Product Data Sheet

NL-1

Cat. No.: HY-135231 CAS No.: 188532-26-5 Molecular Formula: $C_{18}H_{25}NO_3S$ Molecular Weight: 335.46

Target: Mitochondrial Metabolism; Autophagy

Pathway: Metabolic Enzyme/Protease; Autophagy

Storage: Powder -20°C 3 years

4°C 2 years -80°C 2 years

In solvent -80°C 2 years

-20°C 1 year

SOLVENT & SOLUBILITY

In Vitro

DMSO: 250 mg/mL (745.25 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Mass Concentration	1 mg	5 mg	10 mg
	1 mM	2.9810 mL	14.9049 mL	29.8098 mL
	5 mM	0.5962 mL	2.9810 mL	5.9620 mL
	10 mM	0.2981 mL	1.4905 mL	2.9810 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: ≥ 2.08 mg/mL (6.20 mM); Clear solution
- 2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE- β -CD in saline) Solubility: \geq 2.08 mg/mL (6.20 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (6.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	NL-1 is a mitoNEET inhibitor with antileukemic effect. NL-1 inhibits REH and REH/Ara-C cells growth with IC $_{50}$ s of 47.35 μ M and 56.26 μ M, respectively. NL-1-mediated death in leukemic cells requires the activation of the autophagic pathway ^[1] .
IC ₅₀ & Target	$MitoNEET^{[1]}$
In Vitro	NL-1 (10-100 μM; 72 hours; REH, REH/Ara-C and ALL cell lines) treatment reduces the number of viable cells in REH, REH/Ara-C and ALL (SUP-B15, TOM-1, JM1, NALM-1, NALM-6, BV-173) cell lines, in a concentration-dependent manner. NL-1 inhibits

SUPB15, NALM6 with IC₅₀s of 29.48 μ M, 94.26 μ M, respectively. TOM1, BV173, NALM1 and JM1 all have similar IC₅₀ values of around 60 μ M for NL-1^[1].

NL-1 (60 μ M; 6 hours; REH, REH/Ara-C cell lines) treatment mediates autophagy, and inhibition of autophagy partially decreased NL-1-induced tumor cell death^[1].

NL-1 pretreatment inhibits the chemotactic ability of both REH and REH/Ara-C cells to migrate towards multiple chemoattractants. The cells treated with NL1 shows a dose-dependent decrease in chemotaxis both in the REH and the REH/AraC cells^[1].

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

Cell Proliferation Assay^[1]

Cell Line:	REH, REH/Ara-C and ALL (SUP-B15, TOM-1, JM1, NALM-1, NALM-6, BV-173) cell lines	
Concentration:	10 μΜ, 20 μΜ, 30 μΜ, 40 μΜ, 50 μΜ, 60 μΜ, 70 μΜ, 80 μΜ, 100 μΜ	
Incubation Time:	72 hours	
Result:	Reduced the number of viable cells in REH, REH/Ara-C and ALL (SUP-B15, TOM-1, JM1, NALM-1, NALM-6, BV-173) cell lines, in a concentration-dependent manner.	

Cell Autophagy Assay^[1]

Cell Line:	REH, REH/Ara-C cell lines
Concentration:	60 μΜ
Incubation Time:	6 hours
Result:	Induced cell autophagy.

In Vivo

NL-1 (10 mg/kg; intraperitoneal injection; daily; for 5 days; female NSG mice) treatment shows antileukemic activity in an in vivo mouse ALL model^[1].

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Animal Model:	10 female NOD.Cg-Prkdc scid Il2rg tm1Wjl/SzJ (NSG) mice (6-8 month old) injected with TOM-1 ALL cells $^{[1]}$
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection; daily; for 5 days
Result:	Showed antileukemic activity in an in vivo mouse ALL model.

REFERENCES

[1]. Geldenhuys WJ, et al. The MitoNEET Ligand NL-1 Mediates Antileukemic Activity in Drug-Resistant B-Cell Acute Lymphoblastic Leukemia. J Pharmacol Exp Ther. 2019 Jul;370(1):25-34.

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

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