

Product Data Sheet

Product Name: Dihydromyricetin

Cat. No.: GN10583

Chemical Properties

Cas. No. 27200-12-0

Chemical Name (2R,3R)-3,5,7-trihydroxy-2-(3,4,5-trihydroxyphenyl)-2,3-dihydrochromen-4-one

SMILES C1=C(C=C(C(=C1O)O)O)C2C(C(=O)C3=C(C=C(C(=C3O2)O)O)O

Formula $C_{15}H_{12}O_8$

M.Wt 320.25

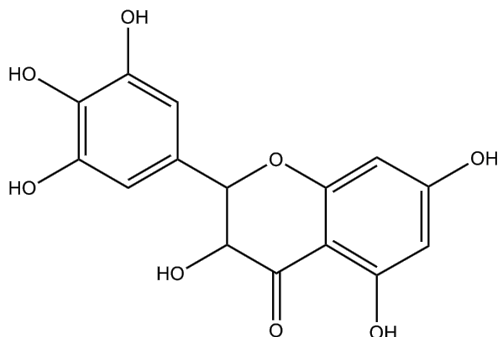
Solubility $\geq 14.6\text{mg/mL}$ in DMSO

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

Cell experiment [1]:

Cell lines MG63 cells

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

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The cells distributed in 100mm dishes at a density of 3×10^7 cells per dish were allowed to attain confluence. After attaining confluence cells were pre-treated with various doses of Dihydromyricetin for 48h or with dimethyl sulfoxide as control. Following incubation, the cells were treated with hydrogen peroxide to induce oxidative stress.

Reaction Conditions 5, 10, 15; 20; 25; 30 μ M; 48h

Applications Pre-treatment of MG63 cells with Dihydromyricetin for 48h prevented hydrogen peroxide induced reduction in cell viability. The reduction in cell viability caused by 100 μ M concentration of hydrogen peroxide was completely prevented on incubation with 30 μ M dose of Dihydromyricetin.

Animal experiment [2]:

Animal models Cute liver injury

Preparation Method Dihydromyricetin was dissolved in 0.5% sodium carboxymethylcellulose (CMC-Na) diluted in ultrapure water, to a final concentration of 37.5mg/mL. Then, mice were treated orally with Dihydromyricetin (150mg/kg; once per day, 4d) 2h after carbon tetrachloride(CCl₄) treatment. JNK inhibitor SP600125 (50mg/kg) was administered by intraperitoneal injection 1h prior to Dihydromyricetin treatment. Mice were dedicated on days 1, 2, 3, 5 and 7 after CCl₄ treatment to examine a series of indicators of liver injury and regeneration.

Dosage form 150mg/kg; po; 4d

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Applications Dihydromyricetin showed a strong anti-inflammatory effect on CCl₄-induced liver injury in mice, Dihydromyricetin could significantly decrease serum ALT, AST, IL-1 β , IL-6 and TNF- α and increase serum albumin, SOD and liver SOD after CCl₄ treatment.

References:

- [1]. Wang Y, Wang W, Qiu E. Protection of oxidative stress induced apoptosis in osteosarcoma cells by Dihydromyricetin through down-regulation of caspase activation and up-regulation of Bcl-2[J]. Saudi Journal of Biological Sciences, 2017, 24(4): 837-842. Xu P, Zhang M, Wang X, et al. Antioxidative effect of Quetiapine on acute ultraviolet-B-induced skin and HaCaT cell damage[J]. International Journal of Molecular Sciences, 2018, 19(4): 953.
- [2]. Xie J, Liu J, Chen T M, et al. Dihydromyricetin alleviates carbon tetrachloride-induced acute liver injury via JNK-dependent mechanism in mice[J]. World Journal of Gastroenterology: WJG, 2015, 21(18): 5473.

Background

Dihydromyricetin is a natural flavanone isolated from *A. grossedentata* and *H. dulcis* that has antioxidant, antiproliferative, anti-apoptotic, and anti-alcohol intoxication properties^[1].

Dihydromyricetin (5, 10, 15; 20; 25; 30 μ M; 48h) pre-treated MG63 cells for 48 hours, preventing hydrogen peroxide-induced reduction in cell viability. 30 μ M of Dihydromyricetin during incubation fully blocked the decrease in cell viability caused by 100 μ M hydrogen peroxide^[2]. Dihydromyricetin (12.5, 25, 50 μ M; 24h) increased the p-STAT3-dependent autophagy by generating ROS-signaling pathways in head and neck squamous cell carcinoma. and inhibiting autophagy could enhance Dihydromyricetin-induced apoptosis in head and neck squamous cell carcinoma^[3].

Dihydromyricetin (150mg/kg; po; 4d) showed a strong anti-inflammatory effect on CCl₄-induced liver injury in mice, Dihydromyricetin could significantly decrease serum ALT, AST, IL-1 β , IL-6 and TNF- α and increase serum albumin, SOD and liver SOD after CCl₄ treatment^[4]. Dihydromyricetin (10mg/kg; ip; 27d) treatment significantly reduced the total number of inflammatory cells in sputum including eosinophils, neutrophils, lymphocytes, and macrophages, as well as the levels of IL-4, IL-5, and IL-13 in BAL fluids, and the secretion of OVA-specific IgE and IgG1 in serum in mice with allergic

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asthma^[5].

- [1]. Liu D, Mao Y, Ding L, et al. Dihydromyricetin: A review on identification and quantification methods, biological activities, chemical stability, metabolism and approaches to enhance its bioavailability[J]. Trends in Food Science & Technology, 2019, 91: 586-597.
- [2]. Wang Y, Wang W, Qiu E. Protection of oxidative stress induced apoptosis in osteosarcoma cells by Dihydromyricetin through down-regulation of caspase activation and up-regulation of Bcl-2[J]. Saudi Journal of Biological Sciences, 2017, 24(4): 837-842. Xu P, Zhang M, Wang X, et al. Antioxidative effect of Quetiapine on acute ultraviolet-B-induced skin and HaCaT cell damage[J]. International Journal of Molecular Sciences, 2018, 19(4): 953.
- [3]. Fan T F, Wu T F, Bu L L, et al. Dihydromyricetin promotes autophagy and apoptosis through ROS-STAT3 signaling in head and neck squamous cell carcinoma[J]. Oncotarget, 2016, 7(37): 59691.
- [4]. Xie J, Liu J, Chen T M, et al. Dihydromyricetin alleviates carbon tetrachloride-induced acute liver injury via JNK-dependent mechanism in mice[J]. World Journal of Gastroenterology: WJG, 2015, 21(18): 5473.
- [5]. Xu B, Huang S, Wang C, et al. Anti-inflammatory effects of Dihydromyricetin in a mouse model of asthma[J]. Molecular Medicine Reports, 2017, 15(6): 3674-3680.

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