

Eriocalyxin B Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: Eriocalyxin B

Catalog No.: CFN97402

Cas No.: 84745-95-9

Purity: > 98%

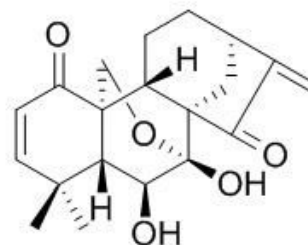
M.F: C₂₀H₂₄O₅

M.W: 344.4

Physical Description: Cryst.

Synonyms:

Kaura-2,16-diene-1,15-dione,7,20-epoxy-6,7- dihydroxy-,(6a,7R)-;Rabdosianone I.



[Intended Use]

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

[Source]

The herb of *Isodon eriocalyx*.

[Biological Activity or Inhibitors]

EriocalyxinB(Eri-B), an ent-kauranoid isolated from *Isodon eriocalyx*, an anti-inflammatory remedy; it inhibits the NF-kappa B transcriptional activity but not that of cAMP response element-binding protein; It suppresses the transcription of NF-kappa B downstream gene products including cyclooxygenase-2 and inducible nitric-oxide synthase induced by tumor necrosis factor-alpha or lipopolysaccharide in macrophages and hepatocarcinoma cells; indicates that Eri-B selectively blocks the binding between NF-kappa B and the response elements in vivo without affecting the nuclear translocation of the transcription factor.^[1]

Eriocalyxin B has antitumor effects via multiple pathways, and these pathways are related to immune responses; EriB exerts potent antiinflammatory effects through selective modulation of pathogenic Th1 and Th17 cells by targeting critical signaling pathways; EriB is a unique therapeutic agent for the treatment of autoimmune diseases.^[2]

Eriocalyxin B induces apoptosis of t(8;21) leukemia cells through NF-B and MAPK signaling pathways and triggers degradation of AML1-ETO oncoprotein in a caspase-3-dependent manner.^[3]

Eriocalyxin B possesses strong antileukemic activity, it can significantly inhibit lymphoma cell proliferation and induce apoptosis in association with caspase activation; suggests that EriB as a promising candidate targeting apoptosis cascade in treatment of hematological malignancies.^[4]

Eriocalyxin B is a specific inhibitor of STAT3, it directly targets STAT3 through a covalent linkage to inhibit the phosphorylation and activation of STAT3 and induces apoptosis of STAT3-dependent tumor cells.^[5]

[Solvent]

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

[HPLC Method]^[6]

Mobile phase: Acetonitrile- 0.1% Triethylamine, gradient elution ;

Flow rate: 1.0 ml/min;

Column temperature: Room Temperature;

The wave length of determination: 233 nm.

[Storage]

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

[References]

[1] Leung C H, Grill S P, Lam W, *et al. Mol. Pharmacol.*, 2006, 70(6):1946-55.

[2] Lu Y, Chen B, Song J H, *et al. P. Natl. Acad. Sci. U.S.A.*, 2013, 110(6):2258-63.

[3] Wang L, Zhao W L, Yan J S, *et al. Cell Death Differ*, 2006, 14(2):306-17.

[4] Zhang Y W, Jiang X X, Chen Q S, *et al. Exp. Hematol*, 2010, 38(3):191-201.

[5] Yu X, Li H, Cao P, *et al. Plos One*, 2015, 10(5): e0128406.

[6] Wang Z, Yuan Q, Sun J H, *et al. Areneimittel Forsch*, 2012, 62(12):666-9.

[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