Columbin

Cat. No.: HY-N0389

CAS No.: 546-97-4

Molecular Formula: $C_{20}H_{22}O_6$

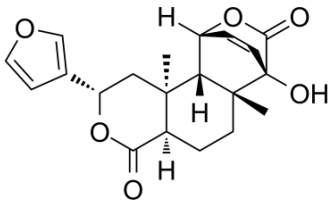
Molecular Weight: 358.39

Target: COX; Parasite

Pathway: Immunology/Inflammation; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (279.03 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.7903 mL	13.9513 mL	27.9026 mL
5 mM	0.5581 mL	2.7903 mL	5.5805 mL
10 mM	0.2790 mL	1.3951 mL	2.7903 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 (6.98 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.98 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Columbin is an orally active diterpenoid furanolactone from Calumbae radix, has anti-inflammatory and anti-trypanosomal effects. Columbin selectively inhibits COX-2 (EC_{50}=53.1 μM) over COX-1 (EC_{50}=327 μM)[1][2].

IC_{50} & Target

COX-2	COX-1
53.1 μM (EC50)	327 μM (EC50)

In Vitro

Treatment with columbin or l-NAME inhibits LPS/IFN-γ-induced NO production without affecting the viability of RAW264.7. Pre-treatment of stimulated cells with columbin does not inhibit the translocation of NF-κB to the nucleus in LPS-stimulated cells. COX-1 and COX-2 inhibitory activities of columbin are 63.7±6.4% and 18.8±1.5% inhibition at 100μM, respectively. The

	interaction of columbin with Tyr385 and Arg120 signifies its higher activity in COX-2, as Tyr385 is reported to be involved in the abstraction of hydrogen from C-13 of arachidonate, and Arg120 is critical for high affinity arachidonate binding[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Columbin inhibits oedema formation in mice paw. At doses of 300 mg/kg and 700 mg/kg, columbin inhibits inflammation from 0 to 5 h and the results are comparable to that of aspirin as a standard anti-inflammatory drug. The inhibitory effect of columbin on carrageenan induced paw oedema in mice may be due to the suppression of the release of mediators responsible for inflammation including prostaglandin[1]. Columbin is poorly bioavailable (2.8% p.o. and 14% i.p.) in rats, but its transport is rapid across the Caco-2 cell monolayers, suggesting that extensive first-pass metabolism in the liver is the likely reason for its poor bioavailability[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1][2]	Rats: Male Wistar rats are treated as following: i.v. injection of columbin in EtOH and PEG-300 (1:1) is administrated through tail vein at dose of 20 mg/kg. Intraperitoneal (i.p.) injection of columbin in EtOH and PEG-300 (1:1) is administrated at dose of 20 mg/kg. An oral gavage of columbin suspended in oral suspension vehicle is given to rats at dose of 50 mg/kg. Blood samples (50-100 μL) are collected by snipping the tail into heparinized tubes at 0, 5, 15, 30, 45, 60, 120, 240, 360, 480 and 1440 min for i.v. administration, or at 0, 15, 30, 60,120, 180, 240, 360, 480, 1440 min for oral dosing or i.p. injection. The blood samples are stored at −20°C until analysis[2]. Mice: Male Balb/c mice (n=60) are randomly divided into six groups. Columbin is intra-peritoneally administered to mice at the dose of 30, 100, 300 and 700 mg/kg. Aspirin, an anti-inflammatory drug, is used as a positive control. To induce acute phase inflammation in paw, rats are injected subcutaneously into the right hind paw with a 1% solution of carrageenan dissolved in saline 30 min after vehicle or columbin treatment. The paw volumes are measured up to 5 h after the injection at intervals of 1 h. Paw volume is measured with a plethysmometer immediately prior to the injection of carrageenan and thereafter at an interval of 1 h for a period of 5 h[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Ibrahim Abdelwahab S, et al. In vitro and in vivo anti-inflammatory activities of columbin through the inhibition of cycloxygenase-2 and nitric oxide but not the suppression of NF-κB translocation. Eur J Pharmacol. 2012 Mar 5;678(1-3):61-70.

[2]. A J Nok, et al. Columbin inhibits cholesterol uptake in bloodstream forms of Trypanosoma brucei-A possible trypanocidal mechanism.J Enzyme Inhib Med Chem. 2005 Aug;20(4):365-8.

[3]. Yang G, et al. Development and validation of an UPLC-MS/MS method for the quantification of columbin in biological matrices: Applications to absorption, metabolism, and pharmacokinetic studies. J Chromatogr B Analyt Technol Biomed Life Sci. 2015 Oct 1;1002:13-8

India Contact:
Life Technologies (India) Pvt. Ltd.
306, Aggarwal City Mall, Opposite M2K Pitampura, Delhi – 110034 (INDIA). Ph: +91-11-42208000, 42208111, 42208222, Mobile: +91-9810521400, Fax: +91-11-42208444
Email: customerservice@lifetechindia.com Website: www.lifetechindia.com