Natural Products

ChemFaces

Cucurbitacin B Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: Cucurbitacin B

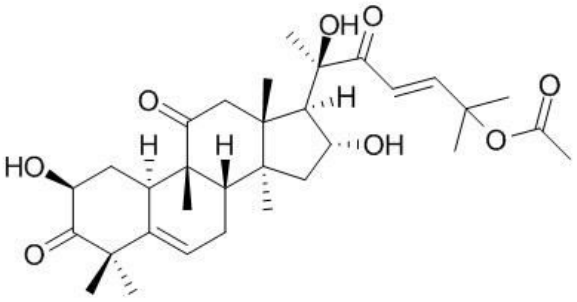
Catalog No.: CFN99129

Cas No.: 6199-67-3

Purity: > 98%

M.F: $C_{32}H_{46}O_8$

M.W: 558.70



Physical Description: Yellow powder

Synonyms: Acetic-acid[(E,6R)-6-[(2S,8S,9R,10R,13R,14S,16R,17R)-2,16-dihydroxy-4,4,9,13,14-pentamethyl-3,11-dioxo-2,7,8,10,12,15,16,17-octahydro-1H-cyclopenta[a]phenanthren-17-yl]-6-hydroxy-2-methyl-5-oxohept-3-en-2-yl] ester.

[Intended Use]

1. Reference standards;
2. Pharmacological research;
3. Food and cosmetic research;;
4. Synthetic precursor compounds;
5. Intermediates & Fine Chemicals;
6. Others.

[Source]

The rhizomes of *Hemsleya amabilis Diels.*

[Biological Activity or Inhibitors]

Cucurbitacin B (cucB) is a triterpenoid constituent of Cucurbitaceae vegetables and a promising phytochemical for cancer prevention, cucB induces G(2) arrest and apoptosis through a STAT3-independent but ROS-dependent mechanism in SW480 cells.[1]

Cucurbitacin B has antiproliferative effect, can induce apoptosis by inhibition of the JAK/STAT pathway and potentiates antiproliferative effects of gemcitabine on pancreatic cancer cells, may be an effective, new approach for the treatment of ER-, Her2/neu amplified, and p53 mutant breast cancers.[2,3]

Combination of cucurbitacin B at a relatively low concentration with either of the chemotherapeutic agents, docetaxel (DOC) or gemcitabine (GEM) , shows prominent antiproliferative activity against breast cancer cells without increased toxicity, this promising combination should be examined in therapeutic trials of breast cancer.[4]

Cucurbitacin B has anti-cutaneous squamous cell carcinoma (CSCC) activity by inhibiting growth, arresting the cell cycle, and synergistically potentiate the anti-proliferative effect of cisplatin in CSCC.[5]

Cucurbitacin B a selective inhibitor of JAK2/STAT3 signaling, could promote dendritic cells (DCs) differentiation and improve antitumor immunity, also significantly reduce the frequency of imCs in patients with lung cancers and enhance the effect of p53-specific CTL on tumor 16HBE/BPDE cells.[6]

[Solvent]

Chloroform, Dichloromethane, DMSO, Acetone.

[HPLC Method][7]

Mobile phase: Methanol:H2O=70:30;

Flow rate: 1.0 ml/min;

Column temperature: 30 ℃;

The wave length of determination: 230 nm.

[Storage]

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

[References]

[1] Yasuda S, Yogosawa S, Izutani Y, *et al. Mol. Nutr. Food Res., 2010, 54(4):559-65.*

[2] Thoennissen N H, Iwanski G B, Doan N B, *et al. Cancer Res., 2009, 69(14):5876-84.*

[3] Wakimoto N, Dong Y, O'Kelly J, *et al. Cancer Sci., 2008, 99(9):1793-7.*

[4] Aribi A, Sigal Gery , Lee D H, *et al. Int. J. Cancer, 2013, 132(12):2730-7.*

[5] Chen W, Leiter A, Yin D, *et al. Int. J. Oncol., 2010, 37(3):737-43.*

[6] Lu P, Yu B, Xu J. *Cancer Biother. Radio, 2012, 27(27):495-503.*

[7] Sun W, Chao Z M, Wang C, *et al. Chinese Journal of Experimental Traditional Medical Formulae ,2014, 20(23):86-8.*

[Contact]

Address:
S5-3 Building, No. 111, Dongfeng Rd.,
Wuhan Economic and Technological Development Zone,
Wuhan, Hubei 430056,
China

Email: info@chemfaces.com
Tel: +86-27-84237783
Fax: +86-27-84254680
Web: www.chemfaces.com
Tech Support: service@chemfaces.com