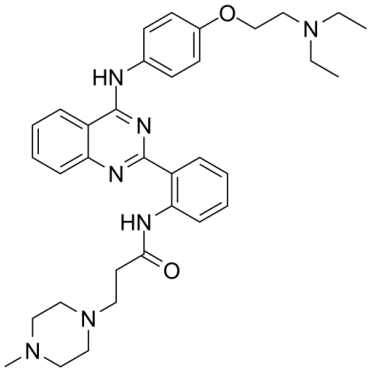
Data Sheet

Product Name:	hVEGF-IN-1
Cat. No.:	CS-7769
CAS No.:	1637443-98-1
Molecular Formula:	C34H43N7O2
Molecular Weight:	581.75
Target:	VEGFR
Pathway:	Protein Tyrosine Kinase/RTK
Solubility:	DMSO : 25 mg/mL (42.97 mM; Need ultrasonic); H2O : < 0.1 mg/mL (insoluble)



BIOLOGICAL ACTIVITY:

hVEGF-IN-1 represses human **VEGF-A** translation and shows antitumor activity. IC50 & Target: VEGFR[1] **In Vitro:** hVEGF-IN-1 shows a significant specific interaction with the G-rich region within the 5′- untranslated regions (5′-UTR) of hVEGF-A mRNA and destabilizes the Gquadruplex structure. hVEGF-IN-1 binds to the IRES-A (WT) with a K_d of 0.928 μM and binds to the hairpin DNA with a K_d of 21.2 μM. The G-rich sequence G774-G790 within the IRES-A of hVEGF-A's 5′-UTR has been shown to be critical for the translation initiation activity of IRES-A. hVEGF-IN-1 hinders BG4 from binding to the IRES-A RNA G-quadruplex in cells. hVEGF-IN-1 down-regulates hVEGF-A's translation via the G-quadruplex within IRES-A mRNA. hVEGF-IN-1 treatment reduces MDA-MB- 231 cell migration to approximately 25%[1]. **In Vivo:** Tumor bearing mice treated with hVEGF-IN-1 have an average tumor volume of less than 300 mm^3. The tumor weight in the presence of hVEGF-IN-1 reduces around 60.1% to a final weight of 0.18 g. No significant change in body weight is observed during the treatment[1].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [1]MDA-MB-231 cells are plated in the top chambers of 0.8 μm pore trans-wells in Opti-MEM reduced serum medium in the presence or absence of hVEGF-IN-1. Meanwhile, 600 μL of DMEM containing 10% fetal bovine serum (FBS) and 100 μM $CoCl_2$ are added to the lower chambers. The cells are allowed to migrate for 24 h. At the end of the assay, the cells in the top chamber are removed, and the cells at the bottom of the filter are treated by adding 500 μL of DMEM containing 2.5 mg/mL MTT to each well. After incubating at 37 °C with 5% CO_2 for 4 h, 500 μL of DMSO is added to each well and the plate is gently rotated for 10 min. Absorbance (570 nm) is measured using a microplate reader[1]. **Animal Administration:** hVEGF-IN-1 is prepared in saline[1].[1]Mouse: Mice are separated into three groups: negative control, compound 1-treated, and positive control (doxorubicin-treated). hVEGF-IN-1, doxorubicin, and saline are administered by ip injection to athymic nude mice with human tumor xenografts established using MCF-7 breast cancer cells. Mice are injected ip once a day for 20 days. Negative controls are injected with 150 μL of saline. The positive control group received doxorubicin by ip injection at a dose of 1 mg/kg. hVEGF-IN-1 is similarly administered to mice at a dose of 7.5 mg/kg. After treating the animals for 20 days, the tumor tissues are collected and IHC assays are conducted using an anti-VEGF-A antibody[1].

References:

[1]. Discovery of Small Molecules for Repressing Cap-Independent Translation of Human VascularEndothelial Growth Factor (hVEGF) as Novel Antitumor Agents. J Med Chem. 2017 Jul 13;60(13):5306-5319.

CAIndexNames:

1-Piperazinepropanamide, N-[2-[4-[[4-[2-(diethylamino)ethoxy]phenyl]amino]-2-quinazolinyl]phenyl]-4-methyl-

SMILES:

CCN(CC)CCOC(C=C1)=CC=C1NC2=NC(C3=C(NC(CCN4CCN(C)CC4)=O)C=CC=C3)=NC5=CC=CC=C52

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

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