Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Crizotinib

Catalog No: tcsc0029

Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg

Size: 500mg

Size: 1g

Size: 2g

Size: 5g

Specifications

CAS No:
877399-52-5

Formula:
$C_{21}H_{22}Cl_2FN_5O$

Pathway:
Protein Tyrosine Kinase/RTK;Autophagy;Protein Tyrosine Kinase/RTK

Target:
c-Met/HGFR;Autophagy;ALK

Purity / Grade:
>98%

Solubility:

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DMSO : 55 mg/mL (122.13 mM; Need ultrasonic)

Alternative Names:
PF-02341066

Observed Molecular Weight:
450.34

Product Description

Crizotinib is a potent inhibitor of **c-Met** and **ALK** with **IC_{50}** of 11 nM and 24 nM in cell-based assays, respectively.

IC50 & Target: IC50: 11 nM (c-Met), 24 nM (ALK)

In Vitro: PF-2341066 displays similar potency against c-Met phosphorylation in mIMCD3 mouse or MDCK canine epithelial cells with IC_{50} of 5 nM and 20 nM, respectivly. PF-2341066 shows improved or similar activity against NIH3T3 cells engineered to express c-Met ATP-binding site mutants V1092I or H1094R or the P-loop mutant M1250T with IC_{50} of 19 nM, 2 nM and 15 nM, respectively, compared with NIH3T3 cells expressing wild-type receptor with IC_{50} of 13 nM. In contrast, a marked shift in potency of PF-2341066 is observed against cells engineered to express c-Met activation loop mutants Y1230C and Y1235D with IC_{50} of 127 nM and 92 nM, respectively, compared with wild-type receptor. PF-2341066 also potently prevents the phosphorylation of c-Met in NCI-H69 and HOP92 cells, with IC_{50} of 13 nM and 16 nM, respectively, which express the endogenous c-Met variants R988C and T1010I, respectively[1]. PF-2341066 also potently inhibits NPM-ALK phosphorylation in Karpas299 or SU-DHL-1 ALCL cells with an IC_{50} of 24 nM. PF-2341066 potently prevents cell proliferation, which is associated with G(1)-S-phase cell cycle arrest and induction of apoptosis in ALK-positive ALCL cells with IC_{50} of 30 nM, but not ALK-negative lymphoma cells[2]. Besides, PF-2341066 prevents osteosarcoma behavior associated with primary tumor growth (i.e., proliferation and survival) as well as metastasis[3].

In Vivo: PF-2341066 reveals the ability to cause marked regression of large established tumors (> 600 mm^3) in both the 50 mg/kg/day and 75 mg/kg/day treatment cohorts, with a 60% decrease in mean tumor volume over the 43-day administration schedule in the GTL-16 model. In an another study, PF-2341066 displays the ability to completely inhibits GTL-16 tumor growth for >3 months, with only 1 of 12 mice exhibiting a significant increase in tumor growth over the 3-month treatment schedule at 50 mg/kg/day. A significant dose-dependent reduction of CD31-positive endothelial cells is observed at 12.5 mg/kg/day, 25 mg/kg/day, and 50 mg/kg/day in GTL-16 tumors, indicating that inhibition of MVD shows a dose-dependent correlation to antitumor efficacy. PF-2341066 displays a significant dose-dependent reduction of human VEGFA and IL-8 plasma levels in both the GTL-16 and U87MG models. Marked inhibition of phosphorylated c-Met, Akt, Erk, PLCλ1, and STAT5 levels is observed in GTL-16 tumors following p.o. administration of PF-2341066[1]. PF-2341066 prevents osteosarcoma behavior associated with primary tumor growth as well as metastasis. In nude mice treated with PF-2341066 via oral gavage, the growth and associated osteolysis and extracortical bone matrix formation of osteosarcoma xenografts are prevented by PF-2341066[3]. Treatment of c-MET-amplified GTL-16 xenografts with 50 mg/kg PF-2341066 elicits tumor regression that is associated with a slow reduction in 18F-FDG uptake and decreases expression of the glucose transporter 1, GLUT-1[4].

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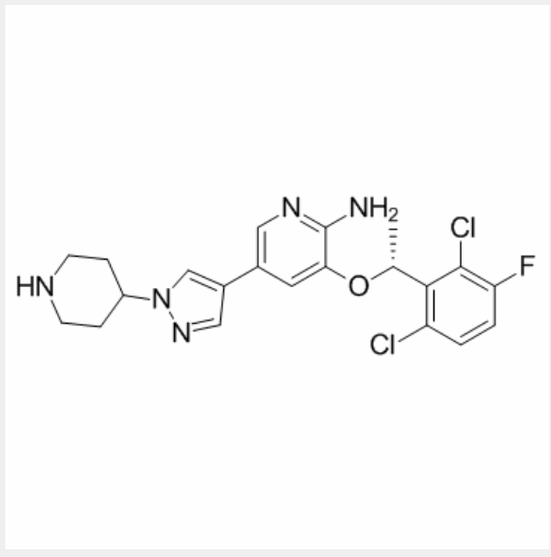

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