MSD® Human Total GIP Kit

For quantitative determination in human plasma

3.

Alzheimer's Disease BioProcess Cardiac Cell Signaling Clinical Immunology Cytokines Growth Factors Hypoxia Immunogenicity Inflammation

Metabolic

Oncology Toxicology Vascular

Catalog Numbers

Human Total GIP Kit		
Kit Size	Catalog #	
1 plate	K151RPD-1	
5 plates	K151RPD-2	
25 plates	K151RPD-4	

Ordering Information

MSD Customer Service Phone: 1-301-947-2085 Fax: 1-301-990-2776 Email: CustomerService@ mesoscale.com

Scientific Support

Phone: 1-301-947-2025 Email: ScientificSupport@ mesoscale.com

Company Address

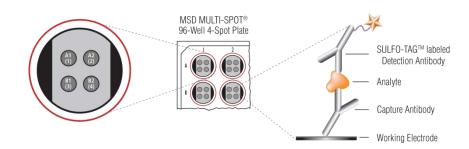
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Total GIP
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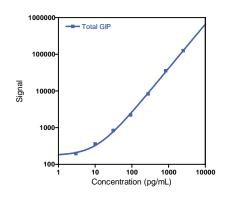
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Glucose-dependent insulinotropic or gastric inhibitory polypeptide (GIP) is a glucose-dependent, insulinotropic peptide that belongs to the incretin family. GIP is synthesized by enteroendocrine K cells in the duodenal and jejunal mucosa and is secreted into plasma in response to nutrient flow through the small intestine. GIP exerts its effects through binding to glycosylated G-protein coupled receptors in the gut, pancreatic islets, adipose tissue, several regions of the brain, testis, pituitary, lung, heart, vascular endothelium, and bone. GIP protects the small intestine from acid damage by reducing gastric acid secretion, inducing neutralization of stomach acid, and inhibiting gastrointestinal motility. GIP also induces insulin secretion from pancreatic β -cells. Additional functions of GIP have recently been proposed: modulation of fatty acid metabolism by stimulating lipoprotein lipase activity in adipocytes, regulation of bone turnover, and regulation of bloodflow. It has been reported that Type 2 diabetics are resistant to GIP. Clinical applications that exploit GIP activity to regulate glucose homeostasis are under investigation. The MSD Human Total GIP assay is available on 96-well, 4-spot plates. Representative data from the assay is presented below.

Assay Sensitivity

The following standard curve illustrates the dynamic range of the Human Total GIP assay.





The lower limit of detection (LLOD) is a calculated concentration based on a signal 2.5 standard deviations above the background (zero calibrator blank).

MSD Advantage

- Multiplexing: Multiple analytes can be measured in one well using typical sample volumes of 25 μL or less without compromising speed or performance
- Large dynamic range: Linear range of up to five logs enables the measurement of native levels of biomarkers in normal and diseased samples without multiple dilutions
- Minimal background: The stimulation mechanism (electricity) is decoupled from the response (light signal), minimizing matrix interference
- > Simple protocols: Only labels bound near the electrode surface are excited, enabling assays with fewer washes
- Flexibility: Labels are stable, non-radioactive, and conveniently conjugated to biological molecules
- > High sensitivity and precision: Multiple rounds of label excitation and emission enhance light levels and improve sensitivity

For Research Use Only. Not for use in diagnostic procedures.

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MSD Metabolic Assays

Tested Samples

Normal human EDTA plasma samples were diluted 2-fold and tested with the Human Total GIP Kit. Median and range of concentrations are displayed below. Concentrations are corrected for sample dilution.

Sample Type	Statistic	Total GIP
EDTA Plasma (N=7)	Median (pg/mL)	146
	Range (pg/mL)	52-368
	Samples in Quantitative Range	7

References

- 1. Holst JJ. On the physiology of GIP and GLP-1. Horm Metab Res. 2440;36:747-754.
- 2. Pederson RA, et al. Inhibition of histamine-, pentagastrin-, and insulin-stimulated canine gastric secretion by pure "gastric inhibitory polypeptide". Gastroenterology, 1972 62:393-400.
- 3. Dupre J, et al. Stimulation of insulin secretion by gastric inhibitory polypeptide in man. J Clin Endocrinol Metab. 1973;37:826-8.
- 4. Ugleholdt R. Glucose-dependent Insulinotropic Polypeptide(GIP): From prohormone to actions in endocrine pancreas and adipose tissue. Dan Med Bull. 2011 Dec;58(12):B4368.
- 5. Diab DL. The contribution of enteroinsular hormones to the pathogenesis of type 2 diabetes mellitus. Curr Diab Rep. 2010 Jun;10(3):192-8.

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