Parthenolide

Cat. No.: HY-N0141

CAS No.: 20554-84-1

Molecular Formula: $C_{15}H_{20}O_3$

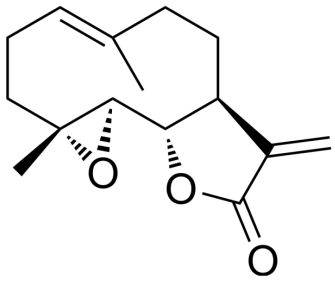
Molecular Weight: 248.32

Target: NF-κB; Autophagy; Mitophagy; Apoptosis

Pathway: NF-κB; Autophagy; Apoptosis

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (402.71 mM)

H_2O : 0.67 mg/mL (2.70 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.0271 mL	20.1353 mL	40.2706 mL
5 mM	0.8054 mL	4.0271 mL	8.0541 mL
10 mM	0.4027 mL	2.0135 mL	4.0271 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.07 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (8.38 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Parthenolide is a sesquiterpene lactone found in the medicinal herb Feverfew. Parthenolide exhibits anti-inflammatory activity by inhibiting NF-κB activation; also inhibits HDAC1 protein without affecting other class I/II HDACs.

IC_{50} & Target			
	NF-κB	Autophagy	Mitophagy

In Vitro

Parthenolide (PTL) has a dose-dependent growth inhibition effect on NSCLC cells Calu-1, H1792, A549, H1299, H157, and H460. Parthenolide can induce cleavage of apoptotic proteins such as CASP8, CASP9, CASP3 and PARP1 both in concentration- and time-dependent manner in tested lung cancer cells, indicating that apoptosis is trigged after Parthenolide exposure. In addition to induction of apoptosis, Parthenolide also induces G_0/G_1 cell cycle arrest in a

	concentration-dependent manner in A549 cells and G_2/M cell cycle arrest in H1792 cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Only Parthenolide, the HDAC inhibitor with anti-inflammatory features, displayed a potent anti-apoptotic effect in Phb1 KO hepatocytes. Indeed, TSA and Parthenolide-treated hepatocytes showed increased levels of FXR, and reduced levels of CYP7A1, HDAC4, TNFα, TRAIL and Bax suggesting a less toxic effect of bile acids as a results of specific HDAC inhibition, resulting in the attenuation of the Phb1 KO hepatocytes apoptotic response. Importantly, Parthenolide exerts a protective effect from the liver injury after BDL in Phb1 KO mice. Indeed, Parthenolide treatment results in a reduction of the mortality rate of this mice after BDL associated with a lower apoptotic response as revealed by a reduction of necrotic areas, Tunel-staining, as well as decreased ALT (8431±957 vs.4225±210 U/L) and AST (4805±300 vs.2242±438 U/L) activities compared to control Phb1 KO mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Human lung cancer cell lines are seeded in 96-well plates and treated on the second day with the given concentration of Parthenolide (0, 5, 10, 20 μM) for another 48 hours and then subjected to SRB or MTT assay. For SRB assay, live cell number is estimated as described earlier. After treatment, the medium is discarded firstly. In order to fix the adherent cells, 100 μL of cold trichloroacetic acid (10% (w/v)) are adding to each well and incubating at 4°C for at least 1 hour. The plates are then washed five times with deionized water and dried in the air. Each well are then added with 50 μL of SRB solution (0.4% w/v in 1% acetic acid) and incubated for 5 min at room temperature. The plates are washed five times with 1% acetic acid to remove unbound SRB and then air dried. The residual bound SRB is solubilized with 100 μL of 10 mM Tris base buffer (pH 10.5), and then read using a microtiter plate reader at 495 nm. The MTT assay is executed. 20 μL MTT (5 mg/mL) are added to each sample and incubate at 37°C for 4 h, then 100 μL solubilization solution are added. Cell viability is determined at 595 nm[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mice[3] ***Phb1*** KO mice are used. Males from 8-12 weeks of age are treated. Parthenolide is intraperitoneally injected at a dose of 3 mg/kg 24 h and 1h before bile duct ligation (BDL) or twice a week during two weeks. Liver specimens are snap-frozen for subsequent analysis[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Gut Microbes. 2023 Jan-Dec;15(1):2221093.
- Proc Natl Acad Sci U S A. 2019 Feb 19;116(8):2961-2966.
- J Pineal Res. 2024 Jan 31.
- Cancer Lett. 2018 Aug 1;428:77-89.
- Phytomedicine. 2022: 154627.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Nakshatri H, et al. NF-κB-dependent and -independent epigenetic modulation using the novel anti-cancer agent DMAPT. Cell Death Dis. 2015 Jan 22;6:e1608.

[2]. Zhao X, et al. Parthenolide induces apoptosis via TNFRSF10B and PMAIP1 pathways in human lung cancer cells. J Exp Clin Cancer Res. 2014 Jan 6;33:3.

[3]. Barbier-Torres L, et al. Histone deacetylase 4 promotes cholestatic liver injury in the absence of prohibitin-1. Hepatology. 2015 Oct;62(4):1237-48.

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