Osthole

Cat. No.: HY-N0054

CAS No.: 484-12-8

Molecular Formula: $C_{15}H_{16}O_3$

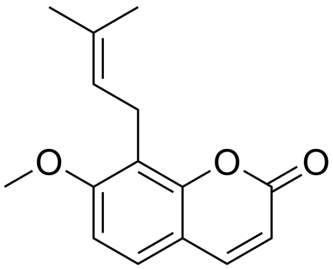
Molecular Weight: 244.29

Target: Histamine Receptor; Apoptosis; Parasite; HBV

Pathway: GPCR/G Protein; Immunology/Inflammation; Neuronal Signaling; Apoptosis; Anti-infection

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 : ≥ 100 mg/mL (409.35 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.0935 mL	20.4675 mL	40.9350 mL
5 mM	0.8187 mL	4.0935 mL	8.1870 mL
10 mM	0.4093 mL	2.0467 mL	4.0935 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: ≥ 2.5 mg/mL (10.23 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.23 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.23 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Osthole (Osthol) is a natural antihistamine alternative. Osthole may be a potential inhibitor of histamine H_1 receptor activity. Osthole also suppresses the secretion of HBV in cells.

IC_{50} & Target

Histamine H_1 receptor[1]

In Vitro	Osthole ($p<0.0001$) and Fexofenadine ($p<0.001$) inhibit increased HRH-1 mRNA expression induced by histamine in the study group. This result is also observed in cells cultured with histamine/Osthole; where combined substances decreased HRH-1 mRNA expression compared to histamine ($p<0.0001$)[1]. Assessment of cell viability does not detect obvious toxicity when Osthole is used at a dose up to 100 μM. However, when the dose reached 500 μM, Osthole started to show toxic effect. Based on these observations, Osthole is used in all in vitro studies at the dose range of 10 to 100 μM. Osthole dose-dependently promotes osteoblast differentiation, as shown by the upregulation of osteoblast differentiation marker genes such as type I collagen (col1), bone sialoprotein (BSP) and osteocalcin (OC) (2 days of culture). Osthole promotes ALP activity in mouse primary osteoblasts in a dose-dependent manner[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Subcutaneous injection of Osthole at a dose of 5 mg/kg per day onto mouse calvariae significantly stimulates local bone formation, as shown by histologic analysis of calvarial samples harvested 2 weeks after the last injection and stained with H&E orange G. Histomorphometric analysis reveals that Osthole has a significant effect on bone formation as potent as the positive control, the microtubule inhibitor TN-16. This effect, however, is not seen when Osthole is used at a dose of 1 mg/kg per day. Intraperitoneal injection of Osthole for 8 weeks significantly reverses bone loss in the ovariectomized rats. Histologic examination of the L4samples stained with trinitrophenol poinsettia demonstrates a partial recovery of the trabecular structure in ovariectomized rats treated with Osthole. Histomorphometric analysis shows that treatment with Osthole significantly increases total BMD, trabecular bone volume, and trabecular thickness and decreases trabecular separation[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Peripheral blood samples are collected from participants between 7.00 and 9.00 a.m. on the first study day and these are concentrated in grouping tubes with K_3EDTA. Fresh PBMCs are then prepared. Isolated cells are seeded on 24-well plates at 1×10^6 per well with RPMI-1640 and supplemented with 1% heat inactivated human AB serum, 1% gentamicin and 0.25% PHA. Active reagents are added to each well after 24 h and pure medium formed the control for each substance. Cells are then harvested after a further three days[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Four-week-old ICR Swiss mice are injected subcutaneously over the calvarial surface with or without the treatment of Osthole twice a day for 5 consecutive days at the doses of 1 and 5 mg/kg per day (3 mice per group). Microtubule inhibitor TN-16 is used as a positive control (5 mg/kg per day, by subcutaneous injection, twice a day for 2 days; 3 mice per group). All mice are euthanized 3 weeks after treatment, and calvariae are dissected, fixed in 10% phosphate-buffered formalin for 2 days, decalcified in 10% EDTA for 2 weeks, and embedded in paraffin. Histologic sections are cut and stained with hematoxylin and eosine orange G. New bone area over the calvarial surface is quantified by histomorphometry using the OsteoMeasure System. To measure mineral appositional rate (MAR) and bone-formation rate (BFR), double calcein labeling is performed at days 7 and 14 by intraperitoneal injection (20 mg/kg), and mice are euthanized 7 days after the second labeling. The labeling is examined in plastic sections. The dissected calvarial samples are fixed in 75% ethanol and embedded in methyl methacrylate. Unstained transverse sections (3 μm thick) are examined with a fluorescent microscope. MAR and BFR are measured using the OsteoMeasure System. Rats[2] Thirty 6-month-old female Sprague-Dawley rats are used. After anesthesia with intraperitoneal nembutal injection (30 mg/kg), the rats are randomized by body weight into three groups for the surgery (n=10/group): group 1: sham surgery followed by PBS vehicle treatment (sham+VEH); group 2: ovariectomy followed by vehicle treatment (OVX+VEH); and group 3: ovariectomy followed by Osthole treatment (OVX+OST). The treatment is started 1 month after surgery and continued for 8 weeks. Vehicle or Osthole (100 mg/kg per day) is administered orally once a day for 8 weeks. Before rats are euthanized at the end of the experiments, the total bone mineral density (BMD, g/m^2) is measured using dual-energy X-ray absorptiometry. The fourth lumbar vertebrae (L4) then are dissected for histomorphometric and micro-computed tomographic (μCT) analysis, and the left femoral shafts are used for biomechanical testing. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Pharmacol Sin. 2019 May;40(5):608-619.
- Acta Pharmacol Sin. 2018 Jan;39(1):74-84.
- Acta Pharmacol Sin. 2017 Aug;38(8):1120-1128.
- Int J Mol Sci. 2023 Aug 25, 24(17), 13210.
- J Funct Foods. 2023 Sep, 108, 105737.

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REFERENCES

[1]. Kordulewska NK, et al. Changes in gene expression induced by histamine, fexofenadine and osthole: Expression of histamine H1 receptor, COX-2, NF-κB, CCR1, chemokine CCL5/RANTES and interleukin-1β in PBMC allergic and non-allergic patients. Immunobiology.

[2]. Tang DZ, et al. Osthole stimulates osteoblast differentiation and bone formation by activation of beta-catenin-BMP signaling. J Bone Miner Res. 2010 Jun;25(6):1234-45.

[3]. Sun W, et al. Osthole pretreatment alleviates TNBS-induced colitis in mice via both cAMP/PKA-dependent and independent pathways. Acta Pharmacol Sin. 2017 Aug;38(8):1120-1128.

[4]. Zhang ZR, et al. Osthole: A Review on Its Bioactivities, Pharmacological Properties, and Potential as Alternative Medicine. Evid Based Complement Alternat Med. 2015;2015:919616.

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