

## Product Data Sheet

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Product Name: (Z)-Orantinib

Cat. No.: GC62291

### Chemical Properties

Cas. No. 210644-62-5

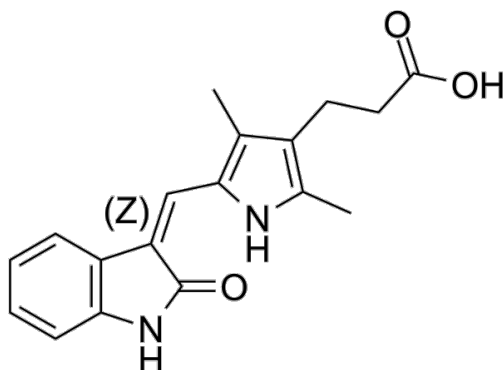
Formula  $C_{18}H_{18}N_2O_3$  M.Wt 310.35

Solubility DMSO : 50 mg/mL (161.11 mM; Need ultrasonic) Storage Store at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure



### Background

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

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(Z)-Orantinib ((Z)-SU6668) is a potent, selective, orally active and ATP competitive inhibitor of Flk-1/KDR, PDGFR $\beta$ , and FGFR1, with IC<sub>50</sub>s of 2.1, 0.008, and 1.2  $\mu$ M, respectively. (Z)-Orantinib is a potent antiangiogenic and antitumor agent that induces regression of established tumors[1][2].

SU6668 (5-15 min) inhibits Flk-1 trans-phosphorylation ( $K_i$ =2.1  $\mu$ M), FGFR1 trans-phosphorylation ( $K_i$ =1.2  $\mu$ M), and PDGFR autophosphorylation ( $K_i$ =0.008  $\mu$ M)[1]. SU6668 (0.03-10  $\mu$ M; 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<sub>50</sub>s of 0.34 and 9.6  $\mu$ M, respectively[1].

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].

[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 PDGFR $\beta$  in vivo, resulting in rapid apoptosis of tumor vasculature and tumor regression in mice. FASEB J. 2002 May;16(7):681-90.

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