

Product Data Sheet

Product Name: MYCi975

Cat. No.: GC38588

Chemical Properties

Cas. No. 2289691-01-4

SMILES OC1=C(C2=CC(C(F)(F)F)=NN2C)C=CC(OCC3=CC=C(Cl)C=C3)=C1C4=CC=C(Cl)C(C(F)(F)F)=C4

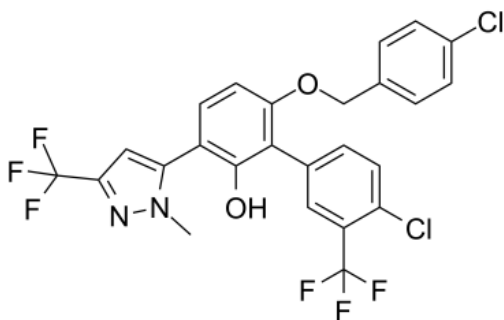
Formula $C_{25}H_{16}Cl_2F_6N_2O_2$ M.Wt 561.3

Solubility DMSO: ≥ 250 mg/mL (445.39 mM) Storage Store at $-20^{\circ}C$

General tips For obtaining a higher solubility, please warm the tube at $37^{\circ}C$ and shake it in the ultrasonic bath for a while. Stock solution can be stored below $-20^{\circ}C$ 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

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

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

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

Product Data Sheet

Cell experiment [1]:

Cell lines HS578T cells

Preparation Method For measuring apoptosis, cells were seeded in 6-well plates at a density of 2×10^5 per well and incubated overnight at 37°C. Cells were then treated with dimethyl sulfoxide (DMSO) or 10μM MYCi975. Following 48h of incubation, they were harvested and stained with annexin-V and propidium iodide using the Annexin-V-FITC Apoptosis Detection Kit.

Reaction Conditions 10μM; 48h

Applications In HS578T cells, MYCi975 treatment induced apoptosis in up to 80% of cells after 48h of incubation.

Animal experiment [2]:

Animal models Head and neck squamous cell carcinoma (HNSCC) nude mouse model

Preparation Method For the orthotopic nude mouse model of HNSCC, EpCAM + ALDH^{high} CSCs from HNSCC PDX were injected into the tongues of nude mice. One week after injection, the mice were divided into four groups and given: 1) control vehicle; 2) cisplatin (3mg/kg body weight once a week); 3) MYCi975 (100mg/kg); and 4) MYCi975 plus cisplatin for 4 weeks.

Dosage form 100mg/kg; ip; 4 weeks

Applications MYCi975 alone or MYCi975 plus cisplatin significantly reduced lymph node metastasis.

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

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

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

Product Data Sheet

References:

- [1]. Tang M, O'Grady S, Crown J, et al. MYC as a therapeutic target for the treatment of triple-negative breast cancer: preclinical investigations with the novel MYC inhibitor, MYCi975. *Breast Cancer Research and Treatment*. 2022 Sep; 195(2): 105-115.
- [2]. Liu S, Qin Z, Mao Y, et al. Pharmacological inhibition of MYC to mitigate chemoresistance in preclinical models of squamous cell carcinoma. *Theranostics*. 2024 Jan 1; 14(2): 622.

Background

MYCi975 is a small molecule inhibitor of c-Myc [1]. MYCi975 can directly bind to c-Myc protein, block its heterodimer formation with Max, and promote phosphorylation and protein degradation of c-Myc at Thr58, thereby inhibiting c-Myc-driven transcriptional activity and cancer cell proliferation [2-3]. MYCi975 is commonly used to treat cancer [4].

In HS578T cells, MYCi975 (10 μ M; 48h) treatment induced apoptosis in up to 80% of cells after 48 h of incubation [5]. In MDA-MB-231 cells, combination treatment of COTI-2 and MYCi975 (0.16-10 μ M; 5d) produced a synergistic growth inhibitory effect on cells [6]. In A549 cells, combination of MYCi975 (2.5-10 μ M; 72h) and TMP269 significantly reduced the viability of NSCLC cell lines [7].

In head and neck squamous cell carcinoma (HNSCC) nude mouse model, MYCi975 (100mg/kg; ip; 4 weeks) alone or MYCi975 plus cisplatin significantly reduced lymph node metastasis [8]. In both HNSCC xenograft and syngeneic murine models, MYCi975(100mg/kg; ip; 4 weeks) inhibited HNSCC growth [9].

References:

- [1]. Makii R, Ned T, Underdown MJ, et al. 582 A comparative approach to understanding the role of oncogenic MYC signaling in the metastatic osteosarcoma tumor immune microenvironment. *Journal of Clinical and Translational Science*. 2025 Apr 1; 9(s1): 171.
- [2]. Holmes AG, Parker JB, Sagar V, et al. A MYC inhibitor selectively alters the MYC and MAX cistromes and modulates the epigenomic landscape to regulate target gene expression. *Science Advances*. 2022 Apr 27; 8(17): eab3635.
- [3]. Vízkeleti L, Spisák S. Rewired metabolism caused by the oncogenic deregulation of MYC as an

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

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

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

Product Data Sheet

attractive therapeutic target in cancers. Cells. 2023 Jun 29; 12(13): 1745.

[4]. Whitfield JR, Soucek L. MYC in cancer: from undruggable target to clinical trials. Nature Reviews Drug Discovery. 2025 Feb 19: 1-3.

[5]. Tang M, O'Grady S, Crown J, et al. MYC as a therapeutic target for the treatment of triple-negative breast cancer: preclinical investigations with the novel MYC inhibitor, MYCi975. Breast Cancer Research and Treatment. 2022 Sep; 195(2): 105-115.

[6]. Tang M, Crown J, Duffy MJ. Degradation of MYC by the mutant p53 reactivator drug, COTI-2 in breast cancer cells. Investigational New Drugs. 2023 Aug; 41(4):5 41-550.

[7]. Park J, Chen YY, Cao JJ, et al. MYC plus class IIa HDAC inhibition drives mitochondrial dysfunction in non-small cell lung cancer. Cell Reports. 2025 Jun 24; 44(6).

[8]. Liu S, Qin Z, Mao Y, et al. Pharmacological inhibition of MYC to mitigate chemoresistance in preclinical models of squamous cell carcinoma. Theranostics. 2024 Jan 1; 14(2): 622.

[9]. Liu S, Qin Z, Mao Y, et al. Therapeutic targeting of MYC in head and neck squamous cell carcinoma. Oncoimmunology. 2022 Dec 31; 11(1): 2130583.

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

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

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