Product Data Sheet

Product Name: N-Acetyl-Ser-Asp-Lys-Pro
Cat. No.: GC33889

Chemical Properties

Cas. No.	127103-11-1		
SMILES	Ac-Ser-Asp-Lys-Pro		
Formula	$C_{20}H_{33}N_5O_9$	M.Wt	487.5
Solubility	Soluble in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	N-Acetyl-Ser-Asp-Lys-Pro		

Protocol

Cell experiment [1]:

Cell lines	Rat cardiac fibroblasts
Preparation Method	Rat cardiac fibroblasts were cultured at 37°C and 5% CO_2 in DMEM medium supplemented with 10% fetal bovine serum (FBS), 1% L-glutamate, 50U/ml penicillin, and 0.1g/l streptomycin. Cells were plated at a density of 1.2×10^4 cells/ml in a 96-well plate with growth medium for 24h, and then were incubated with the different concentrations of N-Acetyl-Ser-Asp-Lys-Pro (0, 0.01, 0.1, and 1nM) for 24h, analyzed the cell viability.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

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Reaction Conditions	0, 0.01, 0.1, and 1nM; 24h
Applications	N-Acetyl-Ser-Asp-Lys-Pro treatment significantly inhibited cell viability of Rat cardiac fibroblasts in a concentration-dependent manner.
Animal experiment [2]:	
Animal models	Male Sprague Dawley rats
Preparation Method	Male Sprague Dawley rats weighing 275 to 300g were housed in an air-conditioned room with a 12h light/dark cycle and received standard laboratory rat chow and tap water. Rats were allowed 7 days to adjust to their new environment. Before all of the surgical procedures, rats were given analgesia (2mg/kg of butorphanol; s.c.) and anesthesia (50mg/kg of sodium pentobarbital; i.p.). Rats were anesthetized and 5/6Nx was performed by unilateral nephrectomy plus ligation of lower and upper renal arterial branches of the contralateral kidney with a 6-0 silk suture. Ligation was deemed successful when two thirds of the kidney turned dark red. The sham-operated group underwent a similar surgical procedure except that the suture around the renal artery was not tightened. An osmotic minipump filled with N-Acetyl-Ser-Asp-Lys-Pro (800μg/kg/day) for 3 weeks or vehicle (0.01M acetic acid saline solution) was implanted s.c. between the shoulder blades. At the end of the research, the kidneys were collected for analysis.
Dosage form	800μg/kg/day for 3 weeks; s.c.
Applications	N-Acetyl-Ser-Asp-Lys-Pro treatment attenuated renal fibrosis and improved the renal function in rats.

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References:
[1] Pokharel S, Rasoul S, Roks A J M, et al. N-acetyl-Ser-Asp-Lys-Pro inhibits phosphorylation of Smad2 in cardiac fibroblasts[J]. Hypertension, 2002, 40(2): 155-161.
[2] Liao T D, Yang X P, D'Ambrosio M, et al. N-acetyl-seryl-aspartyl-lysyl-proline attenuates renal injury and dysfunction in hypertensive rats with reduced renal mass: council for high blood pressure research[J]. Hypertension, 2010, 55(2): 459-467.

Background

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N-Acetyl-Ser-Asp-Lys-Pro is an endogenous tetrapeptide secreted by bone marrow, which is a specific substrate for the N-terminal site of angiotensin-converting enzyme (ACE) [1]. N-Acetyl-Ser-Asp-Lys-Pro inhibits hematopoietic stem cells from entering the S phase, participates in the regulation of hematopoietic stem cell proliferation, and can block a stem cell-specific proliferation stimulating factor, selectively acting on quiescent progenitors [2]. N-Acetyl-Ser-Asp-Lys-Pro has been widely used to inhibit the differentiation of hematopoietic stem cells and tissue fibrosis[3].

In vitro, N-Acetyl-Ser-Asp-Lys-Pro treatment (1nM) for 24 hours significantly inhibited the inhibitory effect of S17092 (100μg/ml) on the proliferation of U87-MG cells, and induced Akt phosphorylation[4]. Treatment with 1nM N-Acetyl-Ser-Asp-Lys-Pro for 24 hours significantly inhibited the cell cycle progression of rat cardiac fibroblasts from the G0/G1 phase to the S phase, and led to a decrease in the phosphorylation and nuclear translocation of Smad2[5]. Treatment with 100nM N-Acetyl-Ser-Asp-Lys-Pro for 72 hours significantly inhibited the increase in MMP-2 and MMP-9 protein levels induced by IL-1β in rat cardiac fibroblasts, and reduced collagenase activity[6].

In vivo, N-Acetyl-Ser-Asp-Lys-Pro treatment via daily subcutaneous injection at a dose of 800μg/kg for 3 weeks alleviated proteinuria and renal fibrosis in the hypertensive rat model induced by 5/6 nephrectomy, and improved renal function[7]. Daily subcutaneous injection of 800μg/kg dose of N-Acetyl-Ser-Asp-Lys-Pro was administered for 12 weeks to prevent inflammatory cell infiltration, collagen deposition, downregulation of renin expression and proteinuria in hypertensive mice [8].

References:

[1] Stéphan J P, Melaine N, Ézan E, et al. Source, catabolism and role of the tetrapeptide N-acetyl-ser-asp-lys-Pro within the testis[J]. Journal of Cell Science, 2000, 113(1): 113-121.
[2] Rousseau A, Michaud A, Chauvet M T, et al. The hemoregulatory peptide N-Acetyl-Ser-Asp-Lys-Pro is a natural and specific substrate of the N-terminal active site of human angiotensin-converting enzyme (∗)[J]. Journal of Biological Chemistry, 1995, 270(8): 3656-3661.
[3] Douglas R G, Ehlers M R, Sturrock E D. Antifibrotic peptide N-acetyl-Ser-Asp-Lys-Pro (Ac-SDKP): opportunities for angiotensin-converting enzyme inhibitor design[J]. Clinical &

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[4] Hu P, Li B, Zhang W, et al. AcSDKP regulates cell proliferation through the PI3KCA/Akt signaling pathway[J]. PloS one, 2013, 8(11): e79321.
[5] Pokharel S, Rasoul S, Roks A J M, et al. N-acetyl-Ser-Asp-Lys-Pro inhibits phosphorylation of Smad2 in cardiac fibroblasts[J]. Hypertension, 2002, 40(2): 155-161.
[6] Rhaleb N E, Pokharel S, Sharma U C, et al. N-acetyl-Ser-Asp-Lys-Pro inhibits interleukin-1β-mediated matrix metalloproteinase activation in cardiac fibroblasts[J]. Pflügers Archiv-European Journal of Physiology, 2013, 465(10): 1487-1495.
[7] Liao T D, Yang X P, D'Ambrosio M, et al. N-acetyl-seryl-aspartyl-lysyl-proline attenuates renal injury and dysfunction in hypertensive rats with reduced renal mass: council for high blood pressure research[J]. Hypertension, 2010, 55(2): 459-467.
[8] Rhaleb N E, Pokharel S, Sharma U, et al. Renal protective effects of N-acetyl-Ser-Asp-Lys-Pro in deoxycorticosterone acetate–salt hypertensive mice[J]. Journal of hypertension, 2011, 29(2): 330-338.

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