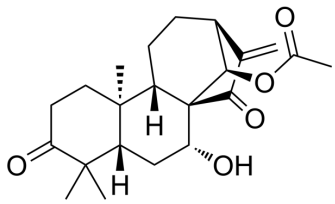
Glaucocalyxin B

Cat. No.:	HY-N2113
CAS No.:	80508-81-2
Molecular Formula:	$C_{22}H_{30}O_5$
Molecular Weight:	374.47
Target:	Autophagy
Pathway:	Autophagy
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (267.04 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6704 mL	13.3522 mL	26.7044 mL
	5 mM	0.5341 mL	2.6704 mL	5.3409 mL
	10 mM	0.2670 mL	1.3352 mL	2.6704 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: ≥ 1.25 mg/mL (3.34 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.25 mg/mL (3.34 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.25 mg/mL (3.34 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Glaucocalyxin B is an ent kaurane diterpenoid isolated from the Chinese traditional medicine Rabdosia japonica with anticancer and antitumor activity; decreases the growth of HL-60 cells with an IC_{50} of approximately 5.86 μM at 24 h.

IC_{50} & Target

IC50: 5.86 μM (HL-60 cell Growth)[1]

In Vitro

Glaucocalyxin A (GlnA) and (GlnB) dose-dependently decrease the growth of HL-60 cells with an IC_{50} of approximately 6.15 and 5.86 μM at 24 h, respectively. Both Gln A and B could induce apoptosis, G2/M-phase cycle arrest, DNA damage and the accumulation of reactive oxygen species (ROS) in HL-60 cells[1]. GlnB inhibits the proliferation of human cervical cancer cells

in vitro through the induction of apoptosis andautophagy, which may be mediated by the phosphatidylinositol 4,5 bisphosphate 3 kinase/Akt signaling pathway. Treatment with GlnB inhibits the proliferation of HeLa and SiHa cervical cancer cell lines in a dose dependent manner. GlnB increases the apoptotic cell population of and enhanced poly (ADP ribose) polymerase 1 cleavage. GlnB also induces increased light chain 3 II/I protein cleavage, indicating the induction of autophagy. GlnB treatment increases the expression of phosphatase and tensin homolog and decreases the expression of phosphorylated protein kinase B[2]. Glaucocalyxin B (GLB), one of five ent-kauranoid diterpenoids, significantly decreased the generation of nitric oxide (NO), tumor necrosis factor (TNF)-α, interleukin (IL)-1β, cyclooxygenase (COX)-2 and inducible nitric oxide synthase (iNOS) in the lipopolysaccharide (LPS)-activated microglia cells[3].

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

PROTOCOL

Cell Assay [3]

The microglia cells viability is assessed by MTT assay. Cells are seeded in 96-well plates at the density of 5×10^4 cells/well. The cell culture supernatant is discarded after treatment with various agents, and then 30 μL of MTT (0.5 mg/mL) solution is added into each well. After incubation for 4 h at 37 °C, 100 μL of DMSO is added into each well to dissolve the formazan dye, and then the absorbance of solubilized formazan is measured by microplate reader[3].

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

REFERENCES

[1]. Yang WH, et al. Glaucocalyxin A and B-induced cell death is related to GSH perturbation in human leukemia HL-60 cells. Anticancer Agents Med Chem. 2013 Oct;13(8):1280-90.

[2]. Pan Y, et al. Glaucocalyxin B induces apoptosis and autophagy in human cervical cancer cells. Mol Med Rep. 2016 Aug;14(2):1751-5.

[3]. Gan P, et al. Anti-inflammatory effects of glaucocalyxin B in microglia cells. J Pharmacol Sci. 2015 May;128(1):35-46.

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