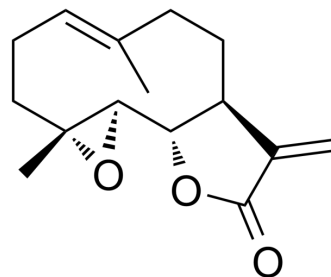


Parthenolide

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)
 H₂O : < 0.1 mg/mL (insoluble)
 * "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	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

- Add each solvent one by one: 10% DMSO >> 90% corn oil
 Solubility: ≥ 2.5 mg/mL (10.07 mM); Clear solution
- 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.

IC₅₀ & Target

NF-κB	Autophagy	Mitophagy
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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 triggered after Parthenolide exposure. In addition to induction of apoptosis, Parthenolide also induces G₀/G₁ cell cycle arrest in a

concentration-dependent manner in A549 cells and G₂/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, TNF α , 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 \pm 957 vs.4225 \pm 210 U/L) and AST (4805 \pm 300 vs.2242 \pm 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 μ M) 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 μ L 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 μ L 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 μ L 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 μ L MTT (5 mg/mL) are added to each sample and incubate at 37°C for 4 h, then 100 μ L solubilization solution are added. Cell viability is determined at 595 nm^[2].

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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].

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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.

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REFERENCES

[1]. Nakshatri H, et al. NF- κ B-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.

Caution: Product has not been fully validated for medical applications. For research use only.

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