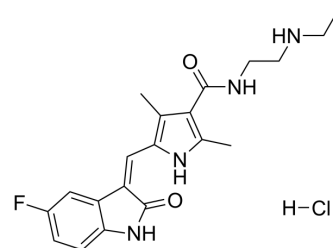


## N-Desethyl Sunitinib hydrochloride

<b>Cat. No.:</b>	HY-138813
<b>Molecular Formula:</b>	C <sub>20</sub> H <sub>24</sub> ClFN <sub>4</sub> O <sub>2</sub>
<b>Molecular Weight:</b>	406.88
<b>Target:</b>	Drug Metabolite
<b>Pathway:</b>	Metabolic Enzyme/Protease
<b>Storage:</b>	4°C, sealed storage, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



### SOLVENT & SOLUBILITY

<b>In Vitro</b>	DMSO : 50 mg/mL (122.89 mM; Need ultrasonic)					
	<b>Preparing Stock Solutions</b>	<b>Solvent</b>	<b>Mass</b>	<b>1 mg</b>	<b>5 mg</b>	<b>10 mg</b>
		<b>Concentration</b>				
		<b>1 mM</b>		2.4577 mL	12.2886 mL	24.5773 mL
		<b>5 mM</b>		0.4915 mL	2.4577 mL	4.9155 mL
<b>10 mM</b>		0.2458 mL	1.2289 mL	2.4577 mL		
Please refer to the solubility information to select the appropriate solvent.						
<b>In Vivo</b>	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: 2.5 mg/mL (6.14 mM); Clear solution; Need ultrasonic  2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 2.5 mg/mL (6.14 mM); Suspended solution; Need ultrasonic					

### BIOLOGICAL ACTIVITY

<b>Description</b>	N-Desethyl Sunitinib (SU-12662) (hydrochloride) is a metabolite of sunitinib. Sunitinib is a potent, ATP-competitive VEGFR, PDGFRβ and KIT inhibitor with K <sub>i</sub> values of 2, 9, 17, 8 and 4 nM for VEGFR -1, -2, -3, PDGFRβ and KIT, respectively <sup>[1]</sup> .
<b>In Vitro</b>	Sunitinib also potently inhibits Kit and FLT-3 <sup>[1]</sup> . Sunitinib is a potent ATP-competitive inhibitor of VEGFR2 (Flk1) and PDGFRβ with K <sub>i</sub> 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 <sub>50</sub> of 10 nM and 10 nM, respectively. Sunitinib inhibits VEGF-induced proliferation of serum-starved HUVECs with IC <sub>50</sub> of 40 nM, and inhibits PDGF-induced proliferation of NIH-3T3 cells overexpressing PDGFRβ or PDGFRα with IC <sub>50</sub> of 39 nM and 69 nM, respectively <sup>[2]</sup> . Sunitinib inhibits phosphorylation of wild-type FLT3, FLT3-ITD, and FLT3-Asp835 with IC <sub>50</sub> of 250 nM, 50 nM, and 30 nM, respectively. Sunitinib inhibits the proliferation of MV4;11 and OC1-AML5 cells with IC <sub>50</sub> of 8 nM and 14 nM, respectively, and induces

	apoptosis in a dose-dependent manner <sup>[3]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>In Vivo</b>	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 approx 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 <sup>[2]</sup> . 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 <sup>[3]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

## CUSTOMER VALIDATION

- Acta Pharmacol Sin. 2016 Jul;37(7):930-40.
- Biol Pharm Bull. 2021;44(10):1565-1570.
- Biomed Chromatogr. 2015 May;29(5):679-88.
- SSRN. 23 Sep 2021.

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## REFERENCES

- [1]. Sun L, et al. Discovery of 5-[5-fluoro-2-oxo-1,2-dihydroindol-(3Z)-ylidenemethyl]-2,4-dimethyl-1H-pyrrole-3-carboxylic acid (2-diethylaminoethyl)amide, a novel tyrosine kinase inhibitor targeting vascular endothelial and platelet-derived growth factor receptor tyrosine kinase. *J Med Chem.* 2003;46(7):1116-1119.
- [2]. Mendel DB, et al. In vivo antitumor activity of SU11248, a novel tyrosine kinase inhibitor targeting vascular endothelial growth factor and platelet-derived growth factor receptors: determination of a pharmacokinetic/pharmacodynamic relationship. *Clin Cancer Res.* 2003;9(1):327-337.
- [3]. O'Farrell AM, et al. SU11248 is a novel FLT3 tyrosine kinase inhibitor with potent activity in vitro and in vivo. *Blood.* 2003;101(9):3597-3605.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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