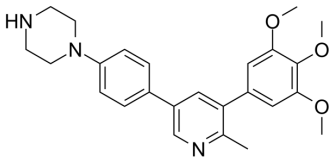
LDN-214117

Cat. No.:	HY-16712
CAS No.:	1627503-67-6
Molecular Formula:	$C_{25}H_{29}N_3O_3$
Molecular Weight:	420
Target:	TGF-β Receptor
Pathway:	TGF-beta/Smad
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 : 20 mg/mL (47.62 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3810 mL	11.9048 mL	23.8095 mL
	5 mM	0.4762 mL	2.3810 mL	4.7619 mL
	10 mM	0.2381 mL	1.1905 mL	2.3810 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 mg/mL (4.76 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2 mg/mL (4.76 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2 mg/mL (4.76 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

LDN-214117 is an orally active ALK2 inhibitor with well-tolerated and good brain penetration. LDN-214117 has a high selectivity and low cytotoxicity for ALK2 with an IC_{50} value of 24 nM. LDN-214117 also is a specific bone morphogenetic proteins (BMPs) signaling inhibitor and has relatively selective inhibition for BMP6 with an IC_{50} value of 100 nM. LDN-214117 can be used for the research of fibrodysplasia ossificans progressiva (FOP), diffuse intrinsic pontine glioma (DIPG) [1][2]

IC_{50} & Target

IC50: 24 nM (ALK2); 27 nM (ALK1); 1,171 nM (ALK3); 3,000 nM (ALK5); 1,022 nM (BMP6); 27 nM (BMP2); 960 nM (BMP4); 16,000 nM (TGF-β1)[1]

In Vitro

LDN-214117 has high inhibition and selectivity for ALK2 kinase proteins with an IC_{50} value of 24 nM[1].
LDN-214117 has kinase activity for ALK1, ALK3 and ALK5 with IC_{50} values of 27 nM, 1,171 nM and 3,000 nM, respectively[1].
LDN-214117 exhibits relatively selective inhibition for BMP6, BMP2 and BMP4 with IC_{50} values of 100 nM, 1,022 nM and 960 nM, respectively[1].
LDN-214117 has inhibition of TGF-β1-induced transcriptional activity with an IC_{50} values of 16,000 nM[1].
LDN-214117 (5 μM, 30 min, 3 h and 24 h) has time-dependent effect activity on gene regulation level and/ or a BMP signaling pathway other than SMAD-dependent that is responsible for ID1 targeting[2].
LDN-214117 (5 μM, 24-120 h) reduces viability, proliferation and causes apoptosis of lung carcinoma cells LCLC-103H[2].
LDN-214117 (5 μM, 0-48 h) suppresses wound healing and chemotactic potential of LCLC-103H cells[2].
LDN-214117 (5 μM, 48 h) hinders growth of multicellular LCLC-103H spheroids[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	LCLC-103H cells
Concentration:	5 μM
Incubation Time:	24 h, 48 h, 72 h and 96 h
Result:	Decreased markedly with time, counting approximately 60% of the vehicle control level at the 96-h measurement point.

Western Blot Analysis[2]

Cell Line:	LCLC-103H cells
Concentration:	5 μM
Incubation Time:	30 min, 3 h and 24 h
Result:	Diminished the increase of ID1 protein.

Apoptosis Analysis[2]

Cell Line:	LCLC-103H cells
Concentration:	5 μM
Incubation Time:	24 h, 48 h, 72 h and 96 h
Result:	Induced considerable death of LCLC-103H cells.

RT-PCR[2]

Cell Line:	LCLC-103H cells
Concentration:	5 μM
Incubation Time:	24 h, 48 h and 72 h
Result:	Induced distinct gene expression patterns for the two EMTTFs.

Cell Migration Assay [2]

Cell Line:	LCLC-103H cells
Concentration:	5 μM
Incubation Time:	0 h, 24 h and 48 h

Result:	Significantly hindered the migration of LCLC-103H cells into the wound area by Inhibiting of BMP signaling.

In Vivo

LDN-214117 (p.o., 25 mg/kg, daily, for 14 days) has well-tolerated in mice[3].

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

Animal Model:	NOD.SCID mice[3]
Dosage:	25 mg/kg
Administration:	p.o., daily, for 14 days
Result:	Showed good-tolerated in mice.

CUSTOMER VALIDATION

- Adv Sci (Weinh). 2024 Jan 16:e2306499.

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REFERENCES

[1]. Agustin H Mohedas, et al. Structure-activity relationship of 3,5-diaryl-2-aminopyridine ALK2 inhibitors reveals unaltered binding affinity for fibrodysplasia ossificans progressiva causing mutants. J Med Chem. 2014 Oct 9;57(19):7900-15.

[2]. Jelena Mihajlović, et al. Inhibition of bone morphogenetic protein signaling reduces viability, growth and migratory potential of non-small cell lung carcinoma cells. J Cancer Res Clin Oncol. 2019 Nov;145(11):2675-2687.

[3]. Diana Carvalho, et al. ALK2 inhibitors display beneficial effects in preclinical models of ACVR1 mutant diffuse intrinsic pontine glioma. Commun Biol. 2019 May 9;2:156.

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