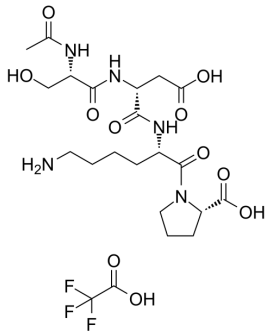
N-Acetyl-Ser-Asp-Lys-Pro TFA



Cat. No.:	HY-P0266A
Molecular Formula:	$C_{22}H_{34}F_3N_5O_{11}$
Molecular Weight:	601.53
Sequence:	Ac-Ser-Asp-Lys-Pro
Sequence Shortening:	Ac-SDKP
Target:	Angiotensin-converting Enzyme (ACE)
Pathway:	Metabolic Enzyme/Protease
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)

BIOLOGICAL ACTIVITY

Description	N-Acetyl-Ser-Asp-Lys-Pro (TFA), an endogenous tetrapeptide secreted by bone marrow, is a specific substrate for the N-terminal site of ACE.
In Vitro	N-Acetyl-Ser-Asp-Lys-Pro is degraded specifically by ACE, and its plasma level rises substantially during ACE inhibitor therapy. Flow cytometry of rat cardiac fibroblasts treated with N-Acetyl-Ser-Asp-Lys-Pro shows significant inhibition of the progression of cells from G0/G1 phase to S phase of the cell cycle. Moreover, phosphorylation and nuclear translocation of Smad2 is decreased in cardiac fibroblasts treated with N-Acetyl-Ser-Asp-Lys-Pro[1]. N-acetyl-seryl-aspartyl-lysyl-proline appears to exert this function by blocking the action of a stem cell-specific proliferation stimulator and acts selectively on quiescent progenitors[2]. N-Acetyl-Ser-Asp-Lys-Pro inhibits collagenase expression and activation is associated with increased expression of TIMP-1 and TIMP-2. N-Acetyl-Ser-Asp-Lys-Pro normalizes the IL-1β-mediated increase in MMP-2 and MMP-9 activities and MMP-13 expression[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	N-Acetyl-Ser-Asp-Lys-Pro prevents hypertension-induced inflammatory cell infiltration, collagen deposition, nephrin downregulation and albuminuria, which could lead to renoprotection in hypertensive mice[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. 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.

[2]. Pokharel S, et al. N-acetyl-Ser-Asp-Lys-Pro inhibits phosphorylation of Smad2 in cardiac fibroblasts. Hypertension. 2002 Aug;40(2):155-61.

[3]. Rhaleb NE, et al. N-acetyl-Ser-Asp-Lys-Pro inhibits interleukin-1β-mediated matrix metalloproteinase activation in cardiac fibroblasts. Pflugers Arch. 2013 Oct;465(10):1487-95.

[4]. Rhaleb NE, et al. Renal protective effects of N-acetyl-Ser-Asp-Lys-Pro in deoxycorticosterone acetate-salt hypertensive mice. J Hypertens. 2011 Feb;29(2):330-8.

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

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