Trovafloxacin

Cat. No.: HY-A0170

CAS No.: 147059-72-1

Molecular Formula: $C_{20}H_{15}F_3N_4O_3$

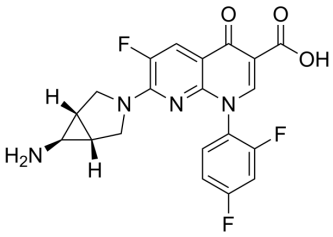
Molecular Weight: 416.35

Target: Bacterial; Topoisomerase; Antibiotic

Pathway: Anti-infection; Cell Cycle/DNA Damage

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 : 10 mg/mL (24.02 mM; ultrasonic and adjust pH to 2 with 1M HCl)
DMSO : 9.09 mg/mL (21.83 mM; ultrasonic and adjust pH to 3 with HCl)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4018 mL	12.0091 mL	24.0183 mL
5 mM	0.4804 mL	2.4018 mL	4.8037 mL
10 mM	0.2402 mL	1.2009 mL	2.4018 mL

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

BIOLOGICAL ACTIVITY

Description

Trovafloxacin is a broad-spectrum quinolone antibiotic with potent activity against Gram-positive, Gram-negative and anaerobic organisms. Trovafloxacin blocks the DNA gyrase and topoisomerase IV activity. Trovafloxacin is also a potent, selective and orally active pannexin 1 channel (PANX1) inhibitor with an IC_{50} of 4 μM for PANX1 inward current. Trovafloxacin does not inhibit connexin 43 gap junction or PANX2. Trovafloxacin leads to dysregulated fragmentation of apoptotic cells by inhibiting PANX1[1][2][3].

IC_{50} & Target

Quinolone

In Vitro

Trovafloxacin (20 μM; 24 hours; HepG2 cells) and tumor necrosis factor (TNF; 4 ng/mL) incubation induces apoptosis and increases leakage of lactate dehydrogenase (LDH) in HepG2 cells[1].
Trovafloxacin (20 μM; 24 hours; HepG2 cells) and TNF (4 ng/mL) incubation increases expression of early NF-κB-related factors A20 and IκBα[1].
Trovafloxacin prolongs TNF-induced activation of MAPKs and IKKα/β activation in HepG2[1].
Trovafloxacin is a potent inhibitor of TO-PRO-3 uptake by apoptotic cells. Trovafloxacin also inhibits ATP release from

apoptotic cells. Trovafloxacin does not inhibit caspase 3/7 activation, or caspase-mediated PANX1 cleavage during apoptosis[2].
Trovafloxacin is equally active against both penicillin-susceptible and -resistant pneumococci, with MICs of 0.06-0.25 mg/mL reported for more than 700 isolates. The MICs of Trovafloxacin at which 90% of isolates are inhibited for 55 isolates of pneumococci is 0.125 μg/mL[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[1]

Cell Line:	HepG2 cells
Concentration:	20 μM
Incubation Time:	24 hours
Result:	Showed a gradual increase of Annexin V-staining and an increased leakage of lactate dehydrogenase (LDH) at 24 h.

RT-PCR[1]

Cell Line:	HepG2 cells
Concentration:	20 μM
Incubation Time:	24 hours
Result:	Caused a higher increase in the transcription of A20 and IκBα in HepG2 cells.

In Vivo

Trovafloxacin (150 mg/kg; oral administration; male C57BL/6 J mice) treatment disrupts TNF-induced p65 nuclear translocation. Trovafloxacin treatment increases expression of early NF-κB-related factors A20 and IκBα[1].
Trovafloxacin, when administered in combination with lipopolysaccharide (LPS) or TNF to mice induces severe liver toxicity associated with vast apoptotic areas in the liver, increased serum levels of alanine amino transferases (ALT) and pro-inflammatory cytokines[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57BL/6 J mice (9-11-week-old) injected with recombinant murine TNF ion[1]
Dosage:	150 mg/kg
Administration:	Oral administration
Result:	Showed a greater number of cells with increased nuclear/cytoplasmic p65 ratio in liver.

CUSTOMER VALIDATION

- Biotechnol Bioeng. 2021 Sep 3.

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REFERENCES

[1]. Giustarini G, et al. The hepatotoxic fluoroquinolone trovafloxacin disturbs TNF- and LPS-induced p65 nuclear translocation in vivo and in vitro. Toxicol Appl Pharmacol. 2020 Mar 15;391:114915.

[2]. Poon IK, et al. Unexpected link between an antibiotic, pannexin channels and apoptosis. Nature. 2014 Mar 20;507(7492):329-34.

[3]. Gootz TD, et al. Activity of the new fluoroquinolone trovafloxacin (CP-99,219) against DNA gyrase and topoisomerase IV mutants of Streptococcus pneumoniae selected in vitro. Antimicrob Agents Chemother. 1996 Dec;40(12):2691-7.

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