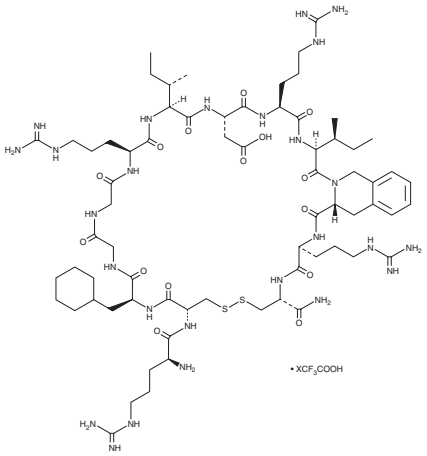
PRODUCT INFORMATION

A71915 (trifluoroacetate salt)

Item No. 43005

Formal Name:	L-arginyl-L-cysteinyl-3-cyclohexyl-L-alanylglycylglycyl-L-arginyl-L-isoleucyl-L-α-aspartyl-L-arginyl-L-isoleucyl-(3R)-1,2,3,4-tetrahydro-3-isoquinolinecarbonyl-L-arginyl-L-cysteinamide, cyclic (2→13)-disulfide, trifluoroacetate salt
Synonyms:	[Arg^6,Cha^8]ANP-(6-15)-D-Tic-Arg-Cys-NH_2; [Arg^6,Cha^8]Atrial Natriuretic Peptide-(6-15)-D-Tic-Arg-Cys-NH_2
Peptide Sequence:	Cyclo(RCX_1GGRIDRIX_2RC)-NH_2 (X_1 = Cyclohexylalanine; X_2 = D-1,2,3,4-Tetrahydro-3-isoquinolinecarbonyl)
MF:	$C_{69}H_{116}N_{26}O_{15}S_2 \bullet XCF_3COOH$
FW:	1,614.0
Purity:	≥98%
Supplied as:	A solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

A71915 (trifluoroacetate salt) is supplied as a solid. A stock solution may be made by dissolving the A71915 (trifluoroacetate salt) in the solvent of choice, which should be purged with an inert gas. A71915 (trifluoroacetate salt) is sparingly soluble (1-10 mg/ml) in ethanol and DMSO.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. Organic solvent-free aqueous solutions of A71915 (trifluoroacetate salt) can be prepared by directly dissolving the solid in aqueous buffers. A71915 (trifluoroacetate salt) is sparingly soluble (1-10 mg/ml) in PBS (pH 7.2). We do not recommend storing the aqueous solution for more than one day.

Description

A71915 is an antagonist of guanylyl cyclase A receptor (GC-A; K_i = 0.66 nM).[1] It inhibits cGMP production induced by atrial natriuretic peptide (ANP) in NB-OK-1 cells (pA_2 = 9.48). A71915 (1 µM) induces dilation and increases glomerular blood flow in rat hydronephrotic renal vascular segments but inhibits ANP-induced dilation in the same preparations.[2] It also prevents increases in spontaneous action potential frequency induced by brain natriuretic peptide (BNP) in isolated mouse sinoatrial node myocytes when used at a concentration of 0.5 µM.[3]

References

1. Delporte, C., Winand, J., Poloczek, P., *et al.* Discovery of a potent atrial natriuretic peptide antagonist for ANP_A receptors in the human neuroblastoma NB-OK-1 cell line. *Eur. J. Pharmacol.* **224(2-3)**, 183-188 (1992).
2. Endlich, K. and Steinhausen, M. Natriuretic peptide receptors mediate different responses in rat renal microvessels. *Kidney Int.* **52(1)**, 202-207 (1997).
3. Springer, J., Azer, J., Hua, R., *et al.* The natriuretic peptides BNP and CNP increase heart rate and electrical conduction by stimulating ionic currents in the sinoatrial node and atrial myocardium following activation of guanylyl cyclase-linked natriuretic peptide receptors. *J. Mol. Cell. Cardiol.* **52(5)**, 1122-1134 (2012).

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