Myristicin

Cat. No.: HY-N2510

CAS No.: 607-91-0

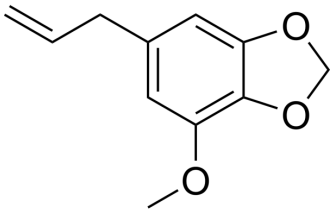
Molecular Formula: $C_{11}H_{12}O_3$

Molecular Weight: 192.21

Target: 5-HT Receptor; EGFR; ERK; Apoptosis; Bacterial

Pathway: GPCR/G Protein; Neuronal Signaling; JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; MAPK/ERK Pathway; Stem Cell/Wnt; Apoptosis; Anti-infection

Storage: 4°C, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (520.26 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	5.2026 mL	26.0132 mL	52.0264 mL
	5 mM	1.0405 mL	5.2026 mL	10.4053 mL
	10 mM	0.5203 mL	2.6013 mL	5.2026 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 (13.01 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (13.01 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (13.01 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Myristicine is an orally bioavailable serotonin receptor antagonist and weak monoamine oxidase (MAO) inhibitor. Myristicine also exerts anti-cancer effects on gastric cancer cells by inhibiting the EGFR/ERK signaling pathway. Myristicine is the main component of nutmeg essential oil and has anti-cancer, anti-proliferative, antibacterial, anti-inflammatory and apoptosis-inducing effects. Myristicine abuse can produce hallucinogenic effects, organ damage, etc[1][2][3][4].

IC_{50} & Target	EGFR	ERK

In Vitro

Myristicin (7.8-31.25 μM; 48 h) can delay the proliferation of gastric cancer cells and induce endoplasmic reticulum (ER)

stress and apoptosis; at the same time, Myristicin reduces the expression of cell cycle proteins, increases the expression of Bax, and activates caspase enzyme, and enhances the release of cytochrome C and mitochondrial ROS levels [3].

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

Western Blot Analysis[3]

Cell Line:	Human gastric cancer cells
Concentration:	7.8125, 15.625, and 31.25 μM
Incubation Time:	48 h
Result:	Significantly decreased expression of cyclins, increased Bax expression.

In Vivo

In animal models, Myristicin can inhibit the growth of gastric cancer cells and the EGFR/ERK signaling pathway[3].

Myristicin (200 mg/kg; po; single dose) treated 6 hours in advance, on acetic acid (AA)-induced small Mouse experimental colitis model has preventive and protective effect[4].

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

Animal Model:	Experimental colitis model in mice[4]
Dosage:	200 mg/kg
Administration:	po; single dose, 6 h before Rectal AA (colitis inducer) instillation
Result:	Reduced cellular oxidative stress induced by AA, and relieved the histopathological colonic damage, alleviated inflammation in the AA-induced colitis.

REFERENCES

[1]. Kalbhen DA, et al. Nutmeg as a narcotic. A contribution to the chemistry and pharmacology of nutmeg (Myristica fragrans).Angew Chem Int Ed Engl. 1971 Jun;10(6):370-4.

[2]. Dawidowicz AL, et al. Simple and rapid determination of myristicin in human serum.Forensic Toxicol. 2013 Jan;31(1):119-123. Epub 2012 Aug 15.

[3]. Badr G, Elsawy H, Amalki M A, et al. Protective effects of myristicin against ulcerative colitis induced by acetic acid in male mice[J]. Food and Agricultural Immunology, 2020, 31(1): 435-446.

[4]. Song J, et al. Myristicin Suppresses Gastric Cancer Growth via Targeting the EGFR/ ERK Signaling Pathway. Curr Mol Pharmacol. 2023;16(7):712-724.

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