Naringin

Cat. No.: HY-N0153

CAS No.: 10236-47-2

Molecular Formula: $C_{27}H_{32}O_{14}$

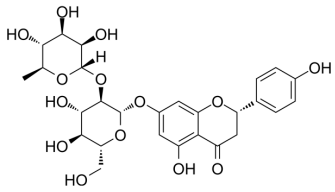
Molecular Weight: 580.53

Target: Cytochrome P450; Autophagy; Mitophagy; Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (215.32 mM; Need ultrasonic)

H_2O : 1 mg/mL (1.72 mM; ultrasonic and warming and heat to 80°C)

Ethanol : < 1 mg/mL (ultrasonic;warming;heat to 80°C) (insoluble)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7226 mL	8.6128 mL	17.2256 mL
	5 mM	0.3445 mL	1.7226 mL	3.4451 mL
	10 mM	0.1723 mL	0.8613 mL	1.7226 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 40 mg/mL (68.90 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.08 mg/mL (3.58 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.58 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.58 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Naringin is a major flavanone glycoside obtained from tomatoes, grapefruits, and many other citrus fruits. Naringin exhibits biological properties such as antioxidant, anti-inflammatory, and antiapoptotic activities.

In Vitro	Naringin suppresses NF-κ B signaling pathway activation. Naringenin inhibits high glucose-induced proliferation, inflammatory reaction and oxidative stress injury in HBZY-1 cells[1]. Naringin inhibits AGS cancer cell proliferation in a dose- and time-dependent manner. Phosphorylation of PI3K and its activated downstream targets p-Akt and p-mTOR are significantly decreased at 2 mM in Naringin-treated AGS cells. Naringin induces autophagic cell death in AGS cells. Naringin activated the autophagy related protein in AGS cells[2]. Naringin protects PC12 cells from 3-NP neurotoxicity. The lactate dehydrogenase release is decreased upon naringin treatment in 3-NP-induced PC12 cells. Naringin treatment enhances the antioxidant defense by increasing the activities of enzymatic antioxidants and the level of reduced glutathione[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Treatment with naringin significantly alleviates renal injury in diabetic rats and increases diabetic rats body weight significantly. Administration of naringin effectively alleviates the collagen deposition and renal interstitial fibrosis in diabetic rats. Treatment with naringin could result in decreased levels of ROS and MDA and increased activities of SOD and GSH-Px[1]. Oral administration of naringin significantly improves the learning and memory abilities. Naringin significantly enhances insulin signaling pathway[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	HBZY-1 cells are plated into 96-well plates and pretreated with various concentrations(1, 5, 10, 25, 50, 100 μM) of naringin for 2 h. Then cells are treated with 30 mM glucose for 24 h. The control group is added sterile normal saline in the same volume. After treatment, all the wells are incubated with 20 μL of 5 mg/ml MTT for 4 h at 37°C. Subsequently, 100 μL of DMSO are used to dissolve the formed formazan crystals after removal of the supernatant. The result is recorded at 490 nm on a microplate reader[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1][4]	Rats: The rats are randomly divided into six groups: control, naringin (80 mg/kg), STZ, STZ+naringin (20 mg/kg), STZ+naringin (40 mg/kg), STZ+naringin(80 mg/kg). The rats in the STZ and STZ+naringin groups are intraperitoneally injected with STZ (65 mg/kg). The control and naringin groups are intraperitoneally injected with 0.1 M citrate buffer of same volume. After injection of STZ for 3 and 5 days, blood glucose levels are measured by tail vein puncture blood sampling[1]. Mice: Sixty 4-week-old male mice are randomized into four groups and fed for 20 weeks with either control diet or high-fat diet chow. Mice are dosed with 100 mg/kg of naringin daily. Mice body weight and food intake are weekly measured. Following behavioral assessment, animals are deeply anesthetized with isoflurane and sacrificed by decapitation after fasting for at least 5 h. Their plasma is collected for further analysis[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Front Pharmacol. 2021 Jul 15;12:696135.
- J Zhejiang Univ Sci B. 2023 Mar 15;24(3):221-231.
- Environ Toxicol. 2022 Feb 18.
- Mol Med Rep. 2024 Feb;29(2):26.
- Mol Med Rep. 2021 Nov;24(5):772.

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REFERENCES

[1]. Chen F, et al. Naringin Alleviates Diabetic Kidney Disease through Inhibiting Oxidative Stress and Inflammatory Reaction. PLoS One. 2015 Nov 30;10(11):e0143868.

[2]. Raha S, et al. Naringin induces autophagy-mediated growth inhibition by downregulating the PI3K/Akt/mTOR cascade via activation of MAPK pathways in AGS cancer cells. Int J Oncol. 2015 Sep;47(3):1061-9.

[3]. Kulasekaran G, et al. Neuroprotective efficacy of naringin on 3-nitropropionic acid-induced mitochondrial dysfunction through the modulation of Nrf2 signaling pathway in PC12 cells. Mol Cell Biochem. 2015 Nov;409(1-2):199-211.

[4]. Wang D, et al. Naringin Improves Neuronal Insulin Signaling, Brain Mitochondrial Function, and Cognitive Function in High-Fat Diet-Induced Obese Mice. Cell Mol Neurobiol. 2015 Oct;35(7):1061-71.

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