Notoginsenoside Ft1

Cat. No.: HY-N0910

CAS No.: 155683-00-4

Molecular Formula: $C_{47}H_{80}O_{17}$

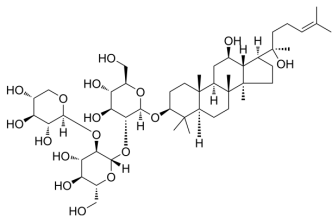
Molecular Weight: 917.13

Target: HIF/HIF Prolyl-Hydroxylase; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage: 4°C, stored under nitrogen

* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (109.04 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.0904 mL	5.4518 mL	10.9036 mL
	5 mM	0.2181 mL	1.0904 mL	2.1807 mL
	10 mM	0.1090 mL	0.5452 mL	1.0904 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (2.73 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (2.73 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.67 mg/mL (0.73 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Notoginsenoside Ft1 is a saponin isolated from Panax notoginseng; stimulator of angiogenesis.IC50 value:Target: angiogenesis stimulatorin vitro: Ft1 increases translocalization of hypoxia-inducible factor-1α (HIF-1α) from cytoplasm to nuclei, where it binds to the vascular endothelial growth factor (VEGF) promoter, increasing the expression of VEGF mRNA and the subsequent secretion of the growth factor. Ft1 induces the activation of PI3K/AKT and Raf/MEK/ERK signaling pathways [1]. Among the saponins examined, Ft1 was the most potent procoagulant and induced dose-dependent platelet aggregation. Ft1 reduced plasma coagulation indexes, decreased tail bleeding time and increased thrombogenesis. Moreover, it potentiated ADP-induced platelet aggregation and increased cytosolic Ca(2+) accumulation, effects that were attenuated by clopidogrel. Ft1 binds to platelet P2Y12 receptors. The increase in intracellular Ca(2+) evoked by Ft1 in HEK293 cells overexpressing P2Y12 receptors could be blocked by ticagrelor [2]. Ft1 caused endothelium-dependent

relaxations, which were abolished by l-NAME (inhibitor of nitric oxide synthases) and ODQ (inhibitor of soluble guanylyl cyclase). Ft1 increased the cGMP level in rat mesenteric arteries. GR and ER were present in the endothelial layer and their antagonism by RU486 and PHTPP, respectively, inhibited Ft1-induced endothelium-dependent relaxations and phosphorylations of eNOS, Akt and ERK1/2 [3]. Ft1 showed the best inhibitory effect on cell proliferation of SH-SY5Y cells with IC50 of 45μM. Ft1 not only arrested the cell cycle at S, G2/M stages, but also promoted cell apoptosis. Ft1 up-regulated the protein expressions of cleaved caspase 3, phospho-p53, p21, and cyclin B1, but down-regulated that of Bcl-2. Moreover, Ft1 enhanced the phosphorylation of ERK1/2, JNK and p38 MAPK [4].in vivo: Ft1 promotes the formation of blood vessels in Matrigel plug and wound healing in mice [1].

REFERENCES

[1]. Shen K, et al. Notoginsenoside Ft1 promotes angiogenesis via HIF-1α mediated VEGF secretion and the regulation of PI3K/AKT and Raf/MEK/ERK signaling pathways. Biochem Pharmacol. 2012 Sep 15;84(6):784-92.

[2]. Gao B, et al. Platelet P2Y?? receptors are involved in the haemostatic effect of notoginsenoside Ft1, a saponin isolated from Panax notoginseng. Br J Pharmacol. 2014 Jan;171(1):214-23.

[3]. Shen K, et al. Notoginsenoside Ft1 activates both glucocorticoid and estrogen receptors to induce endothelium-dependent, nitric oxide-mediated relaxations in rat mesenteric arteries. Biochem Pharmacol. 2014 Mar 1;88(1):66-74.

[4]. Gao B, et al. p38 MAPK and ERK1/2 pathways are involved in the pro-apoptotic effect of notoginsenoside Ft1 on human neuroblastoma SH-SY5Y cells. Life Sci. 2014 Jul 17;108(2):63-70.

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