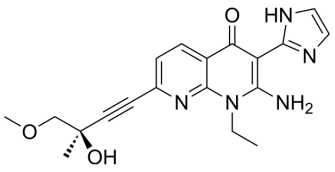
EVT801

Cat. No.:	HY-152293
CAS No.:	1412453-70-3
Molecular Formula:	$C_{19}H_{21}N_5O_3$
Molecular Weight:	367.4
Target:	VEGFR; ERK
Pathway:	Protein Tyrosine Kinase/RTK; MAPK/ERK Pathway; Stem Cell/Wnt
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

EVT801 is an orally active and selective inhibitor of VEGFR-3 (IC_{50}=11 nM), which has antitumor effects. EVT801 inhibits not only VEGF-C-induced human endothelial cell proliferation, but also tumor (lymphatic) angiogenesis in tumor mouse models. EVT801 can reduce tumor hypoxia, immunosuppressive cytokines (CCL4, CCL5) and myeloid derived suppressor cells (MDSC) production. EVT801 has synergistic effect with immune checkpoint therapy (ICT), which improves ICT response rate and has better inhibitory effect on cancer mouse models[1]. EVT801 is a click chemistry reagent, it contains an Alkyne group and can undergo copper-catalyzed azide-alkyne cycloaddition (CuAAc) with molecules containing Azide groups.

IC_{50} & Target

VEGFR3	VEGFR1	VEGFR2	ERK
11 nM (IC_{50})	396 nM (IC_{50})	130 nM (IC_{50})	13 nM (IC_{50})

In Vitro

EVT801 (10 nM-1 μM) dose-dependently inhibits VEGFR-1/2/3 autophosphorylation in HEK293 cells with IC_{50}s of 39 nM (VEGFR-3), 2130 nM (VEGFR-1), 260 nM (VEGFR-2), respectively[1].

EVT801 (1 nM-1 μM) prevents proliferation of VEGFR-3-positive cells, human lymphatic microvascular endothelial cells (hLMVEC) for example. EVT801 inhibits the induction of hLMVECs proliferation dose-dependently with IC_{50}s of 15 nM (VEGF-C), 8 nM (VEGF-D), 155 nM (VEGF-A), respectively[1].

EVT801 (1 μM) inhibits proliferation and tumor growth of VEGFR-3-positive tumor cells[1].

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

Cell Proliferation Assay[1]

Cell Line:	VEGFR-3-positive cells, human lymphatic microvascular endothelial cells (hLMVEC)
Concentration:	1 nM-1 μM
Incubation Time:	
Result:	Showed a maximum inhibition of 74%, 100%, and 65% against VEGF-C, VEGF-D, VEGF-A induction, respectively.

In Vivo

EVT801 (30 mg/kg; p.o.; twice daily for 7 d) shows inhibitory effect on VEGFR-3-positive tumors in mouse models, such as RT-001-HAM Subcutaneous Patient-derived xenograft (PDx) Tumor Mouse Model, 4T1 Mammary Carcinoma Mouse Model, N-diethylnitrosamine-Induced Hepatocarcinoma Mouse Model, NCI-H1703 Subcutaneous Xenograft Tumor Mouse Model, Rip1-Tag2/transgenic Mouse Models, and CT26 Ectopic Tumor Mouse Model. EVT801 is expressed in blood vessels of kidney cancer primary tumors and metastases, and in tumor cells of endothelial malignancies[1].

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

REFERENCES

[1]. Paillasse M R, et al. Targeting Tumor Angiogenesis with the Selective VEGFR-3 Inhibitor EVT801 in Combination with Cancer Immunotherapy[J]. Cancer Research Communications, 2022, 2(11): 1504-1519.

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