Kaempferide

Cat. No.: HY-15449

CAS No.: 491-54-3

Molecular Formula: $C_{16}H_{12}O_6$

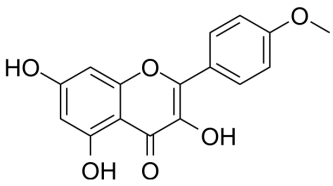
Molecular Weight: 300.26

Target: Estrogen Receptor/ERR; Autophagy; Bacterial; Influenza Virus; Apoptosis; PI3K; Akt; GSK-3; FOXO; β-catenin

Pathway: Vitamin D Related/Nuclear Receptor; Autophagy; Anti-infection; Apoptosis; PI3K/Akt/mTOR; Stem Cell/Wnt; Metabolic Enzyme/Protease

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 20 mg/mL (66.61 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.3304 mL	16.6522 mL	33.3045 mL
	5 mM	0.6661 mL	3.3304 mL	6.6609 mL
	10 mM	0.3330 mL	1.6652 mL	3.3304 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 mg/mL (6.66 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Kaempferide is an orally active flavonol isolated from Hippophae rhamnoides L. Kaempferide has anticancer, anti-inflammatory, antioxidant, antidiabetic, antiobesity, antihypertensive, and neuroprotective activities. Kaempferide induces apoptosis. Kaempferide promotes osteogenesis through antioxidants and can be used in osteoporosis research[1][2][3][4][5][6].

In Vitro

Kaempferid is toxic in HCC cell lines (HepG2:IC_{50} = 27.94 μM; Huh7: IC_{50} = 25.65 μM; N1S1: IC_{50} = 15.18 μM)[1].
Kaempferid (5, 20 μM; 48 h) reduces lipid accumulation and oxidative stress in HepG2 cells induced by oral acid (OA) (0.5 mM) (HY-N1446)[2].
Kaempferide promotes osteogenesis through the FoxO1/β-catenin signaling pathway [3].Kaempferide (10-15 μM; 24 h) is toxic in HeLa cells with an IC_{50} of 16 μM and can induce apoptosis[6].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	HepG2
Concentration:	5 μM, 10 μM, 20 μM . Before treatment with OA (HY-N1446) (0.5 mM; 48 h)
Incubation Time:	48 h
Result:	Lowered the expression of proteins related to fat production, including sterol regulatory element-binding protein 1 (SREBP1), fatty acid synthase (FAS), and stearoyl-CoA desaturase 1 (SCD-1). Reduced the expression of two adipogenic transcription factors, peroxisome proliferator-activated receptor gamma (PPARγ) and CCAAT enhancer-binding protein β (C/EBPβ). Enhanced the expression of heme oxygenase-1 (HO-1) and nuclear factor erythroid 2-related factor 2 (Nrf2).

In Vivo

Kaempferid (25 mg/kg; IV; three times a week) has anticancer activity in SD (Sprague Dawley) rats[1].Kaempferide (10 mg/kg; supplemented in daily diet, once daily for16 weeks) ameliorates oxidative stress and inflammation in obese C57BL/6J mice by inhibiting the TLR4/i-κBα/NF-κB pathway and can alleviate Obesity and glucose and lipid metabolism disorders[4]. Kaempferide (0.1-0.3 mg/kg; injection, single dose) alleviates myocardial ischemia/reperfusion injury by activating PI3K/Akt/GSK-3β pathway in Sprague Dawley (I/R)-induced rats[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	High-fat diet male C57BL/6J mice model[4]
Dosage:	10 mg/kg
Administration:	Supplemented in daily diet, once daily for16 weeks
Result:	Reduced the weight, organ weight, and index of mice. Lowered the levels of glycolipids in mouse serum. Decreased the expression levels of inflammatory-related genes, including NF-κB, IL-6, ICAM-1, VCAM-1, and TNF-α.

Animal Model:	Ischemia/Reperfusion (I/R) SD rat model[5].
Dosage:	0.1 mg/kg, 0.3 mg/kg, 3 mg/kg
Administration:	Injection, Single dose. Before the I/R injury induced by Coronary Artery Ligation (CAL) in SD rats.
Result:	Significantly improved heart function, reduced myocardial injury by reducing myocardial enzyme levels, and dose-dependently reduced the area of myocardial infarction in rats. Significantly decreased serum levels of TNF-α, IL-6, C-reactive protein (CRP), MDA, and ROS, while increasing serum levels of SOD. Downregulated the expression levels of nuclear factor erythroid 2-related factor 2 (Nrf2) and cleaved caspase-3, and upregulated the phosphorylation expression levels of phospho-Akt (p-Akt) and phospho-glycogen synthase kinase-3β (p-GSK-3β).

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2021 Jan;11(1):143-155.
- Evid-Based Compl Alt. 26 Aug 2021.

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REFERENCES

[1]. Chandrababu G, et al. Kaempferide exhibits an anticancer effect against hepatocellular carcinoma in vitro and in vivo[J]. Naunyn-Schmiedeberg's Archives of Pharmacology, 2023: 1-7.

[2]. Tie F, et al. Kaempferol and kaempferide attenuate oleic acid-induced lipid accumulation and oxidative stress in HepG2 cells[J]. International Journal of Molecular Sciences, 2021, 22(16): 8847.

[3]. Ma X, et al. Kaempferide enhances antioxidant capacity to promote osteogenesis through FoxO1/β-catenin signaling pathway[J]. European Journal of Pharmacology, 2021, 911: 174555.

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[5]. Wang D, et al. Kaempferide Protects against Myocardial Ischemia/Reperfusion Injury through Activation of the PI3K/Akt/GSK-3β Pathway. Mediators Inflamm. 2017;2017:5278218.

[6]. Nath L R, et al. Kaempferide, the most active among the four flavonoids isolated and characterized from Chromolaena odorata, induces apoptosis in cervical cancer cells while being pharmacologically safe[J]. RSC advances, 2015, 5(122): 100912-100922.

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