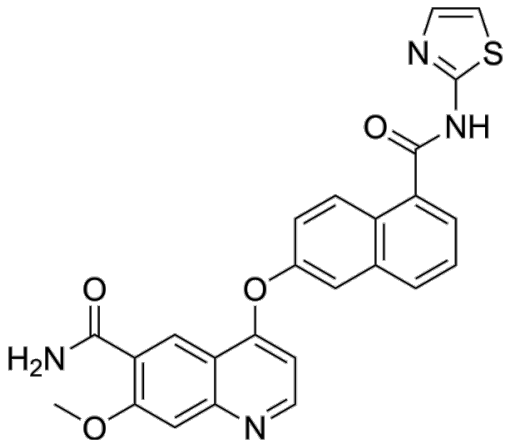
Product Data Sheet

Product Name: VEGFR2-IN-84
Cat. No.: GC81325

Chemical Properties

Cas. No.	861877-12-5		
Formula	$C_{25}H_{18}N_4O_4S$	M.Wt	470.5
Solubility		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

VEGFR2-IN-84 is an orally active, multi-targeted tyrosine kinase inhibitor based on a naphthalene ring scaffold. VEGFR2-IN-84 inhibits VEGFR2 with sub-nanomolar affinity and broadly targets kinases including Kit, FGFR, PDGFR, and Ret. By competitively binding to the ATP -binding pocket, VEGFR2-IN-84 blocks the phosphorylation of VEGFR2 and its downstream AKT/ERK signaling pathway, thereby significantly inhibiting endothelial cell proliferation, migration, and tumor angiogenesis. VEGFR2-IN-84 exhibits broad-spectrum antiproliferative activity against various solid tumors such as liver cancer, lung cancer, and renal cancer, shows weak toxicity to normal cells, and has superior potency to Lenvatinib. VEGFR2-IN-84 possesses favorable pharmacokinetic properties

Caution: Product has not been fully validated for medical applications. For research use only.
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and high safety (LD 50 >2000 mg/kg), and can be used in related studies of various malignant tumors [1].

In Vivo, VEGFR2-IN-84 (E20) (50 mg/kg; p.o.; once daily; 21 days) significantly inhibits tumor growth in a male BALB/c nude mouse HepG2 hepatocellular carcinoma xenograft model, with efficacy superior to that of Lenvatinib at the same dose, and causes no obvious body weight loss[1]. VEGFR2-IN-84 (30-60 mg/kg; p.o.; once daily; 20-30 days) dose-dependently inhibits tumor growth in male BALB/c nude mouse xenograft models of A549 lung cancer, 786-O renal cell carcinoma, and 8305C thyroid carcinoma, respectively, and exhibits superior efficacy over Lenvatinib at the same dose[1]. VEGFR2-IN-84 (2 mg/kg; tail vein injection; single dose; 24 h) exhibits a moderate elimination half-life (T1/2=5.05 h) and plasma exposure (AUC0-∞=1970 ng?h/mL) in healthy ICR mice[1].

In Vitro, VEGFR2-IN-84 (E20) (4.92 nM; 96 h) significantly inhibits VEGF-induced proliferation of HUVEC cells, with approximately 10-fold the activity of Lenvatinib [1]. VEGFR2-IN-84 (10-1000 nM; 48 h) dose-dependently inhibits the phosphorylation of VEGFR2 and its downstream molecules AKT and ERK in HUVEC, A549 and HepG2 cells, and exhibits superior activity to Lenvatinib[1]. VEGFR2-IN-84 (8.53-14.1 μM; 72 h) exhibits weak inhibitory activity against normal cells LO2, HEK293T and BEAS-2B, showing favorable tumor cell selectivity[1].

References:
[1]. Yang T, et al. Structure-Based Discovery of Novel, Highly Potent VEGFR2 Inhibitors with a Naphthalene Scaffold against a Broad Range of Solid Tumors. J Med Chem. Published online March 20, 2026.