Colchicine

Cat. No.: HY-16569

CAS No.: 64-86-8

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

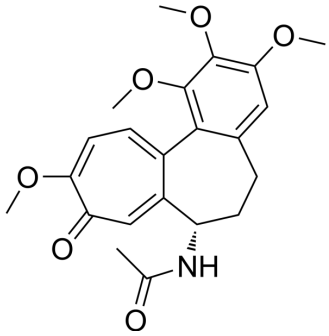
Molecular Weight: 399.44

Target: Microtubule/Tubulin; Autophagy; Apoptosis; NOD-like Receptor (NLR)

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

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



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, an orally active alkaloid, is a potent tubulin inhibitor and a microtubule disrupting agent. Colchicine inhibits microtubule polymerization with an IC_{50} of 3 nM. Colchicine is also a competitive antagonist of the α3 glycine receptors (GlyRs). Colchicine prevents non-steroidal anti-inflammatory drug (NSAID)-induced small intestinal injury by inhibiting activation of the NLRP3 inflammasome. Colchicine has extensive anti-inflammatory, immunosuppressive and strong anti-fibrosis effects and has the potential for gouty arthritis research[1][2][3][4][5].

IC_{50} & Target

Microtubule/Tubulin[1]

In Vitro

Colchicine (5 μM; pretreated 24 h) inhibits MIRI-induced apoptosis of H9C2 by regulating the PI3K/AKT/eNOS pathway[3].

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

Apoptosis Analysis[3]

Cell Line:	H9C2 cells
Concentration:	5 μM
Incubation Time:	Pretreated 24 h
Result:	Caused a significant reduction (4.490%) of early apoptotic cells as compared with the H/R only groups (6.182%).

Western Blot Analysis[3]

Cell Line:	H9C2 cells
Concentration:	5 μM
Incubation Time:	Pretreated 24 h
Result:	Observably increased the decreased phosphorylation levels of PI3K, AKT, and eNOS after H/R injury in H9C2 cell.

In Vivo

Colchicine (5 μM; pretreated 24 h) inhibits MIRI-induced apoptosis of H9C2 by regulating the PI3K/AKT/eNOS pathway[3].

Colchicine (1, 3 mg/kg; orally 30 min prior) causes less mucosal inflammation and ulceration and a decrease in the size and numbers of lesions compared with vehicle-treated mice with Indomethacin (HY-14397)[4].

Induction of Alzheimer's disease (AD)[6]

Background

Colchicine is a tubulin inhibitor that binds irreversibly to tubulin dimers and inhibits microtubule assembly with disruption of their polymerization, leading to neurofibrillary degeneration and synaptic loss. This causes neurotrophic factor intracellular trafficking to be disrupted, as well as axonal excitotoxicity and oxidative damage.

Specific Mmodeling Methods

Rats: Wistar • male • adult, weighing 180-220 g (acclimate: 1 weeks)
Administration: 7.5 μg in 5 μL/site • intracerebroventricular (icv) injection • single dose

(1) For induction of AD, rats are anesthetized using intraperitoneal (i.p.) Thiopental sodium (45 mg/kg) and then positioned in stereotaxic apparatus.
(2) The stereotaxic coordinates are 0.8 mm posterior to bregma, 1.8 mm lateral to sagittal suture and 3.6 mm beneath the cortical surface.
(3) Using a 28-gauge micro-syringe (10 μL) 15 μg Colchicine dissolved in 10 μL ACSF is injected bilaterally

into the lateral ventricle (7.5 μg in 5 μL/site). The injection rate is 2 μL/min, then the micro-syringe is remained in place for 2 min to avoid withdrawal of the injected fluid.
(4) Artificial cerebrospinal fluid (ACSF constituents in milli mole: NaCl 147, $MgCl_2$ 1.6, KCl 2.9, $CaCl_2$ 1.7, and Dextrose 2.2) is freshly prepared.

Modeling Indicators

Molecular changes: Induced indicators: Aβ 1-40 and Aβ 1-42 peptides level, MDA concentration, TNF-α level in both hippocampus and prefrontal cortex
Decrease indicators: SOD activity in both hippocampus and prefrontal cortex

Correlated Product(s) β-Amyloid (1-42), human TFA (HY-P1363)

Opposite Product(s) Sacubitril/Valsartan (HY-18204A)

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

Animal Model:	Specific-pathogen-free 8-week-old male mice (Wild-type C57BL/6 mice)[4]
Dosage:	1 or 3 mg/kg
Administration:	Orally; 30 min prior to Indomethacin administration
Result:	Caused less mucosal inflammation and ulceration and a decrease in the size and numbers of lesions compared with vehicle-treated mice with Indomethacin (10 mg/kg; gavage).

CUSTOMER VALIDATION

- Cell Mol Immunol. 2023 Jan;20(1):51-64.
- Nat Commun. 2020 Sep 29;11(1):4902.
- Acta Pharm Sin B. 2022 Sep;12(9):3618-3638.
- EMBO J. 2020 Jul 1;39(13):e104168.
- Cancer Immunol Res. 2023 May 3;11(5):583-599.

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REFERENCES

[1]. Yuanjun Tang, et al. Network Pharmacology-Based Investigation and Experimental Exploration of the Antiapoptotic Mechanism of Colchicine on Myocardial Ischemia Reperfusion Injury. Front Pharmacol. 2021 Dec 16:12:804030.

[2]. Sami H Hammadi, et al. Effect of sacubitril/valsartan on cognitive impairment in colchicine-induced Alzheimer's model in rats. Fundam Clin Pharmacol. 2023 Apr;37(2):275-286.

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

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

[5]. 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.

[6]. 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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