Ginkgetin

Cat. No.: HY-N0889

CAS No.: 481-46-9

Molecular Formula: $C_{32}H_{22}O_{10}$

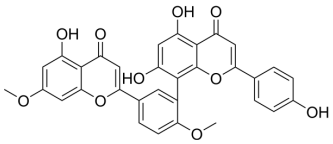
Molecular Weight: 566.51

Target: Wnt; Apoptosis; Autophagy; COX

Pathway: Stem Cell/Wnt; Apoptosis; Autophagy; Immunology/Inflammation

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 : ≥ 20.83 mg/mL (36.77 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7652 mL	8.8260 mL	17.6519 mL
	5 mM	0.3530 mL	1.7652 mL	3.5304 mL
	10 mM	0.1765 mL	0.8826 mL	1.7652 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 2 mg/mL (3.53 mM); Suspended solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 2 mg/mL (3.53 mM); Suspended solution; Need ultrasonic and warming

BIOLOGICAL ACTIVITY

Description

Ginkgetin, a biflavone, is isolated from Ginkgo biloba leaves. Ginkgetin exhibit anti-tumor, anti-inflammatory, neuroprotective, anti-fungal activities. Ginkgetin is also a potent inhibitor of Wnt signaling, with an IC_{50} of 5.92 μM[1][2][3][4][5].

In Vitro

Ginkgetin (2.5-20 μM; 48 h) inhibits the growth of Daoy and D283 cell lines, and induces G_2/M cell cycle arrest in Daoy cells[2].
?Ginkgetin (20-40 μM; 24 h) significantly activates the apoptosis of osteosarcoma cells in a concentration-dependent manner[3].
?Ginkgetin (10-20 μM; 3-24 h) down-regulated the expression of Wnt target genes without affecting the expression of β-catenin in medulloblastoma cells[2].

?Ginkgetin (1-10 μM; 24 or 48 h) significantly inhibits the VEGF-induced endothelial cell proliferation, migration, and wound recovery in a concentration-dependent manner[1].
?Ginkgetin (5-10 μM; 48 h) induces autophagy responsible for cell death in A549[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	Daoy and D283 cell lines
Concentration:	2.5, 5, 10, 20 μM
Incubation Time:	48 hours
Result:	Inhibited the cell growth, with IC_{50}s of 14.65 and 15.81 μM for Daoy and D283 cells, respectively.

Apoptosis Analysis[3]

Cell Line:	Osteosarcoma cells
Concentration:	20, 30, 40 μM
Incubation Time:	24 hours
Result:	Markedly induced the apoptosis of osteosarcoma cells in a concentration-dependent manner.

Cell Cycle Analysis[2]

Cell Line:	Daoy cells
Concentration:	2.5, 5, 10, 20 μM
Incubation Time:	24 hours
Result:	Increased G_2/M phase, compared with that of control, indicating a G_2/M cell phase arrest.

Cell Cycle Analysis[2]

Cell Line:	Daoy and D283 cell lines
Concentration:	10, 20 μM
Incubation Time:	3, 6, 12, 24 hours
Result:	Attenuated the expression of Wnt target genes, Axin2, cyclin D1 and survivin at 20 μM for 24 h in Daoy cells. Unaffected the level of total β-catenin and diminished the level of β-catenin phosphorylation in a time- and concentration-dependent manner.

In Vivo

Ginkgetin (25-100 mg/kg; i.p. 2 hours after the onset of ischemia) exerts anti-inflammatory effects on cerebral ischemia/reperfusion-induced injury in a rat model via the TLR4/NF-κB signaling pathway[4].
?Ginkgetin (30 mg/kg; intragastrically once per day for 42 d) suppresses tumor growth in A549 cells bearing nude mice[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (200-220 g)[4]
Dosage:	25, 50, 100 mg/kg

Administration:	I.p. 2 hours after the onset of ischemia
Result:	Reduced the neurological deficit score. Suppressed the expression of NF-κB, TLR4 and IκBαin ischemic penumbra cortex, and inhibited the degradation of IκBα. Decreased the expressions of ICAM-1, COX-2, and iNOS. Downregulated downstream inflammatory factor PGE2 and TNF-α expression. Decreased IL-1β, IL-6, IL-8, and IL-10 protein expression.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2021 Jan;11(1):143-155.
- Immunol Invest. 2023 May 8;1-15.
- Biomed Res Int. 2020 Dec 28.
- Biomed Res Int. 2020 Nov 4;2020:1928410.

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REFERENCES

[1]. Hu WH, et, al. Synergy of Ginkgetin and Resveratrol in Suppressing VEGF-Induced Angiogenesis: A Therapy in Treating Colorectal Cancer. Cancers (Basel). 2019 Nov 20;11(12):1828.

[2]. Ye ZN, et, al. Biflavone Ginkgetin, a Novel Wnt Inhibitor, Suppresses the Growth of Medulloblastoma. Nat Prod Bioprospect. 2015 Mar 29;5(2):91-97.

[3]. Xiong M, et, al. Ginkgetin exerts growth inhibitory and apoptotic effects on osteosarcoma cells through inhibition of STAT3 and activation of caspase-3/9. Oncol Rep. 2016 Feb;35(2):1034-40.

[4]. Li Q, et, al. Ginkgetin exerts anti-inflammatory effects on cerebral ischemia/reperfusion-induced injury in a rat model via the TLR4/NF-κB signaling pathway. Biosci Biotechnol Biochem. 2019 Apr;83(4):675-683.

[5]. Lou J, et, al. Ginkgetin induces autophagic cell death through p62/SQSTM1-mediated autolysosome formation and redox setting in non-small cell lung cancer. Oncotarget. 2017 Oct 16;8(54):93131-93148.

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