CIL56

Cat. No.: HY-112063

CAS No.: 300802-28-2

Molecular Formula: $C_{23}H_{27}N_3O_5S_2$

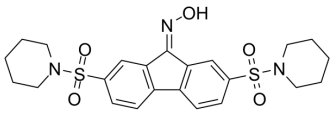
Molecular Weight: 489.61

Target: Ferroptosis

Pathway: Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 41.67 mg/mL (85.11 mM; ultrasonic and warming and heat to 60°C)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0424 mL	10.2122 mL	20.4244 mL
5 mM	0.4085 mL	2.0424 mL	4.0849 mL
10 mM	0.2042 mL	1.0212 mL	2.0424 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (5.11 mM); Suspended solution; Need ultrasonic and warming
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.25 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.25 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CIL56 is a potent and selective ferroptosis inducer. Ferroptosis is an iron-dependent form of regulated cell death (RCD).

In Vitro

Ferroptosis is a non-apoptotic form of regulated cell death observed in cancer cells, kidney cells and neurons.CIL56 induces iron-dependent reactive oxygen species (ROS). Antioxidants and iron chelators only suppress the lethality of low concentrations of CIL56[1]. CIL56 triggers cell death dependent upon the rate-limiting de novo lipid synthetic enzyme ACC1.Using mass spectrometry, the metabolome of HT-1080 cells treated with CIL56 (6.5 μM)±TOFA (4 μM) is analyzed, compared to vehicle-treated controls. Among the 298 polar and nonpolar metabolites identified in this analysis, the levels of

141 metabolites are significantly altered by CIL56 treatment, with 82 metabolites significantly increased and 59 significantly decreased (FDR q<0.01). CIL56 triggers the striking TOFA-sensitive accumulation of all detectable long chain saturated and monounsaturated fatty acids and all detectable polyunsaturated fatty acids. A plausible model to account for the accumulation of both nonessential and essential fatty acids species is that CIL56 inhibits the normal breakdown of fatty acids by mitochondrial β-oxidation. CIL56 accelerates the ACC1-dependent production of malonyl-CoA, a metabolite that acts as a negative regulator of this process[2].

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

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.

[2]. Dixon SJ, et al. Human Haploid Cell Genetics Reveals Roles for Lipid Metabolism Genes in Nonapoptotic Cell Death. ACS Chem Biol. 2015 Jul 17;10(7):1604-9.

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