

Product Data Sheet

Product Name: ML-792
Cat. No.: GC36631

Chemical Properties

Cas. No. 1644342-14-2

SMILES O=S(OC[C@@H]1[C@@H](O)C[C@H](NC2=NC=NC=C2C(C3=NN(CC4=CC=CC(Br)=C4)C=C3)=O)C1)(N)=O

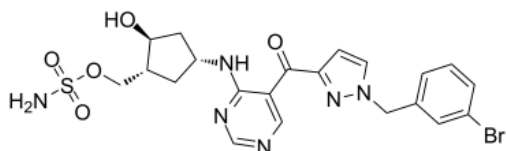
Formula $C_{21}H_{23}BrN_6O_5S$ M.Wt 551.41

Solubility DMSO: 100 mg/mL (181.35 mM) 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.

Structure



Protocol

Cell experiment [1]:

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Cell lines 5-FU-resistant cell line HCT-8/5-FU

Preparation Method 1000 HCT-8/5-FU cells/well were seeded in 6-well plates and incubated with 100μM 5-FU and 0, 1, or 5μM ML-792 for 12-16 days. Colony formation assay was performed.

Reaction Conditions 0, 1, 5μM; 12-16 days

Applications ML-792 significantly reduced colony formation numbers in HCT-8/5-FU cells.

Animal experiment [2]:

Animal models BALB/c-nude mice

Preparation Method HOS cells were suspended in culture media at a density of 1.0×10^6 /mL and subcutaneously transplanted onto the 6-week-old female BALB/c-nude mice (200μL). After weighing, the nude mice were randomly divided into three groups (6 in each group): control group, 50mg/kg ML-792, and 100mg/kg ML-792. Treatment begins when subcutaneous tumors form. The intraperitoneal administration regimen was two days a week. Tumors were measured weekly using calipers. The nude mice were killed before the tumor reached ethical size.

Dosage form 50, 100mg/kg; two days a week; i.p.

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Applications ML-792 significantly inhibited the growth of tumors, and the effect of the high concentration group was more obvious.

References:

- [1] Deng Y, Chen Y, Gao S, et al. RREB1-mediated SUMOylation enhancement promotes chemoresistance partially by transcriptionally upregulating UBC9 in colorectal cancer[J]. *Frontiers in Pharmacology*, 2024, 15: 1381860.
- [2] Jin X, Yin H, Bao J, et al. ML792 inhibits growth and TGF- β 1-induced EMT of osteosarcoma cells via TGF- β 1/Smad and PI3K/AKT pathways[J]. *All Life*, 2022, 15(1): 1222-1235.

Background

ML-792 is a specific inhibitor of small ubiquitin-like modifier (SUMO) activating enzyme (SAE), which can inhibit SAE/SUMO1 and SAE/SUMO2 with IC₅₀ of 3nM and 11nM, respectively^[1]. The SUMO protein pathway can control a series of important biological processes, including cell death, proliferation, differentiation, metabolism and signal transduction, by diversifying the functions, half-lives and partnerships of in situ target proteins^[2]. ML-792 is often used to study the role of SUMOylation in cancer, neurodegenerative diseases and viral infection^[3, 4].

In vitro, ML-792 (0, 1, 5 μ M) treatment of 5-FU-resistant cell lines (HCT-8/5-FU cells) for 12-16 days significantly reduced the number of cell colony formation and reversed the cell's 5-FU resistance^[5].

In vivo, ML-792 (50, 100mg/kg) was intraperitoneally injected into mice bearing HOS cell xenografts, which inhibited tumor growth in a dose-dependent manner^[6]. ML-792 (150, 200mg/kg) was subcutaneously injected into mice bearing HCT-116 cell xenografts, which inhibited tumor growth in a dose-dependent manner^[7].

References:

- [1] He X, Riceberg J, Soucy T, et al. Probing the roles of SUMOylation in cancer cell biology by using a selective SAE inhibitor[J]. *Nature chemical biology*, 2017, 13(11): 1164-1171.
- [2] Connolly J G, Plant L D. SUMO regulation of ion channels in health and disease[J]. *Physiology*,

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[3] Celen A B, Sahin U. Sumoylation on its 25th anniversary: mechanisms, pathology, and emerging concepts[J]. The FEBS journal, 2020, 287(15): 3110-3140.

[4] Sahin U, de Thé H, Lallemand-Breitenbach V. Sumoylation in physiology, pathology and therapy[J]. Cells, 2022, 11(5): 814.

[5] Deng Y, Chen Y, Gao S, et al. RREB1-mediated SUMOylation enhancement promotes chemoresistance partially by transcriptionally upregulating UBC9 in colorectal cancer[J]. Frontiers in Pharmacology, 2024, 15: 1381860.

[6] Jin X, Yin H, Bao J, et al. ML792 inhibits growth and TGF- β 1-induced EMT of osteosarcoma cells via TGF- β 1/Smad and PI3K/AKT pathways[J]. All Life, 2022, 15(1): 1222-1235.

[7] Langston S P, Grossman S, England D, et al. Discovery of TAK-981, a first-in-class inhibitor of SUMO-activating enzyme for the treatment of cancer[J]. Journal of medicinal chemistry, 2021, 64(5): 2501-2520.

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