

Ginsenoside Rg2

Catalog No: tcsc3833



Available Sizes

Size: 5mg

Size: 10mg



Specifications

CAS No:

52286-74-5

Formula:

$C_{42}H_{72}O_{13}$

Pathway:

Neuronal Signaling;NF-κB

Target:

Amyloid-β;NF-κB

Purity / Grade:

>98%

Solubility:

DMSO : ≥ 100 mg/mL (127.39 mM)

Alternative Names:

Chikusetsusaponin I;Panaxoside Rg2;Prosapogenin C2

Observed Molecular Weight:

785.01

Product Description

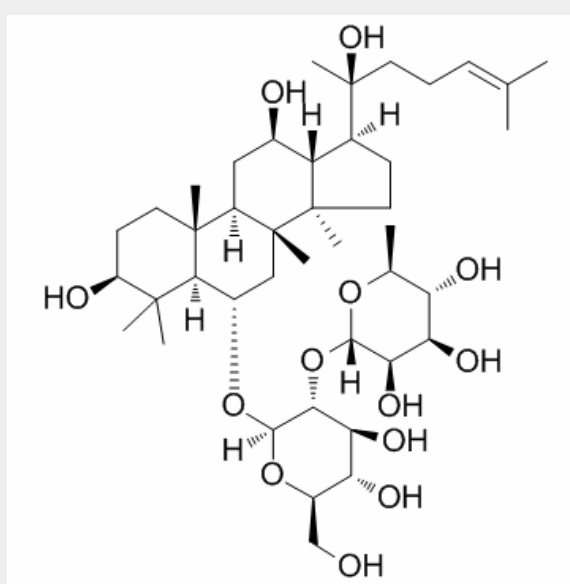
Ginsenoside Rg2 is one of the major active components of ginseng. Ginsenoside Rg2 acts as a **NF-κB** inhibitor. Ginsenoside Rg2 also reduces **Aβ₁₋₄₂** accumulation.

IC50 & Target: NF- κ B^[1]

A β ₁₋₄₂^[2]

In Vitro: Ginsenoside Rg2 prevents the decrease of I κ B expression stimulated with lipopolysaccharide (LPS). I κ B dissociation from RelA-p50 complex is crucial for NF- κ B 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 (1 μ g/mL) decreases I κ B α expression. By 1 hr after LPS treatment, significant decrease of I κ B α is attained. To determine whether LPS-stimulated I κ B α expression is affected by Ginsenoside Rg2, endothelial cells are treated for 1 hr with Ginsenoside Rg2 (1~50 μ M) prior to LPS (1 μ g/mL) stimulation for 1 hr. Ginsenoside Rg2 reverses the decrease of LPS-induced I κ B α 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 μ g/mL) stimulation for 8 hrs. Ginsenoside Rg2 (1~50 μ M) inhibits the adhesion of THP-1 cells to endothelial cells stimulated with LPS, in a concentration-dependent manner^[1].

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 (p1-42 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 A β deposits are visible^[2].



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