

8-Prenylnaringenin Datasheet

4th Edition (Revised in July, 2016)

OH

[Product Information]

Name: 8-Prenylnaringenin

Catalog No.: CFN92016

Cas No.: 53846-50-7

Purity: > 95%

M.F: C₂₀H₂₀O₅

M.W: 340.4

Physical Description: Cryst.

Synonyms:(2S)-2,3-Dihydro-5,7-dihydroxy-2-(4-hydroxyphenyl)-8-(3-methyl-2-butenyl)-4

H-1-benzopyran-4-one.

[Intended Use]

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

[Source]

The herbs of *Humulus Iupulus*.

[Biological Activity or Inhibitors]

8-Prenylnaringenin is a phytoestrogen, it exhibits high estrogenic activity.[1]

8-Prenylnaringenin can inhibit angiogenesis induced by basic fibroblast growth factor

(bFGF), vascular endothelial growth factor (VEGF), or the synergistic effect of the two

cytokines in combination, with an IC(50) of between 3 and 10 microM,

8-prenylnaringenin has potential therapeutic applications for diseases in which

angiogenesis is an important component.[2]

Treatment with 8-prenylnaringenin results in very good biomechanical properties and

shows an increased bone mineral density (BMD), 8-prenylnaringenin may be a safe

alternative for hormone replacement therapy (HRT) to prevent osteoporosis. [3]

8-Prenylnaringenin is an inhibitor of multidrug resistance (MDR)-associated transporters,

P-glycoprotein and MRP1, but 8-prenylnaringenin is not able to modulate MDR in human

adenocarcinoma cell line .[4]

8-Prenylnaringenin can inhibit estrogen receptor-alpha mediated cell growth and induce

apoptosis in MCF-7 breast cancer cells.^[5]

8-Prenylnaringenin exerts anti-aggregatory and anti-adhesive effects on human platelets,

independently of estrogen receptors, acting as an inhibitor of multiple proteins essential

for the morphological and biochemical transformations that occur during platelet activation

and aggregation, it may represent a useful tool in the therapy and prevention of vascular

diseases associated with platelet aggregation, such as atherosclerosis, myocardial

infarction, coronary artery disease, and thrombosis. [6]

[Solvent]

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

[HPLC Method]^[7]

Mobile phase: Acetonitrile-Phosphoric acid aqueous solution (pH 1.6), gradient elution;

Flow rate: 1.5 ml/min;

Column temperature: Room Temperature;

The wave length of determination: 314 nm.

[Storage]

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

[References]

[1] Milligan S R, Kalita J C, Pocock V, et al. J. Clin. Endocr. Metab., 2000, 85(12):4912-5.

[2] Pepper M S, Hazel S J, Hümpel M, et al. J. Cell. Physiol., 2004, 199(1):98-107.

[3] Sehmisch S, Hammer F, Christoffel J, et al. Planta Med., 2008, 74(8):794-801.

[4] Wesołowska O, Wiśniewski J, Sroda K, et al. Eur. J. Pharmacol., 2010, 644(1-3):32-40.

[5] Brunelli E, Minassi A, Appendino G, et al. J. Steroid Biochem., 2007, 107(3-5):140-8.

[6] Di V C, Bertoni A, Nalin M, et al. Biochim. Et Biophy. Acta, 2012, 1820(11):1724-33.

[7] Kao T H, Wu G Y. Food Chem., 2013, 141(2):1218-26.

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