(Z)-Orantinib

Cat. No.: HY-10517A

CAS No.: 210644-62-5

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

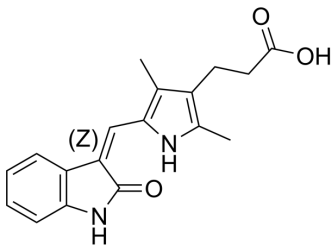
Molecular Weight: 310.35

Target: VEGFR; PDGFR; FGFR

Pathway: Protein Tyrosine Kinase/RTK

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (161.11 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / 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: 2.08 mg/mL (6.70 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (6.70 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

(Z)-Orantinib ((Z)-SU6668) is a potent, selective, orally active and ATP competitive inhibitor of Flk1/KDR, PDGFRβ, and FGFR1, with IC_{50}s of 2.1, 0.008, and 1.2 μM, respectively. (Z)-Orantinib is a potent antiangiogenic and antitumor agent that induces regression of established tumors[1][2].

IC_{50} & Target

Flk-1/KDR	PDGFRβ	FGFR1
2.1 μM (IC_{50})	0.008 μM (IC_{50})	1.2 μM (IC_{50})

In Vitro

SU6668 (5-15 min) inhibits Flk-1 trans-phosphorylation (K_i=2.1 μM), FGFR1 trans-phosphorylation (K_i=1.2 μM), and PDGFR autophosphorylation (K_i=0.008 μM)[1].

SU6668 (0.03-10 μM; 60 min) inhibits the VEGF-stimulated increase of KDR tyrosine phosphorylation in HUVECs[1].

SU6668 inhibits mitogenesis of HUVECs induced by both VEGF and FGF in a dose-dependent manner with IC_{50}s of 0.34 and 9.6 μM, respectively[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

SU6668 (4-200 mg/kg/day; p.o. for 21 d) induces dose-dependent inhibition of A431 tumor growth in athymic mice[1].

SU6668 (75 mg/kg/day; i.p. for 22 d) significantly suppresses tumor angiogenesis and vascularization in mice[1].

SU6668 (200 mg/kg/day; p.o. for 11-27 d) induces striking regression of large established A431 xenografts in athymic mice[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female athymic mice (BALB/c, nu/nu) were implanted A431 tumor cells[1]
Dosage:	4, 40, 75, 200 mg/kg
Administration:	P.o. daily for 21 days
Result:	Induced 97% growth inhibition against A431 tumor at the dose of 97%. No mortality was observed in any treatment group.

REFERENCES

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

[2]. Laird ad, et, al. SU6668 inhibits Flk-1/KDR and PDGFRbeta in vivo, resulting in rapid apoptosis of tumor vasculature and tumor regression in mice. FASEB J. 2002 May;16(7):681-90.

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