Avitinib maleate

Cat. No.: HY-19816A

CAS No.: 1557268-88-8

Molecular Formula: $C_{30}H_{30}FN_7O_6$

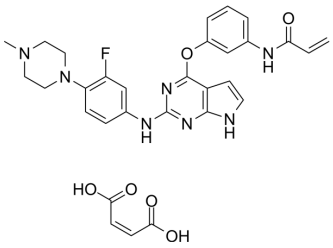
Molecular Weight: 603.6

Target: EGFR; Btk; Apoptosis

Pathway: JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; Apoptosis

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (165.67 mM)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.6567 mL	8.2836 mL	16.5673 mL
5 mM	0.3313 mL	1.6567 mL	3.3135 mL
10 mM	0.1657 mL	0.8284 mL	1.6567 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.14 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.14 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.14 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Avitinib (Abivertinib) maleate is a third-generation, irreversible and orally active selective EGFR inhibitor, with IC_{50} values of 0.18 nM, 0.18 nM, 7.68 nM and against EGFR L858R, EGFR T790M and wild-type EGFR. Avitinib maleate is also a BTK inhibitor that induces apoptosis and inhibits phosphorylation of BTK in mantle cell lymphoma. Avitinib maleate shows anticancer effects[1][2].

IC_{50} & Target			
	EGFR L858R 0.18 nM (IC_{50})	$EGFR^{T790M}$ 0.18 nM (IC_{50})	EGFR (WT) 7.68 nM (IC_{50})

In Vitro

Avitinib (AC0010; 0.13 nM-2 μM; 2 h) maleate selectively inhibits mutant EGFR phosphorylation with IC_{50} values of 7.3 and 2.8 nM in NCI-H1975 and NIH/3T3_TC32T8 cells, about 115- and 298-fold more sensitive than that of the inhibition of wild-type EGFR in A431. Avitinib potently inhibits EGFR-Tyr1068 phosphorylation in NCI-H1975 cells, and the selectivity ratio is at 65-fold for NCI-H1975 cells versus A431 cells. In addition to inhibition of EGFR-Tyr1068 phosphorylation, Avitinib inhibits phosphorylation of the downstream targets Akt and ERK1/2 in NCI-H1975 and HCC827 cells[1].

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

Western Blot Analysis[1]

Cell Line:	NCI-H1975, HCC827, A431 cells
Concentration:	0.13 nM, 0.64 nM, 3.2 nM, 16 nM, 80 nM, 0.4 μM, 2 μM
Incubation Time:	2 h
Result:	Selectively inhibits mutant EGFR phosphorylation with IC50 values of 7.3 and 2.8 nM in NCI-H1975 and NIH/3T3_TC32T8 cells.

In Vivo

Avitinib (AC0010; 12.5-500 mg/kg; orally administration; once daily; for 14 days) maleate inhibits EGFR-mutant tumor growth but not wild-type EGFR tumor growth in xenograft models over extended duration[1].

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

Animal Model:	Nu/Nu nude mice (Six- to 8-week-old) injected with NCI-H1975 and A431 cells[1]
Dosage:	12.5, 50, and 500 mg/kg
Administration:	Orally administration; once daily; for 14 days
Result:	Inhibited EGFR-mutant tumor growth but not wild-type EGFR tumor growth.

CUSTOMER VALIDATION

- Molecules. 2021 May 5;26(9):2717.
- J Pharm Biomed Anal. 2019 Feb 5;164:659-667.

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REFERENCES

[1]. Yan X, et al. Promising efficacy of novel BTK inhibitor AC0010 in mantle cell lymphoma. J Cancer Res Clin Oncol. 2018;144(4):697-706.

[2]. Xu X, et al. AC0010, an Irreversible EGFR Inhibitor Selectively Targeting Mutated EGFR and Overcoming T790M-Induced Resistance in Animal Models and Lung Cancer Patients. Mol Cancer Ther. 2016 Nov;15(11):2586-2597.

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