JK-P3

Cat. No.: HY-108933

CAS No.: 942655-44-9

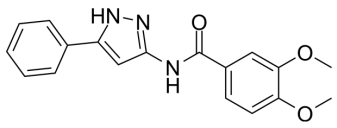
Molecular Formula: $C_{18}H_{17}N_3O_3$

Molecular Weight: 323.35

Target: VEGFR; FGFR

Pathway: Protein Tyrosine Kinase/RTK

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (154.63 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0926 mL	15.4631 mL	30.9262 mL
	5 mM	0.6185 mL	3.0926 mL	6.1852 mL
	10 mM	0.3093 mL	1.5463 mL	3.0926 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: ≥ 2.5 mg/mL (7.73 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.73 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.73 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

JK-P3 is a potent and pan VEGFR2 inhibitor, with IC_{50}s of 7.83 μM, 27 μM and 5.18 μM for VEGFR2, FGFR1 and FGFR3, respectively. JK-P3 can inhibit VEGF-A-stimulated VEGFR2 activation and intracellular signalling, also inhibits endothelial monolayer wound closure and angiogenesis, as well as fibroblast growth factor receptor kinase activity in vitro. JK-P3 has anti-angiogenic activity[1].

IC_{50} & Target			
	VEGFR2	FGFR1	FGFR3
	7.83 μM (IC_{50})	27 μM (IC_{50})	5.18 μM (IC_{50})

In Vitro

JK-P3 (0.01-10 μM; 1 hour) inhibits VEGF-A-mediated VEGFR2 phosphorylation and downstream signalling[1].

JK-P3 (0.01-10 μM; 16 hours) dose not inhibit HUVEC cell proliferation at 0.01~1 μM, and shows slight inhibitory activity at 10 μM[1].

JK-P3 (1 and 10 μM; 1 hour) does not significantly inhibit VEGF-A-stimulated endothelial tube formation at 1 μM, but almost completely inhibits the ability of endothelial cells to form into elongated hollow tubes in the presence of VEGF-A at 10 μM[1].

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

Western Blot Analysis

Cell Line:	Primary endothelial cells (treated for 7.5 min with 25 ng/mL VEGF-A)[1]
Concentration:	0.01, 0.1, 1 and 10 μM
Incubation Time:	1 hour
Result:	Almost completely inhibited VEGFR2 Y1175 phosphorylation, also inhibited VEGF-A-stimulated PLCγ1, Akt and ERK1/2 phosphorylation.

Cell Proliferation Assay

Cell Line:	HUVEC[1]
Concentration:	0.01, 0.1, 1 and 10 μM
Incubation Time:	16 hours
Result:	Failed to inhibit endothelial cell proliferation at 0.01~1 μM but elicited a small but significant increase in cell proliferation at certain lower concentrations.

REFERENCES

[1]. Kankanala J, et al. A combinatorial in silico and cellular approach to identify a new class of compounds that target VEGFR2 receptor tyrosine kinase activity and angiogenesis. Br J Pharmacol. 2012;166(2):737-748.

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