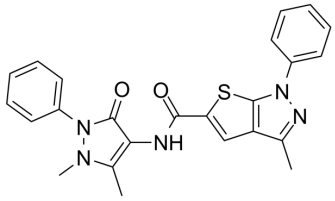
MYLS22

Cat. No.:	HY-136446		
CAS No.:	306959-01-3		
Molecular Formula:	$C_{24}H_{21}N_5O_2S$		
Molecular Weight:	444		
Target:	Others		
Pathway:	Others		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (56.31 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2523 mL	11.2613 mL	22.5225 mL
	5 mM	0.4505 mL	2.2523 mL	4.5045 mL
	10 mM	0.2252 mL	1.1261 mL	2.2523 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: ≥ 1.25 mg/mL (2.82 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.25 mg/mL (2.82 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

MYLS22 is a selective optic atrophy 1OPA1 inhibitor. MYLS22 reduces tumor vascularization and associated lymphatic angiogenesis by inhibiting OPA1, thereby limiting tumor growth and metastasis and effectively normalizing tumor vascular morphology. MYLS22 has anticancer activity[1][2][3].

IC_{50} & Target

OPA1[1]

In Vitro

MYLS22 (10-30 μM, 7-10 days) inhibits the clonal growth of acute myeloid leukemia (AML) cells with an IC_{50} of 12.5 μM[1].
MYLS22 (10-30 μM, 7-10 days) significantly inhibits the mitochondrial fusion of AML cells, thereby reducing the ROS content and inhibiting the cell cycle in transition stage of G_0/G_1 transition stage[1].
MYLS22 (10 nM, 30 min) aggravates necroptosis of AECs under the LPS challenge in vitro in MLE12 cells[4].

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

Western Blot Analysis[4]

Cell Line:	MLE12 cells
Concentration:	10 nM
Incubation Time:	30 min
Result:	Increased the phosphorylation levels of RIPK3 and MLKL protein.

In Vivo

MYLS22 (30 mg/kg, Intraperitoneal injection, Once daily for 7 days) significantly reduces the total tumor load of leukemia mice [1].

MYLS22 (10 mg/kg, Paratumoral injection, Once every two days for six days) inhibits tumor growth in mice with subcutaneous B16F10 melanoma tumors by targeting Opa1[3].

MYLS22 (12.5 mg/kg, i.p., once time) aggravates the pathological injury and inflammatory response in the lungs of LPS induced ALI mice, which also can aggravate necroptosis of AECs[4].

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

Animal Model:	LPSinduced ALI mice[4]
Dosage:	12.5 mg/kg
Administration:	i.p., 2 days
Result:	Increased lung inflammatory score, Tnfα and proIl1β mRNA expression, and total IL1β or IL1β p17 protein expression. Increased the levels of RIPK3, MLKL protein, and their phosphorylation. Decreased the levels of SPC protein.

CUSTOMER VALIDATION

- Cell Metab. 2023 Feb 7;35(2):345-360.e7.
- Leukemia. 2023 Feb 4.
- Pharmacol Res. 2021 Jun 6;170:105712.
- Biomed Pharmacother. 2023 Aug 24;166:115342.
- J Cardiovasc Transl Res. 2023 May 30.

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REFERENCES

[1]. Larrue C, et al. Mitochondrial fusion is a therapeutic vulnerability of acute myeloid leukemia. Leukemia. 2023 Apr;37(4):765-775.

[2]. Schuler MH, et al. OPA1 and Angiogenesis: Beyond the Fusion Function. Cell Metab. 2020 May 5;31(5):886-887.

[3]. Herkenne S, et al. Developmental and Tumor Angiogenesis Requires the Mitochondria-Shaping Protein Opa1. Cell Metab. 2020 May 5;31(5):987-1003.e8.

[4]. Jiang HL, et al. L-OPA1 deficiency aggravates necroptosis of alveolar epithelial cells through impairing mitochondrial function during acute lung injury in mice. J Cell Physiol. 2022 Jul;237(7):3030-3043.

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

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