# Ginsenoside Rg2

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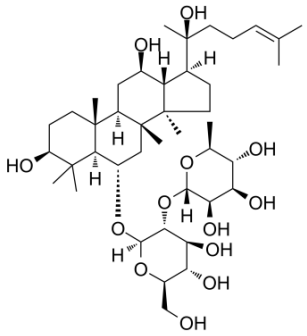
| **Cat. No.:** | HY-N0602 |
| **CAS No.:** | 52286-74-5 |
| **Molecular Formula:** | $C_{42}H_{72}O_{13}$ |
| **Molecular Weight:** | 785.01 |
| **Target:** | NF-κB; Amyloid-β |
| **Pathway:** | NF-κB; Neuronal Signaling |
| **Storage:** | Powder -20°C 3 years<br>4°C 2 years<br>In solvent -80°C 6 months<br>-20°C 1 month |



## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : ≥ 100 mg/mL (127.39 mM)

* "≥" means soluble, but saturation unknown.

**Preparing Stock Solutions**

| Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
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| 1 mM | 1.2739 mL | 6.3693 mL | 12.7387 mL |
| 5 mM | 0.2548 mL | 1.2739 mL | 2.5477 mL |
| 10 mM | 0.1274 mL | 0.6369 mL | 1.2739 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 (3.18 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.18 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.18 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

Ginsenoside Rg2 is one of the major active components of ginseng. Ginsenoside Rg2 acts as a NF-κB inhibitor. Ginsenoside Rg2 also reduces $Aβ_{1-42}$ accumulation.

| **$IC_{50}$ & Target** | NF-κB | $Aβ_{1-42}$ |
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**In Vitro**

Ginsenoside Rg2 prevents the decrease of IκB expression stimulated with lipopolysaccharide (LPS). IκB dissociation from

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| | RelA-p50 complex is crucial for NF-κB activity. Ginsenoside Rg2, protopanaxatriol, inhibits vascular cell adhesion molecule 1 (VCAM-1) and intercellular adhesion molecule 1 (ICAM-1) expression stimulated with LPS from human umbilical vein endothelial cell (HUVEC). The inhibition of VCAM-1 and ICAM-1 expression by Ginsenoside Rg2 is in a concentration-dependent manner, significantly. Treatment of endothelial cells with LPS (1μg/mL) decreases IκBα expression. By 1 hr after LPS treatment, significant decrease of IκBα is attained. To determine whether LPS-stimulated IκBα expression is affected by Ginsenoside Rg2, endothelial cells are treated for 1 hr with Ginsenoside Rg2 (1~50 μM) prior to LPS (1 μg/mL) stimulation for 1 hr. Ginsenoside Rg2 reverses the decrease of LPS-induced IκBα expression in a concentration-dependent manner, significantly. The adhesion of THP-1 cells to endothelial cells is measured using quantitative monolayer adhesion assay. The adhesion of THP-1 cells onto endothelial cells are increased to five folds by LPS (1 μg/mL) stimulation for 8 hrs. Ginsenoside Rg2 (1~50 μM) inhibits the adhesion of THP-1 cells to endothelial cells stimulated with LPS, in a concentration-dependent manner[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **In Vivo** | G-Rg1 and Ginsenoside Rg2 (G-Rg2) reduce the escape latencies on the last two training days compared to the Alzheimer's disease (AD) model group ($p<0.05$). In the spatial exploration test, the total time spent in the target quadrant and the number of mice that exactly crossed the previous position of the platform are clearly shorter and lower, respectively, in the AD model group mice than in the normal control group mice ($p<0.01$), a trend that is reversed by treatment with G-Rg1 and Ginsenoside Rg2 (G-Rg1, $p<0.01$; Ginsenoside Rg2, $p<0.05$). Treatment with G-Rg1 and Ginsenoside Rg2 effectively improve cognitive function of the mice that have declined due to AD. G-Rg1 and Ginsenoside Rg2 reduce $A\beta_{1-42}$ accumulation in APP/PS1 mice. In the G-Rg1 and Ginsenoside Rg2 treated mice, the pathological abnormalities observed in the APP/PS1 mice are gradually ameliorated. Clear nucleoli and light brown, sparsely scattered Aβ deposits are visible[2].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## PROTOCOL

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| **Cell Assay** | HUVECs ares grown in EBM-2 containing 10% FBS at a density of $2.0\times10^5$ cells/well on 24-well plates. Endothelial cells at 90~95% confluence are treated with Ginsenoside Rg2 (1, 20, 50 μM) for 1 hr prior to 1 μg/mL of LPS stimulation for 8 hr. THP-1 cells are labeled with Calcein-AM (5 μM) in RPMI 1640 medium containing 10% FBS for 30 min. After extensive washing with PBS, the labeled THP-1 cells are seeded at a density of $5.0\times10^5$ cells/well onto endothelial cells which are treated with the Rg2 and/or LPS and, then, incubated for 1 hr at 37°C while gentle shaking. After incubation, non-adherent cells are removed by gentle washing two times with PBS. Photograph images are obtained at 485 nm excitation and 538 nm emission using a SPOT II digital camera-attached fluorescence microscope[1].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **Animal Administration [2]** | Mice[2]<br>Male APP/PS1 mice, weighing 20±2 g, and male C57BL/6J mice, weighing 20±2 g, are used. The animals are maintained in an air-conditioned animal center at 23±2°C and a relative humidity of 50±10%, with a natural light-dark cycle. Food and water are available ad libitum. After acclimatization for 1 wk, the mice are divided into four groups (n=10 in each group): the normal control group, the AD model group, the G-Rg1 group, and the Ginsenoside Rg2 group. According to the concentration-response curves, the mice in the G-Rg1 and Ginsenoside Rg2 groups are injected intraperitoneally once daily with G-Rg1 and Ginsenoside Rg2 (30 mg/kg), respectively, dissolved in saline. The mice in the AD model group (APP/PS1 mice) and the normal control group (C57BL/6J nontransgenic littermates) are treated with isodose saline (0.9% w/v). All mice are treated for 1 mo before brain metabolite profiling.<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## REFERENCES

[1]. Cho YS, et al. Ginsenoside rg2 inhibits lipopolysaccharide-induced adhesion molecule expression in human umbilical vein endothelial cell. Korean J Physiol Pharmacol. 2013 Apr;17(2):133-7.

[2]. Li N, et al. A UPLC/MS-based metabolomics investigation of the protective effect of ginsenosides Rg1 and Rg2 in mice with Alzheimer's disease. J Ginseng Res. 2016 Jan;40(1):9-17.



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