Bleomycin sulfate

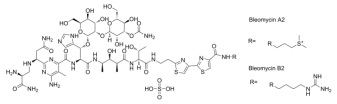
Cat. No.: HY-17565

CAS No.: 9041-93-4

Target: DNA/RNA Synthesis; Antibiotic

Pathway: Cell Cycle/DNA Damage; Anti-infection

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro	H_2O : 255 mg/mL (Need ultrasonic and warming) DMSO : 41.67 mg/mL (Need ultrasonic)
In Vivo	1. Add each solvent one by one: PBS Solubility: 100 mg/mL (Infinity mM); Clear solution; Need ultrasonic and warming and heat to 60°C 2. Add each solvent one by one: Saline Solubility: 100 mg/mL (Infinity mM); Clear solution; Need ultrasonic 3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.08 mg/mL (Infinity mM); Clear solution 4. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.08 mg/mL (Infinity mM); Clear solution 5. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (Infinity mM); Clear solution

BIOLOGICAL ACTIVITY

Description	Bleomycin sulfate is a DNA synthesis inhibitor. Bleomycin hydrochloride is a DNA damaging agent. Bleomycin sulfate is an antitumor antibiotic[1].
IC_{50} & Target	DNA/RNA Synthesis[1]
In Vitro	Bleomycin (BLM) sulfate is chosen as the best-studied micronucleus (MN) inducers in human lymphocytes with different mechanisms of genotoxicity. The most frequent Bleomycin-induced DNA lesions are single and double strand breaks and single apuinic/apyrimidinic sites. At the same time Bleomycin is true radiomimetic compound, resembling almost completely the genetic effect of ionizing radiation[1]. The IC_{50} value of Bleomycin sulfate for UT-SCC-19A cell line is 4.0±1.3 nM. UT-SCC-12A and UT-SCC-12B are both more resistant to Bleomycin (BLM); IC_{50} values are 14.2±2.8 nM and 13.0±1.1 nM, respectively[2]. Bleomycin sulfate (50, 100 μM; for 24, 48 h) induce pulmonary fibrosis in RLE-6TN cell (50 μM) and A549 cell (100 μM)[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Bleomycin suLfate (3.5-4.0 mg/kg; intratracheal instillation) significantly increased lung hydroxyproline levels and increased right caudal lobe mass; body weight decreased on day 4 and then increased on day 7[3].

Bleomycin suLfate (5.0 mg/kg/d; intratracheal instillation) induces pulmonary fibrosis and increases α-SMA and collagen I in BALB/c mice (20-30 g; 8 weeks old; male) The expression level[4].

Bleomycin sulfate (2.5 mg/kg; 1.25 mg/mL, 50 μL; intratracheal instillation) induces pulmonary fibrosis in male C57BL/6 mice (8 weeks old, approximately 24.5 g)[5].

Bleomycin sulfate is quickly absorbed following intramuscular, subcutaneous, intraperitoneal, or intrapleural administration and reaches peak plasma concentrations in approximately 60 min. Less than 1% of the drug given intravenously binds to plasma proteins, leading to high bioavailability. Additionally, a mean plasma drug clearance approaching 70 mL/min/m^2 has been calculated for Bleomycin sulfate. Bleomycin sulfate possesses a high plasma elimination rate and high urinary excretion rate[6].

Bleomycin sulfate can be used to construct model of pulmonary fibrosis.

Induction of Pulmonary Fibrosis[7]

- Background

  Bleomycin sulfate can lead to lung patchy parenchymal inflammation, epithelial cell injury with reactive hyperplasia, epithelial-mesenchymal transition, activation and differentiation of fibroblasts to myofibroblasts, and basement membrane and alveolar epithelium injures. The experimental use of Bleomycin sulfate is to induce pulmonary fibrosis animal models.

- Specific Mmodeling Methods

  Mice: C57BL/6 • 12-week-old

  Administration: 3-5 mg/kg • intratracheal administration • sprays on day one

  Note

  The mice were housed in separate stainless-steel cages (six mice per cage) in a temperature-controlled environment (20-24°C) on 12 h light-dark cycles with unrestricted access to food and water.

- Modeling Indicators

  Body quality changes: The appetite activity is reduced, with the fur less shiny, the spirits being lethargic, and the bodyweight decreasing. Showed shortness of breath, coughing, and noisy.

  Lung changes: Increased fibrotic consolidations, non-aerated lung area, and high-density lung area. Pulmonary function decreased.

  Molecular changes: Increased indicators: TGF-β1, TNF-α, IL-6, and GM-CSF in bronchoalveolar lavage fluid.

Correlated Product(s): Bleomycin hydrochloride (HY-17565A)

Opposite Product(s): Neotuberostemonine (HY-N3196)

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

Animal Model:	Male Fischer 344 rats, 8-10 week old, weighing 150-250 g[3]
Dosage:	3.5-4 mg/kg
Administration:	Intra-tracheal
Result:	Body weights decreased by day 4 then increased by Day 7 through the end of the study.

CUSTOMER VALIDATION

- Nat Metab. 2021 Dec 6.
- Small. 2021 Oct 8;e2103919.
- Redox Biol. 2021 Jul 26;46:102082.
- J Control Release. 2023 Aug;360:365-375.
- MedComm. 2023 Jul 12;4(4):e319.

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REFERENCES

[1]. Brandt JP, et, al. Bleomycin. National Library of Medicine. August 28, 202

[2]. Gul A, et, al. Pulmonary fibrosis model of mice induced by different administration methods of bleomycin. BMC Pulm Med. 2023 Mar 21;23(1):91.

[3]. Hovhannisyan G, et al. Comparative analysis of individual chromosome involvement in micronuclei induced by bleomycin in human leukocytes. Mol Cytogenet. 2016 Jun 21;9:49.

[4]. Jaaskela-Saari HA, et al. Squamous cell cancer cell lines: sensitivity to bleomycin and suitability for animal xenograft studies. Acta Otolaryngol Suppl. 1997;529:241-4.

[5]. Corboz MR, et al. Therapeutic administration of inhaled INS1009, a treprostinil prodrug formulation, inhibits bleomycin-induced pulmonary fibrosis in rats. Pulm Pharmacol Ther. 2018 Apr;49:95-103.

[6]. Ling Peng, et al. Scutellarin ameliorates pulmonary fibrosis through inhibiting NF-κB/NLRP3-mediated epithelial-mesenchymal transition and inflammation. Cell Death Dis. 2020 Nov 13;11(11):978.

[7]. Kang Miao, et al. Scutellarein inhibits BLM-mediated pulmonary fibrosis by affecting fibroblast differentiation, proliferation, and apoptosis. Ther Adv Chronic Dis. 2020 Jul 30;11:2040622320940185.

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