



Vandetanib

Catalog No: tcsc0130



Available Sizes

Size: 25mg

Size: 50mg

Size: 100mg

Size: 500mg

Size: 1g

Size: 2g



Specifications

CAS No:

443913-73-3

Formula:

 $\mathsf{C_{22}H_{24}BrFN_4O_2}$

Pathway:

Protein Tyrosine Kinase/RTK; Autophagy

Target:

VEGFR; Autophagy

Purity / Grade:

>98%

Solubility:

DMSO: 27.5 mg/mL (57.85 mM; Need ultrasonic and warming)

Alternative Names:

ZD6474





Observed Molecular Weight:

475.35

Product Description

Vandetanib is a potent inhibitor of **VEGFR2** with IC_{50} of 40 nM.

IC50 & Target: IC50: 40 nM (VEGFR2)

In Vitro: Vandetanib inhibits VEGFR3 and EGFR with IC $_{50}$ of 110 nM and 500 nM, respectively. Vandetanib is not sensitive to PDGFR β , Flt1, Tie-2 and FGFR1 with IC $_{50}$ of 1.1-3.6 μM, while almost has no activity against MEK, CDK2, c-Kit, erbB2, FAK, PDK1, Akt and IGF-1R with IC $_{50}$ above 10 μM. Vandetanib inhibits VEGF-, EGF- and bFGF-stimulated HUVEC proliferation with IC $_{50}$ of 60 nM, 170 nM and 800 nM, with no effect on basal endothelial cell growth. Vandetanib inhibits tumor cell growth with IC $_{50}$ of 2.7 μM (A549) to 13.5 μM (Calu-6)^[1]. Odanacatib is a weak inhibitor of antigen presentation, measured in a mouse B cell line (IC $_{50}$ =1.5±0.4 μM), compared to the Cat S inhibitor LHVS (IC $_{50}$ =0.001 μM) in the same assay. Odanacatib also shows weak inhibition of the processing of the MHC II invariant chain protein lip10 in mouse splenocytes compared to LHVS (minimum inhibitory concentration 1-10 μM versus 0.01 μM, respectively)^[2]. Vandetanib suppresses phosphorylation of VEGFR-2 in HUVECs and EGFR in hepatoma cells and inhibits cell proliferation^[4].

In Vivo: Vandetanib (15 mg/kg, p.o.) has a superior anti-tumor effect than gefitinib in the H1650 xenograft model, and suppresses tumor growth with IC_{50} of $3.5\pm1.2~\mu M^{[3]}$. In tumor-bearing mice, vandetanib (50 or 75 mg/kg) suppresses phosphorylation of VEGFR-2 and EGFR in tumor tissues, significantly reduces tumor vessel density, enhances tumor cell apoptosis, suppresses tumor growth, improves survival, reduces number of intrahepatic metastases, and upregulates VEGF, TGF- α , and EGF in tumor tissues^[4].

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