

Product Data Sheet

Product Name: NGI-1
Cat. No.: GC33055

Chemical Properties

Cas. No. 790702-57-7

SMILES O=C(NC1=NC=C(C)S1)C2=CC(S(=O)(N(C)C)=O)=CC=C2N3CCCC3

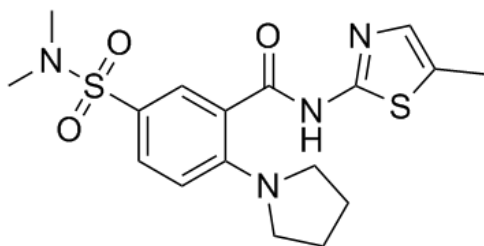
Formula $C_{17}H_{22}N_4O_3S_2$ M.Wt 394.51

Solubility DMSO : 160 mg/mL (405.57 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]:

Cell lines PC9 cells

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

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Address: 10292 Central Ave. #205, Montclair, CA, USA

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Growth rates were determined by CellTiter 96 NonRadioactive Cell Proliferation Assay (Promega) according to the manufacturer's directions. Briefly, NSCLC cells (2×10^3) untreated or treated with 10 μ M NGI-1, 100 μ M gefitinib, or 1 μ M osimertinib were seeded in triplicate in 96-wells plates and grown in culture medium containing 10% serum. The media were changed with or without new inhibitor every 48 hours.

Preparation Method

Reaction Conditions 10 μ M; 48h

Applications NGI-1 reduced the proliferation of both parental and T790M-expressing PC9 cell lines by approximately 90%.

Animal experiment [2]:

Animal models Oral squamous cell carcinoma xenograft model

Preparation Method NGI-1 was diluted with 10% DMSO + 40% PEG300 + 5% Tween 80 + 45% saline to a final concentration of 1mg/mL, and the PDL1 inhibitor was diluted with PBS to a final concentration of 0.5mg/mL. In animal experiments, SPF female C57BL/6 mice (6 weeks old) were used. MTCQ1 cells were used to establish a tumor xenograft model, and cells (5×10^6 cells/100 μ L PBS) were subcutaneously injected into the flank of mice, and tumor size was monitored 3 times a week. NGI-1 and PDL1 inhibitors were administered by abdominal injection 4 times a week at doses of 10mg/kg and 5mg/kg, respectively.

Dosage form 10mg/kg; ip; 14d

Applications Combined inhibition of EREG glycosylation by targeting NGI-1 significantly enhanced the efficacy of anti-PDL1 inhibitors in vivo.

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References:

- [1]. Lopez Sambrooks C, Baro M, Quijano A, et al. Oligosaccharyltransferase inhibition overcomes therapeutic resistance to EGFR tyrosine kinase inhibitors. *Cancer research*. 2018 Sep 1; 78(17): 5094-5106.
- [2]. Xu S, Wang H, Zhu Y, et al. Stabilization of EREG via STT3B-mediated N-glycosylation is critical for PDL1 upregulation and immune evasion in head and neck squamous cell carcinoma. *International Journal of Oral Science*. 2024 Jul 1; 16(1): 47.

Background

NGI-1 (ML414) is an effective oligosaccharyl transferase (OST) inhibitor [1]. NGI-1 selectively inhibits the STT3A and STT3B subunits in the oligosaccharyl transferase (OST) complex, thereby blocking the N-linked glycosylation process of proteins [2-3]. NGI-1 is often used to study the regulation of glycosylation-dependent proteins such as EGFR in tumor cells and has certain anti-tumor potential [4].

In PC9 cells, NGI-1 (10 μ M; 48h) reduced the proliferation of both parental and T790M-expressing PC9 cell lines by approximately 90% [5]. In HEK293 cells, NGI-1 (5 μ M; 60min) treatment results in cell type-dependent dysfunction of HSV-1 [2]. In Caco2 cells, NGI-1 (1 μ M, 5 μ M; 3d) has a significant inhibitory effect on SARS-CoV-2 [6]. In SKMG3 cells, NGI-1 (10 μ M; 48h) disrupts RTK signaling in glioblastoma cells [7].

In oral squamous cell carcinoma xenograft model, combined inhibition of EREG glycosylation by targeting NGI-1 (10mg/kg; ip; 14d) significantly enhanced the efficacy of anti-PDL1 inhibitors in vivo [8]. In lung adenocarcinoma xenograft model, the tumor volume and wet weight of NGI-1 (20mg/kg; ip; 24d) treated mice were significantly smaller than those of the DMSO control group at the end point [9]. In CD24-WT nude mice, NGI-1 (20mg/kg; ip; 24d) treatment significantly inhibited tumor growth [10].

References:

- [1]. Rinis N, Golden JE, Marceau CD, et al. Editing N-glycan site occupancy with small-molecule oligosaccharyltransferase inhibitors. *Cell chemical biology*. 2018 Oct 18; 25(10): 1231-1241.

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- [2]. Lu H, Cherepanova NA, Gilmore R, et al. Targeting STT3A-oligosaccharyltransferase with NGI-1 causes herpes simplex virus 1 dysfunction. The FASEB Journal. 2019 Feb 27;33(6):6801.
- [3]. Harada Y, Ohkawa Y, Kizuka Y, et al. Oligosaccharyltransferase: a gatekeeper of health and tumor progression. International journal of molecular sciences. 2019 Dec 2; 20(23): 6074.
- [4]. Cao Y, Yi W, Zhu Q. Glycosylation in the tumor immune response: the bitter side of sweetness: Glycosylation in the tumor immune response. Acta Biochimica et Biophysica Sinica. 2024 Jun 28; 56(8): 1184.
- [5]. Lopez Sambrooks C, Baro M, Quijano A, et al. Oligosaccharyltransferase inhibition overcomes therapeutic resistance to EGFR tyrosine kinase inhibitors. Cancer research. 2018 Sep 1; 78(17): 5094-5106.
- [6]. Huang YJ, Zhao H, Huang X, et al. Identification of oligosaccharyltransferase as a host target for inhibition of SARS-CoV-2 and its variants. Cell Discovery. 2021 Nov 30; 7(1): 116.
- [7]. Baro M, Lopez Sambrooks C, Quijano A, et al. Oligosaccharyltransferase inhibition reduces receptor tyrosine kinase activation and enhances glioma radiosensitivity. Clinical Cancer Research. 2019 Jan 15; 25(2): 784-795.
- [8]. Xu S, Wang H, Zhu Y, et al. Stabilization of EREG via STT3B-mediated N-glycosylation is critical for PDL1 upregulation and immune evasion in head and neck squamous cell carcinoma. International Journal of Oral Science. 2024 Jul 1; 16(1): 47.
- [9]. Cheng J, Xia L, Hao X, et al, Tavolari S. Targeting STT3A produces an anti-tumor effect in lung adenocarcinoma by blocking the MAPK and PI3K/AKT signaling pathway. Translational Lung Cancer Research. 2022 Jun; 11(6): 1089.
- [10]. Wang J, Zhang HM, Zhu GH, et al. STT3-mediated aberrant N-glycosylation of CD24 inhibits paclitaxel sensitivity in triple-negative breast cancer. Acta Pharmacologica Sinica. 2025 Apr; 46(4): 1097-1110.

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