Product Data Sheet

Product Name: Vatalanib
Cat. No.: GC14464

Chemical Properties

Cas. No.	212141-54-3		
Chemical Name	N-(4-chlorophenyl)-4-(pyridin-4-ylmethyl)phthalazin-1-amine		
SMILES	C1=CC=C2C(=C1)C(=NN=C2NC3=CC=C(C=C3)Cl)CC4=CC=NC=C4		
Formula	$C_{20}H_{15}ClN_4$	M.Wt	346.81
Solubility	≥ 16.85 mg/mL in DMSO, ≥ 3.0125 mg/mL in EtOH with ultrasonic and warming, ≥ 32.53 mg/mL in Water with ultrasonic and warming	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	Vatalanib		

Protocol

Kinase experiment: Each GST-fused kinase is incubated under optimized buffer conditions. ATP in a total volume of 30 µL in the presence or absence of a test substance (Vatalanib) for 10 min at ambient temperature. The reaction is stopped by adding 10 µL of 250 mM EDTA[1].

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Cell experiment:

Subconfluent HUVECs are seeded into 96-well plates coated with 1.5% gelatin. After 24 h, growth medium is replaced by basal medium containing 1.5% FCS and a constant concentration of VEGF (50 ng/mL), bFGF (0.5 ng/mL), or FCS (5%), in the presence or absence of Vatalanib. As a control, wells without growth factor are also included. After 24 h of incubation, BrdUrd labeling solution is added, and cells incubated an additional 24 h before fixation, blocking, and addition of peroxidase-labeled anti-BrdUrd antibody. Bound antibody is then detected using 3,3' 5,5'-tetramethylbenzidine substrate[1].

Animal experiment:

A porous Teflon chamber (volume, 0.5 mL) is filled with 0.8% w/v agar containing heparin (20 units/mL) with or without growth factor (3 μg/mL human VEGF, 2 μg/mL human PDGF) is implanted s.c. on the dorsal flank of C57/C6 mice. The mice are treated with Vatalanib (12.5, 25 or 50 mg/kg dihydrochloride p.o. once daily) or vehicle (water) starting 1 day before implantation of the chamber and continuing for 5 days after. At the end of treatment, the mice are killed, and the chambers are removed. The vascularized tissue growing around the chamber is carefully removed and weighed, and the blood content is assessed by measuring the hemoglobin content of the tissue[1].

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References:

[1]. Wood JM, et al. PTK787/ZK 222584, a novel and potent inhibitor of vascular endothelial growth factor receptor tyrosine kinases, impairs vascular endothelial growth factor-induced responses and tumor growth after oral administration. Cancer Res. 2000, 60(8

[2]. Murakami M, et al. Tyrosine kinase inhibitor PTK/ZK enhances the antitumor effects of interferon-α/5-fluorouracil therapy for hepatocellular carcinoma cells. Ann Surg Oncol. 2011, 18(2), 589-596.

[3]. Wan J, et al. Local recurrence of small cell lung cancer following radiofrequency ablation is induced by HIF-1α expression in the transition zone. Oncol Rep. 2016 Mar;35(3):1297-308.

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Background

Vatalanib is a novel and potent inhibitor of VEGFR with IC50 value of 77 nM, 27 nM and 37 nM for VEGFR-1 (Flt-1), VEGFR-2 (FLK-1) and VEGFR-2 (KDR), respectively [1].
The vascular endothelial growth factor receptors (VEGFRs) are tyrosine kinases and are receptors for VEGF. VEGF acts as a key factor in pathological situations that involve in pathological situations that involve enhancing vascular permeability as well as neovascularization [1].
In CHO and HUVECs cells transfected with the KDR receptor, Vatalanib inhibited the VEGF-induced phosphorylation of KDR with an IC50 of 34 nM and 17 nM for the CHO and HUVECs cells, respectively. Also, Vatalanib inhibited thymidine incorporation induced by VEGF with IC50 value of 7.1 nM in HUVECs cells. Vatalanib inhibited VEGF-induced endothelial cell proliferation in a dose-dependant way [1].
In a growth factor implant mice model, Vatalanib (12.5, 25 or 50 mg/kg, 6 days) inhibited the angiogenic response around the implant induced by VEGF and PDGF [1]. In a xenograft mouse model, treatment mice with Vatalanib through gastric tube daily caused tumor inhibition rate of 76% [2].

References:

[1]. Wood JM, Bold G, Buchdunger E, et al. PTK787/ZK 222584, a novel and potent inhibitor of vascular endothelial growth factor receptor tyrosine kinases, impairs vascular endothelial growth factor-induced responses and tumor growth after oral administration. Cancer Res, 2000, 60(8): 2178-2189.

[2]. Paesler J, Gehrke I, Gandhirajan RK, et al. The vascular endothelial growth factor receptor tyrosine kinase inhibitors vatalanib and pazopanib potently induce apoptosis in chronic lymphocytic leukemia cells in vitro and in vivo. Clin Cancer Res, 2010, 16(13): 3390-3398.

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