Kaempferitrin

Cat. No.: HY-N0628

CAS No.: 482-38-2

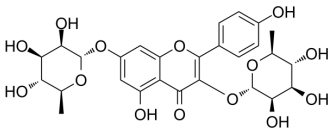
Molecular Formula: $C_{27}H_{30}O_{14}$

Molecular Weight: 578.52

Target: Insulin Receptor

Pathway: Protein Tyrosine Kinase/RTK

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (43.21 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7285 mL	8.6427 mL	17.2855 mL
	5 mM	0.3457 mL	1.7285 mL	3.4571 mL
	10 mM	0.1729 mL	0.8643 mL	1.7285 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 (3.60 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.60 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.60 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Kaempferitrin is a natural flavonoid, possesses antinociceptive, anti-inflammatory, anti-diabetic, antitumoral and chemopreventive effects, and activates insulin signaling pathway.

IC_{50} & Target

Insulin Receptor[1]

In Vitro

Kaempferitrin activates insulin signaling pathway. Kaempferitrin causes survival rates higher than 90% at 1-20 μM in matured 3T3-L1 adipocyte, and the survival rates decline rapidly at 25 and 50 μM. Kaempferitrin (15 μM) increases insulin receptor beta tyrosine phosphorylation and tyrosine phosphorylation of the insulin receptor substrate 1, and such effects are similar to that of 10 nM insulin. Kaempferitrin (15 μM) also stimulates akt phosphorylation on ser473, and the stimulation

	can be blocked by a PI3-K inhibitor wortmannin. Kaempferitrin potently exerts the translocation of GLUT4 to the membrane of adipocytes at 15 μM, and this is suppressed by wortmannin. In addition, Kaempferitrin increases the total levels of Glu4 protein in differentiated cells and secreted adiponectin in mature 3T3-L1 adipocytes[1]. Kaempferitrin is cytotoxic to human cancer cells such as HeLa and MDA-MB231 cells, with IC_{50}s of 45 ± 2.6 and 65 ± 2.6 μM, and shows low toxic effects on non-tumorigenic cells. Kaempferitrin (45 μM) induces apoptosis of HeLa cells after treatment for 24 and 48 h, and causes reactive oxygen species (ROS) generation in HeLa cells. Furthermore, Kaempferitrin (45 μM) exerts G1 arrest, causes the expression of proteins associated with intrinsic pathway of apoptosis and activates caspase 3 in HeLa cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Kaempferitrin (2.5, 10 and 25 mg/kg, i.p.) markedly suppresses the growth of tumor by 40%, 87% and 97%, and decreases tumor weight by 37%, 81% and 95%, respectively in nu/nu mice bearing HeLa tumor. Kaempferitrin also inhibits cell proliferation and extends life span in mice bearing tumor[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Viability assay is carried out by MTT assay. Preconfluent 3T3-L1 preadipocytes are seeded to reach confluence. Kaempferitrin is added in replacement of insulin immediately after confluence (day 0), and the viability is measured 3 h after addition of the compound. Another cell viability assay is performed at day 8. For kaempferitrin combined with insulin treatment, various concentrations of kaempferitrin are added simultaneously with 0.2 nM insulin from day 0 to day 8. Cells without insulin or kaempferitrin treatment during differentiation (day 0 to day 8) are used as control. Finally, to verify the cell viability in matured 3T3-L1 cells treated with insulin or kaempferitrin for 24-48 h, MTT are incubated for 3 h at the end of the 24 h and 48 h period of compound treatments, and survival rates calculated and compared to that without insulin or kaempferitrin[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	The nu/nu mice are injected subcutaneously in their backs with HeLa cells (1.5×10^6). Four hours after tumor implantation, groups of five mice receive doses of Kaempferitrin between 2.5 to 25 mg/kg, dissolved in 0.1 mL of 0.9% saline solution, cisplatin (CDDP) 1 mg/kg or paclitaxel (PCX) 1 mg/kg injected intraperitoneally daily over a period of 32 days. The animal control group received 0.1 mL of vehicle solution. Tumors are measured using a vernier caliper, and their size in mm^3 is calculated Tumor volume = lenght × width × $height^2$. At the end of the experiments, animals are sacrificed and their tumors are excised and weighed[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Biomed Pharmacother. 2020 Sep;129:110369.

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REFERENCES

[1]. Tzeng YM, et al. Kaempferitrin activates the insulin signaling pathway and stimulates secretion of adiponectin in 3T3-L1 adipocytes. Eur J Pharmacol. 2009 Apr 1;607(1-3):27-34.

[2]. Alonso-Castro AJ, et al. Kaempferitrin induces apoptosis via intrinsic pathway in HeLa cells and exerts antitumor effects. J Ethnopharmacol. 2013 Jan 30;145(2):476-89.

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

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