Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

N-Desethyl Sunitinib

Catalog No: tcsc1204

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Specifications

CAS No:
356068-97-8

Formula:
$C_{20}H_{23}FN_4O_2$

Pathway:
Others

Target:
Others

Purity / Grade:
>98%

Solubility:
10 mM in DMSO

Alternative Names:
SU-11662

Observed Molecular Weight:
370.42

Product Description

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N-Desethyl Sunitinib is a metabolite of sunitinib. Sunitinib is a potent, ATP-competitive **VEGFR**, **PDGFRβ** and **KIT** inhibitor with $\mathbf{K_i}$ values of 2, 9, 17, 8 and 4 nM for VEGFR -1, -2, -3, PDGFRβ and KIT, respectively.

In Vitro: Sunitinib also potently inhibits Kit and FLT-3[1]. Sunitinib is a potent ATP-competitive inhibitor of VEGFR2 (Flk1) and PDGFRβ with K_i of 9 nM and 8 nM, respectively, displaying >10-fold higher selectivity for VEGFR2 and PDGFR than FGFR-1, EGFR, Cdk2, Met, IGFR-1, Abl, and src. In serum-starved NIH-3T3 cells expressing VEGFR2 or PDGFRβ, Sunitinib inhibits VEGF-dependent VEGFR2 phosphorylation and PDGF-dependent PDGFRβ phosphorylation with IC_{50} of 10 nM and 10 nM, respectively. Sunitinib inhibits VEGF-induced proliferation of serum-starved HUVECs with IC_{50} of 40 nM, and inhibits PDGF-induced proliferation of NIH-3T3 cells overexpressing PDGFRβ or PDGFRα with IC_{50} of 39 nM and 69 nM, respectively[2]. Sunitinib inhibits phosphorylation of wild-type FLT3, FLT3-ITD, and FLT3-Asp835 with IC_{50} of 250 nM, 50 nM, and 30 nM, respectively. Sunitinib inhibits the proliferation of MV4;11 and OC1-AML5 cells with IC_{50} of 8 nM and 14 nM, respectively, and induces apoptosis in a dose-dependent manner[3].

In Vivo: Sunitinib (20-80 mg/kg/day) exhibits broad and potent dose-dependent anti-tumor activity against a variety of tumor xenograft models including HT-29, A431, Colo205, H-460, SF763T, C6, A375, or MDA-MB-435, consistent with the substantial and selective inhibition of VEGFR2 or PDGFR phosphorylation and signaling in vivo. Sunitinib (80 mg/kg/day) for 21 days leads to complete tumor regression in six of eight mice, without tumor re-growing during a 110-day observation period after the end of treatment. Second round of treatment with Sunitinib remains efficacious against tumors that are not fully regressed during the first round of treatment. Sunitinib treatment results in significant decrease in tumor MVD, with appr 40% reduction in SF763T glioma tumors. SU11248 treatment results in a complete inhibition of additional tumor growth of luciferase-expressing PC-3M xenografts, despite no reduction in tumor size[2]. Sunitinib treatment (20 mg/kg/day) dramatically suppresses the growth subcutaneous MV4;11 (FLT3-ITD) xenografts and prolongs survival in the FLT3-ITD bone marrow engraftment model[3].