity of CYP2A6 in human liver microsomes with an IC_{50} value of 63.6±4.2 μM, and an even weaker inhibition against the activity of CYP2D6 in human liver microsomes with an IC_{50} value of more than 100 μM[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The weight of the collagen-induced arthritis (CIA) mice increases slowly and is significantly less than that of the normal DBA/1 mice beginning on d 3 after injection of the emulsion. Ginsenoside C-K (28, 56, and 112 mg/kg) mice recover their weight by d 32 after the emulsion injection. Ginsenoside C-K (56 and 112 mg/kg) and Methotrexate (MTX)-treated (2 mg/kg) mice show significantly increased body weight on d 50 as compared with CIA mice. Hind paw-swelling began on d 24 post-immunization. CIA mice are treated from d 28 to d 50. Arthritis scores are measured every 4 d beginning on d 24. Ginsenoside C-K (56 and 112 mg/kg) significantly reduces the arthritis scores of the mice on d 51[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	LS174T cells are seeded in cell imaging dish. After overnight incubation, cells are treated with ginseng saponin extract (GSE) (100 μg/mL), Rb1 (10 μM), or Ginsenoside C-K (10 μM) 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-κB 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[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mice[3] Specific pathogen-free DBA/1 mice (male, 18±2 g) are used. DBA/1 mice are injected intradermally twice with 0.1 mL of this emulsion (containing 100 mg of chicken type II collagen (CII)/mouse) in the back and the base of the tail. The day of the first immunization is defined as d 0, and the booster injection is administered into the back on d 21. After the onset of arthritis, animals are randomly divided into five groups, and each experimental group consists of ten mice. Mice with CIA are intragastrically administered Ginsenoside C-K (28, 56, or 112 mg/kg) once per day or MTX (2 mg/kg) once every 3 d from d 28 to d 51 after immunization. Normal and CIA mice are administered an equal volume of vehicle (CMC-Na) at the same time[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Agric Food Chem. 2023 Jan 11.
- Food Funct. 2021 Apr 28.
- J Agric Food Chem. 2020 Jul 29;68(30):8050-8056.

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REFERENCES

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[2]. 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.

[3]. Liu KK, et al. Ginsenoside compound K suppresses the abnormal activation of T lymphocytes in mice with collagen-induced arthritis. Acta Pharmacol Sin. 2014 May;35(5):599-612.

[4]. Liu Y, et al. Ginsenoside metabolites, rather than naturally occurring ginsenosides, lead to inhibition of human cytochrome P450 enzymes. Toxicol Sci. 2006 Jun;91(2):356-64.

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