ML162

Cat. No.: HY-100002

CAS No.: 1035072-16-2

Molecular Formula: $C_{23}H_{22}Cl_2N_2O_3S$

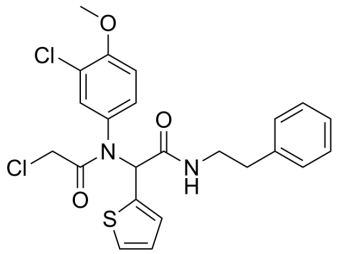
Molecular Weight: 477.4

Target: Ferroptosis; Glutathione Peroxidase

Pathway: Apoptosis; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (209.47 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0947 mL	10.4734 mL	20.9468 mL
	5 mM	0.4189 mL	2.0947 mL	4.1894 mL
	10 mM	0.2095 mL	1.0473 mL	2.0947 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

ML162 is a covalent glutathione peroxidase 4 (GPX4) inhibitor. ML162 has a selective lethal effect on mutant RAS oncogene-expressing cell lines[1][2]

In Vitro

ML162 (compound 1a) shows nanomolar potencies against two $HRAS^{G12V}$ expressing cell lines, with IC_{50} values of 25 nM and 578 nM for HRASG12V-expressing and wild-type BJ fibroblasts, respectively[1].

ML162 (8 μM; 24 hours) treatment increases the expression of p62 and Nrf2 in chemoresistant HN3R and HN3-rslR cells, inactivates Keap1, and increases expression of the phospho-PERK-ATF4-SESN2 pathway[2].

ML162 induces the head and neck cancer (HNC) cell death to varying degrees, with parental HN3 cells more sensitive and cisplatin-resistant (HN3R) and acquired RSL3-resistant (HN3-rslR) cells less sensitive[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	HN3R cells
Concentration:	8 μM
Incubation Time:	24 hours
Result:	Increased the expression of p62 and Nrf2 in chemoresistant HN3R and HN3-rslR cells.

CUSTOMER VALIDATION

- ACS Appl Mater Interfaces. 2022 Nov 18.
- Am J Cancer Res. 2023 Feb 28.

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REFERENCES

[1]. Michel Weïwer, et al. Development of small-molecule probes that selectively kill cells induced to express mutant RAS. Bioorg Med Chem Lett. 2012 Feb 15;22(4):1822-6.

[2]. Daiha Shin, et al. Nrf2 inhibition reverses resistance to GPX4 inhibitor-induced ferroptosis in head and neck cancer. Free Radic Biol Med. 2018 Dec;129:454-462.

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