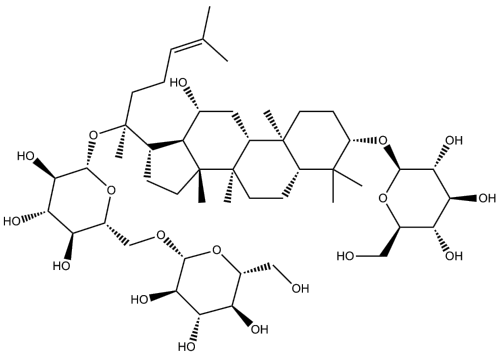
Product Data Sheet

Product Name: Gypenoside XVII
Cat. No.: GN10566

Chemical Properties

Cas. No.	80321-69-3		
Chemical Name	Gypenoside XVII		
SMILES	CC(=CCCC(C)(C1CCC2(C1C(CC3C2(CCC4C3(CCC(C4(C)C)OC5C(C(C(C(O5)CO)O)O)O)C)C)O)C)OC6C(C(C(C(O6)COC7C(C(C(C(O7)CO)O)O)O)O)O)O)C		
Formula	$C_{48}H_{82}O_{18}$	M.Wt	947.15
Solubility		Storage	4°C, protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

Cell lines rat pheochromocytoma PC-12 cells

Caution: Product has not been fully validated for medical applications. For research use only.
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Preparation Method	PC-12 cells were pretreated with Gypenoside XVII (10μM) for 12h, and then washed twice with PBS. Afterwards, the cells were treated with $A\beta_{25-35}$ (20μM) for 24h.
Reaction Conditions	10μM; 12h
Applications	Gypenoside XVII provided significant neuroprotective effects against oxidative stress, autophagy, and apoptosis induced by $A\beta_{25-35}$.

Animal experiment [2]:

Animal models	Sprague Dawley rats
Preparation Method	Excitotoxicity was produced in rats that were injected intraperitoneally (i.p.) with kainic acid (15mg/kg). Gypenoside XVII (30mg/kg) was dissolved in a saline solution containing 1% DMSO and was administered (i.p.) 30min before kainic acid (KA) (15mg/kg in 0.9% NaCl, pH 7.0, i.p.) injection. The rats were divided into the following four groups: control group (i.p. injection of 1% DMSO; n = 10), kainic acid group (only i.p. injection of kainic acid; n = 10), Gypenoside XVII + kainic acid group (i.p. injection of Gypenoside XVII and kainic acid administration; n = 10), and Gypenoside XVII group (only i.p. injection of Gypenoside XVII; n = 3).
Dosage form	30mg/kg; i.p.; 30min before kainic acid injection

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Applications	Pretreatment with Gypenoside XVII inhibited the convulsive effects of kainic acid by increasing the latency to seizure onset and reducing seizure severity. Gypenoside XVII pretreatment also blocked kainic acid-induced neuronal injury, as indicated by an increase in the number of surviving neurons and a decrease in the number of degenerated neurons in the entorhinal cortex of kainic acid-treated rats.

References:
[1] Meng X B, Wang M, Sun G B, et al. Attenuation of Aβ5-35-induced parallel autophagic and apoptotic cell death by gypenoside XVII through the estrogen receptor-dependent activation of Nrf2/ARE pathways. Toxicol Appl Pharmacol. 2014 Aug 15;279(1):63-75.
[2] Lu C W, Lin T Y, Chiu K M, et al. Gypenoside XVII Reduces Synaptic Glutamate Release and Protects against Excitotoxic Injury in Rats. Biomolecules. 2024 May 16;14(5):589.

Background

Gypenoside XVII is a novel phytoestrogen primarily isolated from the plants *Gynostemma pentaphyllum* and *Panax notoginseng* and dose-dependently decreases glutamate release of with a IC_{50} value of 16μM[1]. Gypenoside XVII has neuroprotective and cardiovascular benefits, which make it a potential candidate for the development of treatments for conditions such as atherosclerosis and Alzheimer's disease[2][3][5][7]. Furthermore, the estrogenic properties of Gypenoside XVII could be explored for hormone replacement therapy[4][6].

In vitro, Gypenoside XVII has protective effects against atherosclerosis. Gypenoside XVII(3.125-100μg/mL) incubation for 24h dose-dependently mitigated the toxic effect of oxidized low-density lipoprotein (Ox-LDL) on human umbilical vein endothelial cell (HUVEC) viability. Incubation of HUVECs with Gypenoside XVII (50μg/mL) for 12h before exposure to Ox-LDL markedly reduced the ROS generation and increased Antioxidant Defense via the ERα/PI3K/Akt Pathway[2]. Gypenoside XVII (100μg/mL) treated Ox-LDL-induced THP-1 macrophage-derived foam cells for 24h, exhibiting protective effects by enhancing cholesterol efflux and mitigating inflammation via upregulating ABCA1, ABCG1, and miR-182–5p expression while downregulating HDAC9, and facilitating the M2

macrophage phenotype and curbing the inflammatory response[3]. Pretreated with rat pheochromocytoma PC-12 cells with Gypenoside XVII (10μM) for 12h conferred protection against $A\beta_{25-35}$-induced neurotoxicity through estrogen receptor-dependent activation of PI3K/Akt pathways, inactivation of GSK-3β and activation of Nrf2/ARE/HO-1 pathways[4].

In vivo, Gypenoside XVII (50mg/kg) administered into ApoE –/– mice via intragastric gavage (i.g.) once a day for 10 weeks reduced the serum lipid levels, increase expression of antioxidant enzymes and decrease atherosclerotic lesion formation in ApoE–/– mice[5]. Gypenoside XVII (1, 2.5, 5 and 10mg/kg) orally administrated once produced the antidepressant-like effects in mice, which was mediated by the inhibition of complement C3/C3aR/STAT3/cytokine signaling in the prefrontal cortex[6]. Gypenoside XVII (40mg/kg) administered by gavage for 60 days significantly improved the spatial learning and memory abilities of double-transgenic APPswe/PS1dE9 (APP/PS1) mice, reduced Aβ deposition and AβPP expression, and enhanced autophagy and lysosomal biosynthesis by activating TFEB. These results suggest that Gypenoside XVII may be a promising candidate drug for the prevention and treatment of Alzheimer's disease[7].

References:
[1] Lu C W, Lin T Y, Chiu K M, et al. Gypenoside XVII Reduces Synaptic Glutamate Release and Protects against Excitotoxic Injury in Rats. Biomolecules. 2024 May 16;14(5):589.
[2] Yang K, Zhang H J, Luo Y, 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):77.
[3] Deng W Y, Zhou C L, Zeng M Y. Gypenoside XVII inhibits ox-LDL-induced macrophage inflammatory responses and promotes cholesterol efflux through activating the miR-182-5p/HDAC9 signaling pathway. J Ethnopharmacol. 2024 Jan 30;319(Pt 1):117070.
[4] Meng X B, Wang M, Sun G B, et al. Attenuation of Aβ25-35-induced parallel autophagic and apoptotic cell death by gypenoside XVII through the estrogen receptor-dependent activation of Nrf2/ARE pathways. Toxicol Appl Pharmacol. 2014 Aug 15;279(1):63-75.
[5] Wang J R, Yu Y L, Zhang H R, et al. Gypenoside XVII attenuates renal ischemia-reperfusion injury by inhibiting endoplasmic reticulum stress and NLRP3 inflammasome-triggered pyroptosis. Eur J Pharmacol. 2024 Jan 5:962:176187.
[6] Zhang M M, G H, Cheng J, et al. Gypenoside XVII, an Active Ingredient from Gynostemma Pentaphyllum, Inhibits C3aR-Associated Synaptic Pruning in Stressed Mice. Nutrients. 2022 Jun

10;14(12):2418.
[7] Meng X B, Luo Y, Liang T, et al. Gypenoside XVII Enhances Lysosome Biogenesis and Autophagy Flux and Accelerates Autophagic Clearance of Amyloid-β through TFEB Activation. J Alzheimers Dis. 2016 Apr 5;52(3):1135-50.