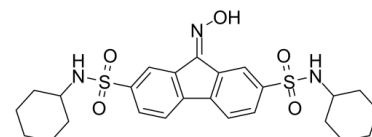


## Data Sheet

<b>Product Name:</b>	FIN56
<b>Cat. No.:</b>	CS-7632
<b>CAS No.:</b>	1083162-61-1
<b>Molecular Formula:</b>	C <sub>25</sub> H <sub>31</sub> N <sub>3</sub> O <sub>5</sub> S <sub>2</sub>
<b>Molecular Weight:</b>	517.66
<b>Target:</b>	Ferroptosis
<b>Pathway:</b>	Apoptosis
<b>Solubility:</b>	DMSO : ≥ 100 mg/mL (193.18 mM); H <sub>2</sub> O : < 0.1 mg/mL (insoluble)



### BIOLOGICAL ACTIVITY:

FIN56 is a specific inducer of ferroptosis. **In Vitro:** FIN56 causes the loss of GPX4 activity in cell lysates. FIN56-induced cell death is suppressed by GFP-GPX4 fusion protein overexpression. FIN56 triggers ferroptosis through a mechanism involving the regulation of GPX4 protein abundance<sup>[1]</sup>.

### PROTOCOL (Extracted from published papers and Only for reference)

**Cell Assay:** <sup>[1]</sup>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 inhibitors (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% CO<sub>2</sub>. 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.

### References:

[1]. Shimada K, et al. Global survey of cell death mechanisms reveals metabolic regulation of ferroptosis. Nat Chem Biol. 2016 Jul;12(7):497-503.

### CAIndexNames:

9H-Fluorene-2,7-disulfonamide, N2,N7-dicyclohexyl-9-(hydroxyimino)-

### SMILES:

O=S(C1=CC(/C2=N\O)=C(C3=C2C=C(C(S(=O)(NC4CCCC4)=O)C=C3)C=C1)(NC5CCCC5)=O

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

Tel: 732-484-9848 Fax: 888-484-5008 E-mail: [sales@ChemScene.com](mailto:sales@ChemScene.com)

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA