Vapreotide acetate

Cat. No.: HY-P0061A

CAS No.: 849479-74-9

Molecular Formula: $C_{59}H_{74}N_{12}O_{11}S_2$

Molecular Weight: 1191.42

Sequence: {d-Phe}-Cys-Tyr-{d-Trp}-Lys-Val-Cys-Trp-NH2 (Disulfide bridge: Cys2-Cys7)

Sequence Shortening: {d-Phe}-CY-{d-Trp}-KVCW-NH2 (Disulfide bridge: Cys2-Cys7)

Target: Neurokinin Receptor

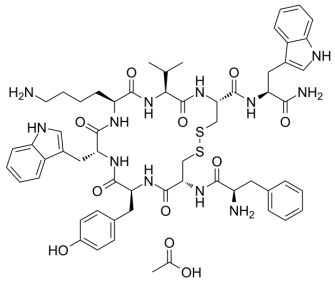
Pathway: GPCR/G Protein; Neuronal Signaling

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)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 14.29 mg/mL (11.99 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.8393 mL	4.1967 mL	8.3933 mL
	5 mM	0.1679 mL	0.8393 mL	1.6787 mL
	10 mM	0.0839 mL	0.4197 mL	0.8393 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Vapreotide acetate (RC-160 acetate; BMY-41606 acetate) is a neurokinin-1 (NK1) receptor antagonist, with an IC_{50} of 330 nM.

IC_{50} & Target

NK1

In Vitro

Vapreotide attenuates the Substance P (SP)-triggered intracellular calcium increases and NF-κB activation in a dose-dependent manner. Vapreotide also inhibits SP-induced IL-8 and MCP-1 production in HEK293-NK1R and U373MG cell lines. Vapreotide inhibits HIV-1 infection of human MDM in vitro, an effect that is reversible by SP pretreatment[1]. Vapreotide significantly inhibits GH-, PRL, and/or alpha-subunit release by human GH-secreting pituitary adenoma cells in concentrations as low as 10^{-12}-10^{-14} M. Vapreotide inhibits GH release with an IC_{50} of 0.1 pM[2]. Vapreotide exhibits moderate-to-high affinities for SSTR2, -3, and -5 (IC_{50}=0.17, 0.1 and 21 nM, respectively) and low affinity for SSTR1 and -4 (IC_{50}=200 and 620 nM, respectively). RC-160 inhibits serum-induced proliferation of CHO cells expressing SSTR2 and SSTR5 (EC_{50}=53 and 150 pM, respectively)[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	In cirrhosis, bleeding by rupture of oesophagogastric varices is a severe complication of portal hypertension. The acute administration of vapreotide to rats decreases collateral circulation blood flow while chronic administration attenuated its development[4]. Tumor volumes and weights are reduced by about 40% by RC-160 by s.c. injections at doses of 100 μg/day/animal. Vapreotide can inhibit the growth of androgen-independent prostate cancer when the therapy is started at an early stage of tumor development[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	CHO cells are cultured in aMEM containing 10% FCS. After overnight attachment, the medium is changed to a MEM containing either 10% FCS or insulin with and without vapreotide. Cell growth is measured after 24 h by counting cells with a Coulter counter model ZM[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4][5]	Rats: Acute effects are evaluated at baseline and 30 min after placebo or vapreotide (8 μg/kg/hr) infusions in DMNA rats. Chronic hemodynamic effects are evaluated using subcutaneous implants for five weeks in anesthetized DMNA rats and in sham rats. Hemodynamic measurements include splenorenal shunt blood flow by the transit time ultrasound method and cardiac output by the combined dilution–TTU method[4]. Mice: Nude mice bearing xenografts of the androgen-independent human prostate-cancer cell line PC-3 are treated for 4 weeks with somatostatin analog vapreotide (20 μg/day/animal), bombesin/gastrin-releasing peptide (GRP) antagonist, or the combination of both peptides. Tumor volumes and weights are measured[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Neurotox Res. 2022 Nov 15.

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REFERENCES

[1]. Spitsin S, et al. Analog of somatostatin vapreotide exhibits biological effects in vitro via interaction with neurokinin-1 receptor. Neuroimmunomodulation. 2013;20(5):247-55.

[2]. Hofland LJ, et al. Relative potencies of the somatostatin analogs octreotide, BIM-23014, and RC-160 on theinhibition of hormone release by cultured human endocrine tumor cells and normal rat anterior pituitary cells. Endocrinology. 1994 Jan;134(1):301-6.

[3]. Buscail L, et al. Inhibition of cell proliferation by the somatostatin analogue RC-160 is mediated by somatostatin receptor subtypes SSTR2 and SSTR5 through different mechanisms. Proc Natl Acad Sci U S A. 1995 Feb 28;92(5):1580-4.

[4]. Veal N, et al. Hemodynamic effects of acute and chronic administration of vapreotide in rats with cirrhosis. Dig Dis Sci. 2003 Jan;48(1):154-61.

[5]. Pinski J, et al. Effect of somatostatin analog RC-160 and bombesin/gastrin releasing peptide antagonist RC-3095 on growth of PC-3 human prostate-cancer xenografts in nude mice.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA