

Product Data Sheet

Product Name: Alectinib Hydrochloride

Cat. No.: GC35280

Chemical Properties

Cas. No. 1256589-74-8

SMILES [H]Cl.N#CC1=CC2=C(C3=C(N2)C(C)(C4=CC(N5CCC(CC5)N6CCOCC6)=C(C=C4C3=O)CC)C)C=C1

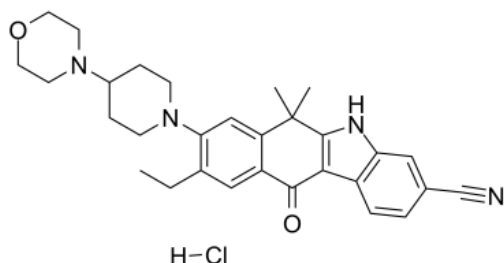
Formula $C_{30}H_{35}ClN_4O_2$ M.Wt 519.08

Solubility DMSO: 6 mg/mL (11.56 mM and warming) Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

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Cell lines U87MG cells

Preparation Method U87MG cells were cultured in RPMI-1640 medium supplemented with 10% fetal bovine serum (FBS) and penicillin-streptomycin (1%) at 37°C in a humidified atmosphere containing 5% CO₂. The cells were cultured in 96-well plates overnight and incubated with various concentrations of Alectinib Hydrochloride (0, 1, 2, 5, and 10µM) for 48 hours and analyzed for viability.

Reaction Conditions 0, 1, 2, 5, and 10µM; 48h

Applications Alectinib Hydrochloride treatment decreased the cell viability of U87MG cells in a dose-dependent manner.

Animal experiment [2]:

Animal models Female BALB/c nude mice

Preparation Method Female BALB/c nude mice (6 weeks old) were fed with sterilized food and water and housed in a barrier facility. The light/dark cycle was 12 hours. H3122 cells (5×10⁶) were injected bilaterally subcutaneously into the mice's backs. The vectors and Alectinib Hydrochloride (10mg/kg) were administered by gavage, once a day, five times a week. The tumor volume was measured twice a week, and the calculation formula was: width²×length/2, for a duration of 4 weeks.

Dosage form 10mg/kg/day for 4 weeks; p.o.

Applications Alectinib Hydrochloride treatment suppressed tumor growth in the H3122 cell xenograft mouse model without affecting body weight.

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- [1] Kawauchi D, Takahashi M, Satomi K, et al. The ALK inhibitors, alectinib and ceritinib, induce ALK-independent and STAT3-dependent glioblastoma cell death[J]. Cancer Science, 2021, 112(6): 2442-2453.
- [2] Ando C, Ichihara E, Nishi T, et al. Efficacy of gilteritinib in comparison with alectinib for the treatment of ALK-rearranged non-small cell lung cancer[J]. Cancer Science, 2023, 114(11): 4343-4354.

Background

Alectinib Hydrochloride is a potent, selective, and orally available ALK inhibitor with an IC₅₀ value of 1.9nM and a K_d value of 2.4nM^[1]. Alectinib Hydrochloride suppresses the L1196M-mutant ALK with an IC₅₀ of 1.56nM^[2]. Alectinib Hydrochloride increases the intracellular accumulation of ABCB1/ABCG2 substrates such as doxorubicin (DOX) and Rhodamine 123 (Rho 123) by inhibiting the efflux function of the transporters in ABCB1- or ABCG2-overexpressing cells, and stimulates ATPase activity and competed with substrates of ABCB1 or ABCG2^[3]. Alectinib Hydrochloride has been widely used to inhibit the growth of lung cancer cells with ALK mutations^[4].

In vitro, Alectinib Hydrochloride treatment for 48 hours significantly inhibited the proliferation of H2228 cells and ABC-11 cells, with IC₅₀ values of 0.03μM and 0.13μM, respectively^[5]. Treatment with 30nM Alectinib Hydrochloride for 48 hours significantly inhibited ALK-mediated neurite growth in PC12 cells^[6]. Treatment with 10μM Alectinib Hydrochloride for 48 hours induced apoptosis in U87MG cells, inhibited the tyrosine phosphorylation of STAT3, and activated the tyrosine phosphorylation of Janus kinase 2 (JAK2)^[7].

In vivo, Alectinib Hydrochloride treatment at a single dose of 50mg/kg via intraperitoneal injection for 24 hours significantly reduced cerebral infarction and neurological deficits in the ischemic stroke mouse model, as well as the loss of cerebral vascular integrity and damage to the blood-brain barrier^[8]. Oral administration of 10mg/kg of Alectinib Hydrochloride daily for 4 weeks significantly inhibited tumor growth in a H3122 cell-xenograft mouse model, without affecting the body weight of the mice^[9].

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- [1] Madloul D T, Al-Ani I, Ata T, et al. Solubility, pH-Solubility Profile, pH-Rate Profile, and Kinetic Stability of the Tyrosine Kinase Inhibitor, Alectinib[J]. Pharmaceuticals, 2024, 17(6): 776.
- [2] Song Z, Wang M, Zhang A. Alectinib: a novel second generation anaplastic lymphoma kinase (ALK) inhibitor for overcoming clinically-acquired resistance[J]. Acta Pharmaceutica Sinica B, 2015, 5(1): 34-37.
- [3] Yang K, Chen Y, To K K W, et al. Alectinib (CH5424802) antagonizes ABCB1-and ABCG2-mediated multidrug resistance in vitro, in vivo and ex vivo[J]. Experimental & molecular medicine, 2017, 49(3): e303-e303.
- [4] Yoshimura Y, Kurasawa M, Yoroze K, et al. Antitumor activity of alectinib, a selective ALK inhibitor, in an ALK-positive NSCLC cell line harboring G1269A mutation: efficacy of alectinib against ALK G1269A mutated cells[J]. Cancer chemotherapy and pharmacology, 2016, 77(3): 623-628.
- [5] Isozaki H, Ichihara E, Takigawa N, et al. Non-small cell lung cancer cells acquire resistance to the ALK inhibitor alectinib by activating alternative receptor tyrosine kinases[J]. Cancer research, 2016, 76(6): 1506-1516.
- [6] Alam M W, Borenäs M, Lind D E, et al. Alectinib, an anaplastic lymphoma kinase inhibitor, abolishes ALK activity and growth in ALK-positive neuroblastoma cells[J]. Frontiers in oncology, 2019, 9: 579.
- [7] Kawauchi D, Takahashi M, Satomi K, et al. The ALK inhibitors, alectinib and ceritinib, induce ALK-independent and STAT3-dependent glioblastoma cell death[J]. Cancer Science, 2021, 112(6): 2442-2453.
- [8] Hu Y, Chang L, Zhu Y, et al. Inhibition of anaplastic lymphoma kinase protects from ischemic stroke[J]. Stroke, 2024, 55(4): 1075-1085.
- [9] Ando C, Ichihara E, Nishi T, et al. Efficacy of gilteritinib in comparison with alectinib for the treatment of ALK-rearranged non-small cell lung cancer[J]. Cancer Science, 2023, 114(11): 4343-4354.

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