Web: www.taiclone.com
Tel: +886-2-2735-9682
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Alectinib

Catalog No: tcsc0518

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg

Specifications

CAS No:
1256580-46-7

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

Pathway:
Protein Tyrosine Kinase/RTK

Target:
ALK

Purity / Grade:
>98%

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

Alternative Names:
CH5424802;AF802

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Observed Molecular Weight:
482.62

Product Description

Alectinib (CH5424802) is a potent, selective, and orally available **ALK** inhibitor with an **IC_{50}** of 1.9 nM.

IC50 & 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 (CH5424802) prevents autophosphorylation of ALK in NCI-H2228 NSCLC cells expressing EML4-ALK, and Alectinib also results in substantial suppression of phosphorylation of STAT3 and AKT, but not of ERK1/2[1]. Alectinib (CH5424802) shows high kinase selectivity and strong anti-proliferative activity against KARPAS-299 with an IC_{50} value of 3 nM[2].

In Vivo: In the NCI-H2228 model, once-daily oral administration of Alectinib (CH5424802) results in dose-dependent tumor growth inhibition (ED_{50}=0.46 mg/kg) and tumor regression. Treatment of 20 mg/kg Alectinib shows rapid tumor regression (168% tumor growth inhibition; p3 after 11 days of treatment (at day 28), a potent antitumor effect is maintained, and tumor re-growth dpes not occur throughout the 4-week drug-free period[1]. Oral administration of Alectinib (CH5424802) at 20 mg/kg displays significant tumor regression without body weight loss in an established ALK fusion gene-positive NSCLC xenograft model in mice[2]. Alectinib (Alectinib) at 60 mg/kg causes tumor regression against EML4-ALK-positive NCI-H2228 xenograft model and decreases the levels of phosphorylated ALK in this model. In addition, in mice at dose levels up to 60 mg/kg of CH5424802, there is no body weight loss, no significant change in peripheral blood cell count, no elevations of aspartate aminotransferase or alanine aminotransferase, and no substantial change in electrolytes. Oral administration of CH5424802 at 60 mg/kg for 4 days results in significant tumor regression seen in the luminescence signal[3].