

Motesanib (Diphosphate)

Catalog No: tcsc0193



Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg

Size: 500mg



Specifications

CAS No:

857876-30-3

Formula:

$C_{22}H_{29}N_5O_9P_2$

Pathway:

Protein Tyrosine Kinase/RTK;Protein Tyrosine Kinase/RTK

Target:

VEGFR;c-Kit

Purity / Grade:

>98%

Solubility:

DMSO : ≥ 110 mg/mL (193.17 mM)

Alternative Names:

Motesanib;AMG 706

Observed Molecular Weight:

569.44

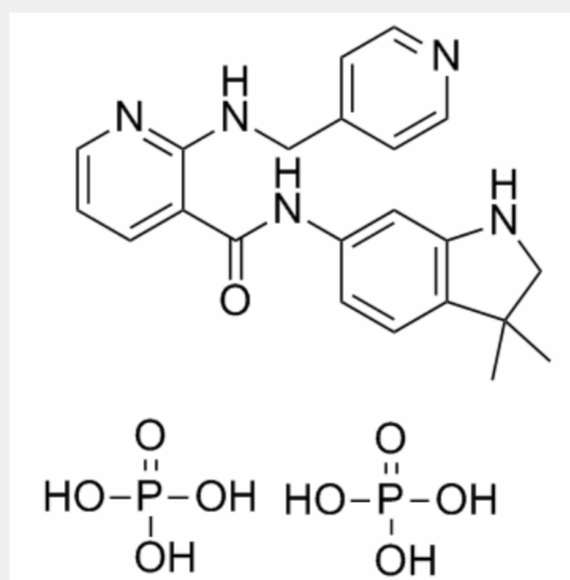
Product Description

Motesanib Diphosphate is a potent ATP-competitive inhibitor of **VEGFR1/2/3** with **IC₅₀**s of 2 nM/3 nM/6 nM, respectively, and has similar activity against Kit, and is approximately 10-fold more selective for VEGFR than PDGFR and Ret.

IC50 & Target: IC50: 2 nM (VEGFR1), 3 nM (VEGFR2), 6 nM (VEGFR3)^[1]

In Vitro: Motesanib has broad activity against the human VEGFR family, and displays over 1000-fold selectivity against EGFR, Src, and p38 kinase. Motesanib significantly inhibits VEGF-induced cellular proliferation of HUVECs with an IC₅₀ of 10 nM, while displaying little effect at bFGF-induced proliferation with an IC₅₀ of >3,000 nM. Motesanib also potently inhibits PDGF-induced proliferation and SCF-induced c-kit phosphorylation with IC₅₀ of 207 nM and 37 nM, respectively, but not effective against the EGF-induced EGFR phosphorylation and cell viability of A431 cells^[1]. Although displaying little antiproliferative activity on cell growth of HUVECs alone, Motesanib treatment significantly sensitizes the cells to fractionated radiation^[2].

In Vivo: Motesanib (100 mg/kg) significantly inhibits VEGF-induced vascular permeability in a time-dependent manner. Oral administration of Motesanib twice daily or once daily potently inhibits, in a dose-dependent manner, VEGF-induced angiogenesis using the rat corneal model with ED₅₀ of 2.1 mg/kg and 4.9 mg/kg, respectively. Motesanib induces a dose-dependent tumor regression of established A431 xenografts by selectively targeting neovascularization in tumor cells^[1]. Motesanib in combination with radiation displays significant anti-tumor activity in head and neck squamous cell carcinoma (HNSCC) xenograft models^[2]. Motesanib treatment also induces significant dose-dependent reductions in tumor growth and blood vessel density of MCF-7, MDA-MB-231, or Cal-51 xenografts, which can be markedly enhanced when combined with docetaxel or tamoxifen^[3].



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