Ginsenoside C-K

Cat. No.: HY-N0904  
CAS No.: 39262-14-1  
Molecular Formula: C₃₆H₆₂O₈  
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 6 months  
-20°C 1 month

SOLVENT & SOLUBILITY

In Vitro  
DMSO : ≥ 100 mg/mL (160.55 mM)  
* "≥" means soluble, but saturation unknown.  

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Concentration</th>
<th>Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td>1.6055 mL</td>
<td>8.0274 mL</td>
<td>16.0547 mL</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.3211 mL</td>
<td>1.6055 mL</td>
<td>3.2109 mL</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.1605 mL</td>
<td>0.8027 mL</td>
<td>1.6055 mL</td>
<td></td>
</tr>
</tbody>
</table>

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

In Vivo  
1. Add each solvent one by one: 10% DMSO >> 90% corn oil  
   Solubility: ≥ 2.75 mg/mL (4.42 mM); Clear solution  
2. 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  
3. 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

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 IC₅₀ values of 32.0±3.6 μM and 63.6±4.2 μM, respectively.

IC₅₀ & Target  
iNOS, COX-2 [1]
**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[4]. 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 IC50 value of 32.0±3.6 μM, a weak inhibition against the activity of CYP2A6 in human liver microsomes with an IC50 value of 63.6±4.2 μM, and an even weaker inhibition against the activity of CYP2D6 in human liver microsomes with an IC50 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].

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**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]

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.
REFERENCES


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