

Product Data Sheet

Product Name: MM-102
Cat. No.: GC14652

Chemical Properties

Cas. No. 1417329-24-8

Chemical Name N-[bis(4-fluorophenyl)methyl]-1-[[[(2S)-5-(diaminomethylideneamino)-2-[[2-ethyl-2-(2-methylpropanoylamino)butanoyl]amino]pentanoyl]amino]cyclopentane-1-carboxamide

SMILES CCC(CC)(C(=O)NC(CCCN=C(N)N)C(=O)NC1(CCCC1)C(=O)NC(C2=CC=C(C=C2)F)C3=CC=C(C=C3)F)NC(=O)C(C)C

Formula C₃₅H₄₉F₂N₇O₄ M.Wt 669.8

Solubility ≥ 67mg/mL in DMSO Storage Store at -20° C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

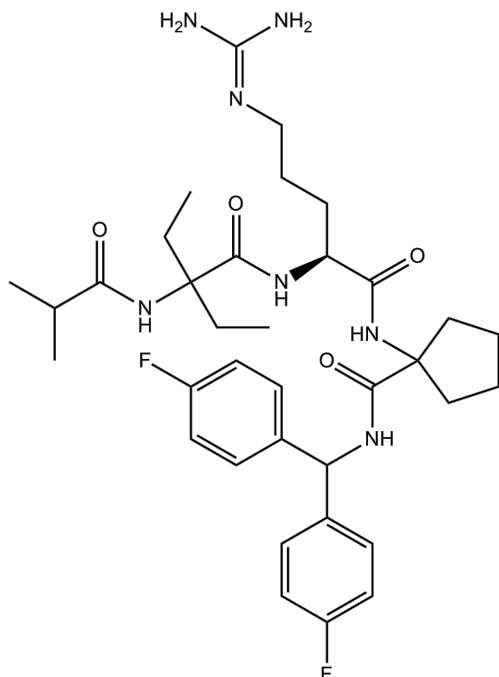
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:

Cell lines TFK1 and RBE cells (human cholangiocarcinoma cell lines)

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TFK1 and RBE cells were cultured in RPMI 1640 medium supplemented with Preparation Method 10% fetal bovine serum (FBS) at 37°C, 5% CO₂. Cells were treated with MM-102 at a concentration of 50µM for 24 hours.

Reaction
Conditions 50µM; 24h.

Applications MM-102 significantly inhibited the expression of ABCB1 in TFK1 and RBE cells, as determined by western blot analysis. MM-102 also suppressed the proliferation, migration, and chemoresistance of cholangiocarcinoma cells, demonstrated by reduced IC₅₀ values for cisplatin in CCK-8 assays and decreased colony formation in plate cloning assays. Additionally, MM-102 disrupted the interaction between WDR5 and KMT2A, leading to reduced H3K4 trimethylation at the ABCB1 promoter, thereby attenuating ABCB1-mediated drug efflux and enhancing cellular sensitivity to cisplatin.

Animal experiment [2]:

Animal models C57BL/6 mice with methylglyoxal (MGO)-induced peritoneal fibrosis

Preparation Method Mice received intraperitoneal injections of 40mM MGO in 2.5mL saline for 3 weeks (5 days/week). MM-102 was administered at 10mg/kg via intraperitoneal injection concurrently with MGO treatment. Mice were sacrificed after 21 days for peritoneal tissue analysis.

Dosage form 10mg/kg; i.p.; daily administration for 3 weeks.

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Applications

MM-102 significantly reduced peritoneal fibrosis by decreasing submesothelial compact zone thickness and cell density. MM-102 suppressed H3K4me3 and p16INK4a expression in peritoneal tissues, inhibited collagen deposition and α -SMA-positive myofibroblast accumulation, and attenuated inflammation by reducing IL-1 β , IL-6, and TNF- α levels. MM-102 also improved peritoneal membrane function by normalizing dialysate/plasma urea nitrogen and glucose absorption ratios.

References:

- [1] Wang D, Chen J, Wu G, et al. MBD2 regulates the progression and chemoresistance of cholangiocarcinoma through interaction with WDR5. J Exp Clin Cancer Res. 2024 Sep 30;43(1):272.
[2] Hara D, Sasaki K, Doi S, et al. Targeting MLL1/WDR5-Mediated Epigenetic Regulation Mitigates Peritoneal Fibrosis by Reducing p16INK4a. FASEB J. 2025 Apr 30;39(8):e70543.

Background

MM-102 is a high-affinity, cell-permeable peptidomimetic that functions as a potent inhibitor of the MLL1-WDR5 protein-protein interaction, with an IC₅₀ of 2.4nM and a K_i<1nM^[1-2]. MM-102 inhibits histone H3K4 methyltransferase activity by blocking the binding of WDR5 to the MLL1 complex. MM-102 is utilized in research areas such as leukemia and renal fibrosis^[3-4].

In vitro, treatment of human cholangiocarcinoma cells (TFK1 and RBE) with MM-102 (50 μ M) for 24 hours. MM-102 significantly inhibited the interaction between WDR5 and KMT2A, downregulated ABCB1 expression, and enhanced the sensitivity of the cholangiocarcinoma cells to cisplatin, while also inhibiting cell proliferation and migration capabilities^[5]. MM-102 (300pM) was used to treat synovial fibroblasts (RASFs) derived from patients with rheumatoid arthritis (RA) for 72 hours. MM-102 significantly reduced the levels of H3K4 trimethylation (H3K4me3) in the promoter regions of specific chemokine genes (CCL5, CXCL9, CXCL10, CXCL11) and correspondingly decreased the mRNA expression levels of these chemokine genes^[6].

In vivo, in a mouse model of renal ischemia-reperfusion injury (IRI), intraperitoneal administration of MM-102 (15mg/kg/day) for 7 consecutive days, starting from the day of surgery, MM-102 significantly

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suppressed the expression of MLL1, WDR5, and H3K4me3, reduced the accumulation of p16INK4a-positive cells, and alleviated renal fibrosis, inflammatory responses, and cellular senescence-related pathological changes^[7]. In a methylglyoxal (MGO)-induced peritoneal fibrosis mouse model using C57BL/6J mice, intraperitoneal administration of MM-102 (10mg/kg) for 3 weeks. MM-102 significantly attenuated MGO-induced peritoneal fibrosis, inflammation, and functional impairment^[8].

References:

- [1] Karatas H, Townsend EC, Cao F, et al. High-affinity, small-molecule peptidomimetic inhibitors of MLL1/WDR5 protein-protein interaction. *J Am Chem Soc.* 2013 Jan 16;135(2):669-82.
- [2] Zhang Z, Zhai Y, Ma X, et al. Down-Regulation of H3K4me3 by MM-102 Facilitates Epigenetic Reprogramming of Porcine Somatic Cell Nuclear Transfer Embryos. *Cell Physiol Biochem.* 2018;45(4):1529-1540.
- [3] Liu C, Qiu S, Li W, et al. MLL1 downregulation drives hair cell ferroptosis via mitochondrial and endoplasmic reticulum stress mechanisms through PERK-eIF2 α -Atf4-Chop and PI3K/Akt-Lrp1 signaling pathway. *Chin Med J (Engl).* 2025 Jul 10.
- [4] Koch MS, Deo M, Schmidt C, et al. KMT2A is a prerequisite of malignant transformation during IDH-mutant gliomagenesis. *Neuro Oncol.* 2026 Jan 14: noag006.
- [5] Wang D, Chen J, Wu G, et al. MBD2 regulates the progression and chemoresistance of cholangiocarcinoma through interaction with WDR5. *J Exp Clin Cancer Res.* 2024 Sep 30;43(1):272.
- [6] Okamoto K, Araki Y, Aizaki Y, et al. Regulation of cytokine and chemokine expression by histone lysine methyltransferase MLL1 in rheumatoid arthritis synovial fibroblasts. *Sci Rep.* 2024 May 9;14(1):10610.
- [7] Shimoda H, Doi S, Nakashima A, et al. Inhibition of the H3K4 methyltransferase MLL1/WDR5 complex attenuates renal senescence in ischemia reperfusion mice by reduction of p16INK4a. *Kidney Int.* 2019 Nov;96(5):1162-1175.
- [8] Hara D, Sasaki K, Doi S, et al. Targeting MLL1/WDR5-Mediated Epigenetic Regulation Mitigates Peritoneal Fibrosis by Reducing p16INK4a. *FASEB J.* 2025 Apr 30;39(8):e70543.

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