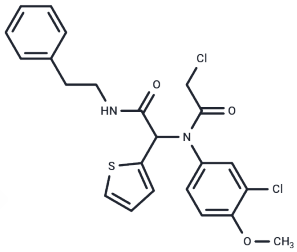
Data Sheet (Cat.No.T8970)

TargetMol

ML162

Chemical Properties



CAS No. : 1035072-16-2

Formula: C23H22Cl2N2O3S

Molecular Weight: 477.4

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	ML162 is a covalent glutathione peroxidase 4 (GPX4) inhibitor that induces ferroptosis. ML162 has antitumor activity and selectively inhibits cell lines expressing mutant RAS oncogenes.
Targets(IC50)	Ferroptosis,Glutathione Peroxidase,GPX
In vitro	**METHODS**: Human lung cancer cells A549 were pretreated with ML162 (0.1-15 μM) for 4-24 h, and TXNRD1 activity was measured with the RX1 activity probe. **RESULTS**: RX1 signaling was significantly inhibited dose-dependently at ML162 concentrations of 0.5 μM or higher. there was also a fairly rapid onset of concentration-dependent inhibition of cellular TXNRD1 activity, and incubation with 1 μM or higher ML162 treatment for 4 h was sufficient to inhibit RX1 signaling. mL162 effectively inhibited cellular TXNRD1 activity. ML162 effectively inhibits cellular TXNRD1 activity. ML162 effectively inhibited cellular TXNRD1 activity.[1] **METHODS**: Melanoma cells A2058 and A375 were pretreated with ferrostatin (10 μM), Z-VAD-FMK (10 μM), or necrosulfonamide (0.5 μM) for 24 h. Cell viability was assayed after ML162 (1-16 μM) treatment. **RESULTS**: ML162 caused cell death in a dose-dependent manner in the A2058 and A375 melanoma cell lines, which could be reversed by ferrostatin-1, a ferritin-specific inhibitor, but not by Z-VAD-FMK, an inhibitor of apoptosis, or necrosulfonamide, an inhibitor of necrotic apoptosis. [2]
In vivo	**METHODS**: To detect anti-tumor activity in vivo, ML162 (40 mg/kg once daily) and anti-PD-1 antibody (200 μg every three days) were intraperitoneally injected into BALB/c mice bearing TS/A tumors once daily for two weeks. **RESULTS**: The combination of GPX4 inhibitor and anti-PD-1 antibody significantly inhibited tumor growth compared with monotherapy. The combination therapy did not result in additional immune cell infiltration compared to monotherapy, but the combination therapy induced a significant immune response with an increased proportion of PRF1+CD8+ T cells and GZMB+CD8+ T cells. [3]

Solubility Information

Solubility	DMSO: 247 mg/mL (517.39 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 0.48 mg/mL (1.01 mM),Solution. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
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
50 mM	0.0419 mL	0.2095 mL	0.4189 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Cheff DM, et al. The ferroptosis inducing compounds RSL3 and ML162 are not direct inhibitors of GPX4 but of TXNRDRedox Biol. 2023 Jun;62:102703.

Li H, Yu K, Hu H, et al.METTL17 coordinates ferroptosis and tumorigenesis by regulating mitochondrial translation in colorectal cancer.Redox Biology.2024: 103087.

Wang H, et al. Targeting Wnt/β-Catenin Signaling Exacerbates Ferroptosis and Increases the Efficacy of Melanoma Immunotherapy via the Regulation of MITF. Cells. 2022 Nov 12;11(22):3580.

Yang F, et al. Ferroptosis heterogeneity in triple-negative breast cancer reveals an innovative immunotherapy combination strategy. Cell Metab. 2023 Jan 3;35(1):84-100.e8.