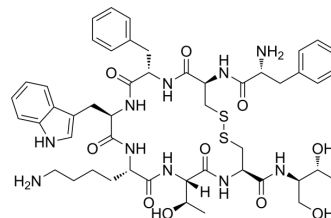


Octreotide

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:	{d-Phe}-Cys-Phe-{d-Trp}-Lys-Thr-Cys-{d-Threoninol} (Disulfide bridge: Cys2-Cys7)
Sequence Shortening:	{d-Phe}-CF{d-Trp}-KTC-{d-Threoninol} (Disulfide bridge: Cys2-Cys7)
Target:	Somatostatin Receptor; Apoptosis
Pathway:	GPCR/G Protein; Neuronal Signaling; Apoptosis
Storage:	Sealed storage, away from moisture and 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 ₂ O : 100 mg/mL (98.11 mM; Need ultrasonic)				
		Solvent Concentration	Mass		
	Preparing Stock Solutions	1 mM	1 mg	5 mg	10 mg
		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.					
In Vivo	1. Add each solvent one by one: PBS Solubility: 100 mg/mL (98.11 mM); Clear solution; Need ultrasonic				

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]} .		
IC₅₀ & Target	SSTR2	SSTR3	SSTR5
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 3 β (GSK3 β) 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 GSK3 β 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 GSK3 β 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

- Nat Commun. 2023 Feb 21;14(1):962.
- J Pharm Sci. 2022 Oct 10;S0022-3549(22)00454-3.
- J Pharm Biomed Anal. 2022: 115156.
- J Pharm Biomed Anal. 11 December 2021, 114518.
- Basic Clin Pharmacol Toxicol. 2022 Jun 10.

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REFERENCES

- [1]. Xiao-Xia Wang, 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]. 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
- [3]. 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.

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

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