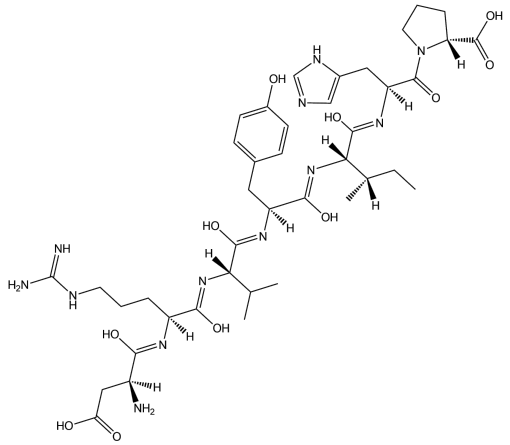
Product Data Sheet

Product Name: (D-Pro^7)-Angiotensin I/II (1-7)
Cat. No.: GA20155

Chemical Properties

Cas. No.	586962-44-9		
Formula	$C_{41}H_{62}N_{12}O_{11}$	M.Wt	899.02
Solubility	1 mg/mL in Water	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C 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 microvascular endothelial cells

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Preparation Method	Human pulmonary microvascular endothelial cells (PMVECs, passages 4–8) were grown to confluence in polycarbonate wells containing gelatin-coated gold microelectrodes. Following a 30 minute recording of baseline resistance, the Ang-(1–7) receptor blocker (D-Pro^7)-Angiotensin I/II (1-7)(100nM), the bradykinin receptor blocker icatibant (1μM), the NO synthase inhibitor *N*ω-nitro-L-arginine methyl ester (l-NAME; 1mM), or respective vehicle controls were added, followed after 15 minutes by Ang-(1–7) (100nM) or vehicle and after an additional 30 minutes by thrombin (0.2U/mL). Transendothelial electrical resistance (TEER) measurements were performed using an electrical cell substrate impedance sensing system. TEER values were recorded continuously up to 75 minutes after thrombin stimulation.
Reaction Conditions	100nM; 30min
Applications	Pretreatment of human pulmonary microvascular endothelial cells (PMVEC) with (D-Pro^7)-Angiotensin I/II (1-7) completely abolished the protective effect of Ang-(1-7) against thrombin induced barrier disruption, reducing the trans-endothelial electrical resistance (TEER).

Animal experiment [2]:

Animal models	Sprague–Dawley rats

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Preparation Method	7–9-month-old male Hanover Sprague–Dawley rats were maintained in collective cages in an appropriate room with controlled temperature and with a 12h light cycle. The vasorelaxant activity of Ang-(1–7) was measured in vessels with or without functional endothelium pre-contracted to the same tension level (approximately 1.5g of tension) with submaximal concentrations of phenylephrine (0.03–0.1μM). Ang-(1–7) was added in increasing cumulative concentrations once the response to phenylephrine had stabilized. In an attempt to determine which receptor is involved in the vasorelaxant effect of Ang-(1–7), experiments were performed in endothelium-containing vessels, in the presence of the Ang-(1–7) antagonists A-779 (0.1, 1.0 or 10μM) or (D-Pro^7)-Angiotensin I/II (1-7) (0.1μM); the B2 receptor antagonist HOE 140 (1μM), the Ang II receptor antagonists CV11974 (0.01μM; AT1 antagonist) or PD123319 (1μM; AT2 antagonist). As a control, another vessel segment from each rat was simultaneously monitored for Ang-(1–7) effects alone.
Dosage form	0.1μM; 20min pre-incubation, vascular-bath perfusion
Applications	(D-Pro^7)-Angiotensin I/II (1-7) blocks an A-779-insensitive novel Ang-(1–7) receptor subtype and completely reverses the endothelium-dependent relaxation induced by Ang-(1–7) in isolated thoracic aortic rings from Sprague–Dawley rats.

References:

[1] Klein N, Gembardt F, Supé S, et al. Angiotensin-(1-7) protects from experimental acute lung injury. *Crit Care Med*. 2013;41(11):e334-e343.
[2] Silva DM, Vianna HR, Cortes SF, Campagnole-Santos MJ, Santos RA, Lemos VS. Evidence for a new angiotensin-(1-7) receptor subtype in the aorta of Sprague-Dawley rats. *Peptides*. 2007;28(3):702-707.

Background

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(D-Pro^7)-Angiotensin I/II (1-7) is a peptide antagonist of the Mas receptor, engineered by replacing the C-terminal Pro^7 of Angiotensin-(1-7) with a D-proline[1]. The Mas receptor, a G-protein-coupled receptor for Ang-(1-7), elicits vasodilatory, anti-proliferative and anti-fibrotic protective signals upon activation[2]. (D-Pro^7)-Angiotensin I/II (1-7) competitively blocks the binding of Angiotensin-(1-7) to Mas and is widely used to selectively interrupt ACE2/Ang-(1-7)/Mas-axis signaling in research[3].

In vitro, pretreatment of human pulmonary microvascular endothelial cells (PMVEC) with 100nM (D-Pro^7)-Angiotensin I/II (1-7)(30min) completely abolished the protective effect of Ang-(1-7) against thrombin induced barrier disruption, reducing the trans-endothelial electrical resistance (TEER)[4]. In rat aortic vascular smooth muscle cells (VSMC) cultured in high glucose (25mM), 72h co-incubation with 1μM (D-Pro^7)-Angiotensin I/II (1-7) reversed the inhibitory effect of Ang-(1-7) on high glucose-induced ErbB2 phosphorylation, restoring the p-ErbB2/t-ErbB2 ratio[5].

In vivo, (D-Pro^7)-Angiotensin I/II (1-7) (10μM; 20min pre-incubation in isolated organ bath) reduced the maximal vasorelaxation evoked by Ang-(1-7) in rat renal arteries, completely blocking Ang-(1-7)-induced vasodilation and abolishing Ang-(1-7)-mediated activation of the NO-sGC-cGMP pathway[6]. (D-Pro^7)-Angiotensin I/II (1-7) (0.1μM; 20min pre-incubation; vascular-bath perfusion) blocks an A-779-insensitive novel Ang-(1–7) receptor subtype and completely reverses the endothelium-dependent relaxation induced by Ang-(1–7) in isolated thoracic aortic rings from Sprague–Dawley rats[7].

References:

[1] Santos RA, Haibara AS, Campagnole-Santos MJ, et al. Characterization of a new selective antagonist for angiotensin-(1-7), D-pro7-angiotensin-(1-7). *Hypertension*. 2003;41(3 Pt 2):737-743.
[2] Santos RAS, Sampaio WO, Alzamora AC, et al. The ACE2/Angiotensin-(1-7)/MAS Axis of the Renin-Angiotensin System: Focus on Angiotensin-(1-7). *Physiol Rev*. 2018;98(1):505-553.
[3] Santos RA, Frézard F, Ferreira AJ. Angiotensin-(1-7): blood, heart, and blood vessels. *Curr Med Chem Cardiovasc Hematol Agents*. 2005;3(4):383-391.
[4] Klein N, Gembardt F, Supé S, et al. Angiotensin-(1-7) protects from experimental acute lung injury. *Crit Care Med*. 2013;41(11):e334-e343.
[5] Akhtar S, Chandrasekhar B, Attur S, Dhaunsi GS, Yousif MH, Benter IF. Transactivation of ErbB Family of Receptor Tyrosine Kinases Is Inhibited by Angiotensin-(1-7) via Its Mas Receptor. *PLoS One*. 2015;10(11):e0141657.

Caution: Product has not been fully validated for medical applications. For research use only.
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[6] Yousif MHM, Benter IF, Diz DI, Chappell MC. Angiotensin-(1-7)-dependent vasorelaxation of the renal artery exhibits unique angiotensin and bradykinin receptor selectivity. *Peptides*. 2017;90:10-16.
[7] Silva DM, Vianna HR, Cortes SF, Campagnole-Santos MJ, Santos RA, Lemos VS. Evidence for a new angiotensin-(1-7) receptor subtype in the aorta of Sprague-Dawley rats. *Peptides*. 2007;28(3):702-707.

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