VSPPLTLGQLLS

Cat. No.: HY-P3695

CAS No.: 1206896-24-3

Molecular Formula: $C_{56}H_{97}N_{13}O_{17}$

Molecular Weight: 1224.45

Sequence Shortening: VSPPLTLGQLLS

Target: FGFR

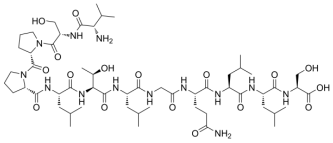
Pathway: Protein Tyrosine Kinase/RTK

Storage: Sealed storage, away from moisture and light

Powder -80°C 2 years

-20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (40.83 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	0.8167 mL	4.0835 mL	8.1669 mL
	5 mM	0.1633 mL	0.8167 mL	1.6334 mL
	10 mM	0.0817 mL	0.4083 mL	0.8167 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

VSPPLTLGQLLS is a small peptide FGFR3 inhibitor, peptide P3, inhibits FGFR3 phosphorylation. VSPPLTLGQLLS inhibits 9-cisRA-induced tracheal lymphangiogenesis and blocks lymphatic endothelial cell (LEC) proliferation, migration, and tubule formation[1][2].

IC_{50} & Target	FGFR3	K650M-FGFR3	K644E FGFR3

In Vitro

VSPPLTLGQLLS (5 μM and 10 μM; 24 h and 48 h) inhibits human primary lymphatic endothelial cell (LEC)s proliferation, migration, and tubule formation[1].

VSPPLTLGQLLS shows effective inhibition in FGFR3 phosphorylation in LECs and also demonstrated to be effective in ATDC5 chondrogenic cells, 293T cells, explanted metatarsal bone cultures, and an in vivo mouse model of thanatophoric dysplasia II[1][2].

VSPPLTLGQLLS (10 μM; 6 h) inhibits tyrosine kinase activity of FGFR3 and its typical downstream molecules, extracellular signal-regulated kinase/mitogen-activated protein kinase[2].

VSPPLTLGQLLS (0, 1, 10, and 50 μM; 24 h and 3 or 7 days, respectively) also promotes proliferation and chondrogenic differentiation of cultured ATDC5 chondrogenic cells[2].
VSPPLTLGQLLS (10 μM; 0-60 min) inhibits the ERK/MAPK pathway in FGFR3-expressing chondrocytic cell line ATDC5[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1][2]

Cell Line:	Human primary lymphatic endothelial cell (LEC)s; ATDC5 chondrogenic cells
Concentration:	10 μM
Incubation Time:	0, 5, 10, 30, 45, 60 min
Result:	Inhibited FGFR3 phosphorylation at Tyr 724 in human primary lymphatic endothelial cell (LEC)s. Inhibited the FGF2-mediated ERK/MAPK phosphorylation in FGFR3-expressing chondrocytic cell line ATDC5.

Cell Proliferation Assay[1]

Cell Line:	Human primary lymphatic endothelial cell (LEC)s
Concentration:	2.5 μM, 5 μM, and 10 μM
Incubation Time:	15 min for pre-incubation and co-incubation with 1 μM 9-cisRA for 48 hr
Result:	Inhibited LEC proliferation.

Cell Migration Assay [1]

Cell Line:	Human primary lymphatic endothelial cell (LEC)s
Concentration:	5 μM
Incubation Time:	15 min for pre-incubation and co-incubation with 1 μM 9-cisRA for 24 hr; observed at 0, 24, 48 hr
Result:	Inhibited LEC migration tubule formation.

In Vivo

VSPPLTLGQLLS (1 mM; intranasal dropping; onced daily for 7 d) blocks 9-cisRA-induced lymphangiogenesis in vivo, while 9-cisRA is an isoform of vitamin A involving in AIDS-related Kaposi Sarcoma[1].
VSPPLTLGQLLS alleviates the bone growth retardation in bone rudiments from mice mimicking human thanatophoric dysplasia type II (TDII), reversed the neonatal lethality of TDII mice[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Lymphatic reporter mice, Prox1-GFP model[1]
Dosage:	1 mM
Administration:	Intranasal dropping; once daily for 7 days; accompanied with 1 mM 9-cisRA or not
Result:	Significantly inhibited total lymphatic vessel length and number of sprouts compared increase induced by 9-cisRA.

Animal Model:	$Fgfr3^{Neo\text{-}K644E/+}$EIIa-Cre mice (TDII mice) from $Fgfr3^{Neo\text{-}K644E/+}$ mice crossed with heterozygous EIIa-Cre mice[2]

Dosage:	10 μM
Administration:	Treated for 7 days
Result:	Suppressed FGFR3-mediated growth inhibition in cultured murine metatarsal bones. Rescued the lethal phenotype in thanatophoric dysplasia type II (TDII) mice. Rescued the abnormal growth plate and the lung phenotypes in the TDII mice.

REFERENCES

[1]. Perrault DP, et al. Small Peptide Modulation of Fibroblast Growth Factor Receptor 3-Dependent Postnatal Lymphangiogenesis. Lymphat Res Biol. 2019 Feb;17(1):19-29.

[2]. Jin M, et al. A novel FGFR3-binding peptide inhibits FGFR3 signaling and reverses the lethal phenotype of mice mimicking human thanatophoric dysplasia. Hum Mol Genet. 2012 Dec 15;21(26):5443-55.

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

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