Natural Products

ChemFaces

Cinobufagin Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: Cinobufagin

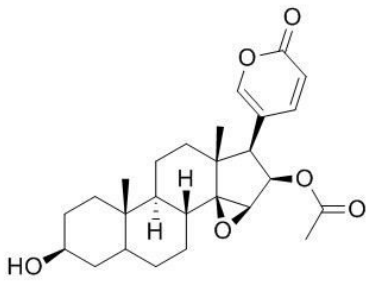
Catalog No.: CFN98544

Cas No.: 470-37-1

Purity: >=98%

M.F: $C_{26}H_{34}O_6$

M.W: 442.55



Physical Description: Powder

Synonyms: Trans-3-phenylpropenoic acid ;Trans-cinnamylic acid ;

22-dienolide,16-(acetyloxy)-14,15-epoxy-3-hydroxy-,(3-beta,5-beta,15-bufa-2.

[Intended Use]

1. Reference standards;

2. Pharmacological research;

3. Synthetic precursor compounds;

4. Intermediates & Fine Chemicals;

5. Others.

[Source]

The glandular body of *Bufo bufo gargarizans Cantor.*

[Biological Activity or Inhibitors]

Cinobufagin(CBG), a major component of cinobufacini (huachansu), is an important cardenolidal steroid, it has potent anti-cancer effects, it can potently inhibit the proliferation of U2OS, MG63 and SaOS-2 cells, significant increases in G2/M cell-cycle arrest and apoptosis in osteosarcoma (OS) cells, suggests that cinobufagin is a promising agent for the treatment of OS.[1]

Cinobufagin and bufalin may inhibit the proliferation of prostate cancer cell lines associated with sustained elevation of the [Ca 2+] i and that of apoptosis.[2]

Cinobufagin can inhibit rectifier potassium current (IK) without noticeable effect on transient potassium current (IA), at 1 μM concentration CBG could alter some channel kinetics and gating properties of IK, such as steady-state activation and inactivation curves, open probability and time constants; suggests that IK is probably a target of bufadienolides, which may explain the mechanisms of CBG' pathological effects on central nervous system. [3]

Cinobufacini and its active components bufalin and cinobufagin have anti-hepatitis B virus activities in HepG2.2.15 cells.[4]

Cinobufagin and bufalin exhibit cardiotonic and natriuretic activities; they also have inhibitory effects on steroidogenesis of aldosterone and cortisol, the effects are associated with inhibition of aldosterone synthase and 11β-hydroxylase, as well as the suppression of StAR protein expression and SF-1 binding to StAR promoter via the phosphorylation of ERK1/2 in H295 cells.[5]

[Solvent]

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

[HPLC Method][6]

Mobile phase: Tetrahydrofuran-Methanol-H2O= 8:31:61;

Flow rate: 1.0 ml/min;

Column temperature: Room Temperature;

The wave length of determination: 299 nm.

[Storage]

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

[References]

[1] Yin J Q, Wen L, Wu L C, *et al. Toxicol. Lett., 2013, 218(2):129-36.*

[2] Yeh J Y, Huang W J, Kan S F, *et al. Prostate, 2003, 54(2):112-24.*

[3] Hao S, Bao Y M, An L J, *et al. Toxicol. in Vitro, 2011, 25(8):1644-53.*

[4] Cui X, Inagaki Y, Xu H, *et al. Biol. Pharmaceut. Bull., 2010, 33(10):1728-32.*

[5] Kau M M, Wang J R, Tsai S C, *et al. Brit. J.Pharmacol., 2012, 165(6):1868–76.*

[6] Song H, Tao G, Bi K, *et al. Biomed. Chromatogr., 2000, 14(2):130-2.*

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