PR-39 TFA

Cat. No.: HY-P1259A

Molecular Formula: $C_{231}H_{347}F_3N_{70}O_{42}$

Molecular Weight: 4833.76

Sequence Shortening: RRRPRPPYLPRPRPPPFFPPRLPPRIPPGFPPRFPPRFP-NH2

RRRPRPPYLPRPRPPPFFPPRLPPRIPPGFPPRFPPRFP-NH_2 (TFA salt)

Target: Proteasome; Bacterial

Pathway: Metabolic Enzyme/Protease; Anti-infection

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)

SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (20.69 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.2069 mL	1.0344 mL	2.0688 mL
	5 mM	0.0414 mL	0.2069 mL	0.4138 mL
	10 mM	0.0207 mL	0.1034 mL	0.2069 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 50 mg/mL (10.34 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

PR-39 TFA, a natural proline- and arginine-rich antibacterial peptide, is a noncompetitive, reversible and allosteric proteasome inhibitor. PR-39 TFAreversibly binds to the α7 subunit of the proteasome and blocks degradation of NF-κB inhibitor IκBα by the ubiquitin-proteasome pathway. PR-39 TFA stimulates angiogenesis, inhibits inflammatory responses and significant reduces myocardial infarct size in mice[1][2].

In Vitro

PR-39 TFA, shown to selectively affect proteasomemediated protein degradation in vivo, alters the shape of the 20S and 26S cylinder and affects the binding of 19S caps in a reversible manner. PR-39 TFA specifically blocks degradation of IκBα and HIF-1α by the proteasome[1].

PR-39 TFA (100 nM) blocks TNF-α-induced (1 ng/mL; for 20 minutes) activation of VCAM-1 (2 hours) and ICAM-1 (8 hours) expression in human umbilical vein endothelial cells (HUVEC)[2].

PR-39 TFA (10 μM) does not affect the ability to proliferate of ECV304 cell. PR39 is able to inhibit IκBα degradation without significantly affecting overall protein degradation in cells[2].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	PR-39 TFA (10 mg/kg, intravenously; 1 hour before Caerulein of 50 μg/kg, ip) blocks IκBα degradation and NF-κB-dependent transcription in the mouse pancreas after induction of acute pancreatitis[2]. PR-39 TFA (1 μg/kg/day; 7-day intraperitoneal infusion) demonstrates significantly small infarct in C57BL/6 mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Maria Gaczynska, et al. Proline- and arginine-rich peptides constitute a novel class of allosteric inhibitors of proteasome activity. Biochemistry. 2003 Jul 29;42(29):8663-70.

[2]. Y Gao, et al. Inhibition of ubiquitin-proteasome pathway-mediated I kappa B alpha degradation by a naturally occurring antibacterial peptide. J Clin Invest. 2000 Aug;106(3):439-48.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA