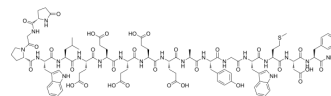


Gastrin I, human

Cat. No.:	HY-P1097
CAS No.:	10047-33-3
Molecular Formula:	C ₉₇ H ₁₂₄ N ₂₀ O ₃₁ S
Molecular Weight:	2098
Sequence:	{pGlu}-Gly-Pro-Trp-Leu-Glu-Glu-Glu-Glu-Glu-Ala-Tyr-Gly-Trp-Met-Asp-Phe-NH ₂
Sequence Shortening:	{pGlu}-GPWLEEEEEAYGWMDF-NH ₂
Target:	Cholecystokinin Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	Sealed storage, away from moisture and light Powder -80°C 2 years -20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro	DMSO : 50 mg/mL (23.83 mM; Need ultrasonic)				
	NH ₄ OH : 50 mg/mL (23.83 mM; ultrasonic and adjust pH to 11 with NH ₃ ·H ₂ O)				
	Preparing Stock Solutions	Solvent \ Mass \ Concentration	1 mg	5 mg	10 mg
		1 mM	0.4766 mL	2.3832 mL	4.7664 mL
		5 mM	0.0953 mL	0.4766 mL	0.9533 mL
10 mM		0.0477 mL	0.2383 mL	0.4766 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 2.5 mg/mL (1.19 mM); Suspended solution; Need ultrasonic 2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (1.19 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	Gastrin I, human is the endogenous peptide produced in the stomach, and increases gastric acid secretion via cholecystokinin 2 (CCK2) receptor.
IC₅₀ & Target	CCKBR

In Vitro	Gastrin I, human is the endogenous peptide produced in the stomach, and acts via cholecystokinin 2 (CCK2) receptor ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Gastrin I (1.5, 5, 15 and 45 nmol/kg, i.v.) increases pepsinogen and acid secretion in rats ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration ^[1]

Rats^[1]

The first set of experiments is carried out on rats with intact vagus nerves which are acutely treated with CCK-8S or Gastrin-1 at the doses of 1.5, 5, 15 and 45 nmol/kg. Both these peptides are administered i.v. as a bolus immediately after the collection of basal effluent samples. In experiments investigating the involvement of muscarinic, histamine H or CCK receptors in the gastric 2 secretory responses elicited by CCK-8S or Gastrin-1, the animals are pretreated with atropine 1 μmol/kg i.v., cimetidine 10 μmol/kg i.v., devazepide 1.25-2.5 μmol/kg i.v. or L-365,260 2.5-5 μmol/kg i.v., 10 min before ending the collection of the second basal effluent sample. Additional experiments are performed in animals pretreated with the irreversible inhibitor of histidine decarboxylase, α-fluoromethylhistidine (450 mmol/kg i.p. twice daily for two consecutive days), in order to suppress endogenous histamine production from digestive enterochromaffin-like cells^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Discov. 2020 Apr 7;6:20.
- FASEB J. 2023 Dec;37(12):e23279.

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REFERENCES

- [1]. Noble F, et al. International Union of Pharmacology. XXI. Structure, distribution, and functions of cholecystokinin receptors. Pharmacol Rev. 1999 Dec;51(4):745-81.
- [2]. Blandizzi C, et al. CCK1 and CCK2 receptors regulate gastric pepsinogen secretion. Eur J Pharmacol. 1999 May 28;373(1):71-84.

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