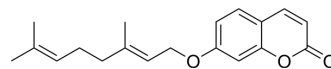


Auraptene

Cat. No.:	HY-N2388
CAS No.:	495-02-3
Molecular Formula:	C ₁₉ H ₂₂ O ₃
Molecular Weight:	298.38
Target:	MMP; PPAR; Bacterial
Pathway:	Metabolic Enzyme/Protease; Cell Cycle/DNA Damage; Vitamin D Related/Nuclear Receptor; Anti-infection
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro	DMSO : 50 mg/mL (167.57 mM; Need ultrasonic)					
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg
		1 mM		3.3514 mL	16.7572 mL	33.5143 mL
		5 mM		0.6703 mL	3.3514 mL	6.7029 mL
		10 mM		0.3351 mL	1.6757 mL	3.3514 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 (6.97 mM); Clear solution 2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.08 mg/mL (6.97 mM); Clear solution 3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (6.97 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	Auraptene is an orally active geranyloxycoumarin that can be isolated from plants in the Brassicaceae family, antibacterial, anti-pathogen, antioxidant, anti-tumor, and neuroprotective effects. Auraptene plays an important role in the treatment of various chronic diseases such as hypertension and cystic fibrosis ^{[1][2]} .
IC ₅₀ & Target	MMP-2
In Vitro	Auraptene (0-20 μM, 2 h) reduces the secretion of inflammatory mediators stimulated by lipopolysaccharides in oral epithelial cells and promotes wound healing by promoting cell migration ^[1] .

Auraptene (10 μM , 24 h) inhibits the cell cycle progression of human breast cancer cell line MCF-7 by reducing the expression of cyclin D1 protein and inhibiting IGF-1^[2].

Auraptene (10 μM , 4 days) exhibits antiviral activity against human coronavirus OC43 in MRC-5 cells^[6].

Auraptene (25-400 μM) protects red blood cells from free radical induced damage by preventing the consumption of intracellular antioxidant GSH and inhibiting protein peroxidation^[7].

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

Cell Viability Assay^[1].

Cell Line:	Oral epithelial cell line GMSM-K
Concentration:	0-20 μM
Incubation Time:	2 h
Result:	Didn't affect the survival rate of epithelial cells.

Real Time qPCR^[2].

Cell Line:	MCF-7 cell
Concentration:	10 μM
Incubation Time:	24 h
Result:	Upregulated gene expression levels of CDKN2B (Cyclin dependent kinase inhibitor 2B), DDIT3 (DNA damage inducible transcript 3), and JUN (JUN oncogene).

Real Time qPCR^[6].

Cell Line:	HCoV-OC43-infected human lung fibroblast MRC-5 cells
Concentration:	10 μM
Incubation Time:	4 days
Result:	Decreased viral RNA levels in HCoV-OC43-infected cells.

In Vivo

Auraptene (200, 500 ppm, mixed in the diet, p.o.) delays the tumor progression of breast cancer rats by inhibiting cyclin D1 protein^[3].

Auraptene (100, 500 ppm, mixed in the diet, p.o.) alleviates gastritis by reducing Helicobacter pylori colonization and pro-inflammatory mediator production in C57BL/6 mice^[4].

Auraptene (5, 50 mg/kg, 6 weeks, p.o.) prevents heart failure caused by myocardial infarction by activating peroxisome proliferator activated receptor alpha (PPAR alpha) in rats^[5].

Auraptene (2, 4, 8, 16 mg/kg, 5 weeks, p.o.) exhibits anti hypertensive effects in hypertensive rats by reducing mean systolic blood pressure^[8].

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

Animal Model:	Mammary carcinogenesis model in female Sprague Dawley rats ^[3] .
Dosage:	200, 500 ppm
Administration:	Oral gavage (p.o.); mixed in the diet
Result:	Delayed median time to tumor by 39 days and reduced Insulin like Growth Factor-1 (IGF-1, 10 ng/mL)-induced cyclin D1 expression by 40% in MCF-7 cells.

Animal Model:	Female C57BL/6 mice ^[4] .
Dosage:	100, 500 ppm
Administration:	Oral gavage (p.o.); mixed in the diet
Result:	Inhibited H. pylori-induced expression and/or production of CD74, macrophage migration inhibitory factor, interleukin-1b, and tumor necrosis factor- α in gastric mucosa, together with serum macrophage inhibitory protein-2.
Animal Model:	Sprague-Dawley rats with moderate myocardial infarction ^[5] .
Dosage:	5, 50 mg/kg
Administration:	Oral gavage (p.o.); 6 weeks
Result:	Suppresses PE-induced hypertrophic responses in cardiomyocytes. Prevented the development of cardiac hypertrophy and fibrosis in rats with myocardial infarction.
Animal Model:	Desoxycorticosterone acetate (DOCA) salt induced hypertensive rats ^[8] .
Dosage:	2, 4, 8, 16 mg/kg
Administration:	Oral gavage (p.o.); 5 weeks
Result:	Reduced the mean systolic blood pressure (MSBP) in DOCA salt treated rats.

REFERENCES

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Caution: Product has not been fully validated for medical applications. For research use only.

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