Pseudoginsenoside F11

Cat. No.: HY-N0541

CAS No.: 69884-00-0

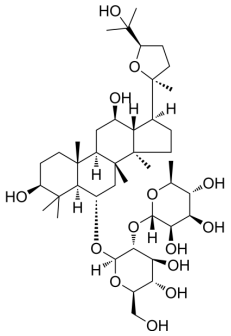
Molecular Formula: $C_{42}H_{72}O_{14}$

Molecular Weight: 801.01

Target: Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (62.42 mM; ultrasonic and warming and heat to 60°C)
H_2O : 0.67 mg/mL (0.84 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.2484 mL	6.2421 mL	12.4842 mL
	5 mM	0.2497 mL	1.2484 mL	2.4968 mL
	10 mM	0.1248 mL	0.6242 mL	1.2484 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 (3.12 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.12 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.12 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pseudoginsenoside F11 (Ginsenoside A1), a component of Panax quinquefolium (American ginseng), has been demonstrated to antagonize the learning and memory deficits induced by scopolamine, morphine and methamphetamine in mice.

In Vitro

Biochemical experiments revealed that Pseudoginsenoside F11 (Ginsenoside A1) could inhibit diprenorphine (DIP) binding with an IC_{50} of 6.1 μM and reduced the binding potency of morphine in Chinese hamster ovary (CHO)-μ cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	One in vivo model of cisplatin-induced acute renal failure was performed. The results showed that pretreatment with Pseudoginsenoside Pseudoginsenoside F11 (Ginsenoside A1) reduced cisplatin-elevated blood urea nitrogen and creatinine levels, as well as ameliorated the histophathological damage [1]. We tested the effects of Pseudoginsenoside Pseudoginsenoside F11 (Ginsenoside A1) on morphine-induced development of behavioral sensitization and alterations in glutamate levels in the medial prefrontal cortex (mPFC) in freely moving mice by using in vivo microdialysis. As the results shown, Pseudoginsenoside Pseudoginsenoside F11 (Ginsenoside A1) antagonized the development of behavioral sensitization and decrease of glutamate in the mPFC induced by morphine[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Front Pharmacol. 29 April 2021.

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REFERENCES

[1]. Wang H, et al. The pseudoginsenoside F11 ameliorates cisplatin-induced nephrotoxicity without compromising its anti-tumor activity in vivo. Scientific Reports [2014, 4:4986]

[2]. Li Zhu, et al. Pseudoginsenoside-F11 attenuates morphine-induced signalling in Chinese hamster ovary-μ cells. Neuroreport, 25 May 2001 - Volume 12 - Issue 7 - pp 1453-1456

[3]. Yue Hao, et al. Pseudoginsenoside-F11 decreases morphine-induced behavioral sensitization and extracellular glutamate levels in the medial prefrontal cortex in mice. Pharmacology Biochemistry and BehaviorVolume 86, Issue 4, April 2007, Pages 660–666

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