Product Datasheet

AZD3759

Catalog No. **BP-01908** · CAS **1626387-80-1** · Form **free base** · Physical state **solid** · Color **White to off-white** · Version **1.0** · Revision **2026-09-04**

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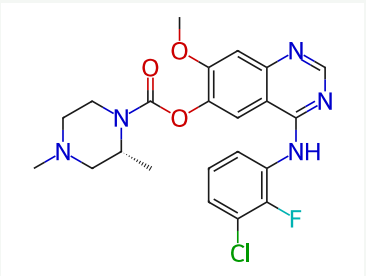
Target	Research area
EGFR; Apoptosis	**Infection; Neurological Disease; Metabolic Disease; Inflammation/Immunology; Cancer**

Molecular formula	$C_{22}H_{23}ClFN_5O_3$
Molecular weight	**459.9010**

STRUCTURE



DESCRIPTION

AZD3759 is a potent, orally active, central nervous system-penetrant, EGFR inhibitor.

BIOLOGICAL ACTIVITY

In Vitro

At 2 mM of ATP concentrations, the IC50s are 102, 7.6, and 2.4 nM for EGFRwt, EGFRL858R, and EGFRexon 19Del, respectively. Zorifertinib (AZD3759) also inhibits pEGFR in H838wt, H3255L858R, and PC-9exon 19Del with IC50 of 64.5, 7.2, and 7.4 nM, respectively. In cellular phosphorylation studies, Zorifertinib also demonstrates 9-fold inhibition selectivity in EGFR-activating mutant cell lines over EGFR wild-type cell lines (H838 cell line).

In Vivo

Following oral dosing in rats at 2 mg/kg, absorption of Zorifertinib (AZD3759) is rapid with blood Cmax of 0.58 μM achieved at 1.0 h. Subsequently, blood concentrations of Zorifertinib decline monoexponentially with a mean elimination half-life of 4.3 h, which is close to the same parameter obtained from intravenous dosing of 4.1 h. The bioavailability following an oral dose in rats is 91%. Blood pharmacokinetic parameters of Zorifertinib in male dogs are determined following both a single dose intravenous infusion and oral administration. Following the IV dose in dogs, Zorifertinib blood clearance is determined as 14 mL/min per kg, and the volume of distribution is 6.4 L/kg. Its elimination half-life is 6.2 h. Absorption of Zorifertinib is rapid with blood Cmax (698 nM) occurring between 0.5 and 1.5 h. The oral bioavailability of Zorifertinib is excellent at 90%. Zorifertinib demonstrated significant dose-dependent antitumor efficacy (78% tumor growth inhibition at 7.5 mg/kg qd and tumor regression at 15 mg/kg qd, respectively, 4 weeks after treatment) with <20% body weight loss, whereas CP-358774 has a limited effect in this model. At the end of the study, brain tissues are collected for histological assessment. Significantly decreased tumor area is observed by Zorifertinib treatment at the doses of 7.5 and 15 mg/kg. In addition, modulation of pEGFR is detected by a single dose of Zorifertinib at 15 mg/kg 1h after dosing, which confirmed target engagement by Zorifertinib.

SOLUBILITY

Soluble in DMSO

REFERENCES

[1]. Zeng Q, et al. Discovery and Evaluation of Clinical Candidate AZD3759, a Potent, Oral Active, Central Nervous System-Penetrant, Epidermal Growth Factor Receptor Tyrosine Kinase Inhibitor. J Med Chem. 2015 Oct 22;58(20):8200-15.

[2]. Chao D, et al. AZD3759 induces apoptosis in hepatoma cells by activating a p53-SMAD4 positive feedback loop. Biochem Biophys Res Commun. 2019 Feb 5;509(2):535-540.

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