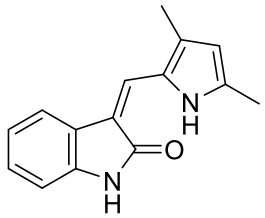
Product data sheet

MedKoo Cat#: 205994 Name: Semaxanib CAS#: 204005-46-9 (free base) Chemical Formula: $C_{15}H_{14}N_2O$ Exact Mass: 238.11061 Molecular Weight: 238.28	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.



1. Product description:
Semaxanib, also known as SU5416, is a quinolone derivative with potential antineoplastic activity. Semaxanib reversibly inhibits ATP binding to the tyrosine kinase domain of vascular endothelial growth factor receptor 2 (VEGFR2), which may inhibit VEGF-stimulated endothelial cell migration and proliferation and reduce the tumor microvasculature. This agent also inhibits the phosphorylation of the stem cell factor receptor tyrosine kinase c-kit, often expressed in acute myelogenous leukemia cells.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	18.28	76.72
DMF	50.0	209.84
Ethanol	2.0	8.39

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	**1 mg**	**5 mg**	**10 mg**
1 mM	4.20 mL	20.98 mL	41.97 mL
5 mM	0.84 mL	4.20 mL	8.39 mL
10 mM	0.42 mL	2.10 mL	4.20 mL
50 mM	0.08 mL	0.42 mL	0.84 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

1. Mezrich JD, Nguyen LP, Kennedy G, Nukaya M, Fechner JH, Zhang X, Xing Y, Bradfield CA. SU5416, a VEGF receptor inhibitor and ligand of the AHR, represents a new alternative for immunomodulation. PLoS One. 2012;7(9):e44547. doi: 10.1371/journal.pone.0044547. Epub 2012 Sep 6. PMID: 22970246; PMCID: PMC3435281.
2. Thill M, Berna MJ, Kunst F, Wege H, Strunnikova NV, Gordiyenko N, Grierson R, Richard G, Csaky KG. SU5416 induces premature senescence in endothelial progenitor cells from patients with age-related macular degeneration. Mol Vis. 2011 Jan 10;17:85-98. PMID: 21245959; PMCID: PMC3021575.

In vivo study

1. Huang X, Zhu J, Jiang Y, Xu C, Lv Q, Yu D, Shi K, Ruan Z, Wang Y. SU5416 attenuated lipopolysaccharide-induced acute lung injury in mice by modulating properties of vascular endothelial cells. Drug Des Devel Ther. 2019 May 23;13:1763-1772. doi: 10.2147/DDDT.S188858. PMID: 31213766; PMCID: PMC6536715.

2. Grailer JJ, Steeber DA. Vascular endothelial growth factor receptor inhibitor SU5416 suppresses lymphocyte generation and immune responses in mice by increasing plasma corticosterone. PLoS One. 2013 Sep 16;8(9):e75390. doi: 10.1371/journal.pone.0075390. PMID: 24066177; PMCID: PMC3774642.

7. Bioactivity

Biological target:

Semaxinib (SU5416) is a potent and selective inhibitor of VEGFR (Flk-1/KDR) with an IC50 of 1.23 μM.

In vitro activity

To analyze the fate of OECs and HUVEC upon long-term inhibition of VEGFR-2 and its downstream signaling pathways, inhibitors were added to the medium every other day for up to 10 days. Treatment with SU5416 resulted in a dose-dependent decrease in proliferation of OECs (Figure 2A). Generally, HUVEC demonstrated a higher proliferation rate when compared to OECs, and proliferation of HUVEC was only decreased or inhibited when higher concentrations of SU5416 were used (Figure 2B).

Reference: Mol Vis. 2011; 17: 85–98. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3021575/

In vivo activity

To determine the biofunction of SU5416 in LPS-induced ALI, this study collected BALF from the mice. In comparison with saline group, LPS significantly increased neutrophil cell numbers in BALF of WT and $TLR4^{-/-}$ mice, while neutrophil cells were significantly diminished in $TLR4^{-/-}$ mice (Figure 1A, $P<0.01$). As a positive control, dexamethasone (DXM) observably inhibited cell population of neutrophil in BALF isolated from two genotype mice ($P<0.01$). In addition, SU5416 exhibited the similar inhibitory effect on the population of neutrophil cell ($P<0.01$). Furthermore, co-treatment with SU5416 and DXM significantly alleviated LPS-induced ALI ($P<0.01$) (Figure 1A). The levels of proinflammatory cytokines (TGF-β, IL-1β, IL-6, and TNF-α) in BALF showed the same trend with the level of neutrophil cells in mice. Moreover, SU5416 and/or DXM significantly reversed LPS-induced proinflammatory factors in BALF (Figure 1B–E).

Reference: Drug Des Devel Ther. 2019; 13: 1763–1772. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6536715/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.