

Ginsenoside Re

Catalog No: tcsc3838



Available Sizes

Size: 5mg

Size: 10mg



Specifications

CAS No:

52286-59-6

Formula:

$C_{48}H_{82}O_{18}$

Pathway:

Neuronal Signaling;MAPK/ERK Pathway;NF-κB

Target:

Amyloid-β;JNK;NF-κB

Purity / Grade:

>98%

Solubility:

10 mM in DMSO

Alternative Names:

Ginsenoside B2; Panaxoside Re; Sanchinoside Re

Observed Molecular Weight:

947.15

Product Description

Ginsenoside Re is an extract from *Panax notoginseng*. Ginsenoside Re decreases the **β-amyloid** protein (**Aβ**). Ginsenoside Re plays a role in antiinflammation through inhibition of **JNK** and **NF-κB**.

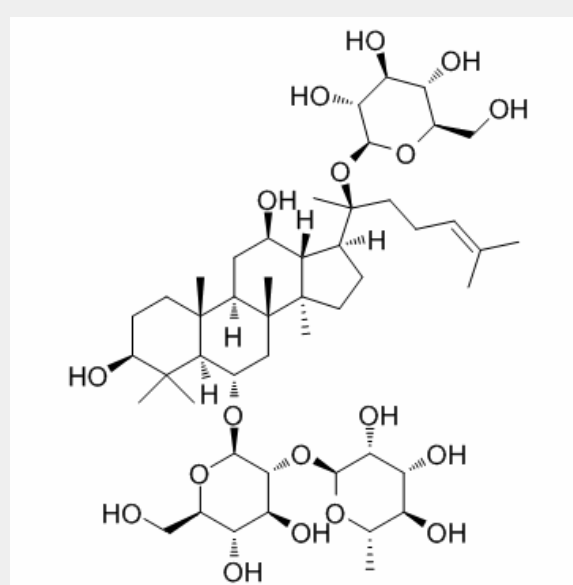
IC50 & Target: A β ₁₋₄₀ and A β ₁₋₄₂^[1]

JNK^[2]

NF- κ B^[2]

In Vitro: Ginsenoside Re is a well-known traditional Chinese medicine, which decreases the β -site amyloid precursor protein cleaving enzyme 1 (BACE1) mRNA and protein levels and inhibits BACE1 activity in the N2a/APP695 cells. Ginsenoside Re also significantly increases the PPAR γ protein and mRNA levels. To prevent Ginsenoside Re from having a cytotoxic effect on the N2a/APP695 cells, the cell viability is first determined by the MTT assay. The N2a/WT and N2a/APP695 cells are treated with increasing concentrations of Ginsenoside Re (0-200 μ M) for 24 h. Ginsenoside Re concentrations under 100 μ M do not affect the viability of the N2a/WT and N2a/APP695 cells, whereas the 150 μ M Ginsenoside Re concentration markedly decreases the survival rate of the N2a/WT and N2a/APP695 cells. Incubation with Ginsenoside Re at a 200 μ M concentration for 24 h reduces the viability of the N2a/WT and N2a/APP695 cells by 15.58% and 26.82%, respectively. These data indicate that Ginsenoside Re treatment within the range of 0-100 μ M for 24 h is safe for the N2a/WT and N2a/APP695 cells ($P > 0.05$)^[1].

In Vivo: Ginsenoside Re reduces insulin resistance in 3T3-L1 adipocytes and high-fat diet (HFD) rats through inhibition of JNK and NF- κ B activation^[2]. Intraperitoneal injection of lipopolysaccharide (LPS) at a dose of 20 mg/kg is lethal to mice, and 70% to 80% of the mice die within 60 h. However, pretreatment of the mice with Rg1 or Ginsenoside Re increases their survival rates in a dose-dependent manner. With the doses of Rg1 or Ginsenoside Re increase from 2.5 to 5 mg/kg, the survival rate is elevated from 60% to 90% (Rg1) or from 30% to 40% (Ginsenoside Re). All the mice administered Rg1 at a minimal dose of 10 mg/kg are protected from death compared to 80% survival of mice treated with an equal dose of Ginsenoside Re. To protect all the mice, 20 mg/kg Ginsenoside Re is needed. To investigate the anti-inflammatory potential of Rg1 and Ginsenoside Re, 1 mg/kg Rg1 or Ginsenoside Re is injected into rats and then challenged the animals with LPS. The injection procedure itself causes a transient stress-induced increase in body temperature of $\sim 1.2^{\circ}\text{C}$ in each group. Thereafter, LPS-challenged rats without pretreatment develop a robust biphasic fever, with the first peak reaching $\sim 1.5^{\circ}\text{C}$ at 2 h and the second peak reaching 1.8°C at 4 h. In contrast, the temperature changes for the Rg1-, Ginsenoside Re-, and TAK-242-treated groups are only 0.9, 1.2, and 0.8°C at 2 h and 1.3, 1.4, and 1.0°C at 4 h, respectively. Pretreatment with Rg1, Ginsenoside Re, or TAK-242 significantly attenuates LPS-induced alterations in body temperature^[3].



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