CKLF1-C19

Cat. No.:	HY-P3982
CAS No.:	960358-79-6
Molecular Formula:	$C_{103}H_{161}N_{27}O_{28}$
Molecular Weight:	2225.54
Sequence Shortening:	FNPSGPYQKKPVHEKKEVL
Target:	CCR
Pathway:	GPCR/G Protein; Immunology/Inflammation
Storage:	Sealed storage, away from moisture Powder -80°C 2 years -20°C 1 year * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)

FNPSGPYQKKPVHEKKEVL

SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 50 mg/mL (22.47 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.4493 mL	2.2466 mL	4.4933 mL
	5 mM	0.0899 mL	0.4493 mL	0.8987 mL
	10 mM	0.0449 mL	0.2247 mL	0.4493 mL

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

BIOLOGICAL ACTIVITY

Description

CKLF1-C19 is the C-terminal peptide of human chemokine-like factor 1 (CKLF1). CKLF1-C19 interacts with CCR4, and inhibits chemotaxis induced by both CKLF1 and CCL17. CKLF1-C19 can suppress allergic lung inflammation via inhibiting chemotaxis mediated by CCR3 and CCR4[1].

IC_{50} & Target		
	CCL17-CCR4	CCR3

In Vitro

CKLF1-C19 (200 ng/mL or 500 ng/mL; 30 min) induces chemotactic migration of human Th2 cells[1].

CKLF1-C19 (100 ng/mL; 30 min) inhibits CCL11-induced chemotaxis of mouse eosinophils and human CCR3-transfected or mouse CCR3-transfected HEK293 cells[1].

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

In Vivo

CKLF1-C19 (0.01-100 μg, 200 μL; i.p.; single dose at day 24-26, 30 min before OVA induced) results in a significant reduction of airway hyperresponsiveness (AHR) compared to ovalbumin (OVA) group with saline[1].

	CKLF1-C19 (0.5 mg or 1 mg, 200 μL; s.c.; single dose) reduces CCL11-mediated recruitment of eosinophils into the peritoneal cavity in a mouse model of asthma[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Tian L, et al. The CKLF1-C19 peptide attenuates allergic lung inflammation by inhibiting CCR3- and CCR4-mediated chemotaxis in a mouse model of asthma. Allergy. 2011 Feb;66(2):287-97.

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