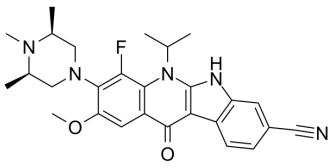
CJ-2360

Cat. No.:	HY-131909
CAS No.:	2226742-61-4
Molecular Formula:	$C_{27}H_{30}FN_5O_2$
Molecular Weight:	475.56
Target:	ALK
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

CJ-2360 is a potent and orally active ALK inhibitor with IC_{50}s of 2.2, 4.0, 8.8, 6.3, and 8.9 nM against wild-type ALK and F1197M, G1269A, L1196M, and S1206Y ALK mutants, respectively. CJ-2360 displays potent inhibitory activity against two clinically reported ALK mutants (C1156Y and L1196M) and a few other kinases (LTK, MERTK, CLK1, DAPK1, and DAPK2) among the 468 kinases evaluated[1].

In Vitro

CJ-2360 achieves an IC_{50} value of 1.8 nM in inhibition of cell growth in the KARPAS-299 cell line. Further tested CJ-2360 for its potency in cell growth inhibition in the H3122 non-small-cell lung cell line carrying EML4-ALK and obtained an IC_{50} value of 3 nM. CJ-2360 inhibits Mer tyrosine-protein kinase (MERTK), CLK1, DAPK1, DAPK2, and DAPK3 with IC_{50}s of 6.3, 11, 31, 23, 22, and 260 nM, respectively. CJ-2360 displays >100-fold selectivity for ALK over the insulin receptor kinase (INSR) and shows no significant activity against IGF1R[1].

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

In Vivo

CJ-2360 (100 mg/kg; p.o.; twice daily for 22 days) is capable of achieving complete and long-lasting tumor regression in the KARPAS-299 xenograft tumor model[1].

CJ-2360 (100 mg/kg; p.o.) is very effective in inhibition of ALK phosphorylation, as well as ERK and STAT3 phosphorylation in KARPAS-299 tumor tissue, with the effect persisting for at least 24 h[1].

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

Animal Model:	KARPAS-299 xenograft model (female SCID mice)[1]
Dosage:	100 mg/kg
Administration:	P.o.; twice daily for 22 days
Result:	Very efficacious in the KARPAS-299 xenograft model. It achieves complete tumor regression in 100% of tumors and all tumors did not return until day 53, 23 days after the last dose.

REFERENCES

[1]. Chen J, et al. Discovery of CJ-2360 as a Potent and Orally Active Inhibitor of Anaplastic Lymphoma Kinase Capable of Achieving Complete Tumor Regression. J Med

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA