Hesperidin methylchalcone

Cat. No.: HY-126382

CAS No.: 24292-52-2

Molecular Formula: $C_{29}H_{36}O_{15}$

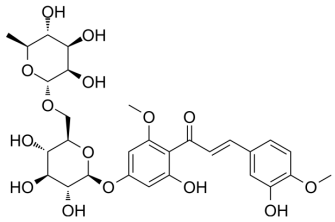
Molecular Weight: 624.59

Target: NF-κB

Pathway: NF-κB

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (80.05 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.6011 mL	8.0053 mL	16.0105 mL
5 mM	0.3202 mL	1.6011 mL	3.2021 mL
10 mM	0.1601 mL	0.8005 mL	1.6011 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (3.33 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.33 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Hesperidin methylchalcone (Hesperidin methyl chalcone) is an orally active flavonoid that has analgesic, anti-inflammatory and antioxidant properties. Hesperidin methylchalcone exhibits vasoprotective activity. Hesperidin methylchalcone inhibits oxidative stress, cytokine production and NF-κB activation. Hesperidin methylchalcone can be used for the research of gout disease[1][2].

IC_{50} & Target

NF-κB

In Vivo

Hesperidin methylchalcone (0.03-3 mg/kg, i.p., once time) inhibits the elevation of renal dysfunction markers (urea and creatinine) induced by SDCF in mice in a dose-dependent manner[1].
Hesperidin methylchalcone (3 mg/kg, i.p., once time) can effectively counteract the oxidative stress induced by SDCF in

renal tissue of mice[1].
Hesperidin methylchalcone (3 mg/kg, i.p., once time) reduces SDCF-induced renal histopathology, swelling and tubular cells cytotoxicity in mice[1].
Hesperidin methylchalcone (3-30 mg/kg, p.o., once time) reduces in a dose-dependent manner the MSU-induced hyperalgesia, edema, and leukocyte infiltration in mice[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Swiss mice[1]
Dosage:	3 mg/kg
Administration:	i.p., once time
Result:	Increased the levels of FRAP and ABTS, and reduced concentration of TBARS. Reduced IL-6, IFN-γ, and IL-33 levels in plasma. Increased the FRAP, ABTS, and GSH levels, reduced Keap1, and inhibited lipid peroxidation in kidneys. Reduced IL-1β, IL-6, IFN-γ, and IL-33, and increased IL-10 levels in renal tissue. Increased Nrf2, Ho-1 and Nqo1 mRNA expression.

Animal Model:	Swiss mice[2]
Dosage:	3-30 mg/kg
Administration:	p.o., once time
Result:	Inhibited MSU-induced infiltration of LysM-eGFP$^+$ cells and synovitis at the dose of 30 mg/kg. Reduced IL-1β, TNF-α, IL-6, and IL-10 levels, and increased GSH, FRAP, and ABTS at the dose of 30 mg/kg. Inhibited MSU-induced NF-κB activation, gp91phox and NLRP3 inflammasome components mRNA expression in vivo (NLRP3, ASC, pro-caspase-1, and pro-IL-1 β). Induced Nrf2/HO-1 mRNA expression.

CUSTOMER VALIDATION

- Biochim Biophys Acta Mol Basis Dis. 2023 Feb;1869(2):166620.

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REFERENCES

[1]. Bussmann AJC,et al. The Flavonoid Hesperidin Methyl Chalcone Targets Cytokines and Oxidative Stress to Reduce Diclofenac-Induced Acute Renal Injury: Contribution of the Nrf2 Redox-Sensitive Pathway. Antioxidants (Basel). 2022 Jun 27;11(7):1261.

[2]. Ruiz-Miyazawa KW, et al. Hesperidin Methylchalcone Suppresses Experimental Gout Arthritis in Mice by Inhibiting NF-κB Activation. J Agric Food Chem. 2018 Jun 27;66(25):6269-6280.

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

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