Product Data Sheet

Product Name: (Pro³)-Gastric Inhibitory Polypeptide (human)
Cat. No.: GA20278

Chemical Properties

Cas. No.	299898-52-5		
Formula	$C_{226}H_{338}N_{60}O_{64}S$	M.Wt	4951.6
Solubility	Soluble in DMSO	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			

Protocol

Cell experiment [1]:

Cell lines	COS-7 cells (African green monkey kidney fibroblast-like cell line) transiently transfected with human GIP receptor cDNA

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Preparation Method	Transfected COS-7 cells were assayed for cAMP accumulation. Cells were stimulated with species-specific (Pro3)-Gastric Inhibitory Polypeptide (4.7nM to 92nM) at the indicated concentration range for 30 minutes.
Reaction Conditions	4.7nM to 92nM; 30 minutes.
Applications	(Pro3)-Gastric Inhibitory Polypeptide acted as a full agonist with similar efficacy as native human GIP at the human GIP receptor.

Animal experiment [2]:

Animal models	Obese diabetic (ob/ob) mice
Preparation Method	Mice were fasted for 18 hours, then intraperitoneally administered a single dose of (Pro3)-Gastric Inhibitory Polypeptide (25nmol/kg). Following injection, the mice were allowed to re-feed for 15 minutes. Blood samples were collected at 0, 15, 30, 60, and 120 minutes post-injection for plasma insulin and glucose analysis.
Dosage form	25nmol/kg; i.p.; single injection.
Applications	Administration of (Pro3)-Gastric Inhibitory Polypeptide significantly decreased the postprandial plasma insulin response in fasted mice, reducing the overall insulin release. (Pro3)-Gastric Inhibitory Polypeptide also lowered the basal hyperinsulinaemia in non-fasted mice. Accompanying changes in plasma glucose concentrations were minimal.

Caution: Product has not been fully validated for medical applications. For research use only.
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References:
[1] Sparre-Ulrich AH, Hansen LS, Svendsen B, et al. Species-specific action of (Pro3)GIP - a full agonist at human GIP receptors, but a partial agonist and competitive antagonist at rat and mouse GIP receptors. Br J Pharmacol. 2016 Jan;173(1):27-38.
[2] Gault VA, O'Harte FP, Harriott P, et al. Effects of the novel (Pro3)GIP antagonist and exendin(9-39)amide on GIP- and GLP-1-induced cyclic AMP generation, insulin secretion and postprandial insulin release in obese diabetic (ob/ob) mice: evidence that GIP is the major physiological incretin. Diabetologia. 2003 Feb;46(2):222-30.

Background

(Pro3)-Gastric Inhibitory Polypeptide (human) is an analog of human glucose-dependent insulinotropic polypeptide (GIP), distinguished by the substitution of proline for glutamate at the third amino acid position. (Pro3)-Gastric Inhibitory Polypeptide acts as a potent, stable, and specific full agonist of the human GIP receptor (hGIPR), exhibiting high binding affinity (Ki/Kd=0.90nM)[1-2]. (Pro3)-Gastric Inhibitory Polypeptide is used in research related to obesity-associated diabetes and type 2 diabetes[3-4].

In vitro, using isolated human atrial preparations, simultaneous stimulation with (Pro3)-Gastric Inhibitory Polypeptide (100nM) and Tirzepatide (100nM) was performed. (Pro3)-Gastric Inhibitory Polypeptide attenuated the positive inotropic effect induced by Tirzepatide. (Pro3)-Gastric Inhibitory Polypeptide antagonized the GIP receptor-mediated enhancement of myocardial contractility[5]. COS-7 cells transiently transfected with the human GIP receptor were stimulated with (Pro3)-Gastric Inhibitory Polypeptide (4.7nM to 92nM) for 30 minutes. (Pro3)-Gastric Inhibitory Polypeptide acted as a full agonist with similar efficacy to native human GIP at the human GIP receptor[6].

In vivo, (Pro3)-Gastric Inhibitory Polypeptide (25nmol/kg) was administered via intraperitoneal injection once or twice daily to high-fat diet-induced obese diabetic mice for 48 days. (Pro3)-Gastric Inhibitory Polypeptide significantly improved glucose tolerance and insulin sensitivity, and reduced weight gain and hyperinsulinemia[7]. In fasted obese diabetic (ob/ob) mice, a single intraperitoneal injection of (Pro3)-Gastric Inhibitory Polypeptide (25nmol/kg) was administered. (Pro3)-Gastric Inhibitory Polypeptide significantly reduced the plasma insulin response 15 minutes after re-feeding,

decreasing overall insulin release by 42%. In non-fasted ob/ob mice, a single intraperitoneal injection of (Pro3)-Gastric Inhibitory Polypeptide (25nmol/kg) ameliorated the basal hyperinsulinemia[8].

References:

[1] Rosenkilde MM, Lindquist P, Kizilkaya HS, et al. GIP-derived GIP receptor antagonists - a review of their role in GIP receptor pharmacology. Peptides. 2024 Jul;177:171212.
[2] McClean PL, Irwin N, Cassidy RS, et al. GIP receptor antagonism reverses obesity, insulin resistance, and associated metabolic disturbances induced in mice by prolonged consumption of high-fat diet. Am J Physiol Endocrinol Metab. 2007 Dec;293(6):E1746-55.
[3] McClean PL, Gault VA, Irwin N, et al. Daily administration of the GIP-R antagonist (Pro3)GIP in streptozotocin-induced diabetes suggests that insulin-dependent mechanisms are critical to anti-obesity-diabetes actions of (Pro3)GIP. Diabetes Obes Metab. 2008 Apr;10(4):336-42.
[4] Cassidy RS, Irwin N, Flatt PR. Effects of gastric inhibitory polypeptide (GIP) and related analogues on glucagon release at normo- and hyperglycaemia in Wistar rats and isolated islets. Biol Chem. 2008 Feb;389(2):189-93.
[5] Neumann J, Hofmann B, Kirchhefer U, et al. Tirzepatide increased force of contraction in the isolated human atrium. Naunyn Schmiedebergs Arch Pharmacol. 2025 Oct;398(10):14451-14459.
[6] Sparre-Ulrich AH, Hansen LS, Svendsen B, et al. Species-specific action of (Pro3)GIP - a full agonist at human GIP receptors, but a partial agonist and competitive antagonist at rat and mouse GIP receptors. Br J Pharmacol. 2016 Jan;173(1):27-38.
[7] McClean PL, Irwin N, Hunter K, et al. (Pro(3))GIP[mPEG]: novel, long-acting, mPEGylated antagonist of gastric inhibitory polypeptide for obesity-diabetes (diabesity) therapy. Br J Pharmacol. 2008 Nov;155(5):690-701.
[8] Gault VA, O'Harte FP, Harriott P, et al. Effects of the novel (Pro3)GIP antagonist and exendin(9-39)amide on GIP- and GLP-1-induced cyclic AMP generation, insulin secretion and postprandial insulin release in obese diabetic (ob/ob) mice: evidence that GIP is the major physiological incretin. Diabetologia. 2003 Feb;46(2):222-30.

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