

Product Data Sheet

Product Name: Lenvatinib (E7080)

Cat. No.: GC15454

Chemical Properties

Cas. No. 417716-92-8

Chemical Name 4-[3-chloro-4-(cyclopropylcarbamoylamino)phenoxy]-7-methoxyquinoline-6-carboxamide

SMILES COC1=CC2=NC=CC(=C2C=C1C(=O)N)OC3=CC(=C(C=C3)NC(=O)NC4CC4)Cl

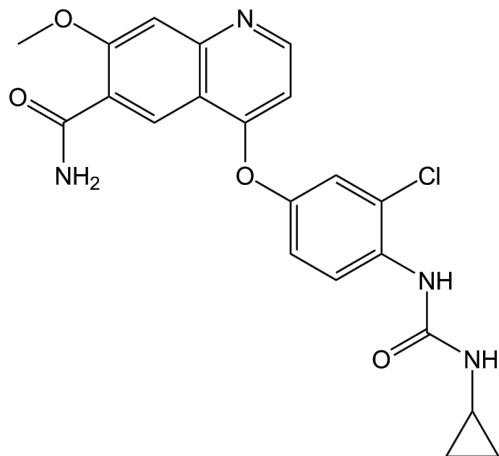
Formula $C_{21}H_{19}ClN_4O_4$ M.Wt 426.85

Solubility DMSO: 17 mg/mL (40 mM) Storage Store at 4°C, protect from light

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



Protocol

Cell experiment [1]:

Cell lines

HCCC-9810, RBE cells

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Preparation Method	HCCC-9810 and RBE cells were maintained in RPMI 1640 medium supplemented with 10% FBS at 37°C, 5% CO ₂ . Cells were treated with 12.5-50µM Lenvatinib for 48h, 72h or 72h, followed by CCK-8 assay, colony formation assay, flow cytometry for cell cycle, Western blot for CDK1/CDK2/CyclinB1/CyclinE/p21/Gadd45a, qRT-PCR for Gadd45a mRNA, and RNA sequencing after 0µM or 25µM Lenvatinib treatment.
Reaction Conditions	12.5-50µM; 48-72h
Applications	Lenvatinib dose- and time-dependently inhibited HCCC-9810 and RBE cell viability and colony formation. Lenvatinib induced G0/G1 phase cell cycle arrest in HCCC-9810 and RBE cells. Lenvatinib upregulated Gadd45a and p21 protein and mRNA expression, and downregulated CDK1, CDK2, CyclinB1 and CyclinE protein expression in HCCC-9810 and RBE cells.
Animal experiment [2]:	
Animal models	6-week-old C57BL/6 mice
Preparation Method	5×10 ⁵ Hepa1-6 cells were implanted subcutaneously into mice. Once tumors reached approximately 50mm ³ , mice were randomized into groups (6 mice per group) and administered 4mg/kg Lenvatinib by gavage daily for 18 days. Tumor volumes were recorded every 3 days. At the end of treatment, mice were sacrificed and tumors were harvested for immunohistochemistry staining of Ki67 and cleaved caspase-3.
Dosage form	4mg/kg; oral gavage; daily for 18 days

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Applications	Lenvatinib administration inhibited Hepa1-6 subcutaneous tumor growth and reduced final tumor weight. Lenvatinib administration did not significantly reduce mouse body weight. Lenvatinib administration decreased Ki67-positive tumor cell proportion and increased cleaved caspase-3-positive tumor cell proportion in harvested tumors.
References: [1] Yan X, Wang D, Ning Z, et al. Lenvatinib inhibits intrahepatic cholangiocarcinoma via Gadd45a-mediated cell cycle arrest. <i>Discov Oncol.</i> 2023 Feb 23;14:26. [2] Yan S, Chen L, Zhuang H, et al. HDAC Inhibition Sensitize Hepatocellular Carcinoma to Lenvatinib via Suppressing AKT Activation. <i>Int J Biol Sci.</i> 2024;20(8):3046-3060.	

Background

Lenvatinib (E7080) is a multi-targeted receptor tyrosine kinase inhibitor. Lenvatinib inhibits the kinase activities of VEGFR1-3, FGFR1-4, PDGFR α , KIT and RET. Lenvatinib blocks VEGF and FGF signaling pathways to suppress tumor angiogenesis and directly inhibits tumor cell proliferation. Lenvatinib can be used for research on radioactive iodine-refractory differentiated thyroid cancer, unresectable hepatocellular carcinoma, advanced renal cell carcinoma and advanced endometrial cancer^[1-4].

In vitro, treatment of HCCC-9810 or RBE intrahepatic cholangiocarcinoma cells with 12.5 μ M-50 μ M Lenvatinib for 48-72 hours dose-dependently inhibited cell viability and colony formation and induced G0/G1 phase cell cycle arrest^[5]. Treatment of multiple liver cancer cell lines (KIM-1, KYN-1, KYN-2, etc.) with 1.875-30 μ M Lenvatinib for 24-72 hours time- and dose-dependently inhibited cell proliferation. Treatment with 7.5 μ M or 30 μ M Lenvatinib for 48 hours did not induce apoptosis^[6]. Treatment of ATC cells with 1nM-50 μ M Lenvatinib for 12-48 hours inhibited ATC cell proliferation, increased the proportion of apoptotic cells, and suppressed cell migration and invasion ability^[7].

In vivo, daily oral gavage or intraperitoneal administration of 4mg/kg Lenvatinib for 18 days in C57BL/6 mice subcutaneously implanted with Hepa1-6 cells inhibited subcutaneous tumor growth

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and reduced final tumor weight without significantly reducing mouse body weight^[8]. Daily oral gavage of 10mg/kg Lenvatinib in BALB/c nude mice subcutaneously implanted with GBC-SD cells inhibited GBC-SD xenograft tumor growth, reduced Ki-67-positive cell proportion and CD31 microvessel density in tumor tissues, and decreased phosphorylated ERK1/2 staining intensity^[9]. Daily oral gavage of 1-100mg/kg Lenvatinib for 14 days in C57BL/6 nude mice subcutaneously implanted with thyroid cancer cells dose-dependently inhibited thyroid cancer xenograft tumor growth and reduced xenograft tumor microvessel density^[10].

References:

- [1] Chen Y, Dai S, Cheng CS, et al. Lenvatinib and immune-checkpoint inhibitors in hepatocellular carcinoma: mechanistic insights, clinical efficacy, and future perspectives. J Hematol Oncol. 2024 Dec 21;17(1):130.
- [2] Wang L, Yang Q, Zhou Q, et al. METTL3-m⁶A-EGFR-axis drives lenvatinib resistance in hepatocellular carcinoma. Cancer Lett. 2023 Apr 10;559:216122.
- [3] Al-Salama ZT, Syed YY, Scott LJ. Lenvatinib: A Review in Hepatocellular Carcinoma. Drugs. 2019 Apr;79(6):665-674.
- [4] Hao Z, Wang P. Lenvatinib in Management of Solid Tumors. Oncologist. 2020 Feb;25(2):e302-e310.
- [5] Yan X, Wang D, Ning Z, et al. Lenvatinib inhibits intrahepatic cholangiocarcinoma via Gadd45a-mediated cell cycle arrest. Discov Oncol. 2023 Feb 23;14:26.
- [6] Ogasawara S, Mihara Y, Kondo R, et al. Antiproliferative effect of lenvatinib on human liver cancer cell lines in vitro and in vivo. Anticancer Res. 2019;39(11):5973-5982.
- [7] Ferrari SM, Bocci G, Di Desidero T, et al. Lenvatinib exhibits antineoplastic activity in anaplastic thyroid cancer in vitro and in vivo. Oncol Rep. 2018;39(5):2225-2234.
- [8] Yan S, Chen L, Zhuang H, et al. HDAC Inhibition Sensitize Hepatocellular Carcinoma to Lenvatinib via Suppressing AKT Activation. Int J Biol Sci. 2024;20(8):3046-3060.
- [9] Sun H, Li S, Zhu Z, et al. Lenvatinib enhances the response of biliary tract cancer to anti-HER2

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therapy. Signal Transduct Target Ther. 2026;11:303.

[10] Tohyama O, Matsui J, Kodama K, et al. Antitumor activity of lenvatinib (E7080): an angiogenesis inhibitor that targets multiple receptor tyrosine kinases in preclinical human thyroid cancer models. J Thyroid Res. 2014;2014:638747.

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