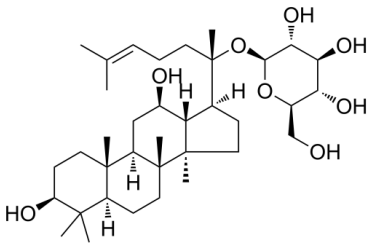
Data Sheet

Product Name:	Ginsenoside C-K
Cat. No.:	CS-3840
CAS No.:	39262-14-1
Molecular Formula:	$C_{36}H_{62}O_8$
Molecular Weight:	622.87
Target:	COX; Cytochrome P450; NO Synthase
Pathway:	Immunology/Inflammation; Metabolic Enzyme/Protease
Solubility:	DMSO : ≥ 100 mg/mL (160.55 mM)



BIOLOGICAL ACTIVITY:

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. IC50 & Target: iNOS, COX-2[1]

IC50: 32.0±3.6 μM (CYP2C9), 63.6±4.2 μM (CYP2A6), >100 μM (CYP1A2), >100 μM (CYP2D6), >100 μM (CYP3A4)[4] ***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]. ***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].

PROTOCOL (Extracted from published papers and Only for reference)

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].

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].

References:

[1]. Kim JH, et al. Role of ginsenosides, the main active components of *Panax ginseng*, in inflammatory responsesand diseases. J Ginseng Res. 2017 Oct;41(4):435-443.

[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.

CAIndexNames:

β-D-Glucopyranoside, (3β,12β)-3,12-dihydroxydammar-24-en-20-yl

SMILES:

C[C@@]([C@@]12C)(CC[C@@]3([H])C4(C)C)[C@@](C[C@@H](O)[C@]1([H])[C@]([C@@](CC/C=C(C)/C)(C)O[C@@H]([C@@H]([C@@H](O)[C@@H]5O)O)O[C@@H]5CO)([H])CC2)([H])[C@]3(CC[C@@H]4O)C

Tel: 610-426-3128 Fax: 888-484-5008 E-mail: sales@ChemScene.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA