SANTA CRUZ BIOTECHNOLOGY, INC.

GIP (021-04): sc-57162



BACKGROUND

Glucose-dependent Insulinotropic polypeptide (GIP) is a major physiologic factor in the augmentation of the Insulin response to oral glucose. GIP is a peptide hormone that is released postprandially from the small intestine and acts in concert with glucagon-like peptide (GLP)-1 to potentiate glucoseinduced Insulin secretion from the pancreatic β-cell. GIP has been shown to increase adenylyl cyclase activity, elevate intracellular calcium levels and stimulate a mitogen-activated protein kinase pathway in the pancreatic β cell. Additionally, nutrient protein provides a potent stimulus for GIP expression, an effect that occurs at the posttranslational level and may be mediated in part through the acid-stimulatory properties of protein. GIP release is demonstrated predominantly after ingestion of carbohydrate and fat and the effects of acid on GIP are consistent with a role for GIP as an enterogastrone.

REFERENCES

- 1. Sarson, D.L., et al. 1984. Glucose-dependent Insulinotropic polypeptide augmentation of Insulin. Physiology or pharmacology? Diabetes 33: 389-393.
- 2. Meneilly, G.S., et al. 2000. Effect of ageing and diabetes on glucose-dependent insulinotropic polypeptide and dipeptidyl peptidase IV responses to oral glucose. Diabet. Med. 17: 346-350.
- 3. Wolfe, M.M., et al. 2000. Regulation of glucose-dependent insulinotropic polypeptide release by protein in the rat. Am. J. Physiol. Gastrointest. Liver Physiol. 279: G561-G566.
- 4. Lynn, F.C., et al. 2001. Defective glucose-dependent insulinotropic polypeptide receptor expression in diabetic fatty Zucker rats. Diabetes 50: 1004-1011.
- 5. Ehses, J.A., et al. 2001. A new pathway for glucose-dependent insulinotropic polypeptide (GIP) receptor signaling. evidence for the involvement of phospholipase $\alpha 2$ in GIP-stimulated Insulin secretion. J. Biol. Chem. 276: 23667-23673.
- 6. Irwin, N., et al. 2006. Biological activity and antidiabetic potential of synthetic fragment peptides of glucose-dependent insulinotropic polypeptide, GIP(1-16) and (Pro3)GIP(1-16). Regul. Pept. 135: 45-53.
- 7. Parker, J.C., et al. 2006. Effects of sub-chronic exposure to naturally occurring N-terminally truncated metabolites of glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1), GIP(3-42) and GLP-1(9-36) amide, on Insulin secretion and glucose homeostasis in ob/ob mice. J. Endocrinol. 191: 93-100.

CHROMOSOMAL LOCATION

Genetic locus: GIP (human) mapping to 17q21.32.

SOURCE

GIP (021-04) is a mouse monoclonal antibody raised against synthetic GIP of human origin.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PRODUCT

Each vial contains 100 μ g lgG₁ in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

GIP (021-04) is recommended for detection of GIP of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for GIP siRNA (h): sc-72038, GIP shRNA Plasmid (h): sc-72038-SH and GIP shRNA (h) Lentiviral Particles: sc-72038-V.

Molecular Weight of GIP: 5 kDa.

Positive Controls: A549 cell lysate: sc-2413.

DATA



GIP (021-04): sc-57162. Western blot analysis of GIP expression in A549 whole cell lysate

SELECT PRODUCT CITATIONS

- 1. El-Salhy, M., et al. 2010. Abnormal small-intestinal endocrine cells in patients with irritable bowel syndrome. Dig. Dis. Sci. 55: 3508-3513.
- 2. El-Salhy, M., et al. 2015. Reduction in duodenal endocrine cells in irritable bowel syndrome is associated with stem cell abnormalities. World J. Gastroenterol. 21: 9577-9587.
- 3. Mazzawi, T. and El-Salhy, M. 2017. Changes in duodenal enteroendocrine cells in patients with irritable bowel syndrome following dietary guidance. Exp. Biol. Med. 242: 1355-1362.
- 4. Mazzawi, T., et al. 2021. The effects of fecal microbiota transplantation on the symptoms and the duodenal neurogenin 3, musashi 1, and enteroendocrine cells in patients with diarrhea-predominant irritable bowel syndrome. Front. Cell. Infect. Microbiol. 11: 524851.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.