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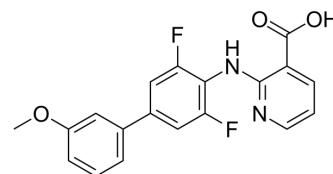
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Farudodstat

Cat. No.:	HY-129239
CAS No.:	1035688-66-4
Molecular Formula:	C ₁₉ H ₁₄ F ₂ N ₂ O ₃
Molecular Weight:	356.32
Target:	Dihydroorotate Dehydrogenase; DNA/RNA Synthesis; Apoptosis
Pathway:	Metabolic Enzyme/Protease; Cell Cycle/DNA Damage; Apoptosis
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 : 125 mg/mL (350.81 mM; Need ultrasonic)					
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg
		1 mM		2.8065 mL	14.0323 mL	28.0647 mL
		5 mM		0.5613 mL	2.8065 mL	5.6129 mL
		10 mM		0.2806 mL	1.4032 mL	2.8065 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: ≥ 2.17 mg/mL (6.09 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.17 mg/mL (6.09 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	Farudodstat (ASLAN003) is an orally active and potent Dihydroorotate Dehydrogenase (DHODH) inhibitor with an IC ₅₀ of 35 nM for human DHODH enzyme. Farudodstat inhibits protein synthesis via activation of AP-1 transcription factors. Farudodstat induces apoptosis and substantially prolongs survival in acute myeloid leukemia (AML) xenograft mice ^{[1][2]} .
IC ₅₀ & Target	IC ₅₀ : 35 nM (human DHODH enzyme) ^[1]
In Vitro	Farudodstat (0.01-100 μM; for 48 hours) inhibits leukemic cell proliferation. The cell viability is maintained at ~50% at Farudodstat 1 μM and higher ^[1] . Farudodstat (0.5, 1 μM; for 48 hours) significantly increases cleaved caspase 8 ^[1] . Farudodstat (2, 4 μM; for 96 hours) decreases viability and induces differentiation in primary acute myeloid leukemia blasts

and myelodysplastic syndrome samples^[1].

Farudodstat (1, 2 μ M; pretreatment 1 h before OPP for 1 h) inhibits protein synthesis, as demonstrated by the reduced incorporation of O-propargyl-puromycin (OPP) at protein translation sites in both MOLM-14 and KG-1 cells. Farudodstat causes the downregulation of EIF4B, and RPL6 proteins^[1].

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

Cell Proliferation Assay^[1]

Cell Line:	THP-1, MOLM-14 and KG-1 cells
Concentration:	0.01, 0.1, 1, 10, 100 μ M
Incubation Time:	For 48 hours
Result:	Inhibited leukemic cell proliferation of THP-1, MOLM-14 and KG-1 with IC ₅₀ values of 152 nM, 582 nM, and 382 nM, respectively.

Western Blot Analysis^[1]

Cell Line:	KG-1 and MOLM-14 cells
Concentration:	0.5, 1 μ M
Incubation Time:	For 48 hours
Result:	Significantly increased cleaved caspase 8, increased leakage of cytochrome c from mitochondria into the cytosol and induced cleaved caspase-3 and -7.

In Vivo

Farudodstat (50 mg/kg; oral gavage; once daily; from the day 3 to 30) substantially reduces the number of disseminated tumors and prolongs survival^[1].

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

Animal Model:	Female NOD.Cg-Prkdc ^{scid} Il2rg ^{tm1Wjl} /SzJ, NGS mice (4-6 weeks old) with MOLM-14 cells ^[1]
Dosage:	50 mg/kg
Administration:	Oral gavage; once daily; from the day 3 to 30
Result:	Substantially reduced the number of disseminated tumors and the size of these tumors. Survival was significantly prolonged.

CUSTOMER VALIDATION

- Biochem Pharmacol. 2022 Aug 27;204:115232.

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REFERENCES

- [1]. Marco L. Lolli, et al. Human Dihydroorotate Dehydrogenase (hDHODH) as a new target on Acute Myelogenous Leukemia (AML): Targeting Myeloid Differentiation using Potent and Innovative hDHODH Inhibitors. 23rd Swedish Conference on Macromolecular Structure an
- [2]. Jianbiao Zhou, et al. ASLAN003, a potent dihydroorotate dehydrogenase inhibitor for differentiation of acute myeloid leukemia. Haematologica. 2019

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

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