Gastrin-1, human

Cat. No.: HY-P1097

CAS No.: 10047-33-3

Molecular Formula: $C_{97}H_{124}N_{20}O_{31}S$

Molecular Weight: 2098.2

Sequence: {pGlu}-Gly-Pro-Trp-Leu-Glu-Glu-Glu-Glu-Glu-Ala-Tyr-Gly-Trp-Met-Asp-Phe-NH2

pE-GPWLEEEEEAYGWMDF-NH_2

Sequence Shortening: {pGlu}-GPWLEEEEEAYGWMDF-NH2

Target: Cholecystokinin Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: Protect from light

Powder -80°C 2 years

-20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)

SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (23.83 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	0.4766 mL	2.3830 mL	4.7660 mL
	5 mM	0.0953 mL	0.4766 mL	0.9532 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 >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (1.19 mM); Clear solution
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-1, human is the endogenous peptide produced in the stomach, and increases gastric acid secretion via cholecystokinin 2 (CCK2) receptor.

IC_{50} & Target

CCK2 receptor[1]

In Vitro

Gastrin-1, 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-1 (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 μ mol/kg i.v., cimetidine 10 μmolrkg 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.
- bioRxiv. 2019 Dec.

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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.

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