Clindamycin

Cat. No.: HY-B1455

CAS No.: 18323-44-9

Molecular Formula: $C_{18}H_{33}ClN_2O_5S$

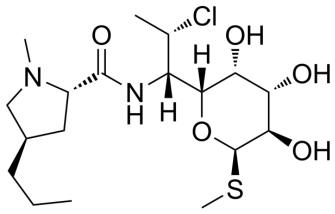
Molecular Weight: 424.98

Target: Bacterial; Antibiotic; Parasite

Pathway: Anti-infection

Storage: -20°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (294.13 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3531 mL	11.7653 mL	23.5305 mL
	5 mM	0.4706 mL	2.3531 mL	4.7061 mL
	10 mM	0.2353 mL	1.1765 mL	2.3531 mL

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

BIOLOGICAL ACTIVITY

Description

Clindamycin is an orally active and broad-spectrum bacteriostatic lincosamide antibiotic. Clindamycin can inhibit bacterial protein synthesis, possessing the ability to suppress the expression of virulence factors in Staphylococcus aureus at sub-inhibitory concentrations (sub-MICs). Clindamycin resistance results from enzymatic methylation of the antibiotic binding site in the 50S ribosomal subunit (23S rRNA). Clindamycin decreases the production of Panton-Valentine leucocidin (PVL), toxic-shock-staphylococcal toxin (TSST-1) or alpha-haemolysin (Hla). Clindamycin also can be used for researching malaria [1][2].

IC_{50} & Target

Plasmodium

In Vitro

Clindamycin (25 μg/mL, 1-18 h) inhibits ceftazidime (HY-B0593)-induced endotoxin release pretreatment in E. coli O55:B5[3].

Clindamycin (50-100 μg/mL, 0-12 min) enhances antibody- and complement-dependent phagocytosis in Staphylococcus aureus[4].

Clindamycin (0-500 μg/mL, 24-72 h) inhibits the cell proliferation in osteoblast cell culture model, stimulates the cell metabolism of osteoblast at the contratibution of 10 μg/mL[5].

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

Cell Viability Assay[3]

Cell Line:	THP-1 cells with stimulated E. coli O55:B5
Concentration:	3.13-50 μg/mL
Incubation Time:	1-18 h
Result:	Reduced TNF-α concentrations after pretreatment for 4 or 18 h at 25 and 50 μg/mL.

Cell Viability Assay[5]

Cell Line:	Osteoblast cell culture model
Concentration:	0-500 μg/mL
Incubation Time:	24-72 h
Result:	Increased alkaline phosphatase (ALP)-activity at 10 μg/mL at 24 and 48 h. Increased LDH values at 500 μg/mL at 24 and 48 h, indicating cytotoxicity. Increased calcification at 10 and 25 μg/mL,decreased or no calcification was found at 50 μg/ml.

In Vivo

Clindamycin (50-300 mg/kg; i.v., p.o.) dosage doesn't affect pharmacokinetic parameters in rats[6].
Clindamycin (160-600 mg/kg; i.v.) improves survival in a dose-dependent manner in endotoxic shock mouse model[7].
Clindamycin (17-50 mg/kg, i.v.) has good penetration into rat muscle tissue that can be applied to inhibit the main bacteria causing odontogenic infections[8].

Pharmacokinetic Analysis in Rat Model[8]

Route	Dose (mg/kg)	$t_{1/2}$ (h)	C_{max} (mg/mL)	T_{max} (h)a	$AUC_{0\text{-}inf}$ (h.mg/L)	⊠T
i.v.	51(Plasma)	2.51 (2.37–2.83)	35.21 (25.04–42.65)	0.08 ± 0.00	44.78 (28.82–65.65)	/
i.v.	51(Tissue)	2.82 (2.57–3.05)	14.20 (10.63–14.89)	0.25 ± 0.00	16.54 (13.83–18.35)	1.10
i.v.	17(Tissue)	3.25 (3.13–3.44)	4.82 (3.35–6.66	0.25 ± 0.00	/	/

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

Animal Model:	Endotoxic shock mouse model[7]
Dosage:	160-600 mg/kg
Administration:	Intravenous injection (i.v.)
Result:	Decreased survival rates to 92 and 36% treated with 520- and 600-mg/kg doses. Lowered the peak concentrations of tumor necrosis factor alpha (TNF-α) and interleukin-1 β (IL-1β) in serum. Increased the the peak concentrations of IL-6 in the sera.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2021 Mar 11.
- Water Res. 2023 May 21, 120110.
- EBioMedicine. 2022 Apr;78:103943.
- ACS Omega. March 3, 2022.
- bioRxiv. 2024 Jan 18.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Kremsner PG. Clindamycin in malaria treatment. J Antimicrob Chemother. 1990 Jan;25(1):9-14.

[2]. Kishi K, et al. Clindamycin suppresses endotoxin released by ceftazidime-treated Escherichia coli O55:B5 and subsequent production of tumor necrosis factor alpha and interleukin-1 beta. Antimicrob Agents Chemother. 1999 Mar;43(3):616-22.

[3]. Veringa EM, et al. Clindamycin at subinhibitory concentrations enhances antibody- and complement-dependent phagocytosis by human polymorphonuclear leukocytes of Staphylococcus aureus. Chemotherapy. 1987;33(4):243-9.

[4]. Naal FD, et al. The effects of clindamycin on human osteoblasts in vitro. Arch Orthop Trauma Surg. 2008 Mar;128(3):317-23.

[5]. Faggion PI, et al. Is the penetration of clindamycin into the masseter muscle really enough to treat odontogenic infections? Clin Oral Investig. 2021 May;25(5):3257-3266.

[6]. Yang SH, Lee MG. Dose-independent pharmacokinetics of clindamycin after intravenous and oral administration to rats: contribution of gastric first-pass effect to low bioavailability. Int J Pharm. 2007 Mar 6;332(1-2):17-23.

[7]. Hirata N, et al. Pretreatment of mice with clindamycin improves survival of endotoxic shock by modulating the release of inflammatory cytokines. Antimicrob Agents Chemother. 2001 Sep;45(9):2638-42.

[8]. Hodille E, et al. Clindamycin suppresses virulence expression in inducible clindamycin-resistant Staphylococcus aureus strains. Ann Clin Microbiol Antimicrob. 2018 Oct 20;17(1):38.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA