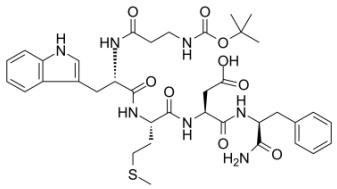
Pentagastrin

Cat. No.:	HY-A0261
CAS No.:	5534-95-2
Molecular Formula:	$C_{37}H_{49}N_7O_9S$
Molecular Weight:	767.89
Target:	Cholecystokinin Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	-20°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 34 mg/mL (**44.28 mM**)

H_2O : < 0.1 mg/mL (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Mass / Solvent / Concentration	1 mg	5 mg	10 mg
1 mM	1.3023 mL	6.5113 mL	13.0227 mL
5 mM	0.2605 mL	1.3023 mL	2.6045 mL
10 mM	0.1302 mL	0.6511 mL	1.3023 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: 1.98 mg/mL (2.58 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: **10% DMSO >> 90% corn oil**
   Solubility: ≥ 2.08 mg/mL (2.71 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pentagastrin (ICI-50123) is a selective agonist of **Cholecystokinin B (CCK_B) receptor** with an **IC_{50}** of 11 nM[1]. Pentagastrin enhances gastric mucosal defence mechanisms against acid and protects the gastric mucosa from experimental injury[2].

IC_{50} & Target

IC50: 11 nM (CCK_B receptor)[1]

In Vitro

Cholecystokinin receptors on GH3 rat anterior pituitary cells have been characterised using radioligand binding and Ca^{2+} mobilisation. Competition curve with Pentagastrin (**IC_{50}** of 25 nM) is consistent with a population predominantly of CCK_B receptors. The selective CCK_B receptor agonist, Pentagastrin, (0.1 nM-100 μM) dose dependently increased

	intracellular Ca^{2+} with a maximal increase of 2.77-fold. Binding of 50 pM [^{125}I]BHCCK to GH 3 cells is dose dependently inhibited by Pentagastrin **IC_{50}** of 45 nM. Response to a submaximal dose of the CCK_B receptor agonist Pentagastrin (100 nM) was dose dependently blocked by the CCK_B receptor antagonist L-365,260[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Pentagastrin (80 μg/kg/h; intravenous injection; male Sprague-Dawley rats) treatment protects rat gastric mucosa from acidified aspirin injury. Pentagastrin induces a hyperaemic response to luminal acid challenge, increases mucus gel thickness, and elevates intracellular pH (pH_i) during acid challenge[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (approximately 200 g)[2]
Dosage:	80 μg/kg/h
Administration:	Intravenous injection
Result:	Protected rat gastric mucosa from acidified aspirin injury. Induced a hyperaemic response to luminal acid challenge, increased mucus gel thickness, and elevated pH_i during acid challenge.

REFERENCES

[1]. Smith AJ, et al. Characterisation of CCKB receptors on GH3 pituitary cells: receptor activation is linked to Ca2+ mobilisation. Eur J Pharmacol. 1994 Apr 15;267(2):215-23.

[2]. Tanaka S, et al. Pentagastrin gastroprotection against acid is related to H2 receptor activation but not acid secretion. Gut. 1998 Sep;43(3):334-41.

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