

**Cat#** SP-100253-1

**Description:** Gastric Inhibitory Polypeptide (6-30) amide (human) (AA: Phe-Ile-Ser-Asp-Tyr-Ser-Ile-Ala-Met-Asp-Lys-Ile-His-Gln-Gln-Asp-Phe-Val-Asn-Trp-Leu-Leu-Ala-Gln-Lys-NH<sub>2</sub>) (MW: 3010.49)

**Size:** 1 mg

**Purity:** >95%

**Store:** Desiccated at -20oC.

**Glucagon** is a member of a multigene family comprising of Secretin, Vasoactive Intestinal Peptide (VIP), Gastric Inhibitory Peptide (GIP) and others like Glicentin and Oxyntomodulin (OXM), which differs from glucagon by C-terminal octapeptide. The glucagon precursor contains at least 3 intervening sequences that divide the protein-coding portion into 4 regions corresponding to the signal peptide and part of the N-terminal peptide, the remainder of the N-terminal peptide and glucagon, glucagon-like peptide-1 (GLP1), and GLP2. GIP, along with glucagon-like peptide-1 (GLP-1), belongs to a class of molecules referred to as incretins.

**GIP**, Gastric inhibitory polypeptide, also known as glucose-dependent insulinotropic polypeptide (GIP), is a 42-amino acid hormone (chr 17q21.3) that stimulates insulin secretion in the presence of glucose. GIP is derived by proteolytic processing of a 153-residue precursor, preproGIP; it is a member of a family of structurally related hormones that includes secretin, glucagon, vasoactive intestinal peptide, and growth hormone-releasing factor. Like all endocrine hormones, it is transported by blood. Gastric inhibitory polypeptide receptors are seven-transmembrane proteins found on beta-cells in the pancreas. The GIP receptor is a member of the B-family of G protein-coupled receptors and activation results in the stimulation of adenylyl cyclase and Ca (2+)-independent phospholipase A and activation of protein kinase (PK) A and PKB.

The function of GIP is to induce insulin secretion, which is stimulated primarily by hyperosmolarity of glucose in the duodenum. In addition to its insulinotropic activity, GIP exerts a number of additional actions including promotion of growth and survival of the pancreatic beta-cell and stimulation of adipogenesis. The brain, bone, cardiovascular system, and gastrointestinal tract are additional targets of GIP. GIP is also thought to have significant effects on fatty acid metabolism through stimulation of lipoprotein lipase activity in adipocytes. GIP secretion has been demonstrated in humans only at approx 10 days of age. GIP is part of the diffuse endocrine system and, as a consequence, difficult to demonstrate physiological or clinical effects. In comparison to insulin its effects are very subtle.

GIP recently appeared as a major player in bone remodeling. Researchers at Universities of Angers and Ulster evidenced that genetic ablation of the GIP receptor in mice resulted in profound alterations of bone micro architecture through modification of the adipokine network. Furthermore, deficiency in GIP receptors has also been associated in mice with a dramatic decrease in bone quality and a subsequent increase in fracture risk. In adipose tissue, GIP interacts with insulin to increase lipoprotein lipase activity and lipogenesis. There is significant interest in potential clinical applications for GIP analogs and both agonists and antagonists have been developed for preclinical studies.

**Reference:** McIntosh CH (2009) Vitam Horm.80:409-71; Meier JJ (2005) Diabetes Metab. Res. Rev. 21 (2): 91–117. Meier JJ et al, Regulatory peptides (2003), 95-100; Inagaki N et al, Mol Endocrinology (1989) 1014-21.

\*This product is for In vitro research use only.

**Related items:**

SP-100253-1	Gastric Inhibitory Polypeptide (6-30) amide (human) (MW: 3010.49)
SP-101812-1	[Tyr0] Gastric Inhibitory Peptide (23-42), human; [Tyr22] Gastric Inhibitory Peptide (22-42), human MW 2584.9]
SP-101816-1	Gastric Inhibitory Polypeptide (1-30) (porcine) (MW: 3552.1)
SP-55416-1	Gastric Inhibitory Peptide (GIP), Human MW: 4983.64]