Natural Products

ChemFaces

Hydroxysafflor yellow A Datasheet

4th Edition (Revised in July, 2016)

[Product Information]

Name: Hydroxysafflor yellow A

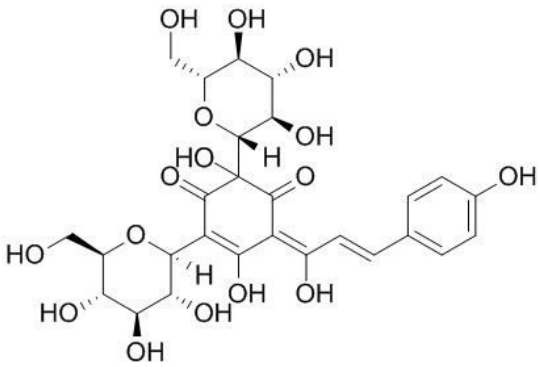
Catalog No.: CFN99950

Cas No.: 78281-02-4

Purity: > 98%

M.F: $C_{27}H_{32}O_{16}$

M.W: 612.53



Physical Description: Yellow powder

Synonyms:(6E)-2,5-dihydroxy-6-[(E)-1-hydroxy-3-(4-hydroxyphenyl)prop-2-enylidene]-2,4-bis[(2S,3R,4R,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)-2-oxanyl]cyclohex-4-ene-1,3-dione.

[Intended Use]

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

[Source]

The stigmas of *Carthamus tinctorius L.*

[Biological Activity or Inhibitors]

Hydroxysafflor yellow A (HSYA), isolated from the dried flower of Carthamus tinctorius L. , which is extensively used in traditional Chinese medicine to treat cirrhosis, can protect against chronic carbon tetrachloride-induced liver fibrosis, might be a promising antifibrotic agent in chronic liver disease.[1]

Hydroxysafflor yellow A can provide protection to H9c2 cardiomyocytes against A/R-induced apoptosis, and this protective effect largely depends on the upregulation of HO-1 expression through the PI3K/Akt/Nrf2 signaling pathway.[2]

Hydroxysafflor yellow A may provide neuroprotection against cerebral ischemia/ reperfusion injury through its suppression of inflammatory responses following focal ischemia reperfusion, and its antioxidant action by inhibiting the opening of mtPTP by a free radical scavenging action in the brain.[3-5]

Hydroxysafflor yellow A can enhance the survival of ECs under hypoxia, which may be correlated with its effect of upregulating the bcl-2/bax ratio and promoting HIF-1 alpha protein accumulation, which increases VEGF, these findings provide evidence for the mechanisms by which HSYA maintains EC survival under hypoxia.[6]

Hydroxysafflor yellow A suppresses inflammatory responses of BV2 microglia after oxygen-glucose deprivation, which is probably associated with the inhibition of the NF-κB signaling pathway and phosphorylation of p38.[7]

[Solvent]

Pyridine, Methanol, Ethanol, Hot water, etc.

[HPLC Method][8]

Mobile phase: Acetonitrile : 0.1% Phosphoric acid H2O=11:89;

Flow rate: 1.0 ml/min;

Column temperature: 35 ℃;

The wave length of determination: 403 nm.

[Storage]

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

[References]

[1] Zhang Y, Guo J, Dong H, *et al. Eur. J. Pharmacol., 2011, 660(2–3):438-44.*

[2] Liu S X, Zhang Y, Wang Y F, *et al. Int. J. Cardiol., 2012, 160(2):95-101.*

[3] Wei X, Liu H, Sun X, *et al. Neurosci. Lett., 2005, 386(1):58-62.*

[4] Tian J, Li G, Liu Z, *et al. Pharmacology, 2008, 82(2):121-6.*

[5] Ye S Y, Gao W Y. *Arch. Pharm. Res., 2008, 31(8):1010-5.*

[6] Ji D B, Zhu M C, Zhu B, *et al. J. Cardiovasc Pharm., 2008, 52(2):191-202.*

[7] Li J. *Neurosci. Lett., 2013, 535(1):51-6.*

[8]Tan S J, Chi J P, He Y Q, *et al. Pharm. J. Chinese Peoples Liberation Army, 2013(05): 464-5.*

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