Web: www.taiclone.com
Tel: +886-2-2735-9682
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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 **$A\beta_{1\text{-}42}$** accumulation.

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IC50 & Target: NF-κB [1]

$A\beta_{1-42}$ [2]

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

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 Aβ deposits are visible[2].