

# Colchicine

**Catalog No: tcsc1141**



## Available Sizes

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**Size:** 200mg

**Size:** 500mg



## Specifications

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**CAS No:**

64-86-8

**Formula:**

$C_{22}H_{25}NO_6$

**Pathway:**

Cell Cycle/DNA Damage;Cytoskeleton;Autophagy

**Target:**

Microtubule/Tubulin;Microtubule/Tubulin;Autophagy

**Purity / Grade:**

>98%

**Solubility:**

DMSO :  $\geq 48$  mg/mL (120.17 mM); H<sub>2</sub>O :  $\geq 33.33$  mg/mL (83.44 mM)

**Observed Molecular Weight:**

399.44

## Product Description

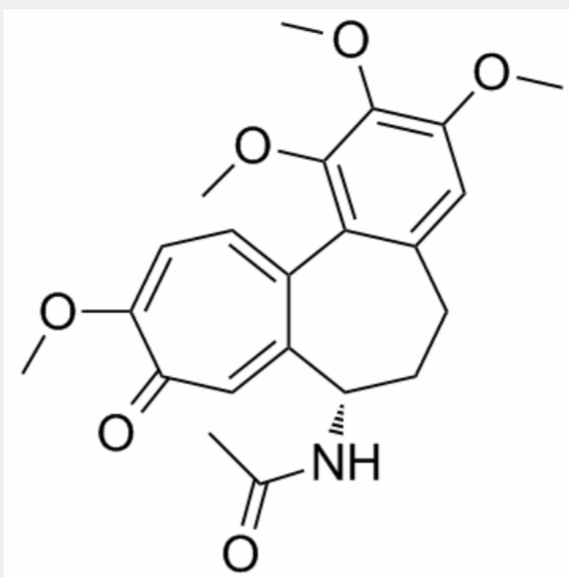
Colchicine is a **tubulin** inhibitor and a **microtubule**

disrupting agent. Colchicine triggers apoptosis.

IC50 & Target: Microtubule/Tubulin<sup>[1]</sup>

**In Vitro:** Exposure to 1 $\mu$ M Colchicine, a microtubule disrupting agent, triggered apoptosis in rat cerebellar granule cells (CGC). Colchicine treatment also causes alterations in Ca<sup>2+</sup> responses to chemical depolarization and a moderate, but progressive, increase in the resting intracellular Ca<sup>2+</sup> concentration<sup>[1]</sup>. 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<sup>[2]</sup>. 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-1 $\beta$ , without affecting the mRNA expression of NLRP3 and IL-1 $\beta$ <sup>[3]</sup>.

**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-1 $\beta$  at doses of 1 mg/kg and 3 mg/kg (by 56% and 69%, respectively) without affecting those of pro-IL-1 $\beta$ . 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<sup>[3]</sup>.



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!