

Product Data Sheet

Product Name: THZ-P1-2

Cat. No.: GC39823

Chemical Properties

Cas. No. 2058075-45-7

SMILES O=C(C(C=C1)=CC=C1NC(/C=C/CN(C)C)=O)NC2=CC=CC(NC3=CC(C4=CNC5=CC=C(C=C45)=NC=N3)=C2

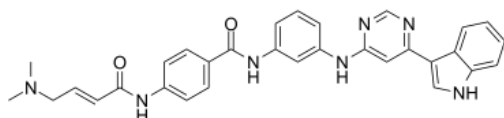
Formula $C_{31}H_{29}N_7O_2$ M.Wt 531.61

Solubility Soluble in DMSO Storage 4°C, protect from light

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 NB4 cells

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

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Preparation Method A total of 2×10^4 NB4 cells/well were seeded into the appropriate medium in 96-well plates and incubated for 48 hours with various concentrations of THZ-P1-2 (1.6, 3.2, and $6.4 \mu\text{M}$). Next, $10 \mu\text{l}$ of methyl thiazolyl tetrazolium (MTT) solution (5mg/mL) was added and incubated at 37°C and 5% CO_2 for 4 hours. The reaction was terminated with $100 \mu\text{l}$ of anhydrous isopropanol solution of 0.1M HCl. Cell viability was assessed by measuring the absorbance at 570nm.

Reaction Conditions 1.6, 3.2, and $6.4 \mu\text{M}$; 48h

Applications THZ-P1-2 treatment inhibited the cell viability of NB4 cells in a dose-dependent manner.

References:

[1] Lima K, Carvalho M F L, Pereira-Martins D A, et al. Pharmacological inhibition of PIP4K2 potentiates venetoclax-induced apoptosis in acute myeloid leukemia[J]. International Journal of Molecular Sciences, 2023, 24(23): 16899.

Background

THZ-P1-2 is a potent inhibitor of PI5P4K kinase, with the IC_{50} values of $0.95 \mu\text{M}$ and $5.9 \mu\text{M}$ for PI5P4K α and PI5P4K β , respectively [1]. THZ-P1-2 binds in the active site and covalently modifies cysteine residues of the PI5P4K kinases[2]. THZ-P1-2 has been widely used in cell models to regulate autophagy[3].

In vitro, THZ-P1-2 treatment for 4 hours inhibited SARS-CoV-2 replication in Calu-3 cells with an EC_{50} value of $0.2 \mu\text{M}$ and inhibited cell viability ($\text{CC}_{50} = 16 \mu\text{M}$)[4]. THZ-P1-2 treatment ($6.4 \mu\text{M}$) for 48h significantly inhibited NB4 cell viability and enhanced venetoclax-induced apoptosis[5]. Treatment with $6.4 \mu\text{M}$ THZ-P1-2 for 72h significantly reduced Jurkat cell viability and induced DNA damage, triggered apoptosis, and disrupted mitochondrial homeostasis and autophagic flux in cells[6].

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- [1] Manz T D, Sivakumaren S C, Yasgar A, et al. Structure–activity relationship study of covalent pan-phosphatidylinositol 5-phosphate 4-kinase inhibitors[J]. ACS medicinal chemistry letters, 2019, 11(3): 346-352.
- [2] Sivakumaren S C, Shim H, Zhang T, et al. Targeting the PI5P4K lipid kinase family in cancer using covalent inhibitors[J]. Cell chemical biology, 2020, 27(5): 525-537. e6.
- [3] Lima K, Nogueira F L, Cipelli M, et al. Potency and efficacy of pharmacological PIP4K2 inhibitors in acute lymphoblastic leukemia[J]. European Journal of Pharmacology, 2024, 977: 176723.
- [4] Karim M, Mishra M, Lo C W, et al. PIP4K2C inhibition reverses autophagic flux impairment induced by SARS-CoV-2[J]. Nature Communications, 2025, 16(1): 6397.
- [5] Lima K, Carvalho M F L, Pereira-Martins D A, et al. Pharmacological inhibition of PIP4K2 potentiates venetoclax-induced apoptosis in acute myeloid leukemia[J]. International Journal of Molecular Sciences, 2023, 24(23): 16899.
- [6] Lima K, Pereira-Martins D A, de Miranda L B L, et al. The PIP4K2 inhibitor THZ-P1-2 exhibits antileukemia activity by disruption of mitochondrial homeostasis and autophagy[J]. Blood cancer journal, 2022, 12(11): 151.

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