Chloramphenicol

Cat. No.: HY-B0239

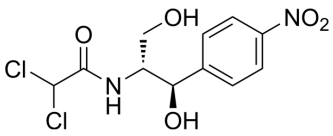
CAS No.: 56-75-7

Molecular Formula: $C_{11}H_{12}Cl_2N_2O_5$

Molecular Weight: 323.13

Target: Bacterial; Antibiotic; HIF/HIF Prolyl-Hydroxylase; VEGFR; Autophagy; Apoptosis; Beclin1; JNK; Akt; MMP

Pathway: Anti-infection; Metabolic Enzyme/Protease; Protein Tyrosine Kinase/RTK; Autophagy; Apoptosis; MAPK/ERK Pathway; PI3K/Akt/mTOR



Storage:

Powder	-20°C	3 years
	4°C	2 years

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

SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 150 mg/mL (464.21 mM)

Ethanol : 100 mg/mL (309.47 mM; Need ultrasonic)

H_2O : 3.06 mg/mL (9.47 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.0947 mL	15.4736 mL	30.9473 mL
5 mM	0.6189 mL	3.0947 mL	6.1895 mL
10 mM	0.3095 mL	1.5474 mL	3.0947 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.74 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.74 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.74 mM); Clear solution
4. Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.74 mM); Clear solution
5. Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.74 mM); Clear solution
6. Add each solvent one by one: 10% EtOH >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.74 mM); Clear solution
7. Add each solvent one by one: PBS

	Solubility: 2.5 mg/mL (7.74 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Chloramphenicol is an orally active, potent and broad-spectrum antibiotic. Chloramphenicol shows antibacterial activity. Chloramphenicol represses the oxygen-labile transcription factor and hypoxia inducible factor-1 alpha (HIF-1α) in hypoxic A549 and H1299 cells. Chloramphenicol suppresses the mRNA levels of vascular endothelial growth factor (VEGF) and glucose transporter 1, eventually decreasing VEGF release. Chloramphenicol can be used for anaerobic infections and lung cancer research[1][2][3].

IC_{50} & Target		
	JNK	MMP13

In Vitro

Chloramphenicol (1-100 μg/mL, 18-24 h) inhibits the HIF-1α pathway in NSCLC cells in a concentration-dependent manner[1].

Chloramphenicol (100 μg/mL, 0-24 h) induces autophagy in NSCLC cells, substantially increases the levels of autophagic biomarkers (beclin-1, Atg12-Atg5 conjugates, and LC3-II)[1].

Chloramphenicol induces abnormal differentiation and inhibits apoptosis in activated T cells[2].

Chloramphenicol can inhibit both bacterial and mitochondrial protein synthesis, causing mitochondrial stress and decreased ATP biosynthesis[3].

chloramphenicol (1-100 μg/mL) can induce matrix metalloproteinase (MMP)-13 expression and increase MMP-13 protein[3].

chloramphenicol (1-100 μg/mL) can activate c-Jun N-terminal kinases (JNK) and phosphatidylinositol 3-kinase (PI-3K)/Akt signaling, leading to c-Jun protein phosphorylation[3].

Chloramphenicol acts primarily on the 50S subunit of bacterial 70S rihosomes and inhibits peptide bond formation by suppressing peptidyl transferase activity[5].

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

Cell Viability Assay[1]

Cell Line:	A549 and H1299 cells
Concentration:	0, 1, 10, 100 μg/mL
Incubation Time:	3 h and 24 h
Result:	In the 3-h-treated group, the viability of A549 and H1299 cells at 100 μg/mL concentration was 97.0 ± 3.9% and 98.1 ± 5.0%, respectively. The viability of A549 cells was 102.9 ± 1.3% and 99.2 ± 0.9%, whereas the viability of H1299 cells was 103.3 ± 1.9% and 93.8 ± 4.5%, under hypoxia and treatment with CoCl2, respectively.

Western Blot Analysis[1]

Cell Line:	A549 and H1299 cells
Concentration:	0, 1, 10, 50, 100 μg/mL
Incubation Time:	18-24 h
Result:	Inhibited HIF-1α protein accumulation in NSCLC cells in a concentration-dependent manner, while the expression levels of ARNT remained unaltered. Had no effect on CoCl2 (250 μM, 3 h treatment)-mediated HIF-1α protein accumulation and SENP-1 protein reduction.

Western Blot Analysis[1]

Cell Line:	A549 and H1299 cells
Concentration:	100 μg/mL
Incubation Time:	0, 6, 12, 24 h
Result:	Induced autophagy in NSCLC cells in a time-dependent manner. Upregulats the expression of beclin-1 and increased the levels of Atg12-Atg5 conjugates in both NSCLC cell lines, both in a time dependent and concentration-dependent manner. Augmented LC3-II and downregulated p62/STSQM1 in A549 cells. Induced an augmentation of p62/STSQM1, and a decrease in LC3-II levels in H1299 cells.

In Vivo

Chloramphenicol (0-3500 mg/kg, Gavage, daily, for 5 days) decreases erythrocytes and erythrocyte precursors and reduces marrow erythroid cells were at day 1 post-dosing, and returns to normal by 14 days post-dosing[4].

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

Animal Model:	Female B6C3F1 mice (12-14 weeks old)[4]
Dosage:	0, 2500 and 3500 mg/kg
Administration:	Gavage, daily, for 5 days
Result:	Cessation of erythropoiesis was evident at day 1 post-dosing. A recovery was seen at day 7 post-dosing at the 2500 mg/kg dose level and at between 7 and 14 days at the 3500 mg/kg dose level. Myelotoxicity was most pronounced in the erythroid series at each dose level. Depressed femoral marrow BFU-E and CFU-E at day 1 post-dosing. All the blood and marrow parameters in the present study returned to normal by 14 days post-dosing.

CUSTOMER VALIDATION

- Nat Commun. 2022 Mar 2;13(1):1116.
- Sci Adv. 2023 Feb 17;9(7):eade4770.
- Theranostics. 2022 Jan 1;12(3):1187-1203.
- J Biomed Sci. 2024 May 13;31(1):50.
- Environ Pollut. 2020 Feb;257:113614.

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REFERENCES

[1]. Hsu HL, et al. Chloramphenicol Induces Autophagy and Inhibits the Hypoxia Inducible Factor-1 Alpha Pathway in Non-Small Cell Lung Cancer Cells. Int J Mol Sci. 2019 Jan 3;20(1):157.

[2]. Yuan ZR, et al. Chloramphenicol induces abnormal differentiation and inhibits apoptosis in activated T cells. Cancer Res. 2008 Jun 15;68(12):4875-81.

[3]. Li CH, et al. Chloramphenicol causes mitochondrial stress, decreases ATP biosynthesis, induces matrix metalloproteinase-13 expression, and solid-tumor cell invasion. Toxicol Sci. 2010 Jul;116(1):140-50.

[4]. Turton JA, et al. Characterization of the myelotoxicity of chloramphenicol succinate in the B6C3F1 mouse. Int J Exp Pathol. 2006 Apr;87(2):101-12.

[5]. Bartlett JG. Chloramphenicol. Med Clin North Am. 1982;66(1):91-102.

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