Aprotinin

Cat. No.:	HY-P0017
CAS No.:	9087-70-1
Molecular Formula:	$C_{284}H_{432}N_{84}O_{79}S_7$
Molecular Weight:	6511.44
Sequence:	Arg-Pro-Asp-Phe-Cys-Leu-Glu-Pro-Pro-Tyr-Thr-Gly-Pro-Cys-Lys-Ala-Arg-Ile-Ile-Arg-Tyr-Phe-Tyr-Asn-Ala-Lys-Ala-Gly-Leu-Cys-Gln-Thr-Phe-Val-Tyr-Gly-Gly-Cys-Arg-Ala-Lys-Arg-Asn-Asn-Phe-Lys-Ser-Ala-Glu-Asp-Cys-Met-Arg-Thr-Cys-Gly-Gly-Ala(Disulfide bridge: Cys5-Cys55,Cys14-Cys38,Cys30-Cys51)
Sequence Shortening:	RPDFCLEPPYTGPCKARIIRYFYNAKAGLCQTFVYGGCRAKRNNFKSAEDCMRTCGGA(Disulfide bridge: Cys5-Cys55,Cys14-Cys38,Cys30-Cys51)
Target:	Influenza Virus; Ser/Thr Protease
Pathway:	Anti-infection; Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

RPDFCLEPPYTGPCKARIIRYFYNAKAGLCQTFVYG
GCRAKRNNFKSAEDCMRTCGGA(Disulfide bridge:
Cys_5-Cys_{55},Cys_{14}-Cys_{38},Cys_{30}-Cys_{51})

SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (15.36 mM; Need ultrasonic)
DMSO : 66.67 mg/mL (10.24 mM; ultrasonic and adjust pH to 3 with HCl)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.1536 mL	0.7679 mL	1.5358 mL
5 mM	0.0307 mL	0.1536 mL	0.3072 mL
10 mM	0.0154 mL	0.0768 mL	0.1536 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 (7.68 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description	Aprotinin is a bovine pancreatic trypsin inhibitor (BPTI) inhibitor which inhibits trypsin and chymotrypsin with K_is of 0.06 pM and 9 nM, respectively.
IC_{50} & Target	Ki: 0.06 pM (Trypsin), 9 nM (Chymotrypsin)[1]
In Vitro	Aprotinin, a serine protease inhibitor isolated from bovine lung, is a complex protease inhibitor that is an antifibrinolytic, inhibits contact activation, and decreases the inflammatory response to cardiopulmonary bypass[2]. Aprotinin inhibits

	trypsin (bovine, K_i= 0.06 pM), chymotrypsin (bovine, K_i= 9 nM), plasmin (human, 0.23 nM)[1]. Aprotinin is also a competitive protein inhibitor of NOS activity. It inhibits NOS-I and NOS-II with K_i values of 50 μM and 78 μM, respectively[3]. Aprotinin significantly inhibits fibrinolysis with an IC_{50} of 0.16±0.05 μM[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	High dose aprotinin can reduce blood loss and transfusion requirements associated with primary cardiac procedures such as coronary artery bypass graft (CABG) or heart valve replacement surgery[5]. Aprotinin inhibits thrombus formation in a dose-dependent manner. Aprotinin at a dose of 1.5 mg kg^{-1} (bolus) and 3 mg kg^{-1} h^{-1} infusion (maintenance infusion) causes a tendency towards a reduction in bleeding time. Aprotinin significantly reduces the bleeding time starting at a dose of 3 mg kg^{-1} bolus plus 6 mg kg^{-1} h^{-1} showing a reduction of approximately 84%±2.9%. At the highest dose of 5 mg kg^{-1} and 10 mg kg^{-1} h^{-1}, the strongest effects are observed[4]. Aprotinin may affect tumor necrosis factor-alpha (TNF) levels. Soluble TNFRI levels are significantly increased following I/R in the aprotinin treated wild type mice and not detected in all TNFRInull mice [6]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [4][6]	Rats: Male Wistar rats (180-220 g) are used in the study. Aprotinin is dissolved in physiological saline. Aprotinin is administered by bolus injection followed by a maintenance infusion. The doses given are 1.5 mg kg^{-1} and 3 mg kg^{-1} h^{-1}, 3mg kg^{-1} and 6 mg kg^{-1} h^{-1} up to 5 mg kg^{-1} and 10 mg kg^{-1} h^{-1}. Plasma concentrations for the two agents are assessed by pharmacokinetic studies in rats[4]. Mice: An intact mouse model of ischemia/reperfusion (30 min-I/60 min-R) is used and left ventricular peak + dP/dt is measured in wild type mice (WT, C57BL/6; n=10), WT mice with aprotinin (4mL/kg; n=10), transgenic mice devoid of the TNFRI (TNFRInull; n=10), and TNFRInull with aprotinin (n=10)[6]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2023 Jul 26;14(1):4487.
- Nat Commun. 2023 May 2;14(1):2523.
- Proc Natl Acad Sci U S A. 2022 Jul 26;119(30):e2208211119.
- Cell Rep. 2021 Nov 2;37(5):109931.
- Am J Physiol Cell Physiol. 2017 Dec 1;313(6):C632-C643.

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REFERENCES

[1]. Fritz H, et al. Biochemistry and applications of aprotinin, the kallikrein inhibitor from bovine organs. Arzneimittelforschung. 1983;33(4):479-94.

[2]. Levy JH, et al. Efficacy and safety of aprotinin in cardiac surgery. Orthopedics. 2004 Jun;27(6 Suppl):s659-62.

[3]. Venturini G, et al. Aprotinin, the first competitive protein inhibitor of NOS activity.Biochem Biophys Res Commun. 1998 Aug 10;249(1):263-5

[4]. Sperzel M, et al. Evaluation of aprotinin and tranexamic acid in different in vitro and in vivo models of fibrinolysis, coagulation and thrombus formation. J Thromb Haemost. 2007 Oct;5(10):2113-8. Epub 2007 Jul 31.

[5]. Davis R, et al. Aprotinin. A review of its pharmacology and therapeutic efficacy in reducing blood loss associated withcardiac surgery. Drugs. 1995 Jun;49(6):954-83.

[6]. Sabbagh MJ, et al. Aprotinin exacerbates left ventricular dysfunction after ischemia/reperfusion in mice lacking tumor necrosis factor receptor I. J Cardiovasc Pharmacol. 2008 Oct;52(4):355-62.

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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