Acacetin

Cat. No.: HY-N0451

CAS No.: 480-44-4

Molecular Formula: $C_{16}H_{12}O_5$

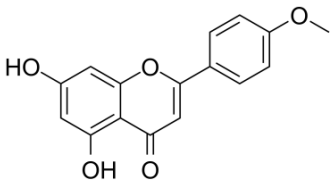
Molecular Weight: 284.26

Target: Apoptosis; Autophagy

Pathway: Apoptosis; Autophagy

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 : ≥ 37 mg/mL (130.16 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.5179 mL	17.5895 mL	35.1791 mL
5 mM	0.7036 mL	3.5179 mL	7.0358 mL
10 mM	0.3518 mL	1.7590 mL	3.5179 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (7.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Acacetin (5,7-Dihydroxy-4'-methoxyflavone) is an orally active flavonoid derived from Tephroseris kirilowii (Turcz.) Holub. Acacetin docks in the ATP binding pocket of PI3Kγ. Acacetin causes cell cycle arrest and induces apoptosis and autophagy in cancer cells. Acacetin has potent anti-cancer and anti-inflammatory activity and has the potential for pain-related diseases research[1][2].

In Vitro

Acacetin (5,7-Dihydroxy-4'-methoxyflavone; 10-200 μM; 24 hours) decreases cell viabilities in a dose-dependent manner. Acacetin has little effect on human normal glial cell line HEB and non-tumorigenic epithelial cell line MCF-10A[1].

Acacetin (50-150 μM; 24 hours) causes G2/M cell cycle arrest and induces apoptosis and autophagy[1].

Acacetin (50-150 μM; 24 hours) leads to decreases in levels of PI3Kγ-p110, p-AKT, p-mTOR, p-p70S6K, and p-ULK in a dose-dependent manner[1].

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

Cell Viability Assay[1]

Cell Line:	Breast cancer MCF-7 cells, hepatocellular carcinoma SMMC-7721 cells, lung adenocarcinoma A549 cells, esophageal carcinoma Eca109 cells
Concentration:	10, 20, 40, 60, 80, 100, 150, 200 μM
Incubation Time:	24 hours
Result:	Decreased cancer cell viabilities in a dose-dependent manner. Had IC_{50} values of 82.75 μM, 103.9 μM, 157.4 μM, 54.7 μM in MDA-MB-231, MCF-7, A549, Eca109 cells, respectively.

Cell Cycle Analysis[1]

Cell Line:	MDA-MB-231 cells
Concentration:	50, 100, 150 μM
Incubation Time:	24 hours
Result:	Resulted in increase in percentage of cells at G2/M phase and decrease in percentage of cells at G1 and S phase in a dose-dependent manner.

Apoptosis Analysis[1]

Cell Line:	MDA-MB-231 cells
Concentration:	50, 100, 150 μM
Incubation Time:	24 hours
Result:	Induced apoptosis.

Cell Autophagy Assay[1]

Cell Line:	MDA-MB-231 cells
Concentration:	50, 100, 150 μM
Incubation Time:	24 hours
Result:	Induced autophagy. Resulted in marked increases in EGFP-LC3 puncta formation and a dose-dependent accumulation of LC3-II.

Western Blot Analysis[1]

Cell Line:	MDA-MB-231 cells
Concentration:	50, 100, 150 μM
Incubation Time:	24 hours
Result:	Resulted in decrease in levels of Bcl-2 and Bcl-xL and increase in levels of p53. Led to decreases in levels of PI3Kγ-p110, p-AKT, p-mTOR, p-p70S6K, and p-ULK in a dose-dependent manner. Had little or no effect on expression of PI3Kα, PI3Kβ, PI3Kδ, p-ERK, p-p38, and p-JNK.

In Vivo

Acacetin (5,7-Dihydroxy-4'-methoxyflavone; 5, 20 mg/kg/day; orally; for 3 days) significantly suppresses microglial activation in an LPS-induced neuroinflammation mouse model[2].
Acacetin (25 mg/kg/day; orally; for 3 days) reduces neuronal cell death in an animal model of ischemia[2].
Acacetin (1.8-56.2 mg/kg/day; ip; single dose) decreases visceral and inflammatory nociception and prevented the formalin-induced oedema[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57BL/6 mice, 7 weeks of age[2]
Dosage:	5, 20 mg/kg
Administration:	Orally; once a day for 3 days
Result:	Significantly suppressed microglial activation in an LPS-induced (ip; 5mg/kg) neuroinflammation mouse model.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2021 Jan;11(1):143-155.
- Pharmacol Res. 2020 May;155:104751.
- Future Microbiol. 2020 May;15:485-496.

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REFERENCES

[1]. Hong-Wei Zhang, et al. Flavonoids inhibit cell proliferation and induce apoptosis and autophagy through downregulation of PI3Kγ mediated PI3K/AKT/mTOR/p70S6K/ULK signaling pathway in human breast cancer cells. Sci Rep. 2018 Jul 26;8(1):11255.

[2]. Sang Keun Ha, et al. Acacetin attenuates neuroinflammation via regulation the response to LPS stimuli in vitro and in vivo. Neurochem Res. 2012 Jul;37(7):1560-7.

[3]. A I Carballo-Villalobos, et al. Evidence of mechanism of action of anti-inflammatory/antinociceptive activities of acacetin. Eur J Pain. 2014 Mar;18(3):396-405.

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