Betulinic acid

Cat. No.: HY-10529

CAS No.: 472-15-1

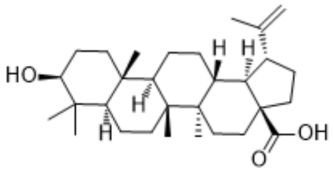
Molecular Formula: $C_{30}H_{48}O_3$

Molecular Weight: 456.7

Target: Apoptosis; Topoisomerase; HIV; Autophagy; Mitophagy; NF-κB; Endogenous Metabolite; Parasite

Pathway: Apoptosis; Cell Cycle/DNA Damage; Anti-infection; Autophagy; NF-κB; Metabolic Enzyme/Protease

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 35.71 mg/mL (78.19 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1896 mL	10.9481 mL	21.8962 mL
	5 mM	0.4379 mL	2.1896 mL	4.3792 mL
	10 mM	0.2190 mL	1.0948 mL	2.1896 mL

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

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 4 mg/mL (8.76 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.55 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Betulinic acid is a natural pentacyclic triterpenoid, acts as a eukaryotic topoisomerase I inhibitor, with an IC_{50} of 5 μM, and possesses anti-HIV, anti-malarial, anti-inflammatory and anti-tumor properties[1][2][3][4].

IC_{50} & Target

Topoisomerase I	HIV-1	NF-κB
5 μM (IC_{50})	1.4 μM (EC50)	

In Vitro

Betulinic acid is a eukaryotic topoisomerase I inhibitor, with an IC_{50} of 5 μM, and prevents topoisomerase I-DNA interaction [1]. Betulinic acid (10, 20, 40, 80, and 160 μM) significantly suppresses MDA-MB-231 cell viability in a time- and dose-dependent manner after treatment for 24 or 48 h.? Betulinic acid (20, 40 μM) causes decrease in Bcl-2 expression of MDA-MB-231 cells. Betulinic acid also induces morphological changes of MDA-MB-231 cells at 20 μM, and leads to ultrastructure

	changes of MDA-MB-231 cells at 40 μM[2]. Betulinic acid shows anti-HIV activities, with an EC_{50} of 1.4 μM in acutely infected H9 lymphocytes[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Betulinic acid (10 and 30 mg/kg, p.o.) significantly abrogates colon shortening, and reduces malondialdehyde (MDA) and lipid hydroperoxide levels in dextran sulfate sodium (DSS)-induced colitis in mice. Betulinic acid (30 mg/kg, p.o.) restores superoxide dismutase (SOD), catalase activity and glutathione (GSH) content to control levels in DSS-induced colitis in mice. Betulinic acid (30 mg/kg, p.o.) also inhibits the DSS-induced increase in inflammatory markers. Betulinic acid (3, 10, 30 mg/kg, p.o.) suppresses acetic acid-induced writhing responses and mustard oil (MO)-induced visceral nociception in mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	CCK-8 is used in the assay. MDA-MB-231 cells are cultured in 96-well plates at a density of 2×10^3 cells/well and then treated with DMSO vehicle or various concentrations of Betulinic acid ranging from 5 μM to 160 μM in 100 μL of medium for the indicated times. After the treatment period, the medium in each well is replaced with 110 μL of medium containing 10 μL of the CCK-8 mixture, and the plates are incubated for 1 h and 30 min at 37°C. The absorbance at a wavelength of 450 nm is measured with a microplate reader[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Female Swiss albino mice are administered vehicle (5% v/v DMSO in peanut oil) or Betulinic acid (3, 10 or 30 mg/kg) in vehicle, orally. After 1 h, acetic acid (300 mg/kg) is administered by intraperitoneal route and number of writhing response of each animal is counted for 20 min by an observer who is blind to the treatments. Writhing response is when animal rubs its abdomen on surface of table/floor with elongation of the body and extension of the hind limbs[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Bull. 2023 Nov 14.
- Theranostics. 2022; 12(8): 3656-3675.
- Green Chem. 2019, 21, 3370-3382.
- Int J Biol Sci. 2021 Mar 11;17(4):1138-1152.
- Fuel. 2018 Dec 15, 234:110-119.

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REFERENCES

[1]. Chowdhury AR, et al. Betulinic acid, a potent inhibitor of eukaryotic topoisomerase I: identification of the inhibitory step, the major functional group responsible and development of more potent derivatives. Med Sci Monit. 2002 Jul;8(7):BR254-65.

[2]. Gao Y, et al. Betulinic acid induces apoptosis and ultrastructural changes in MDA-MB-231 breast cancer cells. Ultrastruct Pathol. 2018 Jan-Feb;42(1):49-54.

[3]. Kalra J, et al. Betulinic acid alleviates dextran sulfate sodium-induced colitis and visceral pain in mice. Naunyn Schmiedebergs Arch Pharmacol. 2017 Dec 26.

[4]. Hashimoto F, et al. Anti-AIDS agents--XXVII. Synthesis and anti-HIV activity of betulinic acid and dihydrobetulinic acid derivatives. Bioorg Med Chem. 1997 Dec;5(12):2133-43.

[5]. Kasperczyk H, et al. Betulinic acid as new activator of NF-kappaB: molecular mechanisms and implications for cancer therapy. Oncogene. 2005 Oct 20;24(46):6945-56.

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