

# **Betulin Datasheet**

4<sup>th</sup> Edition (Revised in July, 2016)

### [ Product Information ]

Name: Betulin

Catalog No.: CFN98710

Cas No.: 473-98-3

**Purity: >=98%** 

**M.F:** C<sub>30</sub>H<sub>50</sub>O<sub>2</sub>

M.W: 442.72

Physical Description: Powder

Synonyms: Lup-20(29)-ene-3b,28-diol.

# HO LE HOOH

### [ Intended Use ]

- 1. Reference standards;
- 2. Pharmacological research;
- 3. Synthetic precursor compounds;
- 4. Intermediates & Fine Chemicals;
- 5. Others.

### [Source]

The barks of Betula alba L.

## [ Biological Activity or Inhibitors]

Betulin is an abundant naturally occurring triterpene and it is found predominantly in

bushes and trees forming the principal extractive (up to 30% of dry weight) of the bark of

birch trees, it has antimalarial and anti-inflammatory activities; it and its derivatives have

especially shown anti-HIV activity and cytotoxicity against a variety of tumor cell lines

comparable to some clinically used drugs [1]

Betulinic acid and its derivatives have cytotoxicity, they have potent anti-tumor activity

especially in combination with cholesterol.<sup>[2]</sup>

Betulinic acid has anti-AIDS activity.[3]

Betulin has inhibition of sterol regulatory element-binding proteins (SREBPs), SREBPs

are major transcription factors activating the expression of genes involved in biosynthesis

of cholesterol, fatty acid and triglyceride, inhibition SREBP pathway can be employed as a

therapeutic strategy to treat metabolic diseases including type II diabetes and

atherosclerosis; thus betulin can improves hyperlipidemia and insulin resistance and

reduce atherosclerotic plaques.[4]

Betulin and betulinic acid inhibit ethanol-induced activation of hepatic stellate cells (HSCs)

on different levels, acting as antioxidants, inhibitors of cytokine production, and inhibitors

of tumor growth factor-  $\beta$  (TGF-  $\beta$ ), and nuclear factor-  $\kappa$  B (NF  $\kappa$  B)/I  $\kappa$  B transduction

signaling; betulin is also inhibitor of both JNK and p38 MAPK signal transduction, while

betulinic acid inhibits only c-Jun N-terminal kinase (JNK), suggests that they are

promising agents for anti-fibrotic combination therapies.<sup>[5]</sup>

[Solvent]

Chloroform, Dichloromethane, Ethyl Acetate, DMSO, Acetone, etc.

[ HPLC Method ]<sup>[6]</sup>

Mobile phase: Acetonitrile- H2O=86:14;

Flow rate: 1.0 ml/min:

Column temperature: Room Temperature;

The wave length of determination: 210 nm.

### [Storage]

2-8°C, Protected from air and light, refrigerate or freeze.

### [References]

- [1] Alakurtti S, Mäkelä T, Koskimies S, et al. Eur. J. Pharm. Sci., 2006, 29(1):1-13.
- [2] Franziska B. Mullauer, Jan H. Kessler, Jan Paul Medema. Plos One, 2009, 4(4):e1-e1.
- [3] Sun C, Wang H K, Yoshiki Kashiwada, et al. J. Med. Chem., 1998, 41(23):4648-57.
- [4] Tang J J, Li J G, Qi W, et al. Cell Metab., 2011, 13(1):44-56.
- [5] Szuster-Ciesielska A, Plewka K, Daniluk J, et al. Toxicology, 2011, 280(3):152-63.
- [6] Zhao G, Yan W, Dan C. J. Pharmaceut. Biomed. Anal., 2007, 43(3):959-62.

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