Ciprofloxacin monohydrochloride

Cat. No.: HY-B0356A

CAS No.: 93107-08-5

Molecular Formula: $C_{17}H_{19}ClFN_3O_3$

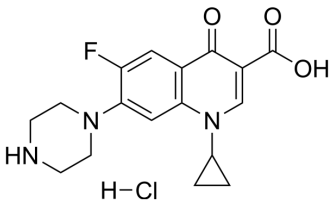
Molecular Weight: 367.8

Target: Bacterial; Antibiotic; Topoisomerase; Apoptosis; Mitochondrial Metabolism; Reactive Oxygen Species

Pathway: Anti-infection; Cell Cycle/DNA Damage; Apoptosis; Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB

Storage: 4°C, sealed storage, away from moisture and light

* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

H_2O : 12.5 mg/mL (33.99 mM; Need ultrasonic)
DMSO : 5 mg/mL (13.59 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7189 mL	13.5943 mL	27.1887 mL
5 mM	0.5438 mL	2.7189 mL	5.4377 mL
10 mM	0.2719 mL	1.3594 mL	2.7189 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 0.5 mg/mL (1.36 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.5 mg/mL (1.36 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ciprofloxacin (Bay-09867) monohydrochloride is a potent, orally active topoisomerase IV inhibitor. Ciprofloxacin monohydrochloride induces mitochondrial DNA and nuclear DNA damage and lead to mitochondrial dysfunction, ROS production. Ciprofloxacin monohydrochloride has anti-proliferative activity and induces apoptosis. Ciprofloxacin monohydrochloride is a fluoroquinolone antibiotic, exhibiting potent antibacterial activity[1][2][3][4].

IC_{50} & Target

Quinolone

In Vitro

Ciprofloxacin (Bay-09867) monohydrochloride (5-50 μg/mL; 0-24 h; tendon cells) inhibits cell proliferation and causes cell cycle arrest at the G2/M phase[1].

?Ciprofloxacin (Bay-09867) monohydrochloride shows potent activity against Y. pestis and B. anthracis with MIC_{90} of 0.03 μg/mL and 0.12 μg/mL, respectively[2].

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

Cell Viability Assay[1]

Cell Line:	Tendon cells
Concentration:	5, 10, 20 and 50 μg/mL
Incubation Time:	24 hours
Result:	Decreased the cellularity of tendon cells.

Cell Cycle Analysis[1]

Cell Line:	Tendon cells
Concentration:	50 μg/mL
Incubation Time:	24 hours
Result:	Arrested cell cycle at the G2/M phase and inhibited cell division in tendon cells.

Western Blot Analysis[1]

Cell Line:	Tendon cells
Concentration:	50 μg/mL
Incubation Time:	0, 6, 12, 17 and 24 hours
Result:	Down-regulated the expression of CDK-1 and cyclin B protein and mRNA. Up-regulated the expression of PLK-1 protein.

In Vivo

Ciprofloxacin (Bay-09867) monohydrochloride (30 mg/kg; i.p.; for 24 hours; BALB/c mice) has protection against Y. pestis in murine model of pneumonic plague[3].

?Ciprofloxacin (Bay-09867) monohydrochloride (100 mg/kg; i.g.; daily, for 4 weeks; C57BL/6J mice) accelerates aortic root enlargement and increases the incidence of aortic dissection and rupture by decreases LOX level and increases MMP levels and activity in the aortic wall[4].

?Ciprofloxacin (Bay-09867) monohydrochloride (100 mg/kg; i.g.; daily, for 4 weeks; C57BL/6J mice) induces DNA damage and release of DNA to the cytosol, mitochondrial dysfunction, and activation of cytosolic DNA sensor signaling. Ciprofloxacin lactate increases apoptosis and necroptosis in the aortic wall[4].

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

Animal Model:	BALB/c mice[3]
Dosage:	30 mg/kg
Administration:	Intraperitoneal injection; for 24 hours
Result:	Reduced the lung bacterial load in murine model of pneumonic plague.

Animal Model:	C57BL/6J mice[4]
Dosage:	100 mg/kg

Administration:	Oral gavage; daily, for 4 weeks
Result:	Had aortic destruction that was accompanied by decreased LOX expression and increased MMP expression and activity.

Animal Model:	C57BL/6J mice[4]
Dosage:	100 mg/kg
Administration:	Oral gavage; daily, for 4 weeks
Result:	Caused mitochondrial DNA and nuclear DNA damage, leading to mitochondrial dysfunction and ROS production. Increased apoptosis and necroptosis in the aortic wall.

CUSTOMER VALIDATION

- Nat Commun. 2022 Mar 2;13(1):1116.
- Adv Sci (Weinh). 2020 Jul 21;7(17):2001374.
- Water Res. 2023 May 21, 120110.
- Genome Biol. 2023 Apr 30;24(1):98.
- EBioMedicine. 2022 Apr;78:103943.

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REFERENCES

[1]. Tsai WC, et, al. Ciprofloxacin-mediated cell proliferation inhibition and G2/M cell cycle arrest in rat tendon cells. Arthritis Rheum. 2008 Jun;58(6):1657-63.

[2]. Steenbergen J, et, al. In Vitro and In Vivo Activity of Omadacycline against Two Biothreat Pathogens, Bacillus anthracis and Yersinia pestis. Antimicrob Agents Chemother. 2017 Apr 24;61(5):e02434-16.

[3]. Hamblin KA, et, al. Inhaled Liposomal Ciprofloxacin Protects against a Lethal Infection in a Murine Model of Pneumonic Plague. Front Microbiol. 2017 Feb 6;8:91.

[4]. LeMaire SA, et, al. Effect of Ciprofloxacin on Susceptibility to Aortic Dissection and Rupture in Mice. JAMA Surg. 2018 Sep 1;153(9):e181804.

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