Icaritin

Cat. No.: HY-N0678

CAS No.: 118525-40-9

Molecular Formula: $C_{21}H_{20}O_6$

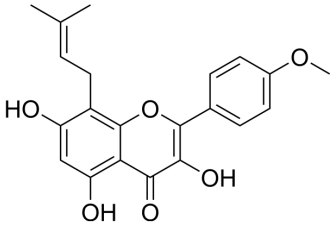
Molecular Weight: 368.38

Target: Autophagy; Apoptosis; JAK

Pathway: Autophagy; Apoptosis; Epigenetics; JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; Stem Cell/Wnt

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 15.62 mg/mL (42.40 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7146 mL	13.5729 mL	27.1459 mL
5 mM	0.5429 mL	2.7146 mL	5.4292 mL
10 mM	0.2715 mL	1.3573 mL	2.7146 mL

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

In Vivo

1. Add each solvent one by one: 15% Cremophor EL >> 85% Saline
   Solubility: 10 mg/mL (27.15 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.56 mg/mL (4.23 mM); Clear solution
3. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 1.51 mg/mL (4.10 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Icaritin (Anhydroicaritin) is a prenylflavonoid derivative from Epimedium brevicornuMaxim. and potently inhibits proliferation of K562 cells (IC_{50} of 8 μM) and primary CML cells (IC_{50} of 13.4 μM for CML-CP and 18 μM for CML-BC). Icaritin can regulate MAPK/ERK/JNK and JAK2/STAT3 /AKT signalings, also enhances osteogenesis[1][2][3].

In Vitro

Icaritin (4-64 μM; 48 hours; K562, imatinib-resistant cells and primary CML cells) treatment inhibits proliferation of K562,

imatinib-resistant cells and primary CML cells [1].
Icaritin (0-64 μM; 48 hours; K562 and primary cells) treatment induces K562 or primary cells apoptosis in an concentration dependent manner[1].
Icaritin (32 μM; K562 cells) treatment increases cell population in the sub-G1 phase in K562 cells[1].
Icaritin (0-64 μM; 48 hours; K562 cells) treatment inhibits MAPK/ERK/JNK downstream signaling and diminishes Jak2/Stat3/Akt expression. Icaritin treatment also significantly inhibits Bcl-2 protein expression and up-regulated Bax protein expression in K562 with a dose-dependent manner accompanied by the cleavage activation of caspase-3 or caspase-9, and a down-regulated expression of Apaf-1[1].

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

Cell Proliferation Assay[1]

Cell Line:	K562, imatinib-resistant cells and primary CML cells
Concentration:	4 μM, 8 μM, 16 μM, 32 μM and 64 μM
Incubation Time:	48 hours
Result:	Inhibited cell proliferation.

Apoptosis Analysis[1]

Cell Line:	K562 or primary cells
Concentration:	0 μM, 4 μM, 8 μM, 16 μM, 32 μM and 64 μM
Incubation Time:	48 hours
Result:	Induced K562 or primary cells apoptosis.

Cell Cycle Analysis[1]

Cell Line:	K562 cells
Concentration:	32 μM
Incubation Time:	
Result:	Cell population in the sub-G1 phase was increased.

Western Blot Analysis[1]

Cell Line:	K562 cells
Concentration:	0 μM, 4 μM, 8 μM, 16 μM, 32 μM and 64 μM
Incubation Time:	48 hours
Result:	Inhibited MAPK/ERK/JNK downstream signaling and diminishes Jak2/Stat3/Akt expression.

In Vivo

Icaritin (4-8 mg/kg; intraperitoneal injection; daily; for 10 weeks; female NOD-SCID nude mice) treatment could prolong lifespan of NOD-SCID nude mice inoculated with K562 cells without suppression of bone marrow in mouse leukemia model [1].

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

Animal Model:	Female NOD-SCID nude mice (6-8 weeks old) with K562 cells[1]

Dosage:	4 mg/kg and 8 mg/kg
Administration:	Intraperitoneal injection; daily; for 10 weeks
Result:	Could prolong lifespan of NOD-SCID nude mice inoculated with K562 cells without suppression of bone marrow.

CUSTOMER VALIDATION

- J Pharm Pharmacol. 2023 Nov 16:rgad103.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Zhu Jf, et al. Icaritin shows potent anti-leukemia activity on chronic myeloid leukemia in vitro and in vivo by regulating MAPK/ERK/JNK and JAK2/STAT3 /AKT signalings. PLoS One. 2011;6(8):e23720.

[2]. Yao D, et al. Icaritin, an exogenous phytomolecule, enhances osteogenesis but not angiogenesis--an in vitro efficacy study. PLoS One. 2012;7(8):e41264.

[3]. Guo Y, et al. An anticancer agent icaritin induces sustained activation of the extracellular signal-regulated kinase (ERK) pathway and inhibits growth of breast cancer cells. Eur J Pharmacol. 2011 May 11;658(2-3):114-22.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA