Octreotide

Cat. No.: HY-P0036

CAS No.: 83150-76-9

Molecular Formula: $C_{49}H_{66}N_{10}O_{10}S_2$

Molecular Weight: 1019.24

Sequence: Phe-Cys-Phe-Trp-Lys-Thr-Cys-Thr (Disulfide bridge: Cys2-Cys7)

FCFWKTCT(Disulfide bridge: Cys2-Cys7)

Sequence Shortening: FCFWKTCT (Disulfide bridge: Cys2-Cys7)

Target: Somatostatin Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: Protect from light

Powder -80°C 2 years

-20°C 1 year

* The compound is unstable in solutions, freshly prepared is recommended.

SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (98.11 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.9811 mL	4.9056 mL	9.8112 mL
5 mM	0.1962 mL	0.9811 mL	1.9622 mL
10 mM	0.0981 mL	0.4906 mL	0.9811 mL

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

BIOLOGICAL ACTIVITY

Description

Octreotide is a somatostatin analog that binds to the somatostatin receptor, mainly subtypes 2, 3, and 5, increases Gi activity, and reduces intracellular cAMP production.

In Vitro

Octreotide reverses the PA-induced alterations in Akt and GSK3β phosphorylation and expression of GS mRNA in HepG2 cells[1].

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

In Vivo

Octreotide significantly lowers the plasma glucose levels in the obese rats of the HFD group. Octreotide intervention significantly decreases the serum insulin concentration; however, there is no marked reduction in serum TG, TC, FFA, ALT and AST levels. Octreotide significantly inhibits the HOMA index. Octreotide decreases ipGTT and ipITT AUCs, but not significantly. Octreotide improves fat degeneration in rats with HFD-induced obesity and lipid droplet accumulation in PA-treated HepG2 cells. Octreotide promotes the phosphorylation of Akt and GSK3β and the expression of GS mRNA in rats with

HFD-induced obesity[1]. Octreotide reduces body weight and wet kidney weight compared with the vehicle-treated (CONT) group. PAS and Octreotide/PAS treatment decrease cAMP levels, but Octreotide alone does not in PCK rats. In the Octreotide/PAS group, there are a significantly fewer pS6-positive cells than in the PAS alone group[2].

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

PROTOCOL

Animal Administration [2]

AMale PCK rats (n = 24) are assigned randomly to 1 of 4 groups (n = 6 per group): treatment by subcutaneous injection every 4 weeks treatment with 8 mg/kg Octreotide-LAR alone, 8 mg/kg PAS-LAR alone, co-application of 8 mg/kg Octreotide and 8 mg/kg PAS, or vehicle (microparticles liquid; CONT) from 4 to 16 weeks of age. The vehicle contains copolymer microparticles with polylactic-co-glycolic acid (PLGA). In 4- and 15-week-old conscious rats, heart rate (HR), diastolic blood pressure (DBP), and systolic blood pressure (SBP) are determined using a tail-cuff sphygmomanometer. Twenty-four-hour urine volume and food consumption are measured using metabolic cages after 15.5 weeks of age. After body weight measurement, the animals are anesthetized with sodium pentobarbital at 16 weeks of age, and the kidneys and liver are removed rapidly, causing lethal exsanguination. Total wet kidney weight and wet liver weight are measured, and blood samples are collected for measurements of serum urea nitrogen (SUN), aspartate amino transferase (AST), alanine aminotransferase (ALT), insulin-like growth factor-1 (IGF-1), glucose, insulin, glucagon, and cortisol.

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

CUSTOMER VALIDATION

- Faculty of Biological and Environmental Sciences. University of Helsinki Finland. 2018 Dec.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Wang XX, et al. Effects of octreotide on hepatic glycogenesis in rats with high fat diet?induced obesity. Mol Med Rep. 2017 Jul;16(1):109-118

[2]. Kugita M, et al. Beneficial effect of combined treatment with octreotide and pasireotide in PCK rats, an orthologous model of human autosomal recessive polycystic kidney disease. PLoS One. 2017 May 18;12(5):e0177934.

India Contact:
Life Technologies (India) Pvt. Ltd.
306, Aggarwal City Mall, Opposite M2K Pitampura, Delhi – 110034 (INDIA). Ph: +91-11-42208000, 42208111, 42208222, Mobile: +91-9810521400, Fax: +91-11-42208444
Email: customerservice@lifetechindia.com Website: www.lifetechindia.com