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

FCFWKTCT(Disulfide bridge: Cys2-Cys7)

Sequence: Phe-Cys-Phe-Trp-Lys-Thr-Cys-Thr (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 (SMS 201-995) is a somatostatin receptor agonist and synthetic octapeptide endogenous somatostatin analogue. Octreotide (SMS 201-995) can bind to the somatostatin receptor and mainly subtypes 2, 3, and 5, increases Gi activity, and reduces intracellular cAMP production. Octreotide (SMS 201-995) has antitumor activity, mediates apoptosis and may also be used in disease studies in acromegaly[1][2].

In Vitro

Octreotide reverses the PA-induced alterations in Akt and GSK3β phosphorylation and expression of GS mRNA in HepG2 cells[1].Octreotide (10⁻⁸mM, 6 hours) induces phosphorylated glycogen synthase kinase 3β (GSK3β) phosphorylation and increases glycogen synthase (GS) activity[3].

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

Western Blot Analysis[3]

Cell Line:	Human hepatoblastoma HepG2 cell line

Concentration:	10^{-8}mM
Incubation Time:	6 hours
Result:	Increased the protein expression levels of phosphorylated Akt and GSK3β by 140.8% and 12.2%, respectively and the mRNA level of GS also increased.

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.

CUSTOMER VALIDATION

- J Pharm Biomed Anal. 11 December 2021, 114518.
- Basic Clin Pharmacol Toxicol. 2022 Jun 10.
- Research Square Print. 2022 Aug.
- Faculty of Biological and Environmental Sciences. University of Helsinki Finland. 2018 Dec.

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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.

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