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

Semaxinib

Catalog No: tcsc1225

Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
204005-46-9

Formula:
$C_{15}H_{14}N_2O$

Pathway:
Protein Tyrosine Kinase/RTK

Target:
VEGFR

Purity / Grade:
>98%

Solubility:
DMSO : 22.5 mg/mL (94.43 mM; Need ultrasonic and warming)

Alternative Names:
SU5416

Observed Molecular Weight:
238.28

Product Description

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Semaxinib (SU5416) is a potent and selective inhibitor of the vascular endothelial growth factor receptor (**Flk-1/KDR**) with an **IC_{50}** of 1.23 μM.

IC50 & Target: IC50: 1.23±0.2 μM (Flk-1 receptor)[1]

In Vitro: Semaxinib (SU5416) inhibits VEGF-driven mitogenesis in a dose-dependent manner with an IC_{50} of 0.04±0.02 μM (n=3). In contrast, Semaxinib (SU5416) blocked FGF-dependent mitogenesis of HUVECs with an IC_{50} of 50 μM (n=10). The selective activity of Semaxinib (SU5416) on Flk-1 is supported by the fact that testing of Semaxinib (SU5416) using NIH 3T3 cells overexpressing either the EGF or insulin receptors indicated a complete lack of activity (IC_{50}>100 μM). This observation is confirmed by immunoblotting after ligand stimulation. An IC_{50} of 20.26±5.2 μM (n=7), which is about 20-fold less in potency on PDGF-dependent autophosphorylation, is observed when SU5416 is tested in NIH 3T3 cells overexpressing the human PDGF receptor β[1].

In Vivo: Daily administration of Semaxinib (SU5416) (i.p., 3 mg/kg/day) inhibits the local growth of C6 tumors in the colon. A comparable level of growth inhibition (62% by day 16; P=0.001) is observed for tumors growing in the colon in comparison with ones growing in the hindflank region (54% by day 18; P=0.001). These results indicate that Semaxinib (SU5416) could inhibit tumor growth at a site other than the subcutaneous implantation site, where the preexisting vasculature may be different[1]. Daily treatment with Semaxinib (SU5416) (25 mg/kg) results in a significantly lower tumor growth rate with tumor masses of up to 8% of that present in control animals by day 22 after implantation. Inhibition of tumor growth is clearly preceded by a marked reduction of the tissue area covered by the newly formed glioma microvasculature in the Semaxinib-treated group, indicating a reduced initial tumor vascularization[2].