Colchicine

Cat. No.: HY-16569

CAS No.: 64-86-8

Molecular Formula: $C_{22}H_{25}NO_6$

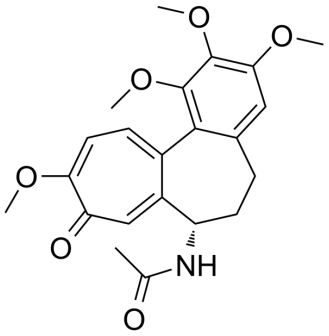
Molecular Weight: 399.44

Target: Microtubule/Tubulin; Autophagy; Apoptosis

Pathway: Cell Cycle/DNA Damage; Cytoskeleton; Autophagy; Apoptosis

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 : ≥ 48 mg/mL (120.17 mM)

H_2O : ≥ 33.33 mg/mL (83.44 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5035 mL	12.5175 mL	25.0350 mL
5 mM	0.5007 mL	2.5035 mL	5.0070 mL
10 mM	0.2504 mL	1.2518 mL	2.5035 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 2.78 mg/mL (6.96 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

Colchicine is a tubulin inhibitor and a microtubule disrupting agent. Colchicine inhibits microtubule polymerization with an IC_{50} of 3 nM[1][2][3]. Colchicine is also a competitive antagonist of the α3 glycine receptors (GlyRs)[4].

IC_{50} & Target

Microtubule/Tubulin[1]

In Vitro

Exposure to 1μM Colchicine, a microtubule disrupting agent, triggered apoptosis in rat cerebellar granule cells (CGC). Colchicine treatment also causes alterations in Ca^{2+} responses to chemical depolarization and a moderate, but progressive, increase in the resting intracellular Ca^{2+} concentration[1]. Colchicine exerts its biological effects through binding to the soluble tubulin heterodimer, the major component of the microtubule. The Colchicine binding abilities of tubulins from a variety of sources are summarized, and the mechanism of Colchicine binding to brain tubulin is explored in depth. The

	relationship between colchicinoid structure and tubulin binding activity provides insight into the structural features of Colchicine responsible for high affinity binding to tubulin and is reviewed for analogs in the Colchicine series. The thermodynamic and kinetic aspects of the association are described and evaluated in terms of the binding mechanism. Colchicine binding to tubulin results in unusual alterations in the low energy electronic spectra of Colchicine. The spectroscopic features of Colchicine bound to tubulin are discussed in terms of the nature of the Colchicine-tubulin complex. Attempts to locate the high affinity Colchicine binding site on tubulin are presented[2]. Colchicine treatment inhibits indomethacin-induced small intestinal injury by 86% (1 mg/kg) and 94% (3 mg/kg) as indicated by the lesion index 24 h after indomethacin administration. Colchicine inhibits the protein expression of cleaved caspase-1 and mature IL-1β, without affecting the mRNA expression of NLRP3 and IL-1β[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Vehicle or Colchicine (1 mg/kg) is administered orally 30 min prior to indomethacin administration. The lesions stained with Evans blue in the small intestine are smaller in Colchicine-treated mice than those in vehicle-treated mice 24 h after indomethacin administration. In addition, histological examination shows that Colchicine-treated mice have less mucosal inflammation and ulceration and a decrease in the size and numbers of lesions compared with vehicle-treated mice. Colchicine treatment significantly reduces the lesion index at doses of 1 mg/kg and 3 mg/kg (by 86% and 94%, respectively) compared with vehicle treatment. Colchicine treatment significantly inhibits the protein levels of mature IL-1β at doses of 1 mg/kg and 3 mg/kg (by 56% and 69%, respectively) without affecting those of pro-IL-1β. Colchicine treatment also significantly inhibits the protein levels of cleaved caspase-1 at doses of 1 mg/kg and 3 mg/kg (by 26% and 39%, respectively) without affecting those of pro-caspase-1[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [3]	Mice[3] Specific-pathogen-free 8-week-old male mice are used. Wild-type C57BL/6 mice and NLRP3$^{-/-}$ mice on a C57BL/6 background are used. To examine the effects of Colchicine on NSAID-induced small intestinal injury, vehicle or Colchicine (1 or 3 mg/kg) is administered orally 30 min prior to indomethacin administration. Mice received intraperitoneal injections of sterilized phosphate buffered saline or mouse recombinant IL-1β (0.1 μg/kg) 3 h after indomethacin treatment. Vehicle or Colchicine (1 or 3 mg/kg) is also administered to NLRP3$^{-/-}$ mice before indomethacin administration. The lesion index is evaluated 24 h after indomethacin administration, and examined mRNA and protein expression of inflammasome components 6 h after indomethacin administration. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2020 Sep 29;11(1):4902.
- EMBO J. 2020 Jul 1;39(13):e104168.
- Acta Pharm Sin B. 31 March 2022.
- Mol Oncol. 2022 Mar;16(6):1347-1364.
- Int J Nanomedicine. 2019 Nov 27;14:9217-9234.

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REFERENCES

[1]. Bonfoco E, et al. Colchicine induces apoptosis in cerebellar granule cells. Exp Cell Res. 1995 May;218(1):189-200.

[2]. Hastie SB. Interactions of colchicine with tubulin. Pharmacol Ther. 1991;51(3):377-401

[3]. Otani K, et al. Colchicine prevents NSAID-induced small intestinal injury by inhibiting activation of the NLRP3 inflammasome. Sci Rep. 2016 Sep 2;6:32587.

[4]. Carola Muñoz-Montesino, et al. Inhibition of the Glycine Receptor alpha 3 Function by Colchicine. Front Pharmacol. 2020 Jul 30;11:1143.

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