Product Data Sheet

Product Name: GIP (human)
Cat. No.: GC17838

Chemical Properties

Cas. No.	100040-31-1		
SMILES	CC[C@]([C@@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/C/N=C(O)/[C@](/N=C(O)/[C@](/N=C(O)/[C@](N)([H])CC1=CC=C(O)C=C1)([H])C)([H])CCC(O)=O)([H])[C@@](O)([H])C)([H])CC2=CC=CC=C2)([H])[C@@](CC)([H])C)		
Formula	$C_{226}H_{338}N_{60}O_{66}S$	M.Wt	4983.58
Solubility	20 mg/mL in Water	Storage	Desiccate at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	GIP (human)		

Protocol

Kinase experiment
[1]:

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com
Address: 10292 Central Ave. #205, Montclair, CA, USA

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Preparation Method	The cells were grown and transfected as described in GIP Receptor Signaling.125I labeled GIP was used as radioligand, and the competition binding analysis was performed on intact cells, seeded at a concentration of 50,000 cells/well, on the second day after transfection, the cells were incubated for 16 h at 4°C in 0.25 ml of buffer consisting of 25 mM Tris HCl, pH 7.4, and 5 mM MgCl2, using 15 pM 125I-GIP as radioligand. Increasing concentrations of unlabeled GIP, ranging from 10-11 to 10-6 M, were used as competitors. The competition binding was terminated by washing the cells once with 1 ml of binding buffer, and subsequently, the cells were lysed by the addition of 1 ml of lysis buffer (8 M carbamide, 3 M acetic acid, 2% Nonidet-P 40). The binding data were analyzed and IC50 values determined using nonlinear regression analysis.
Reaction Conditions	16 h at 4°C
Applications	For the homologous competition binding, IC50 value of GIP was observed to be 5.2 nM.

Cell experiment [2]:

Cell lines	3T3-L1 cells
Preparation Method	Following incubation of 3T3-L1 cells, for the time periods indicated in figure legends, GIP in the incubation medium were precipitated with 1/10 vol. of 7% (wt/vol) ZnSO4, incubated on ice for 10 min, and 1/5 vol. 0.1 m NaOH added at room temp. The mixture was centrifuged and the supernatant assayed for glycerol with an enzymatic assay.

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Reaction Conditions	10 min
Applications	GIP stimulated glycerol release with an EC50 of 3.28 +/- 0.63 Nm.

Animal experiment [1]:

Animal models	Male Wistar rats (390–440 g, 12 wk old)
Preparation Method	GIP was dissolved in 0.9% NaCl containing 1% HSA and infused into the arterial line with the use of a syringe pump to give final perfusate concentrations of 1 nM GIP. After a basal period, peptides were infused alone for 10-min periods separated by 15-min rest periods, during which time endocrine secretion returned to basal levels.
Dosage form	1 nM GIP
Applications	Perfusion of the pancreas with 1 nM GIP increased insulin secretion significantly (P<0.001) relative to basal secretion during perfusion with 10 mM glucose alone.

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References:

[1]. Deacon CF, Plamboeck A, Rosenkilde MM, de Heer J, Holst JJ. GIP-(3-42) does not antagonize insulinotropic effects of GIP at physiological concentrations. Am J Physiol Endocrinol Metab. 2006 Sep;291(3):E468-75.

[2]. McIntosh CH, Bremsak I, Lynn FC, Gill R, Hinke SA, Gelling R, Nian C, McKnight G, Jaspers S, Pederson RA. Glucose-dependent insulinotropic polypeptide stimulation of lipolysis in differentiated 3T3-L1 cells: wortmannin-sensitive inhibition by insulin. Endocrinology. 1999 Jan;140(1):398-404.

Background

Gastric inhibitory polypeptide, also known as glucose-dependent insulinotropic

polypeptide (GIP), is a 42-amino acid peptide that plays an important role in maintaining glucose and lipid homeostasis. Although originally discovered as an inhibitor of gastric acid secretion, its principal physiological property is its role as an incretin peptide, in which it mediates the enteroinsular axis. GIP is synthesized by enteroendocrine K-cells of the duodenum/proximal jejunum, and its secretion is stimulated postprandially. GIP signaling stimulates glucose absorption in enterocytes, potentiates endogenous glucose-dependent insulin release from islet beta-cells, increases glucose uptake while inhibiting lipolysis in adipocytes, increases nutrient uptake into bone, and inhibits bone resorption [1].

The competition binding experiments were carried out in transiently transfected COS-7 cells using ^{125}I GIP as radioligand. For the homologous competition binding, an IC_{50} value of 5.2 nM was observed [2]. GIP receptor messenger RNA was detected by RT-PCR and RNase protection assay. Receptors were detected in binding studies (IC_{50} 26.7 +/- 0.7 nM). GIP stimulated glycerol release with an EC50 of 3.28 +/- 0.63 nM [3].

Perfusion of the pancreas with 1 nM GIP increased insulin secretion significantly (P[2]. The effects of GIP on fat metabolism in vivo may depend upon the circulating insulin level, and that meal-released GIP may elevate circulating fatty acids, thus optimizing pancreatic β-cell responsiveness to stimulation by glucose and GIP [3].

References:
[1].Zhang CY, Boylan MO, Arakawa H, Wolfe MM. Effects of gastric inhibitory polypeptide (GIP) immunoneutralization on mouse motor coordination and memory. Peptides. 2020 Mar;125:170227.
[2].Deacon CF, Plamboeck A, Rosenkilde MM, de Heer J, Holst JJ. GIP-(3-42) does not antagonize insulinotropic effects of GIP at physiological concentrations. Am J Physiol Endocrinol Metab. 2006 Sep;291(3):E468-75.
[3].McIntosh CH, Bremsak I, Lynn FC, Gill R, Hinke SA, Gelling R, Nian C, McKnight G, Jaspers S, Pederson RA. Glucose-dependent insulinotropic polypeptide stimulation of lipolysis in differentiated 3T3-L1 cells: wortmannin-sensitive inhibition by insulin. Endocrinology. 1999 Jan;140(1):398-404.