Product Data Sheet

Product Name:	Angiotensin (1-7)
Cat. No.:	GP10077

Chemical Properties

Cas. No.	51833-78-4		
Chemical Name	Angiotensin (1-7)		
SMILES	CCC(C)C(C(=O)NC(CC1=CN=CN1)C(=O)N2CCCC2C(=O)O)NC(=O)C(CC3=CC=C(C=C3)O)NC(=O)C(C(C)C)NC(=O)C(CCCN=C(N)N)NC		
Formula	$C_{41}H_{62}N_{12}O_{11}$	M.Wt	899
Solubility	≥ 89.9mg/mL in DMSO	Storage	Desiccate 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 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	Angiotensin (1-7)		

Protocol

Cell experiment [1]:

Cell lines	DU-145 cell
Preparation Method	The DU-145 cells underwent two or more passages following recovery from liquid nitrogen storage and cultured in DMEM medium under humidity-controlled conditions at 37 °C with 5% CO_2. The standard supplements were also used, including 10% heat-inactivated Fetal Bovine Serum (FBS), 1mM Sodium Pyruvate, 10mM HEPES buffer, and antibiotics (penicillin 50U/ml; streptomycin 50mg/ml; neomycin 100mg/ml). Angiotensin (1-7) was added to the cell culture medium at concentration 1nM. Four hours before the end of incubation period (48 hours) a MTT working solution at final concentration of 0.5mg/ml was added to each well. Formazan crystals formed by viable cells were dissolved in 10% sodium deodecyl sulfate (SDS) solution in 0.01M HCl. The absorbance was measured at 570nm using a microplate reader. Cell viability (% of control) was calculated in relation to untreated cells. The changes in cell proliferation after Angiotensin (1-7) treatment (1nM; 48 hours) were determined using a BrdU Cell Proliferation Assay.
Reaction Conditions	1nM; 48h
Applications	Angiotensin (1-7) significantly inhibited the viability and proliferation of DU-145 cells.

Animal experiment [2]:

Animal models	Male Wistar rats
Preparation Method	Male Wistar rats (10 weeks old, 200-250g) were given free access to standard rat chow and water during the experiment. Following a 5-day period of acclimatization, 120 rats were randomly divided into two groups: control group (n=15), receiving an intraperitoneal injection of normal saline, and diabetic group (n=105), receiving intraperitoneal injection of 65mg/kg streptozotocin (STZ) dissolved in sodium citrate buffer (pH 4.5). All diabetic rats received an intraperitoneal injection of insulin (2-3U) every 3 days to maintain blood glucose levels between 16.7 and 25mM to avoid death induced by excessively high blood glucose level. Twelve weeks after STZ injection, the diabetic rats were randomly divided into 7 treatment groups (n=15 per group): no-treatment group and Angiotensin (1-7) group that received subcutaneous injection of 800ng/kg/min of Angiotensin (1-7) by an embedded mini-osmotic pump. After treatment for 4 weeks, all rats were killed. Plasma and kidney samples from rats were collected for cytokine and histological analysis.

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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Dosage form	800ng/kg/min for 4 weeks; s.c.
Applications	Angiotensin (1-7) treatment ameliorated streptozotocin-induced renal injury, attenuated glomerulosclerosis, and reduced the expression of TGF-β1, VEGF and NOX4.

References:
[1] Domi?ska K, Ok?a P, Kowalska K, et al. Angiotensin 1-7 modulates molecular and cellular processes central to the pathogenesis of prostate cancer[J]. Scientific Reports, 2018, 8(1): 15772.
[2] Zhang K, Meng X, Li D, et al. Angiotensin (1-7) attenuates the progression of streptozotocin-induced diabetic renal injury better than angiotensin receptor blockade[J]. Kidney international, 2015, 87(2): 359-369.

Background

Angiotensin (1-7), a heptapeptide found in the heart and kidney, inhibits angiotensin-converting enzyme (ACE) in plasma and atrial tissue with an IC_{50} of 3.0 and 4.0μM, respectively[1]. Angiotensin (1-7) has been identified as regulating blood pressure, cardiac function and smooth muscle and myocardial cell growth as well as renal function regulation[2]. Angiotensin (1-7) has been extensively studied for modulation of vascular function in various cellular and animal models[3].

In vitro, Angiotensin (1-7) treatment (1nM; 48h) effectively reduced the proliferation of DU-145 prostate cancer cells and induced a significant reduction in the expression of MKI67[4]. Treatment of mouse podocytes with Angiotensin (1-7) at 10μM for 24 hours reversed high glucose-induced reduction of podocyte viability, increase of podocyte apoptosis, reduction of nephrin, podocin, WT-1 and MasR protein expression, and up-regulation of AT1R expression[5]. Angiotensin (1-7) treatment (1.7μM; 18 hours) effectively reduced tube formation in human umbilical vein endothelial cells[6].

In vivo, Angiotensin (1-7) treatment via daily intraperitoneal injection at 2mg/kg ameliorated sepsis-induced cardiomyopathy in mice by reducing inflammatory response and mitochondrial damage through NF-κB and MAPK pathways[7]. A single dose of 45μg/kg Angiotensin (1-7) administration via gavage promoted the resolution of lipopolysaccharide-induced pleurisy in mice by reducing neutrophil numbers and M1 inflammatory macrophage frequency after 24h treatment[8].

References:
[1] Roks A J M, Van Geel P P, Pinto Y M, et al. Angiotensin-(1–7) is a modulator of the human renin-angiotensin system[J]. Hypertension, 1999, 34(2): 296-301.
[2] Padda R S, Shi Y, Lo C S, et al. Angiotensin-(1-7): a novel peptide to treat hypertension and nephropathy in diabetes?[J]. Journal of diabetes & metabolism, 2015, 6(10): 10.4172/2155-6156.1000615.
[3] Durand M J, Zinkevich N S, Riedel M, et al. Vascular actions of angiotensin 1–7 in the human microcirculation: novel role for telomerase[J]. Arteriosclerosis, Thrombosis, and Vascular Biology, 2016, 36(6): 1254-1262.
[4] Domińska K, Okła P, Kowalska K, et al. Angiotensin 1–7 modulates molecular and cellular processes central to the pathogenesis of prostate cancer[J]. Scientific Reports, 2018, 8(1): 15772.

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[5] Lu J, Chen G, Shen G, et al. Ang-(1-7) attenuates podocyte injury induced by high glucose in vitro[J]. Archives of Endocrinology and Metabolism, 2023, 67: e000643.
[6] Anton L, Merrill D C, Neves L A A, et al. Angiotensin-(1–7) inhibits in vitro endothelial cell tube formation in human umbilical vein endothelial cells through the AT1–7 receptor[J]. Endocrine, 2007, 32(2): 212-218.
[7] Chen X S, Cui J R, Meng X L, et al. Angiotensin-(1–7) ameliorates sepsis-induced cardiomyopathy by alleviating inflammatory response and mitochondrial damage through the NF-κB and MAPK pathways[J]. Journal of Translational Medicine, 2023, 21(1): 2.
[8] de Carvalho Santuchi M, Dutra M F, Vago J P, et al. Angiotensin-(1-7) and alamandine promote anti-inflammatory response in macrophages in vitro and in vivo[J]. Mediators of Inflammation, 2019, 2019(1): 2401081.

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