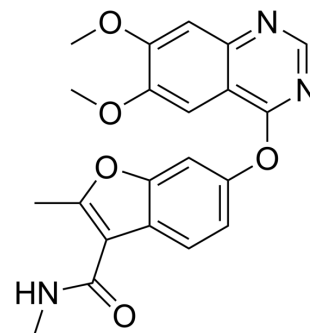


Fruquintinib

Cat. No.:	HY-19912		
CAS No.:	1194506-26-7		
Molecular Formula:	C ₂₁ H ₁₉ N ₃ O ₅		
Molecular Weight:	393.39		
Target:	VEGFR		
Pathway:	Protein Tyrosine Kinase/RTK		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	1 year
		-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro	DMSO : 5.83 mg/mL (14.82 mM; Need ultrasonic)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	2.5420 mL	12.7100 mL	25.4201 mL
		5 mM	0.5084 mL	2.5420 mL	5.0840 mL
10 mM		0.2542 mL	1.2710 mL	2.5420 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: ≥ 0.59 mg/mL (1.50 mM); Clear solution				
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 0.59 mg/mL (1.50 mM); Clear solution				
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 0.59 mg/mL (1.50 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	Fruquintinib (HMPL-013) is a highly potent and selective VEGFR 1/2/3 inhibitor with IC ₅₀ s of 33, 0.35, and 35 nM, respectively.		
IC ₅₀ & Target	VEGFR1 33 nM (IC ₅₀)	VEGFR2 35 nM (IC ₅₀)	VEGFR3 0.5 nM (IC ₅₀)
In Vitro	Fruquintinib demonstrates potent inhibition on VEGF-A dependent KDR phosphorylation in HEK293-KDR cells and VEGF-A induced proliferation in primary HUVECs with IC ₅₀ s of 0.6±0.2 nM and 1.7 nM, respectively. Similarly, potent VEGFR3		

attenuation by fruquintinib is observed in primary HLECs, with IC₅₀s of 1.5 nM and 4.2 nM for VEGF-C stimulated VEGFR3 phosphorylation and proliferation, respectively. Fruquintinib suppresses the tube branching, tube length and area in a concentration-dependent manner. The tubule length of primary HUVECs decreased by 74% and 94% at 0.03 and 0.3 μM of fruquintinib, respectively. Fruquintinib inhibits HUVEC tubule growth and CAM angiogenesis. Tube formation is suppressed significantly after treatment with fruquintinib at 0.3 μM for 18 hours^[1].

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

In Vivo

Gastric cancer BGC-823 model is found to be most sensitive to fruquintinib. In this model, fruquintinib inhibits tumor growth by 62.3% and 95.4% at 0.5 and 2 mg/kg once daily dosing, respectively. When the dose is elevated to 5 mg/kg and 20 mg/kg, the tumors regress by 24.1% and 48.6%, respectively. The level of anti-tumor growth activity of fruquintinib varies in different tumor xenograft models. Fruquintinib significantly decreases the micro-vessel density even at the lowest dose of 0.8 mg/kg^[1].

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PROTOCOL

Cell Assay ^[1]

Primary HUVECs or HLECs in exponential phase are suspended in 100 μL of RPMI-1640 media containing 0.5% FBS, and seeded at 5000 cell/well in 96-well plates pre-coated with 0.2% gelatin or fibronectin, and incubated overnight in a 5% CO₂, 37°C incubator. Fruquintinib and VEGF-A165 or VEGF-C (50 ng/mL) are added and incubated for 48 hours. Viability of the cells is determined using CCK-8 assay format^[1].

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Animal Administration ^[1]

Mice: The patient derived xenograft models are established after the primary tumor adopted serial passages in vivo. Once tumors have grown to 100-300 mm³, the animals are randomly assigned with 6-8 animals per group. The mice are treated orally with the vehicle (control group) or fruquintinib at a dose range of 0.5-20 mg/kg suspended in the vehicle (treated group) once daily for 3 weeks. In combination studies, docetaxel (Taxotere, 5 mg/kg) or oxaliplatin (10 mg/kg) is administered to nude mouse via intravenous injection, once a week. Tumor size and body weights are measured 3 times a week. Tumor volumes (TV) are calculated^[1].

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

CUSTOMER VALIDATION

- Cell Death Differ. 2024 Oct 15.
- Mol Syst Biol. 2023 Dec 18.
- Eur J Med Chem. 2023 Nov 5, 259, 115703.
- Chemotherapy. 2023 Jan 9.
- Biochem Biophys Res Commun. 2023 Apr 10.

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REFERENCES

[1]. Sun Q, et al. Discovery of fruquintinib, a potent and highly selective small molecule inhibitor of VEGFR 1, 2, 3 tyrosine kinases for cancer therapy. Cancer Biol Ther. 2014;15(12):1635-45.

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

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