RAGE antagonist peptide TFA

Cat. No.: HY-P2268A

Molecular Formula: $C_{59}H_{102}F_3N_{13}O_{19}S$

Molecular Weight: 1386.58

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 (TFA salt)

SOLVENT & SOLUBILITY

In Vitro

H_2O : 25 mg/mL (18.03 mM; Need ultrasonic)

Preparing Stock Solutions	Concentration / Solvent / Mass	1 mg	5 mg	10 mg
	1 mM	0.7212 mL	3.6060 mL	7.2120 mL
	5 mM	0.1442 mL	0.7212 mL	1.4424 mL
	10 mM	0.0721 mL	0.3606 mL	0.7212 mL

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

BIOLOGICAL ACTIVITY

Description

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

In Vitro

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

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

In mice bearing asthma, RAGE antagonist peptide TFA (RAP; 4 mg/kg; i.p.) blunts airway reactivity, airway inflammation and goblet cell metaplasia, and decreases release of Th2 cytokines. RAGE antagonist peptide TFA also reduces total, cytoplasmic and nuclear levels of β-catenin, enhances 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].

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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.

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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