RAGE antagonist peptide

Cat. No.: HY-P2268

CAS No.: 1092460-91-7

Molecular Formula: $C_{57}H_{101}N_{13}O_{17}S$

Molecular Weight: 1272.56

Sequence Shortening: Ac-ELKVLMEKEL-NH2

Target: Amyloid-β

Pathway: Neuronal Signaling

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)

Ac-ELKVLMEKEL-NH_2

SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (78.58 mM; Need ultrasonic)

Preparing Stock Solutions	Concentration / Solvent / Mass	1 mg	5 mg	10 mg
	1 mM	0.7858 mL	3.9291 mL	7.8582 mL
	5 mM	0.1572 mL	0.7858 mL	1.5716 mL
	10 mM	0.0786 mL	0.3929 mL	0.7858 mL

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

BIOLOGICAL ACTIVITY

Description

RAGE antagonist peptide is an advanced glycation end products (RAGE) antagonist. RAGE antagonist peptide prevents RAGE from binding with several of its most important ligands, including HMGB-1, S100P, and S100A4. RAGE antagonist peptide (RAP) possesses anti-tumor and anti-inflammatory activities[1][2].

In Vitro

RAGE antagonist peptide (RAP) reduces the ability of the ligands to stimulate RAGE activation of NFκB in cancer cells in vitro [1].

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

In Vivo

RAGE antagonist peptide (RAP, 100 μg) inhibits RAGE-mediated Basal NFκB Activity in PDAC cells in vivo[1].

RAGE antagonist peptide (RAP) reduces the growth and metastasis of pancreatic tumors and also inhibited glioma tumor growth[1].

In mice bearing asthma, RAGE antagonist peptide (RAP; 4 mg/kg; i.p.) blunts airway reactivity, airway inflammation and goblet cell metaplasia, and decreases release of Th2 cytokines. RAGE antagonist peptide also reduces total, cytoplasmic and nuclear levels of β-catenin, enhanced β-catenin phosphorylation at Ser33/37/Thr41, which triggers ubiquitination, down-

regulated expression of β-catenin targeted genes, and tends to keep β-catenin at the cytomembrane, shifting β-catenin from a signalling active pattern to an adhesive function[2].

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

Animal Model:	Cancer cells expressing the NFκB-luc reporter implanted into immune-deficient mice[1].
Dosage:	100 μg.
Administration:	Intratumoral delivery (or intraperitoneally).
Result:	Systemic administration caused a substantial reduction ($p<0.05$) in the NFκB signal 5 h after injection.

CUSTOMER VALIDATION

- Cell Commun Signal. 2023 May 1;21(1):86.

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REFERENCES

[1]. Thiruvengadam Arumugam, et al. S100P-derived RAGE antagonistic peptide reduces tumor growth and metastasis. Clin Cancer Res. 2012 Aug 15;18(16):4356-64.

[2]. Lihong Yao, et al. The receptor for advanced glycation end products is required for β-catenin stabilization in a chemical-induced asthma model. Br J Pharmacol. 2016 Sep;173(17):2600-13.

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