

Product Data Sheet

Product Name: (±)-Naringenin

Cat. No.: GC39271

Chemical Properties

Cas. No. 67604-48-2

SMILES O=C1CC(C2=CC=C(O)C=C2)OC3=CC(O)=CC(O)=C13

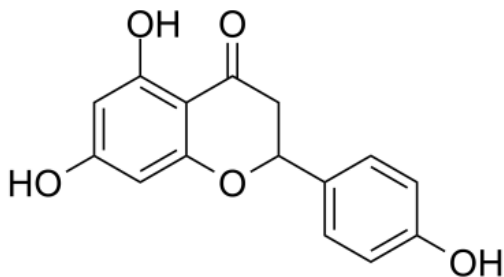
Formula $C_{15}H_{12}O_5$ M.Wt 272.25

Solubility DMF: 10 mg/ml, DMF:PBS(pH 7.2) (1:1): 0.50 mg/ml, DMSO: 5 mg/ml, Ethanol: 2.5 mg/ml
Storage Store at RT

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Caution: Product has not been fully validated for medical applications. For research use only.

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Cell experiment [1]:	
Cell lines	MCF-7 cells (human breast adenocarcinoma cell line)
Preparation Method	MCF-7 cells were maintained in RPMI-1640 medium supplemented with 10% fetal bovine serum (FBS) and 1% penicillin-streptomycin at 37°C, 5% CO₂. MCF-7 cells were treated with (±)-Naringenin at 50-150µM for 24 hours, then cell viability was assessed by MTT, colony formation was assessed after 10-day recovery, migration was assessed by wound healing, cell cycle was assessed by PI flow cytometry, apoptosis was assessed by AO/EB and DAPI staining and Annexin V/PI flow cytometry, ROS was assessed by DCFDA flow cytometry, and autophagy/apoptosis proteins were assessed by Western blot (LC3-II, p62, C-PARP) and LC3-II/LAMP1 confocal co-localization; MCF-7 cells were also treated with 150µM (±)-Naringenin plus 3-MA for 24 hours and analysed by Western blot for C-PARP, LC3-II and p62.
Reaction Conditions	50-150µM; 24h
Applications	(±)-Naringenin decreased MCF-7 cell viability and colony number, attenuated wound closure, arrested cells in G0/G1 and S phases, increased early and late apoptotic and necrotic populations, induced chromatin condensation, raised intracellular ROS median fluorescence intensity, upregulated C-PARP and LC3-II, degraded p62, and increased LC3-II-LAMP1 co-localization; co-treatment with 3-MA reduced C-PARP and LC3-II and restored p62.
Animal experiment [2]:	
Animal models	APP/PS1 transgenic C57BL/6 mice

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Preparation Method	Mice were intragastrically administered 50mg/kg/day ($\pm$)-Naringenin (suspended in 0.5% sodium carboxymethyl cellulose) once daily for 3 months; after treatment, novel object recognition test and Morris water maze test were performed, then brains were collected for immunohistochemistry/immunofluorescence staining (6E10, IBA-1, GFAP), ELISA (A β 40, A β 42, TNF- α , IL-1 β), qPCR (TNF- α , IL-1 β) and Western blot (APP, BACE1, Iba-1, GFAP, p-p38, p38, p-JNK, JNK).
Dosage form	50mg/kg; intragastric; once daily for 3 months
Applications	($\pm$)-Naringenin improved recognition index in novel object recognition test and decreased escape latency on day 2 and day 3 in Morris water maze test. ($\pm$)-Naringenin reduced 6E10-positive A β plaque area in hippocampus and cerebral cortex, decreased soluble A β 42 in hippocampus, downregulated BACE1 protein in hippocampus, reduced IIBA-1 and GFAP positive area and protein in hippocampus and cortex, reduced TNF- α mRNA and IL-1 β mRNA in hippocampus, and decreased TNF- α and IL-1 β protein in hippocampus. ($\pm$)-Naringenin reduced p-p38/p38 and p-JNK/JNK ratios in hippocampus.
References: [1] Mir SA, Bhat BA, Shenoy PS, et al. Naringenin inhibits cellular proliferation, arrests cell cycle and induces pro-apoptotic autophagy in MCF-7 breast cancer cells. J Cell Mol Med. 2025;29:e70747. [2] Zhu Y, Guo X, Li S, et al. Naringenin ameliorates amyloid-β pathology and neuroinflammation in Alzheimer's disease. Commun Biol. 2024 Jul 28;7(1):912.	

Background

($\pm$)-Naringenin is a flavonoid compound with multiple pharmacological activities including antioxidant,

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anti-inflammatory, anticancer, cardioprotective and hepatoprotective effects. (±)-Naringenin scavenges reactive oxygen species and inhibits lipid peroxidation. (±)-Naringenin reduces the production of pro-inflammatory cytokines such as TNF- α and IL-1 β and inflammatory mediators such as COX-2 and iNOS through inhibition of NF- κ B and MAPK signaling pathways. (±)-Naringenin regulates PI3K/Akt and p53 signaling pathways to induce tumor cell apoptosis and inhibit proliferation. (±)-Naringenin can be used in studies related to prostate cancer, metabolic diseases, neurodegenerative diseases and COVID-19^[1-4].

In vitro, treatment of MCF-7 human breast cancer cells with 50-150 μ M (±)-Naringenin for 24 hours inhibited cell proliferation and colony formation, attenuated cell migration, arrested cells in G0/G1 and S phases, increased early and late apoptotic cell proportions and elevated C-PARP expression^[5]. Treatment of HepG2 cells with 1-9 μ g/mL (±)-Naringenin for 24-48 hours inhibited cell proliferation^[6]. Treatment of H1299 and A549 cells with 25-500 μ M (±)-Naringenin for 24 hours reduced cell viability, arrested cell cycle in G2/M phase and downregulated CDK1 and cyclin B1, increased intracellular ROS generation and decreased mitochondrial membrane potential^[7].

In vivo, daily intragastric administration of APP/PS1 transgenic C57BL/6 mice with 50mg/kg/day (±)-Naringenin for 3 months improved novel object recognition ability, learning ability and memory ability, and reduced 6E10-positive A β plaque area in hippocampus and cerebral cortex^[8]. Feeding of C57BL/6 mice with methionine-choline deficient diet (MCD) combined with daily intragastric administration of 50-100mg/kg/day (±)-Naringenin for 7 consecutive days recovered body weight and liver weight, reduced plasma ALT and AST levels, and decreased hepatic lipid droplet area and triglyceride content^[9]. Daily intragastric administration of C57BL/KsJ db/+ gestational diabetes mice with 100mg/kg/day (±)-Naringenin for 4 weeks (until gestational day 18) reduced maternal body weight and body weight gain at GD18, decreased serum glucose levels at GD9 and GD18, improved glucose tolerance and insulin tolerance, reduced fasting insulin levels and HOMA-IR, and increased QUICKI values and adiponectin levels^[10].

References:

[1] Shin JH, Shin SH. A Comprehensive Review of Naringenin, a Promising Phytochemical with Therapeutic Potential. J Microbiol Biotechnol. 2024 Dec;34(12):2371-2385.

[2] Arafah A, Rehman MU, Mir TM, et al. Multi-Therapeutic Potential of Naringenin (4',5,7-

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[3] Motallebi M, Bhia M, Rajani HF, et al. Naringenin: A potential flavonoid phytochemical for cancer therapy. Life Sci. 2022 Sep 15;305:120752.

[4] Atoki AV, Aja PM, Shinkafi TS, et al. Naringenin: its chemistry and roles in neuroprotection. Nutr Neurosci. 2024 Jun;27(6):637-666.

[5] Mir SA, Bhat BA, Shenoy PS, et al. Naringenin Inhibits Cellular Proliferation, Arrests Cell Cycle and Induces Pro-Apoptotic Autophagy in MCF-7 Breast Cancer Cells. J Cell Mol Med. 2025;29:e70747.

[6] Elnawasany S, Haggag YA, Shalaby SM, et al. Anti-cancer effect of nano-encapsulated boswellic acids, curcumin and naringenin against HepG-2 cell line. BMC Complement Med Ther. 2023 Jul 29;23(1):270.

[7] Chang TM, Chi MC, Chiang YC, et al. Promotion of ROS-mediated apoptosis, G2/M arrest, and autophagy by naringenin in non-small cell lung cancer. Int J Biol Sci. 2024;20(3):1093-1109.

[8] Zhu Y, Guo X, Li S, et al. Naringenin ameliorates amyloid- β pathology and neuroinflammation in Alzheimer's disease. Commun Biol. 2024 Jul 28;7(1):912.

[9] Wang Q, Ou Y, Hu G, et al. Naringenin attenuates non-alcoholic fatty liver disease by down-regulating the NLRP3/NF- κ B pathway in mice. Br J Pharmacol. 2020;177(8):1806-1821.

[10] Li S, Zhang Y, Sun Y, et al. Naringenin improves insulin sensitivity in gestational diabetes mellitus mice through AMPK. Nutr Diabetes. 2019 Oct 7;9:28.

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