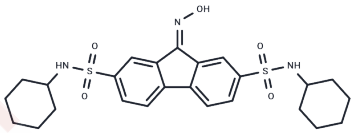
Data Sheet (Cat.No.T4066)

TargetMol

FIN56

Chemical Properties



CAS No. : 1083162-61-1

Formula: C25H31N3O5S2

Molecular Weight: 517.66

Storage: The compound is unstable in solution. Please use soon
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	FIN56 is an iron death-specific inducer that binds to and activates squalene synthase, inducing ferroptosis by triggering GPX4 degradation. FIN56 can be used in studies of ferroptosis regulatory mechanisms, autophagy-ferroptosis crosstalk, and tumor therapy.
Targets(IC50)	Ferroptosis
In vitro	**Methods**: Human bladder cancer cell lines J82, 253J, T24, and RT-112 were treated with FIN56 at concentration gradients (0.3, 1, 3, 10 μM) for 3, 6, 9, 24, and 72 hours, and cell viability was assessed using the CCK-8 assay. **Results**: FIN56 induced cell death in all four cell lines. [1] **Methods**: FIN56 (10 μM), α-tocopherol (ferroptosis inhibitor), and Liproxstatin-1 (ferroptosis inhibitor) were added to 253J and T24 cells and incubated for 24 hours; cell viability was assessed using the CCK-8 assay. **Results**: Both ferroptosis inhibitors significantly inhibited FIN56-induced cell death, confirming that FIN56 exerts its effects via ferroptosis. [1] **Methods**: Human glioblastoma LN229 and U118 cells. The cells were treated with 1 μM FIN56 for 24 h, or with 1 μM Ferrostatin-1 for 1 h followed by co-treatment with FIN56. Lipid peroxidation and ferroptosis levels were assessed using BODIPY 581/591 C11, CellRox Green, 4-HNE immunofluorescence, and transmission electron microscopy. **Results**: FIN56 significantly induced lipid peroxidation and ferrocytosis in LN229 and U118 cells, and this effect was blocked by Ferrostatin-1. [2]
In vivo	**Methods**: A subcutaneous tumor xenograft model was established in LN229-bearing nude mice. FIN56 (10 mg/kg) or a control agent was administered intraperitoneally for 30 consecutive days. Tumor volume, Ki67 (proliferation), and 4-HNE (ferroptosis) were assessed. **Results**: FIN56 significantly reduced tumor volume, decreased the proportion of Ki67-positive cells, and increased 4-HNE levels, thereby inhibiting glioblastoma growth and inducing ferroptosis in vivo. [2]
Cell Research	1000 cells/36 μL are seeded in each well in 384-well plates. Lethal compounds are dissolved and a 2-fold, 12-point dilution series are prepared in DMSO. Compound solutions are further diluted with media at 1:25 and 4 μL/well of the diluted solutions are added to cell cultures immediately after cells are seeded. When ferroptosis

Cell Research	(100 μM α-tocopherol, 152 μM deferoxamine, or 10 μM U-0126) are co-treated with lethal inducers, they are supplemented to cell culture at the same time as lethal compounds are added, and the cells are incubated for 24 hrs. When other cell death modulating compounds (100 nM sodium selenite, 1 μM cerivastatin, 100 μg/mL mevalonic acid) are co-treated, they are first supplemented to cell culture for 24 hrs before lethal compounds are added to cell culture and further incubated for 24 hrs at 37°C under 5% CO2. On the day of the viability measurement, 10 μL/well of 50% Alamar Blue diluted in media is added and further incubated at 37°C for 6 hrs. Fluorescence intensity (ex/em: 530/590) is measured with a Victor 3 plate reader and the normalized viability is calculated by VL = (IL-I0)/(IV-I0), where VL, I0, IV, and IL are the normalized viability, raw fluorescence intensities from the wells containing media, cells treated with a vehicle (negative control), and cells with the lethal compound (L), respectively. When the effect of a chemical modulator (M) on L is calculated, we instead used the equation: VL|M = (IM, L-I0)/(IM, V-I0), where VL|M, IM, L and IM, V are the normalized viability, and fluorescence intensity from cells treated with M and V, and from cells with M and L. respectively. The viability is typically measured in biological triplicates unless otherwise specified. A representative dose-response curve, the mean and standard error of normalized viability from one replicate are plotted.

Solubility Information

Solubility	DMSO: 247.5 mg/mL (478.11 mM),The compound is unstable in solution, please use soon. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+90% Corn Oil: 3.3 mg/mL (6.37 mM),Sonication is recommended. *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	1.9318 mL	9.6588 mL	19.3177 mL
5 mM	0.3864 mL	1.9318 mL	3.8635 mL
10 mM	0.1932 mL	0.9659 mL	1.9318 mL
50 mM	0.0386 mL	0.1932 mL	0.3864 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

Sun Y, et al. Fin56-induced ferroptosis is supported by autophagy-mediated GPX4 degradation and functions synergistically with mTOR inhibition to kill bladder cancer cells. Cell Death Dis. 2021 Oct 29;12(11):1028.
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Zhang X, et al. FIN56, a novel ferroptosis inducer, triggers lysosomal membrane permeabilization in a TFEB-dependent manner in glioblastoma. J Cancer. 2021 Sep 13;12(22):6610-6619.
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In vivo vulnerabilities to GPX4 and HDAC inhibitors in drug-persistent versus drug-resistant BRAFV600E lung adenocarcinoma