Web: www.taiclone.com
Tel: +886-2-2735-9682
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Ginsenoside Ro

Catalog No: tcsc3828

Available Sizes

Size: 5mg

Size: 10mg

Specifications

CAS No:
34367-04-9

Formula:
$C_{48}H_{76}O_{19}$

Pathway:
Membrane Transporter/Ion Channel;GPCR/G Protein

Target:
Calcium Channel;Prostaglandin Receptor

Purity / Grade:
>98%

Solubility:
10 mM in DMSO

Alternative Names:
Polysciasaponin P3; Chikusetsusaponin 5; Chikusetsusaponin V

Observed Molecular Weight:
957.11

Product Description

Ginsenoside Ro exhibits a **Ca^{2+}**-antagonistic antiplatelet effect with an IC_{50} of 155 μM. Ginsenoside Ro reduces the production of **TXA_2** more than it reduces the activities of COX-1 and TXAS.

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IC50 & Target: IC50:155 μM (Ca^{2+})[1]

TXA_2[2]

In Vitro: Ginsenoside Ro in *Panax ginseng* is a beneficial novel Ca^{2+}-antagonistic compound and may prevent platelet aggregation-mediated thrombotic disease. Ginsenoside Ro dose-dependently reduces thrombin-stimulated platelet aggregation, and IC_{50} is approximately 155 μM[1]. Ginsenoside Ro inhibits TXA_2 production to abolish thrombin-induced platelet aggregation. Thromboxane A $_2$ (TXA_2) induces platelet aggregation and promotes thrombus formation. Ginsenoside Ro dose-dependently (50-300 μM) reduces the TXB_2 level that is induced by thrombin; Ginsenoside Ro (300 μM) inhibits the thrombin-mediated elevation in TXB_2 level by 94.9%. COX-1 activity in the absence of Ginsenoside Ro (negative control) is 2.3±0.1 nmol/mg protein. However, Ginsenoside Ro dose-dependently (50-300 μM) reduces its activity; at 300 μM, COX-1 activity is reduced by 26.4% of that of the negative control. TXA_2 synthase (TXAS) activity in the absence of Ginsenoside Ro (negative control) is 220.8±1.8 ng/mg protein/min. However, Ginsenoside Ro dose-dependently (50-300 μM) reduces its activity; at 300 μM, TXAS activity is reduced by 22.9% of that of the negative control. The inhibitory effect of Ginsenoside Ro (300 μM) on TXB_2 production (94.9%) is significantly higher than those on COX-1 (26.4%) and TXAS (22.9%) activities[2]. To assess the toxicity of Ginsenoside Ro in Raw 264.7 cells, they are first treated with various concentrations (10 μM, 50 μM, 100 μM, and 200 μM) of Ginsenoside Ro for 24 h. Ginsenoside Ro exhibits no significant dose dependent toxicity. The effect of Ginsenoside Ro is next determined on cell viability and ROS levels, a marker of oxidative stress, following treatment with 1 μg/mL LPS. LPS reduces cell viability by ~70% compared with nontreated controls. Pretreatment with 100 μM and 200 μM Ginsenoside Ro for 1 h prior to 1 μg/mL LPS incubation for 24 h leads to a significant increase in cell viability. The changes in ROS levels and NO production are consistent with the effects of Ginsenoside Ro on viability[3].

In Vivo: Ginsenoside Ro dissolved in water is administrated by gavage to mice at doses of 25 and 250 mg/kg/day for 4 days before i.v. injection of HT29 in order to keep blood concentrations of Ginsenoside Ro above a certain level before HT29 i.v. injection followed by 40 days of oral administration of Ginsenoside Ro to the mice. After 38 days of treatment, the animals are euthanized, and the number of pulmonary metastatic nodules is counted in addition to evaluation of toxicity of Ginsenoside Ro and mouse pathology by HT29. Ginsenoside Ro (250 mg/kg/day) produces a significant decrease in the number of tumor nodules on the lung surface, yielding inhibition rates of 88% (P [4].