Product Data Sheet

Product Name: Atrial Natriuretic Peptide (ANP) (1-28), human, porcine Acetate
Cat. No.: GC34025

Chemical Properties

Cas. No.	1366000-58-9		
SMILES	Ser-Leu-Arg-Arg-Ser-Ser-Cys-Phe-Gly-Gly-Arg-Met-Asp-Arg-Ile-Gly-Ala-Gln-Ser-Gly-Leu-Gly-Cys-Asn-Ser-Phe-Arg-Tyr (Disulfide bridge: Cys7-Cys23)		
Formula	$C_{127}H_{203}N_{45}O_{39}S_3.C_2H_4O_2$	M.Wt	3140.5
Solubility	25mg/ml in Water	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			

Protocol

Cell experiment [1]:

Cell lines	Human pulmonary artery endothelial cells (HPAECs)
Preparation Method	HPAECs were seeded into 96-well plates or on glass coverslips coated with biotinylated gelatin (0.25mg/mL) and cultured for 48–72h. The cells were then pretreated with Atrial Natriuretic Peptide (ANP) (1-28) (100nM) for 20 minutes and subsequently challenged with thrombin (0.5units/mL) for 15 minutes. FITC-avidin (25μg/mL) was added to the culture medium for 3 minutes before termination of the experiment.

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Reaction Conditions	Atrial Natriuretic Peptide (ANP) (1-28) (100nM); 20 minutes
Applications	Atrial Natriuretic Peptide (ANP) (1-28) significantly attenuated thrombin-induced endothelial cell permeability and preserved the pool of stabilized microtubules.

Animal experiment [2]:

Animal models	Wild-type C57BL/6J mice and heterozygous Nppa knockout mice ($Nppa^{+/-}$)
Preparation Method	8- to 10-week-old wild-type C57BL/6J mice and $Nppa^{+/-}$ mice were subjected to cardiac ischemia-reperfusion (I/R) injury. Ischemia was induced by ligating the left anterior descending artery (LAD) for 30 minutes, followed by 24 hours of reperfusion. Tat-Beclin D11 (15mg/kg) was injected intraperitoneally 6 hours before ischemia to induce autophagy. In other experiments, exogenous Atrial Natriuretic Peptide (ANP) (1-28)(2.5μg/kg) was administered via tail vein injection 1 hour before the I/R protocol, with or without pre-treatment of chloroquine (50mg/kg, intraperitoneal injection) for 1 hour.
Dosage form	2.5μg/kg of Atrial Natriuretic Peptide (ANP) (1-28), i.v.
Applications	Atrial Natriuretic Peptide (ANP) (1-28) significantly increased the levels of the autophagy marker LC3-II in the mouse heart and reduced infarct size in I/R injury.

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References:
[1] Tian X, Tian Y, Gawlak G, et al. Control of vascular permeability by atrial natriuretic peptide via a GEF-H1-dependent mechanism. J Biol Chem. 2014 Feb 21;289(8):5168-83.
[2] Forte M, Marchitti S, Di Nonno F, et al. NPPA/atrial natriuretic peptide is an extracellular modulator of autophagy in the heart. Autophagy. 2023 Apr;19(4):1087-1099.

Background

Atrial Natriuretic Peptide (ANP) (1-28), human, porcine Acetate is a bioactive peptide that plays a crucial role in the body's homeostatic mechanisms. Atrial Natriuretic Peptide (ANP) (1-28) inhibits angiotensin II)-induced endothelin-1 secretion in a dose-dependent manner[1]. Atrial Natriuretic Peptide (ANP) (1-28) is primarily secreted by atrial myocytes in response to increased atrial stretch, such as during volume overload. The 28-amino-acid sequence is highly conserved between human and porcine forms. The acetate modification may affect its solubility, stability, and biological activity to some extent[2]. Atrial Natriuretic Peptide (ANP) (1-28) binds to its specific receptors, mainly

the guanylyl cyclase-linked natriuretic peptide receptor-A (NPR-A). The binding activates guanylyl cyclase, leading to an increase in intracellular cyclic guanosine monophosphate (cGMP) levels. Elevated cGMP then mediates a series of physiological effects. In the kidneys, Atrial Natriuretic Peptide (ANP) (1-28) promotes natriuresis (increased sodium excretion) and diuresis (increased urine output), which helps to reduce blood volume. The peptide achieves this by increasing the glomerular filtration rate, inhibiting sodium reabsorption in the renal tubules, and suppressing the renin-angiotensin-aldosterone system (RAAS)[3]. Atrial Natriuretic Peptide (ANP) (1-28) causes vasodilation of blood vessels, reducing peripheral vascular resistance and thus lowering blood pressure. Atrial Natriuretic Peptide (ANP) (1-28) also has anti-fibrotic and anti-hypertrophic effects on the heart, helping to prevent cardiac remodeling and maintain normal cardiac function[4].

In vitro, Atrial Natriuretic Peptide (ANP) (1-28) (10nmol/L) pretreated human embryonic kidney cells (HEK 293) for 30 minutes, significantly inhibited the aldosterone (500pmol/L)-induced nuclear translocation of the mineralocorticoid receptor (MR)[5]. Atrial Natriuretic Peptide (ANP) (1-28)(100nM) pretreated human pulmonary artery endothelial cells (HPAECs) for 20 minutes, significantly inhibited the increase in endothelial cell permeability induced by thrombin (0.5units/mL). Atrial Natriuretic Peptide (ANP) (1-28) inhibits the secretion of endothelin-1 in a dose-dependent manner, thereby suppressing the Rho signaling pathway, reducing myosin light chain (MLC) phosphorylation, and inhibiting stress fiber formation, thus alleviating cell contraction and increased permeability[6].

In vivo, Atrial Natriuretic Peptide (ANP) (1-28)(2μg in 400μL PBS per mouse) was intraperitoneally injected into mice with DSS-induced colitis. Atrial Natriuretic Peptide (ANP) (1-28) treatment significantly alleviated colonic inflammation by inhibiting the STING pathway, reducing the expression of inflammatory cytokines such as TNF-α, IL-1β, IL-6, IFN-α, and IFN-β in colonic tissues and epithelial cells. Atrial Natriuretic Peptide (ANP) (1-28) also repaired gut barrier function by promoting the expression of tight junction proteins (ZO-1 and occludin) and inhibited ER stress-induced autophagy via the STING pathway[7]. Atrial Natriuretic Peptide (ANP) (1-28)(2.5μg/kg) was administered to wild-type mice via tail vein injection. Atrial Natriuretic Peptide significantly increased the levels of the autophagy marker LC3-II in the mouse heart and reduced infarct size in

ischemia-reperfusion (I/R) injury[8].

References:

[1] Kohno M, Yokokawa K, Horio T, et al. Atrial and brain natriuretic peptides inhibit the endothelin-1 secretory response to angiotensin II in porcine aorta. Circ Res. 1992 Feb;70(2):241-7.
[2] Potter LR, Yoder AR, Flora DR, et al. Natriuretic peptides: their structures, receptors, physiologic functions and therapeutic applications. Handb Exp Pharmacol. 2009; (191):341-66.
[3] Santhekadur PK, Kumar DP, Seneshaw M, et al. The multifaceted role of natriuretic peptides in metabolic syndrome. Biomed Pharmacother. 2017 Aug;92:826-835.
[4] Sadoshima J, Jahn L, Takahashi T, et al. Molecular characterization of the stretch-induced adaptation of cultured cardiac cells. An in vitro model of load-induced cardiac hypertrophy. J Biol Chem. 1992 May 25;267(15):10551-60.
[5] Nakagawa H, Oberwinkler H, Nikolaev VO, et al. Atrial natriuretic peptide locally counteracts the deleterious effects of cardiomyocyte mineralocorticoid receptor activation. Circ Heart Fail. 2014 Sep;7(5):814-21.
[6] Tian X, Tian Y, Gawlak G, et al. Control of vascular permeability by atrial natriuretic peptide via a GEF-H1-dependent mechanism. J Biol Chem. 2014 Feb 21;289(8):5168-83.
[7] Chen C, Zhang Y, Tao M, et al. Atrial Natriuretic Peptide Attenuates Colitis via Inhibition of the cGAS-STING Pathway in Colonic Epithelial Cells. Int J Biol Sci. 2022 Feb 7;18(4):1737-1754.
[8] Forte M, Marchitti S, Di Nonno F, et al. NPPA/atrial natriuretic peptide is an extracellular modulator of autophagy in the heart. Autophagy. 2023 Apr;19(4):1087-1099.

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