Orantinib

Cat. No.: HY-10517

CAS No.: 252916-29-3

Molecular Formula: $C_{18}H_{18}N_2O_3$

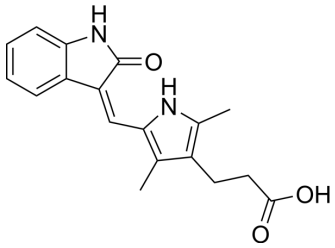
Molecular Weight: 310.35

Target: PDGFR; FGFR; VEGFR; Apoptosis

Pathway: Protein Tyrosine Kinase/RTK; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (322.22 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2222 mL	16.1108 mL	32.2217 mL
	5 mM	0.6444 mL	3.2222 mL	6.4443 mL
	10 mM	0.3222 mL	1.6111 mL	3.2222 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 10 mg/mL (32.22 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Orantinib (SU6668; TSU-68) is a multi-targeted receptor tyrosine kinase inhibitor with K_is of 2.1 μM, 8 nM and 1.2 μM for Flt-1, PDGFRβ and FGFR1, respectively.

IC_{50} & Target

PDGFRβ	FGFR1	Flt-1
8 nM (Ki)	1.2 μM (Ki)	2.1 μM (Ki)

In Vitro

Orantinib (SU6668; 0.03-10 μM) shows inhibitory activity against tyrosine phosphorylation of KDR in VEGF stimulated HUVECs, and also blocks PDGF-stimulated PDGFRβ tyrosine phosphorylation in NIH-3T3 cells overexpressing PDGFRβ. Orantinib (≥10 μM) inhibits acidic FGF-induced phosphorylation of the FGFR1 substrate 2. However, Orantinib (up to 100 μM) has no effect on EGF-stimulated EGFR tyrosine phosphorylation in NIH-3T3 cells overexpressing EGFR. Furthermore, Orantinib inhibits VEGF-driven and FGF-driven mitogenesis of HUVECs with mean IC_{50} of 0.34 μM and 9.6 μM, respectively[1]. In human myeloid leukemia MO7E cells, Orantinib (SU6668) inhibits the tyrosine autophosphorylation of stem cell factor

	(SCF) receptor, c-kit, with IC_{50} of 0.1-1 μM, as well as ERK1/2 phosphorylation. In addition, Orantinib suppresses SCF-induced proliferation of MO7E cells with an IC_{50} of 0.29 μM, and induces apoptosis[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Orantinib (SU6668; 75-200 mg/kg) causes tumor growth inhibition on several tumor types in xenograft models in athymic mice, such as A375, Colo205, H460, Calu-6, C6, SF763T, and SKOV3TP5 cells. Orantinib (75 mg/kg) also inhibits tumor angiogenesis of C6 glioma xenografts[1]. In a tumor model of HT29 human colon carcinoma, Orantinib (200 mg/kg) decreases the average vessel permeability and average fractional plasma volume in the tumor rim and core. Orantinib enhances abnormal stromal development at the periphery of carcinomas[3]. Moreover, Orantinib (TSU-68; 200 mg/kg) augments the effect of chemotherapeutic infusion in a rabbit VX2 liver tumor model[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- PLoS Genet. 2022 Jun 27;18(6):e1010292.
- Patent. US20180263995A1.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Laird AD, et al. SU6668 is a potent antiangiogenic and antitumor agent that induces regression of established tumors. Cancer Res, 2000, 60(15), 4152-4160.

[2]. Smolich BD, et al. The antiangiogenic protein kinase inhibitors SU5416 and SU6668 inhibit the SCF receptor (c-kit) in a human myeloid leukemia cell line and in acute myeloid leukemia blasts. Blood, 2001, 97(5), 1413-1421.

[3]. Marzola P, et al. In vivo assessment of antiangiogenic activity of SU6668 in an experimental colon carcinoma model. Clin Cancer Res, 2004, 10(2), 739-750.

[4]. Kim HC, et al. Augmentation of chemotherapeutic infusion effect by TSU-68, an oral targeted antiangiogenic agent, in a rabbit VX2 liver tumor model. Cardiovasc Intervent Radiol. 2012 Feb;35(1):168-75

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA