Natural Products

ChemFaces

Rutaecarpine Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: Rutaecarpine

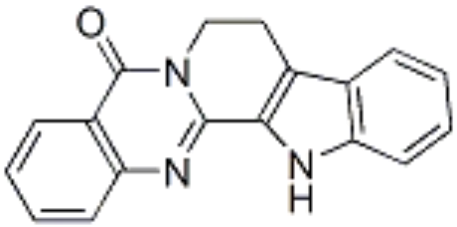
Catalog No.: CFN97337

Cas No.: 84-26-4

Purity: >=98%

M.F: $C_{18}H_{13}N_3O$

M.W: 287.32



Physical Description: Yellow powder

Synonyms: 8,13,13b,14-tetrahydroindolo[2',3':3,4]pyrido[2,1-b]quinazolin-5(7H)-one.

[Intended Use]

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

[Source]

The fruits of *Evodia rutaecarpa (Juss.) Benth.*

[Biological Activity or Inhibitors]

Rutaecarpine, evodiamine, and dehydroevodiamine are quinazolinocarboline alkaloids

isolated from a traditional Chinese medicine, *Evodia rutaecarpa*, rutaecarpine is a potent inhibitor of CYP1A2 in both mouse and human liver microsome.[1]

Rutaecarpine inhibits COX-2-dependent conversion of exogenous arachidonic acid to PGE2 in a dose-dependent manner by the COX-2-transfected HEK293 cells, it inhibits neither PLA2 and COX-1 activity nor COX-2 protein and mRNA expression up to the concentration of 30 microM in BMMC, indicating that rutaecarpine directly inhibits COX-2 activity; it shows in vivo anti-inflammatory activity on rat lambda-carrageenan induced paw edema by intraperitoneal administration, suggests that the anti-inflammatory activity of rutaecarpine could be attributed at least in part by inhibition of COS-2.[2]

Rutaecarpine and evodiamine have positive inotropic and chronotropic effects on the guinea-pig isolated right atria, possible involvement of vanilloid receptors.[3]

Rutaecarpine exerts both antihypertensive and anti-platelet effects through stimulating the synthesis and release of CGRP in spontaneously hypertensive rats (SHR), and calcitonin gene-related peptide (CGRP)-mediated anti-platelet effect is related to inhibiting the release of platelet-derived tissue factor (TF), it may be a potential therapeutic agent for arterial thrombosis.[4,5]

Rutaecarpine has vasorelaxing action, it has direct paradoxical effects on intracellular calcium concentration of vascular smooth muscle and endothelial cells.[6]

Rutaecarpine enhances preservation with cardioplegia in guinea-pig hearts and that the protective effects of rutaecarpine are due to stimulation of endogenous CGRP release via activating vanilloid receptors.[7]

Rutaecarpine protects the gastric mucosa against injury induced by acetylsalicylic acid (ASA)and stress, and that the gastroprotective effect of rutaecarpine is related to a stimulation of endogenous calcitonin gene-related peptide (CGRP) release via activation of the vanilloid receptor.[8]

Rutaecarpine and evodiamine have inhibitory effect on LIGHT-induced migration in human monocytes, the inhibitory effect and the activation of chemokine receptor (CCR) 1, CCR2, ICAM-1, ERK, and p38 MAPK occurs via decreased ROS production and NADPH oxidase activation, indicates that they have the potential for use as an anti-atherosclerosis agent.[9]

Rutaecarpine inhibits ultraviolet A-induced reactive oxygen species generation, resulting in the enhanced expression of matrix metalloproteinase (MMP)-2 and MMP-9 in human skin cells, suggests that it may be useful in the prevention of ultraviolet A-induced photoaging.[10]

A single bolus intravenous injection of rutaecarpine from 20 mg/kg might cause immunosuppressive effects, and that rutaecarpine-induced immunosuppression may be mediated, at least in part, through the inhibition of cytokine production and cell cycle arrest in G0 + G1 phase, and causes possibly by mechanisms associated with metabolic activation.[11]

[Solvent]

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

[HPLC Method][12]

Mobile phase: n-Hexane–2-Propanol–Ethanol =70:20:10 ;

Flow rate: 0.7 ml/min;

Column temperature: Room Temperature;

The wave length of determination: 225 nm.

[Storage]

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

[References]

[1] Ueng Y F, Jan W C, Lin L C, *et al. Drug Metab. Dispos., 2002, 30(3):349-53.*

[2] Moon T C, Murakami M, Kudo I, *et al. Inflamm. Res., 1999, 48(12):621-5.*

[3] Kobayashi Y, Hoshikuma K, Nakano Y, *et al. Planta Med., 2001, 67(3):244-8.*

[4] Sheu J R, Hung W C, Wu C H, *et al. Brit. J. Haematol., 2000, 110(1):110-5.*

[5] Li D, Peng J, Xin H Y, *et al. Peptides, 2008, 29(10):1781-8.*

[6] Wang G J, Shan J, Pang P K, *et al. J. Pharm. Exp. Ther., 1996, 276(3):1016-21.*

[7] Hu C P, Xiao L, Deng H W, *et al. Planta Med., 2002, 68(8):705-9.*

[8] Wang Li, Hu CP, Deng PY, *et al. Planta Med., 2005, 71(5):416-9.*

[9] Heo S K, Yun H J, Yi H S, *et al. J.Cell. Biochem., 2009, 107(1):123-33.*

[10] Beak S M, Paek S Y, Lee Y S, *et al. Eur. J. Pharmacol., 2004, 498(498):19-25.*

[11] Jeon T W, Jin C H, Sang K L, *et al. Toxicol. Lett., 2006, 164(2):155-66.*

[12] Nguyen N V T, Lee K R, Yong J L, *et al. J. Pharm. Biomed.Anal., 2013, 81-82(7):151-9.*

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