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Lieferung & Zahlungsart

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Zuschläge

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Avicularin

Cat. No.: HY-N0222

CAS No.: 572-30-5

Molecular Formula: $C_{20}H_{18}O_{11}$

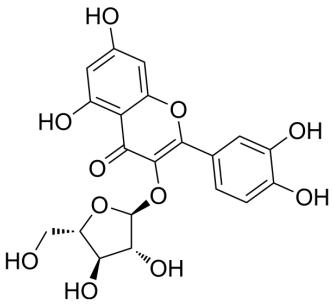
Molecular Weight: 434.35

Target: COX; NF-κB; PPAR; ERK; GLUT; Apoptosis

Pathway: Immunology/Inflammation; NF-κB; Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor; MAPK/ERK Pathway; Stem Cell/Wnt; Membrane Transporter/Ion Channel; Apoptosis

Storage: 4°C, protect from light

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (230.23 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3023 mL	11.5115 mL	23.0229 mL
	5 mM	0.4605 mL	2.3023 mL	4.6046 mL
	10 mM	0.2302 mL	1.1511 mL	2.3023 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 (4.79 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.79 mM); Clear solution

BIOLOGICAL ACTIVITY

Description Avicularin is an orally active flavonoid. Avicularin inhibits NF-κB (p65), COX-2 and PPAR-γ activities. Avicularin has anti-inflammatory, anti-infectious anti-allergic, anti-oxidant, hepatoprotective, and anti-tumor activities[1][3].

IC_{50} & Target			
	GLUT4	PPARγ	COX-2

In Vitro

Avicularin (10-300 μM, 1 h) suppresses NO and PGE_2 production in LPS-stimulated RAW 264.7 cells[1].
Avicularin (10-300 μM, 1 h) exhibits anti-inflammatory activity through the suppression of ERK signaling pathway in LPS-stimulated RAW 264.7 cells[1].
Avicularin (25-100 μg/mL, 48 h) decreases cell proliferation, cell migration and invasion in Huh7 cells[2].
Avicularin (25-100 μg/mL, 48 h) induces apoptosis via the downregulation of NF κB (p65) and COX 2 and the upregulation of

PPAR γ[2].
Avicularin (50 μM, 6 days) decreases the intracellular lipids, along with decreased PPARγ, C/EBPα, and aP2 mRNA levels in 3T3-L1 cells[3].
Avicularin (50 μM, 6 days) suppresses GLUT4-Mediated glucose uptake in 3T3-L1 cells[3].
Avicularin (2.5-10 μM, 2 h) inhibits ECM degradation and inflammation via TRAF6/MAPK activation in rat and human chondrocytes[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	Huh7 cells
Concentration:	25, 50, 100 μg/mL
Incubation Time:	12, 24, 36 and 48 h
Result:	Inhibited cell proliferation in a dose-dependent manner.

Western Blot Analysis[1]

Cell Line:	RAW 264.7 cells
Concentration:	10, 30, 100, 300 μM
Incubation Time:	1 h
Result:	Inhibited LPS-induced protein expression of iNOS and COX-2, release of pro-inflammatory cytokine IL-1β, degradation of cytosolic IκB, and phosphorylation of ERK.

RT-PCR[3]

Cell Line:	3T3-L1 cells
Concentration:	50 μM
Incubation Time:	6 days
Result:	Decreased PPARγ, C/EBPα, and aP2 mRNA levels approximately 28.7, 69.5, and 18.3%, respectively.

In Vivo

Avicularin (injected in articular cavity of the knee, 0.5-2 mg/kg, twice a week for 4 weeks) attenuates the development of OA (osteoarthritis) in ACLT-induced rats[4].
Avicularin (oral administration, 50 and 100 mg/kg, for 21 days) attenuates memory Impairment in rats with amyloid Beta-induced Alzheimer's disease[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	ACLT (anterior cruciate ligament transection)-induced rats[4]
Dosage:	0.5, 1, 2 mg/kg
Administration:	Injected into the articular cavity of the right knee, twice a week for 4 weeks.
Result:	Alleviated the tibial subchondral osteolysis, reduces bone loss, and increases the bone mass of tibial subchondral bone. Attenuated ECM degradation, the loss of Aggrecan and Collagen II in ACLT-induced rats. Decreased the MMP3 and MMP13 protein level.

Animal Model:	Rats with amyloid Beta-induced Alzheimer's disease[5]
Dosage:	25, 50, and 100 mg/kg
Administration:	Oral administration, for 21 days
Result:	Enhanced cognition activity, and reversed the effects of amyloid beta-induced inflammatory response and excessive oxidative stress.

CUSTOMER VALIDATION

- Biomed Pharmacother. 2023, 158: 114113.
- J Agric Food Chem. 2022 Dec 20.
- Aging (Albany NY). 2021 Nov 25;13(22):24753-24767.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Ko Fujimori, et al. Avicularin, a plant flavonoid, suppresses lipid accumulation through repression of C/EBPα-activated GLUT4-mediated glucose uptake in 3T3-L1 cells. J Agric Food Chem. 2013 May 29;61(21):5139-47.

[2]. Zi-Ling Zou, et al. Avicularin suppresses cartilage extracellular matrix degradation and inflammation via TRAF6/MAPK activation. Phytomedicine. 2021 Oct;91:153657.

[3]. Nikita Patil Samant, et al. Avicularin Attenuates Memory Impairment in Rats with Amyloid Beta-Induced Alzheimer's Disease. Neurotox Res. 2022 Feb;40(1):140-153.

[4]. Vo VA, et al. Avicularin Inhibits Lipopolysaccharide-Induced Inflammatory Response by Suppressing ERK Phosphorylation in RAW 264.7 Macrophages. Biomol Ther (Seoul). 2012 Nov;20(6):532-7.

[5]. Wang Z, et al. Avicularin ameliorates human hepatocellular carcinoma via the regulation of NF□κB/COX□2/PPAR□γ activities. Mol Med Rep. 2019 Jun;19(6):5417-5423.

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