Natural Products

ChemFaces

Eupatilin Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: Eupatilin

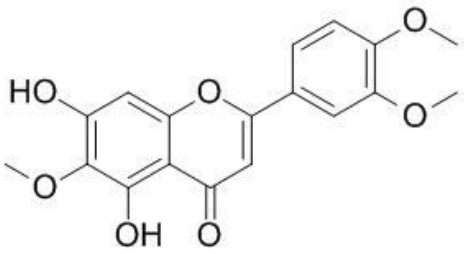
Catalog No.: CFN90190

Cas No.: 22368-21-4

Purity: >= 98%

M.F: $C_{18}H_{16}O_7$

M.W: 344.31



Physical Description: Yellow cryst.

Synonyms: 2-(3,4-Dimethoxyphenyl)-5,7-dihydroxy-6-methoxy-4H-1-benzopyran-4-one; 2-(3,4-Dimethoxyphenyl)-5,7-dihydroxy-6-methoxychromen-4-one; 5,7-Dihydroxy-3',4',6-trimethoxyflavone.

[Intended Use]

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

[Source]

The herbs of *Eupatorium semiserratum.*

[Biological Activity or Inhibitors]

Eupatilinthe has anti-proliferative effect in MCF10A- ras cells, is associated with its blockade of cell cycle progression which appears to be attributable in part to inhibition of ERK1/2 activation.[1]

Eupatilin attenuates bile acid-induced hepatocyte apoptosis by suppressing bile acid-induced kinase activation, it might be therapeutically efficacious in a variety of human liver diseases associated with cholestasis.[2]

Eupatilin and jaceosidin have effects on cytochrome p450 enzyme activities in human liver microsomes, have potential pharmacokinetic drug interactions in vivo due to inhibition of CYP1A2 and CYP2C9.[3]

Eupatilin is a potent anti-atherogenic agent that inhibits PDGF-BB-induced proliferation and migration in HASMCs as well as aortic sprouting, which is likely mediated through the attenuation of PI3K, MKK3/6, and MKK4 activation.[4]

Eupatilin suppresses oxidative damage and reciprocally enhances extracellular matrix production in articular chondrocytes, making eupatilin a promising therapeutic option for the treatment.[5]

Eupatilin improves the acute hepatic IRI by reducing inflammation and apoptosis, it is a promising therapeutic agent against acute IR-induced hepatic damage.[6]

Eupatilin protects against tumor necrosis factor-α-mediated inflammation inhuman umbilical vein endothelial cells.[7]

Eupatilin induces Sestrin2-dependent autophagy to prevent oxidative stress.[8]

[Solvent]

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

[HPLC Method][9]

Mobile phase: Acetonitrile : 0.2% Phosphoric acid H2O=37:63;

Flow rate: 1.0 ml/min;

Column temperature: 25 ℃;

The wave length of determination: 330 nm.

[Storage]

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

[References]

[1] Kim D H, Na H K, Oh T Y, *et al. Biochem. Pharmacol., 2004, 68(6):1081-7.*

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[3] Ji H Y, Kim S Y, Kim D K, *et al. Molecules, 2010, 15(9):6466-75*.

[4] Son J E, Lee E, Seo S G, *et al. Planta Med., 2013, 79(12):1009-16*.

[5] Jeong J H, Moon S J, Jhun J Y, *et al. Plos One, 2015, 10(6):e0130882.*

[6] Lee H M, Jang H J, Kim S S, *et al. Transpl P., 2016, 48(4):1226-33.*

[7] Yu K, Li X M, Xu X L, *et al. Int. J. Clin. Exp. Med., 2016, 8(12):22191-7.*

[8] Jegal K H, Ko H L, Sang M P, *et al. Apoptosis, 2016, 21(5):1-15.*

[9] Zhou Q, Sun L L, Jiang B, *et al. China Pharm., 2013, 24(47):4464-6.*

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