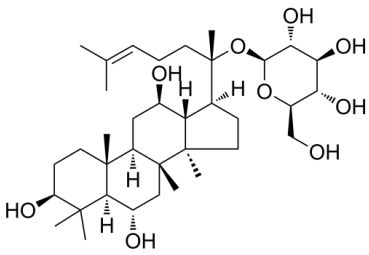
Data Sheet

Product Name:	Ginsenoside F1
Cat. No.:	CS-3842
CAS No.:	53963-43-2
Molecular Formula:	$C_{36}H_{62}O_9$
Molecular Weight:	638.87
Target:	Cytochrome P450; Endogenous Metabolite
Pathway:	Metabolic Enzyme/Protease
Solubility:	DMSO : ≥ 100 mg/mL (156.53 mM)



BIOLOGICAL ACTIVITY:

Ginsenoside F1, an enzymatically modified derivative of Ginsenoside Rg1, demonstrates competitive inhibition of **CYP3A4** activity and weaker inhibition of CYP2D6 activity. IC50 & Target: CYP3A4，CYP2D6[1] ***In Vitro:*** Ginsenoside F1 has been shown to flaunt anticancer, anti-aging, and antioxidant effects and has demonstrated competitive inhibition of CYP3A4 activity and weaker inhibition of CYP2D6 activity. The cell viabilities are 68% at the highest concentration of ginsenoside F1 (200 μM) in MTT assays[1]. ***In Vivo:*** ApoE$^{-/-}$ mice are fed a high fat diet and orally treated with Ginsenoside F1 (50 mg/kg/day) for 8 weeks. Ginsenoside F1 treated mice significantly reduce the lesion size compared with model group mice[2].

PROTOCOL (Extracted from published papers and Only for reference)

Kinase Assay: [1]Glycosylation ability is assayed with overexpressed BSGT1 enzyme and F1. The reaction mixtures contain 100 μL of 0.5 mM F1 and 100 μL of 2.5 mM UDP-glucose and 800 μL of purified enzyme (final concentration at 0.1 mg/mL) (pH 7.0). The mixtures are incubated at 30°C for 24 h. Moreover, three groups of controls are incubated under the same conditions: (1) control 1 (C1) consists of Ginsenoside F1 with BSGT1; (2) control 2 (C2) consists of BSGT1 with UDP-glucose; and (3) control 3 (C3) consists of Ginsenoside F1 with UDP-glucose[1].

Cell Assay: [1]**B16BL6 cells** are cultured in Dulbecco's modified Eagles medium supplemented with 10% fetal bovine serum and 1% Penicillin-Streptomycin at 37°C in a humidified 95% air/5% CO_2 atmosphere. Cell viability is determined for Ginsenoside F1 and metabolite 1 using MTT conversion to formazan. Cells are seeded at a density of 1×10^5 cells/well in a 96-well plate, cultured for 24 h, and treated with various concentrations from **1 μM to 200 μM of Ginsenoside F1** and metabolite 1 for 5 d. Finally, 10 μL of MTT (5 mg/mL in PBS) is added to each well. Cells are incubated at 37°C for 3 h, and then DMSO (100 μL) is added to dissolve the formazan crystals. The absorbance is measured at 570 nm with the reference wavelength of 630 nm using an ELISA reader[1].

Animal Administration: Ginsenoside F1 is dissolved in carboxymethyl cellulose sodium (CMC-Na)[2].[2]Mice[2]

Six-week-old (17±1 g) male C57BL/6 mice and ApoE$^{-/-}$ mice with a C57BL/6 background are maintained in a temperature-controlled facility (temperature: 22±1°C; humidity: 60%) with a 14 h light/10 h dark cycle in conventional cages. Forty mice are randomly divided into four experimental groups (n=10/group): (I) C57BL/6 N mice, the control group; (II) ApoE$^{-/-}$ mice group; (III) ApoE$^{-/-}$ mice+ Ginsenoside F1 group; (IV) ApoE$^{-/-}$ mice+Probucol group. All mice are fed with a high fat diet (HFD, 0.3% cholesterol and 20% pork fat) for 8 weeks. **Ginsenoside F1 (50 mg/kg/day, i.g.)** and Probucol (2 g/kg, i.g.) are dissolved in carboxymethyl cellulose sodium (CMC-Na). Oral administration is given to mice every day for 8 weeks. The control and model groups receive the aseptic 0.5% CMC-Na treatment every day (i.g., 0.1 mL/10g) [2].

References:

[1]. Wang DD, et al. Rare ginsenoside Ia synthesized from F1 by cloning and overexpression of the UDP-glycosyltransferase gene from Bacillus subtilis: synthesis, characterization, and in vitromelanogenesis inhibition activity in BL6B16 cells. J Ginseng Res. 2018 Jan;42(1):42-49.

[2]. Qin M, et al. Ginsenoside F1 Ameliorates Endothelial Cell Inflammatory Injury and Prevents Atherosclerosis in Mice through A20-Mediated Suppression of NF-kB Signaling. Front Pharmacol. 2017 Dec 22;8:953.

CAIndexNames:

β-D-Glucopyranoside, (3β,6α,12β)-3,6,12-trihydroxydammar-24-en-20-yl (9CI)

SMILES:

C[C@]([C@@](C[C@H]1O)([H])[C@]2(CC[C@@H]3O)C)(C[C@H](O)[C@@]2([H])C3(C)C)[C@]4([C@@]1([H])[C@]([C@@](CC/C=C(C)/C)(C)O[C@@H]([C@@H]([C@@H](O)[C@@H]5O)O)O[C@@H]5CO)([H])CC4)C

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