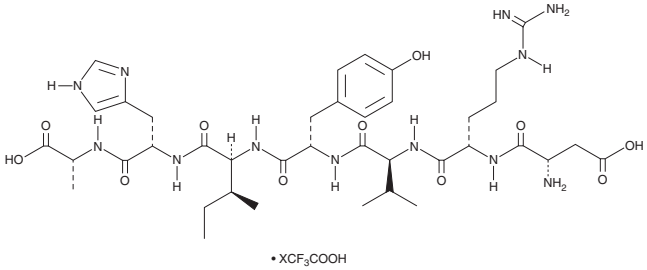
PRODUCT INFORMATION

A-779 (trifluoroacetate salt)

Item No. 23396

Formal Name:	5-L-isoleucine-7-D-alanine-1-7-angiotensin II, trifluoroacetate salt
MF:	$C_{39}H_{60}N_{12}O_{11} \bullet XCF_3COOH$
FW:	873.0
Purity:	≥98%
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥2 years



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

Laboratory Procedures

A-779 (trifluoroacetate salt) is supplied as a crystalline solid. A stock solution may be made by dissolving the A-779 (trifluoroacetate salt) in the solvent of choice, which should be purged with an inert gas. A-779 (trifluoroacetate salt) is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of A-779 (trifluoroacetate salt) in these solvents is approximately 33 and 10 mg/ml, respectively.

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 A-779 (trifluoroacetate salt) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of A-779 (trifluoroacetate salt) in PBS, pH 7.2, is approximately 1 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

A-779 is a peptide antagonist of the Mas receptor, also known as the angiotensin (1-7) (Ang (1-7)) receptor (IC_{50} = 0.3 nM in a radioligand binding assay).[1] It does not compete with angiotensin 1 (AT_1) and AT_2 receptor agonists for binding in adrenocortical membranes when used at a concentration of 1 µM and exhibits an IC_{50} value greater than 10 µM in adrenomedullary membranes.[2] *In vitro*, A-779 inhibits Ang (1-7)-induced release of arachidonic acid from CHO cells transfected with Mas.[1] A-779 (0.01 mg/kg) prevents the antidiuretic effects of Ang (1-7) in water-loaded rats. It also inhibits the Ang (1-7)-induced decrease in mean arterial pressure (MAP) when administered by microinjection into the nucleus of the solitary tract with no effect on basal MAP. A-779 increases urine flow rate and sodium excretion in male, but not female, rats when AT_1 and AT_2 receptors are blocked by the selective antagonists losartan (Item No. 10006594) and PD 123319 (Item No. 16099), respectively.[3]

References

1. Santos, R.A., Simoes e Silva, A.C., Maric, C., *et al.* Angiotensin-(1-7) is an endogenous ligand for the G protein-coupled receptor Mas. *Proc. Natl. Acad. Sci. U.S.A.* **100(14),** 8258-8263 (2003).
2. Santos, R.A., Campagnole-Santos, M.J., Baracho, N.C., *et al.* Characterization of a new angiotensin antagonist selective for angiotensin-(1-7): Evidence that the actions of angiotensin-(1-7) are mediated by specific angiotensin receptors. *Brain Res. Bull.* **35(4)**, 293-298 (1994).
3. Mansoori, A., Oryan, S., and Nematbakhsh, M. Role of Mas receptor antagonist (A779) on pressure diuresis and natriuresis and renal blood flow in the absence of angiotensin II receptors type 1 and 2 in female and male rats. *J. Physiol. Pharmacol.* **65(5)**, 633-639 (2014).

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