

Bioactive Molecules, Building Blocks, Intermediates

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Product Name:	Ginsenoside Rh3
Cat. No.:	CS-3836
CAS No.:	105558-26-7
Molecular Formula:	C36H60O7
Molecular Weight:	604.86
Target:	Keap1-Nrf2
Pathway:	NF-ĸB
Solubility:	10 mM in DMSO

Data Sheet

BIOLOGICAL ACTIVITY:

Ginsenoside Rh3 is a bacterial metabolite of Ginsenoside Rg5. Ginsenoside Rh3 treatment in human retinal cells induces Nrf2 activation. IC50 & Target: Nrf2^[1] In Vitro: Ginsenoside Rh3 inhibits UV-induced oxidative damages in retinal cells via activating nuclear-factor-E2-related factor 2 (Nrf2) signaling. Ginsenoside Rh3 treatment in retinal cells induces Nrf2 activation. The potential activity of Ginsenoside Rh3 is tested on Nrf2 signaling in the retinal pigment epithelium cells (RPEs). The qRT-PCR assay results demonstrate that treatment with Ginsenoside Rh3 dose-dependently increases mRNA transcription and expression of key Nrf2-regulated genes, including HO1, NQO1 and GCLC. Consequently, protein expressions of these Nrf2-dependent genes (HO1, NQO1 and GCLC) are also significantly increased in Ginsenoside Rh3 (3-10 μ M)-treated RPEs. Notably, although Nrf2 mRNA level is unchanged after Ginsenoside Rh3 treatment, its protein level is significantly increased by Rh3^[1]. EZ-Cytox assay is used to assess the effect of ginsenoside-Rh3 on SP 1-keratinocytes viability. Ginsenoside Rh3 (0.01, 0.1, 1 and 10 μ M) shows no cytotoxic effect at all concentrations^[2]. In Vivo: The potential effect of Ginsenoside Rh3 is examined on mouse retina, using the light-induced retinal damage model. Ginsenoside Rh3 intravitreal injection (5 mg/kg body weight, 30 min pre-treatment) significantly attenuates light-induced decrease of both a- and b-wave amplitude. The electroretinography (ERG)'s a-wave decreases to 46.03±1.62% % of control level after light exposure, which is back to 71.84±7.51% with Ginsenoside Rh3 administration. The b-wave is 40.19±3.34% of control level by light exposure, and Rh3 intravitreal injection brings back to 80.01±2.37% of control level^[1].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: ^[2]**SP-1 keratinocytes** are seeded in 96 well plates (2×10^4 cells/well). After 24 h, the media is replaced with media containing various concentrations of (A) SKRG, or (B) **Ginsenoside Rh3 (0.01, 0.1, 1 and 10 µM)**. Control cells are treated with DMSO at a final concentration of 0.1%. After 24 h, the media containing the compounds or DMSO is replaced with media containing 10% EZ-Cytox. The cells are then incubated at 37°C for 1 h, and the absorbance is measured using a microplate reader at a wavelength of 450 nm. All assays are performed in triplicate^[2].

Animal Administration: ^[1]Mice^[1]

The BALB/c mice (Male, 5-6 week old, 17-18 g weight) are used. The pupillary dilation is performed before exposure to 5000 lx of white fluorescent light. Thirty min before light exposure, Ginsenoside Rh3 (at 5 mg/kg body weight) are injected intravitreally to the right eye. ERG recording after light exposure is also reported early. The b-wave amplitude is measured from the trough of the a-wave to the peak of the b-wave, and the amplitude of the a-wave is measured from the initial baseline.

References:

[1]. Tang CZ, et al. Activation of Nrf2 by Ginsenoside Rh3 protects retinal pigment epithelium cells and retinal ganglion cells from UV. Free Radic Biol Med.

2018 Mar;117:238-246.

[2]. Chung I, et al. Inhibitory mechanism of Korean Red Ginseng on GM-CSF expression in UVB-irradiated keratinocytes. J Ginseng Res. 2015 Oct;39(4):322-30.

CAIndexNames:

 $\beta\text{-D-Glucopyranoside, (3\beta,12\beta,20Z)-12-hydroxydammara-20(22),24-dien-3-yl}$

SMILES:

 $C[C@@]([C@@]12C)(CC[C@@]3([H])C4(C)C)[C@@](C[C@@H](O)[C@]1([H])[C@@H](/C(C)=C\C/C=C(C)/C)CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C)(C))CC2)([H])[C@]3(CC[C@@H]4O[C@]([C@@H](C))CC2)([H])[C@]([H](C))CC2)([H])[C@]([C@@](C)(C))CC2)([H](C))CC2)([H](C))CC2)([H](C)(C))CC2)([H](C))CC2)$

Caution: Product has not been fully validated for medical applications. For research use only.

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