SZABO SCANDIC

Part of Europa Biosite

Produktinformation

- Forschungsprodukte & Biochemikalien
- Zellkultur & Verbrauchsmaterial
- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

Weitere Information auf den folgenden Seiten!
See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

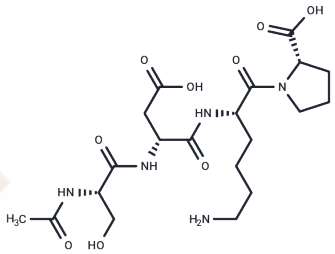
linkedin.com/company/szaboscandic

Data Sheet (Cat.No.TP1828)

TargetMol

N-Acetyl-Ser-Asp-Lys-Pro

Chemical Properties



CAS No. : 127103-11-1

Formula: C20H33N5O9

Molecular Weight: 487.5

Appearance: no data available

Storage: keep away from moisture
Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	Acetyl Ser-Asp-Lys-Pro is formed in bone marrow cells by enzymatic processing of thymosin β4. It inhibits the entry of pluripotent hemopoietic stem cells into S-phase of the cell cycle and protects against Ara-C lethality in mice.
Targets(IC50)	RAAS
In vitro	N-Acetyl-Ser-Asp-Lys-Pro (AcSDKP) is a specific substrate for the N-terminal site of ACE and increases five-fold during ACE inhibitor therapy. AcSDKP inhibited the proliferation of isolated cardiac fibroblasts (P<0.05) but significantly stimulated the proliferation of vascular smooth muscle cells. Flow cytometry of rat cardiac fibroblasts treated with AcSDKP showed significant inhibition of cell progression from G0/G1 phase to S phase. In cardiac fibroblasts transfected with a Smad-sensitive luciferase reporter construct, AcSDKP decreased luciferase activity by 55±9.7% (P=0.01). Additionally, AcSDKP decreased phosphorylation and nuclear translocation of Smad2 in cardiac fibroblasts. In conclusion, AcSDKP inhibits the growth of cardiac fibroblasts and TGFbeta1-stimulated phosphorylation of Smad2. Because AcSDKP increases substantially during ACE inhibitor therapy, this suggests a novel pathway independent of angiotensin II by which ACE inhibitors can inhibit cardiac fibrosis.

Solubility Information

Solubility	DMSO: 10 mM, (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.0513 mL	10.2564 mL	20.5128 mL
5 mM	0.4103 mL	2.0513 mL	4.1026 mL
10 mM	0.2051 mL	1.0256 mL	2.0513 mL
50 mM	0.041 mL	0.2051 mL	0.4103 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

Rousseau A, et al. The hemoregulatory peptide N-acetyl-Ser-Asp-Lys-Pro is a natural and specificsubstrate of the N-terminal active site of human angiotensin-converting enzyme. J Biol Chem. 1995 Feb 24;270(8):3656-61.