Alectinib

Cat. No.: HY-13011

CAS No.: 1256580-46-7

Molecular Formula: $C_{30}H_{34}N_4O_2$

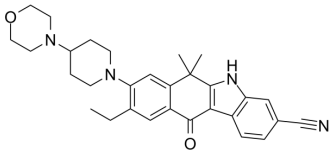
Molecular Weight: 482.62

Target: ALK

Pathway: Protein Tyrosine Kinase/RTK

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 : 6.2 mg/mL (12.85 mM; Need warming)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0720 mL	10.3601 mL	20.7202 mL
	5 mM	0.4144 mL	2.0720 mL	4.1440 mL
	10 mM	0.2072 mL	1.0360 mL	2.0720 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 20 mg/mL (41.44 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 0.38 mg/mL (0.79 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.38 mg/mL (0.79 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Alectinib (CH5424802) is a potent, selective, and orally available ALK inhibitor with an IC_{50} of 1.9 nM and a K_d value of 2.4 nM (in an ATP-competitive manner), and also inhibits ALK F1174L and ALK R1275Q with IC_{50}s of 1 nM and 3.5 nM, respectively[1]. Alectinib demonstrates effective central nervous system (CNS) penetration[2].

IC_{50} & Target

IC50: 1.9 nM(ALK), 1 nM (ALK^{F1174L}), 3.5 nM (ALK^{R1275Q})[1]
Kd: 2.4 nM (ALK)[1]

In Vitro

Alectinib (0-1000 nM; 2 hours; NCI-H2228 cells) treatment could prevent autophosphorylation of ALK in NCI-H2228 cells expressing EML4-ALK, and it also resulted in substantial suppression of phosphorylation of STAT3 and AKT[1].
Alectinib (0-1000 nM; 5 days; HCC827, A549, or NCIH522 cells) treatment reduces cell activity in a dose-dependent manner[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	NCI-H2228 cells
Concentration:	0 nM,10 nM,100 nM, 1000 nM
Incubation Time:	2 hours
Result:	Inhibition of ALK phosphorylation and signal transduction.

Cell Viability Assay[1]

Cell Line:	HCC827, A549, or NCIH522 cells
Concentration:	0-1000 nM
Incubation Time:	5 days
Result:	Reduced cell activity in a dose-dependent manner.

In Vivo

Alectinib (0.2-20 mg/kg; oral administration; once daily; for 11 days; SCID or nude mice bearing NCI-H2228 cells) treatment can result in dose-dependent tumor growth inhibition (EC_{50} of 0.46 mg/kg) and tumor regression. At any dose level, no differences in body weight or gross signs of toxicity are observed[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	SCID or nude mice bearing NCI-H2228 cells[1]
Dosage:	0.2 mg/kg, 0.6 mg/kg, 2 mg/kg, 6 mg/kg, 20 mg/kg
Administration:	Oral administration; once daily; for 11 days
Result:	Resulted in dose-dependent tumor growth inhibition (EC_{50} of 0.46 mg/kg) and tumor regression.

CUSTOMER VALIDATION

- Science. 2017 Dec 1;358(6367):eaan4368.
- Science. 2014 Oct 3;346(6205):1255784.
- Cancer Discov. 2018 Jun;8(6):714-729.
- Cancer Discov. 2016 Oct;6(10):1118-1133.
- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Sakamoto H, et al. CH5424802, a selective ALK inhibitor capable of blocking the resistant gatekeeper mutant. Cancer Cell. 2011, 19(5), 679-690.

[2]. Gadgeel S, et al. Alectinib versus crizotinib in treatment-naive anaplastic lymphoma kinase-positive (ALK+) non-small-cell lung cancer: CNS efficacy results from the ALEX study. Ann Oncol. 2018 Nov 1;29(11):2214-2222.

India Contact:
Life Technologies (India) Pvt. Ltd.
306, Aggarwal City Mall, Opposite M2K Pitampura, Delhi – 110034 (INDIA). Ph: +91-11-42208000, 42208111, 42208222, Mobile: +91-9810521400, Fax: +91-11-42208444
Email: customerservice@lifetechindia.com Website: www.lifetechindia.com