

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

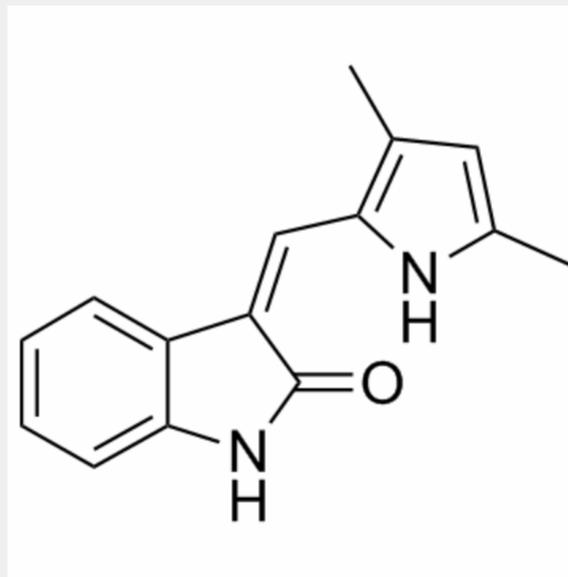
Product Description

Semaxinib (SU5416) is a potent and selective inhibitor of the vascular endothelial growth factor receptor (**Flk-1/KDR**) with an **IC₅₀** of 1.23 μ M.

IC50 & Target: IC50: 1.23 \pm 0.2 μ M (Flk-1 receptor)^[1]

In Vitro: Semaxinib (SU5416) inhibits VEGF-driven mitogenesis in a dose-dependent manner with an IC₅₀ of 0.04 \pm 0.02 μ M (n=3). In contrast, Semaxinib (SU5416) blocked FGF-dependent mitogenesis of HUVECs with an IC₅₀ of 50 μ M (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 (IC₅₀>100 μ M). This observation is confirmed by immunoblotting after ligand stimulation. An IC₅₀ of 20.26 \pm 5.2 μ M (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].



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