Galangin

Cat. No.: HY-N0382

CAS No.: 548-83-4

Molecular Formula: $C_{15}H_{10}O_5$

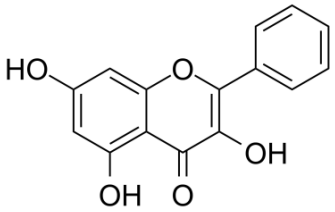
Molecular Weight: 270.24

Target: Cytochrome P450; Autophagy; Autophagy

Pathway: Metabolic Enzyme/Protease; Autophagy

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 36 mg/mL (133.21 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7004 mL	18.5021 mL	37.0041 mL
5 mM	0.7401 mL	3.7004 mL	7.4008 mL
10 mM	0.3700 mL	1.8502 mL	3.7004 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: ≥ 2.08 mg/mL (7.70 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Galangin (Norizalpinin) is an agonist/antagonist of the arylhydrocarbon receptor. Galangin (Norizalpinin) also shows inhibition of CYP1A1 activity.

In Vitro

Galangin (Norizalpinin) inhibits the catabolic breakdown of DMBA, as measured by thin-layer chromatography, in a dose-dependent manner. Galangin also inhibits the formation of DMBA-DNA adducts, and prevents DMBA-induced inhibition of cell growth. Galangin causes a potent, dose-dependent inhibition of CYP1A1 activity, as measured by ethoxyresorufin-O-deethylase activity, in intact cells and in microsomes isolated from DMBA-treated cells. Analysis of the inhibition kinetics by double-reciprocal plot demonstrates that galangin inhibits CYP1A1 activity in a noncompetitive manner. Galangin causes an increase in the level of CYP1A1 mRNA, indicating that it may be an agonist of the aryl hydrocarbon receptor, but it inhibits the induction of CYP1A1 mRNA by DMBA or by 2,3,5,7-tetrachlorodibenzo-p-dioxin (TCDD). Galangin also inhibits the DMBA- or TCDD-induced transcription of a reporter vector containing the CYP1A1 promoter[1]. Galangin treatment inhibits cell

proliferation and induced autophagy (130 μM) and apoptosis (370 μM). In particular, galangin treatment in HepG2 cells causes (1) an accumulation of autophagosomes, (2) elevated levels of microtubule-associated protein light chain 3, and (3) an increased percentage of cells with vacuoles. p53 expression is also increased. The galangin-induced autophagy is attenuated by the inhibition of p53 in HepG2 cells, and overexpression of p53 in Hep3B cells restored the galangin-induced higher percentage of cells with vacuoles to normal level[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]

Cells (5.0×10^3) are seeded and treated with different concentrations of galangin for different periods of time in 96-well plates. The number of viable cells in each well is determined by adding 10 μL of 5 mg/mL MTT solution. Following the 4 hour incubation at 37°C, the cells are dissolved in a 100 μL solution containing 20% SDS and 50% dimethy formamide. The optical densities are quantified at a test wavelength of 570 nm with a reference wavelength of 630 nm using a Varioskan Flash Reader spectrophotometer.

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.
- Pharmacol Res. 2020 May;155:104751.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Ciolino HP, et al. The flavonoid galangin is an inhibitor of CYP1A1 activity and an agonist/antagonist of the aryl hydrocarbon receptor. Br J Cancer. 1999 Mar;79(9-10):1340-6.

[2]. Wen M, et al. Galangin induces autophagy through upregulation of p53 in HepG2 cells. Pharmacology. 2012;89(5-6):247-55.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA