Puerarin

Cat. No.: HY-N0145

CAS No.: 3681-99-0

Molecular Formula: $C_{21}H_{20}O_9$

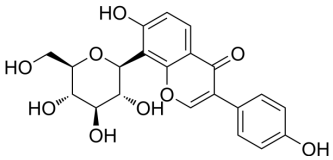
Molecular Weight: 416.38

Target: 5-HT Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (120.08 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4017 mL	12.0083 mL	24.0165 mL
	5 mM	0.4803 mL	2.4017 mL	4.8033 mL
	10 mM	0.2402 mL	1.2008 mL	2.4017 mL

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

In Vivo

1. Add each solvent one by one: 20% SBE-β-CD in saline
   Solubility: 20 mg/mL (48.03 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 3 mg/mL (7.20 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 3 mg/mL (7.20 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 3 mg/mL (7.20 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Puerarin, an isoflavone extracted from Radix puerariae, is a 5-HT2C receptor antagonist.

IC_{50} & Target

5-HT_{2C} Receptor

In Vitro

Puerarin inhibits the expression of LPS-induced iNOS, COX-2 and CRP proteins and also suppresses their mRNAs from RT-

	PCR experiments in RAW264.7 cells. The inhibition of iNOS, COX-2 and CRP expression is due to a dose-dependent inhibition of phosphorylation and degradation of I-κB, which resulted in the reduction of p65NF-κB nuclear translocation. The effect of puerarin-mediated inhibition of LPS-induced iNOS, COX-2 and CRP expression is attributed to suppressed NF-κB activation at the transcriptional level[1]. Puerarin is a novel open-channel blocker of IK1, which may underlie the antiarrhythmic action of puerarin. Puerarin competes with barium, an open-channel blocker of IK1, to inhibit IK1 currents[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Both genistein and puerarin effectively alleviate hepatic damage induced by chronic alcohol administration through potential antioxidant, anti-inflammatory, or anti apoptotic mechanisms. However, genistein is more effective than puerarin in decreasing levels of malondialdehyde (1.05±0.0947 vs. 1.28±0.213 nmol/mg pro, p< 0.05), tumor necrosis factor α (3.12±0.498 vs. 3.82±0.277 pg/mg pro, p < 0.05), interleukin-6 (1.46±0.223 vs. 1.88±0.309 pg/mg pro, p < 0.05), whereas puerarin is more effective than genistein in ameliorating serum activities or levels of alanine transaminase (35.8±3.95 vs. 42.6±6.56 U/L, p < 0.05) and low-density lipoprotein cholesterol (1.12±0.160 vs. 1.55±0.150 mmol/L, p < 0.05) [3]. Early-stage renal damages can be significantly improved by puerarin, possibly via its suppression of ICAM-1 and TNF-α expression in diabetic rat kidneys[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	RAW264.7 cells are maintained at subconfluence in 95% air and 5% CO_2 humidified atmosphere maintained at 37°C. The medium used for routine subculture is Dulbecco's Modified Eagle's Medium supplemented with 10% fetal bovine serum, penicillin (100 units/mL) and streptomycin (100 μg/ mL). An MTT assay is used to measure the viability of the cells after treatment with puerarin. After the supernatants are removed for nitrite determination, cells are incubated at 37°C with MTT (0.05 mg/mL) for 4 h, and the optical density is measured at 540 nm. The concentrations of puerarin are10, 20, 40 and 100 μM[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3][4]	Rats: A cohort of healthy male SD rats (7 weeks old) are randomLy divided into a control group, a model group, and a puerarin treatment group with high (H), moderate (M), and low (L) dosage. Puerarin is re-suspended in 0.9% saline and is given by intra-gastric intubation at various concentrations (0.25 mg/(kg×d) for L group, 0.5 mg/(kg×d) for M group, and 1.0 mg/(kg×d) for H group) each day for 8 consecutive days. An equal volume of saline is administered to control and model rats during the same time period[4]. Mice: Forty male ICR mice (weight: 20-22 g) are acclimatized with a daily 12 h light:12 h dark cycle at 22±2 °C room temperature and 55%±5% relative humidity. After 1 week of adaption, the mice are randomLy divided into four groups with ten mice per group. Genistein and puerarin are applied to the mice in sodium carboxymethyl cellulose solution with an equimolar concentration of 0.1 M (gastric volume: 3 mL kg^{-1} body weight)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2021 Jan;11(1):143-155.
- Int J Nanomedicine. 2023 Sep 8;18:5095-5117.
- Free Radic Biol Med. 2022 Aug 5;S0891-5849(22)00508-1.
- Phytother Res. 2022 Jul 24.
- J Ethnopharmacol. 8 November 2021, 114786.

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REFERENCES

[1]. Hu W, et al. Puerarin inhibits iNOS, COX-2 and CRP expression via suppression of NF-κB activation in LPS-induced RAW264.7 macrophage cells. Pharmacol Rep. 2011;63(3):781-9.

[2]. Zhang H, et al. Puerarin: a novel antagonist to inward rectifier potassium channel (IK1). Mol Cell Biochem. 2011 Jun;352(1-2):117-23.

[3]. Zhao L,et al. Protective Effects of Genistein and Puerarin against Chronic Alcohol-Induced Liver Injury in Mice via Antioxidant, Anti-inflammatory, and Anti-apoptotic Mechanisms.J Agric Food Chem. 2016 Sep 28;64(38):7291-7.

[4]. Pan X, et al. Effect of Puerarin on Expression of ICAM-1 and TNF-α in Kidneys of Diabetic Rats. Med Sci Monit. 2015 Jul 23;21:2134-40.

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