Betulin

Cat. No.: HY-N0083

CAS No.: 473-98-3

Molecular Formula: $C_{30}H_{50}O_2$

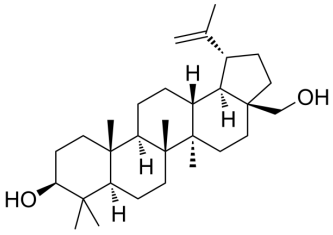
Molecular Weight: 442.72

Target: Apoptosis; Fatty Acid Synthase (FASN); Endogenous Metabolite

Pathway: Apoptosis; Metabolic Enzyme/Protease

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 : 3.33 mg/mL (7.52 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2588 mL	11.2938 mL	22.5876 mL
5 mM	0.4518 mL	2.2588 mL	4.5175 mL
10 mM	---	---	---

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: 3 mg/mL (6.78 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: ≥ 0.2 mg/mL (0.45 mM); Clear solution
3. Add each solvent one by one: 5% DMSO >> 95% corn oil
   Solubility: ≥ 0.2 mg/mL (0.45 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Betulin (Trochol), is a sterol regulatory element-binding protein (SREBP) inhibitor with an IC_{50} of 14.5 μM in K562 cell line.

IC_{50} & Target

IC50: 14.5 μM (SREBP, K562 cell), 74.1 μM (SREBP, HeLa cell), 17.1 μM (SREBP, GOTO cell)[1], 21.09 μM (SREBP, 181P cell), 20.62 μM (SREBP, HeLa cell)[2]

In Vitro

Betulin (BE) displays a broad spectrum of biological and pharmacological properties, among which the anticancer and

	chemopreventive activity attract most of the attention. BE has been shown to elicit anticancer properties by inhibiting cancer cells growth. BE has exhibited quite a different range of its antiproliferative activity, depending on cancer cells type, from a weak inhibition of cell proliferation in human erythroleukaemia cell line (K562) to a strong inhibition in human neuroblastoma cells (SK-N-AS), where the effect has been most pronounced. Additionally, BE has also been found to express significant cytotoxicity against primary cancer cells cultures isolated from tumour samples obtained from ovarian, cervical carcinoma, and glioblastoma patients, where the IC_{50} values have ranged from 2.8 to 3.4 μM, being significantly lower, when compared with established cell lines[1]. The cytotoxic activity of crude birch bark extract and purified betulin and betulinic acid towards human gastric carcinoma (EPG85-257) and human pancreatic carcinoma (EPP85-181) drug-sensitive and drug-resistant (daunorubicin and mitoxantrone) cell lines are compared. Significant differences in sensitivity between cell lines depending on the compound used are shown, suggesting that both betulin and betulinic acid can be considered as a promising leads in the treatment of cancer[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Betulin could improve glucose intolerance and modify basal learning performance. Treatment with betulin significantly restores SOD activity and decreased MDA content in hippocampus. Betulin also markedly reduces the contents of inflammatory cytokines in serum and hippocampus. Furthermore, administration of BE effectively upregulated the expressions of Nrf2, HO-1 and blocked the phosphorylations of IκB, NF-κB. In summary, BE might exhibit protective effect on cognitive decline in STZ-induced diabetic rats through HO-1/Nrf-2/ NF-κB pathway[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Chemoresistance is tested using a proliferation assay based on sulphorhodamine B staining. Briefly, 800 cells per well are seeded in triplicate in 96-well plates. After attachment for 24 h, substances are added in dilution series for a 5-day incubation, before SRB staining is performed. Incubation is terminated by replacing the medium with 10% trichloroacetic acid, followed by further incubation at 4°C for 1h. Subsequently, the plates are ished five times with water and stained by adding 100 μL 0.4% SRB in 1% acetic acid for 10 min at room temperature. Ishing the plates five times with 1% acetic acid eliminated unbound dye. After air-drying and re-solubization of the protein bound dye in 10 mM Tris-HCl (pH=8.0), absorbance is read at 562 nm[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Rats: The rats are randomLy divided into five groups (n=10): control group, STZ group, STZ+betulin (20 mg/kg) group, STZ+betulin (40 mg/kg) group. Diabetes is induced by STZ (30 mg/kg, i.p.) dissolved in citrate buffer (pH 4.4, 0.1 M) using 1 mL syringe for 4 weeks, meanwhile the control rats receive an equal volume of citrate buffer. Thereafter, the diabetic rats are treated with betulin (20 mg/kg, 40 mg/kg) for another 4 weeks[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2022 Jul 30;13(1):4428.
- Autophagy. 2021 Jul;17(7):1592-1613.
- Redox Biol. 2023 Jun.
- Cell Death Dis. 2019 Sep 11;10(9):672.
- Mol Metab. 2021 Dec 30;101428.

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REFERENCES

[1]. Król SK, et al. Comprehensive review on betulin as a potent anticancer agent. Biomed Res Int. 2015;2015:584189.

[2]. Drag M, et al. Comparision of the Cytotoxic Effects of Birch Bark Extract, Betulin and Betulinic Acid Towards Human Gastric Carcinoma and Pancreatic Carcinoma Drug-sensitive and Drug-Resistant Cell Lines. Molecules. 2009 Apr 24;14(4):1639-51.

[3]. Ma C, et al. Protective effect of betulin on cognitive decline in streptozotocin (STZ)-induced diabetic rats. Neurotoxicology. 2016 Dec;57:104-111.

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