

Vasonatin Peptide (VNP) (TFA)

Cat. No.:	HY-P1556A
Molecular Formula:	C ₁₂₆ H ₁₉₉ N ₃₆ O ₃₈ S ₃ F ₃
Molecular Weight:	2979.39
Sequence:	Gly-Leu-Ser-Lys-Gly-Cys-Phe-Gly-Leu-Lys-Leu-Asp-Arg-Ile-Gly-Ser-Met-Ser-Gly-Leu-Gly-Cys-Asn-Ser-Phe-Arg-Tyr (Disulfide bridge: Cys6-Cys22)
Sequence Shortening:	GLSKGCFGLKLDRIKMSGLGCNSFRY (Disulfide bridge: Cys6-Cys22)
Target:	Others
Pathway:	Others
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	Vasonatin Peptide (VNP) TFA is a chimera of atrial natriuretic peptide (ANP) and C-type natriuretic peptide (CNP). Vasonatin peptide TFA possesses the venodilating actions of CNP, the natriuretic actions of ANP, and unique arterial vasodilating actions not associated with either ANP or CNP. Vasonatin Peptide TFA protects the diabetic heart against ischemia-reperfusion injury by inhibiting ER stress via the cGMP-PKG signaling pathway ^{[1][2][3]} .								
In Vitro	Vasonatin Peptide (VNP) markedly enhances adiponectin mRNA expression, as well as protein secretion, however, suppresses IL-6 production in mature adipocytes. In addition, VNP significantly increases the intracellular levels of cGMP. The effects of VNP are mimicked by 8-br-cGMP, whereas inhibits by HS-142-1, or KT-5823 ^[2] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.								
In Vivo	Vasonatin peptide (100 µg/kg; i.v. ; 10 min before reperfusion) attenuates myocardial ischemia-reperfusion injury in diabetic rats ^[3] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.								
	<table> <tr> <td>Animal Model:</td> <td>High-fat diet-fed streptozotocin-induced diabetic Sprague-Dawley rats^[3]</td> </tr> <tr> <td>Dosage:</td> <td>100 µg/kg</td> </tr> <tr> <td>Administration:</td> <td>I.v. ; 10 min before reperfusion</td> </tr> <tr> <td>Result:</td> <td>Significantly improved the instantaneous first derivation of left ventricle pressure (±LV dP/d_{tmax}) and LV systolic pressure and reduced LV end-diastolic pressure, apoptosis index, caspase-3 activity, plasma creatine kinase (CK), and lactate dehydrogenase (LDH) activities. Inhibited endoplasmic reticulum (ER) stress by suppressing glucose-regulated protein 78 (GRP78) and C/EBP homologous protein (CHOP).</td> </tr> </table>	Animal Model:	High-fat diet-fed streptozotocin-induced diabetic Sprague-Dawley rats ^[3]	Dosage:	100 µg/kg	Administration:	I.v. ; 10 min before reperfusion	Result:	Significantly improved the instantaneous first derivation of left ventricle pressure (±LV dP/d _{tmax}) and LV systolic pressure and reduced LV end-diastolic pressure, apoptosis index, caspase-3 activity, plasma creatine kinase (CK), and lactate dehydrogenase (LDH) activities. Inhibited endoplasmic reticulum (ER) stress by suppressing glucose-regulated protein 78 (GRP78) and C/EBP homologous protein (CHOP).
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REFERENCES

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- [1]. Wei CM, et al. Vasonatrin peptide: a unique synthetic natriuretic and vasorelaxing peptide. J Clin Invest. 1993;92(4):2048-2052.
- [2]. Chen BY, et al. Vasonatrin peptide, a new regulator of adiponectin and interleukin-6 production in adipocytes. J Endocrinol Invest. 2011;34(10):742-746.
- [3]. Shi Z, et al. Vasonatrin peptide attenuates myocardial ischemia-reperfusion injury in diabetic rats and underlying mechanisms. Am J Physiol Heart Circ Physiol. 2015;308(4):H281-H290.
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Caution: Product has not been fully validated for medical applications. For research use only.

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