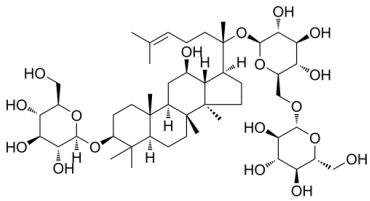
Data Sheet

Product Name:	Gypenoside XVII
Cat. No.:	CS-4163
CAS No.:	80321-69-3
Molecular Formula:	$C_{48}H_{82}O_{18}$
Molecular Weight:	947.15
Target:	Endogenous Metabolite; Estrogen Receptor/ERR
Pathway:	Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor
Solubility:	DMSO : ≥ 100 mg/mL;H_2O : 41.67 mg/mL (ultrasonic)



BIOLOGICAL ACTIVITY:

Gypenoside XVII, a novel phytoestrogen belonging to the gypenosides, can activate **estrogen receptors**. IC50 & Target: Estrogen receptor[1] ***In Vitro:*** The ability of Gypenoside XVII (GP-17) to prevent Ox-LDL-induced cytotoxicity is detected by cell viability assays. Gypenoside XVII does not demonstrate any cytotoxicity in HUVECs. Gypenoside XVII can protect HUVECs against Ox-LDL-induced apoptosis. Gypenoside XVII dose-dependently mitigates the toxic effect of Ox-LDL on HUVEC viability. The viability of HUVECs is significantly higher than that of other groups at 50 μg/mL Gypenoside XVII [1]. ***In Vivo:*** Body weights are measured as physical measures of hormone bioactivity. Mean body weights are significantly higher in every group compared to that of the control, but there is no significant difference in body weight between the different treatments during the 10-week feeding. The mouse plasma lipid levels are also measured at the end of 10 weeks of a high-fat diet. Circulating levels of total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) are significantly increased in the treated groups of $ApoE^{-/-}$ mice compared with those of the C57BL/6J control group; Gypenoside XVII (GP-17) and Probucol treatment substantially decreases both of these parameters relative to those of the $ApoE^{-/-}$ model group[1].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [1]For the establishment of an Ox-LDL-induced apoptosis model and measurement of Gypenoside XVII's protective effect, the **HUVECs** are seeded in 96-well plates at a density of 10^5 cells per mL and grown for 24 h. Then, the HUVECs are pretreated with **Gypenoside XVII (6.25, 12, 25, 50, 100 μg/mL)** for 12 h in serum-free endothelial cell basal medium, followed by incubation with Ox-LDL (100 μg/mL, 24 h) which does not have Gypenoside XVII. After 24 h, the treated HUVECs are incubated with 5 mg/mL MTT in fresh medium for an additional 4 h. Absorbance is measured at 570 nm using a plate reader[1].

Animal Administration: Gypenoside XVII (GP-17) is prepared in vehicle (0.5% sodium carboxymethylcellulose, CMC-Na)[1].[1]Mice [1]

A total of 42 $ApoE^{-/-}$ mice and 12 male C57BL/6J mice with the same age and body weight (approximately 20 g in body weight and 6 weeks old) are randomly divided into 4 groups (12 mice for each group) and orally administered the following once a: C57 control group (vehicle; 0.5% sodium carboxymethylcellulose, CMC-Na); $ApoE^{-/-}$ model group (vehicle; 0.5% CMC-Na); $ApoE^{-/-}$ +Gypenoside XVII group (**Gypenoside XVII, 50 mg/kg via i.g.**); $ApoE^{-/-}$+probucol group (Probucol, 2 mg/kg via i.g.). Probucol is an antioxidant drug used as a positive control. After two weeks of acclimatization, all the mice are fed a high-fat diet including 0.3% cholesterol and 20% pork fat for 10 weeks. They are maintained in pathogen-free conditions at approximately 22±1°C on a 12 h light-dark cycle with free access to food and water. The body weights are determined every two weeks. After 10 weeks of the treatments, all animals are anesthetized with pentobarbital sodium and killed after being deprived of food overnight. Serum is immediately separated from blood samples by centrifugation at 3600 rpm for 15 min, and the tissue samples (heart and aorta) are rapidly removed

and frozen in -8°C.

References:

[1]. Yang K, et al. Gypenoside XVII Prevents Atherosclerosis by Attenuating Endothelial Apoptosis and Oxidative Stress: Insight into the ERα-Mediated PI3K/Akt Pathway. Int J Mol Sci. 2017 Feb 9;18(2). pii: E77.

CAIndexNames:

β-D-Glucopyranoside, (3β,12β)-3-(β-D-glucopyranosyloxy)-12-hydroxydammar-24-en-20-yl 6-O-β-D-glucopyranosyl-

SMILES:

C[C@]([C@@](C[C@H]1O)([H])[C@]2(CC[C@@H]3O[C@]([C@@H]([C@@H](O)[C@@H]4O)O)([H])O[C@@H]4CO)C)(CC[C@@]2([H])C3(C)C)[C@]5([C@@]1([H])[C@]([C@@](CC/C=C(C)/C)(C)O[C@@H]([C@@H]([C@@H](O)[C@@H]6O)O)O[C@@H]6CO[C@@H]([C@@H]([C@@H](O)[C@@H]7O)O)O[C@@H]7CO)([H])CC5)C

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