Aurantio-obtusin

Cat. No.: HY-N0261

CAS No.: 67979-25-3

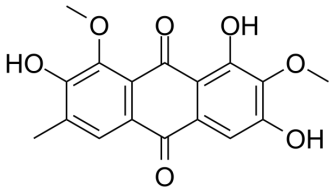
Molecular Formula: $C_{17}H_{14}O_7$

Molecular Weight: 330.29

Target: TNF Receptor; COX

Pathway: Apoptosis; Immunology/Inflammation

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 (75.69 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0276 mL	15.1382 mL	30.2764 mL
	5 mM	0.6055 mL	3.0276 mL	6.0553 mL
	10 mM	0.3028 mL	1.5138 mL	3.0276 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 (6.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Aurantio-obtusin is a anthraquinone compound that can be extracted from cassia seed. Aurantio-obtusin has the effects of decreasing blood pressure, decreasing blood lipids and anti-inflammatory.Aurantio-obtusin is an orally active vasodilator. Aurantio-obtusin ameliorates hepatic steatosis through AMPK/ autophagy- and AMPK/TFEB mediated inhibition of lipid accumulation[1][2][3].

In Vitro

Aurantio-obtusin (6.25-100 μM; 24 h) significantly decreases the production of NO and PGE2, and inhibits the expression of IL-6, TNF-α, COX-2 protein in RAW264.7 cells treated with LPS (0.2μg/mL)[2].
Aurantio-obtusin (6.25-100 μM; 12 h) inhibits the activation of NF-κ B by inhibiting the phosphorylation of i-κB and IKK in RAW264.7 cells treated with LPS (0.2μg/mL)[1].
Aurantioobtusin (1-10000 nM) induces small resistance vasodilation in rat MA in a concentration-dependent manner[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Aurantio-obtusin (5-15 mg/kg; Oral administration; Single dose) in HFSW-induced mice reduces HFSW-induced lipid droplet accumulation and extensive steatosis in a dose-dependent manner[3].

Aurantio-obtusin (5-15 mg/kg; Oral administration; A single dose) inhibits fatty acid synthesis, promotes FAO and activates AMPK signaling and autophagy in HFSW-induced mice [3].

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

Animal Model:	C57BL/6J mice model[3]
Dosage:	5 mg/kg, 10 mg/kg, 15 mg/kg
Administration:	Oral gavage (p.o.), feeding with HFSW diet for 4 weeks then giving different dose of Aurantio-obtusin for another 4 weeks.
Result:	Decreased the levels of TG and TC in liver and TG, ALT and AST in serum induced by HSFW. Reduced the number and size of fat droplets in liver cells. Significantly increased AMPK phosphorylation in HFSW-induced mice.

CUSTOMER VALIDATION

- Biochem Genet. 2023 Aug 26.

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REFERENCES

[1]. Li, Shuzhen, et al. Aurantio-obtusin relaxes systemic arteries through endothelial PI3K/AKT/eNOS-dependent signaling pathway in rats. Journal of pharmacological sciences vol. 128,3 (2015): 108-15.

[2]. Hou Jingyi, et al. Anti-Inflammatory Effects of Aurantio-Obtusin from Seed of Cassia obtusifolia L. through Modulation of the NF-κB Pathway. Molecules (Basel, Switzerland) vol. 23,12 3093. 27 Nov. 2018.

[3]. Zhou Fei, et al. Aurantio-Obtusin Attenuates Non-Alcoholic Fatty Liver Disease Through AMPK-Mediated Autophagy and Fatty Acid Oxidation Pathways. Frontiers in pharmacology vol. 12 826628. 11 Jan. 2022.

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