

Human Interferon Beta (IFN β) ELISA Kit | IFN β elisa kit

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CATALOG #
AAA281417

REACTIVITY
Human

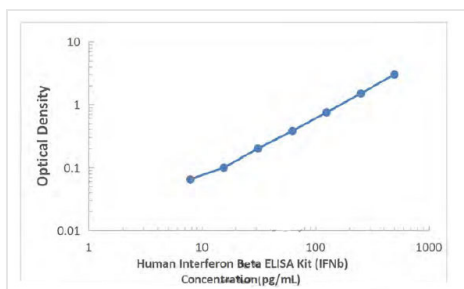
PRODUCT SYNONYMS

Interferon Beta (IFN β); Human Interferon Beta ELISA Kit (IFN β); IFN β elisa kit

PRODUCT SPECIFICATIONS

Reactivity	Human
Specificity	This assay recognizes natural Glucagon. The factors listed below were prepared at 50ng/ml in Standard /sample Diluent and assayed for cross-reactivity and no significant cross-reactivity or interference was observed.
Sequence Length	187
Samples	Cell Culture Supernates, Serum, And Plasma
Assay Type	Quantitative Sandwich
Detection Range	31.25 pg/ml-2000 pg/ml
Sensitivity	7pg/mL.
Preparation and Storage	Store at 4 degree C.

IMAGES



Standard Curve (Sample)

ADDITIONAL INFORMATION

Related Product Information for IFNβ elisa kit	Principle of the Assay: This assay employs the quantitative sandwich enzyme immunoassay technique. A monoclonal antibody specific for Glucagon has been pre-coated onto a microplate. Standards and samples are pipetted into the wells and any Glucagon present is bound by the immobilized antibody. Following incubation unbound samples are removed during a wash step, and then a detection antibody specific for Glucagon is added to the wells and binds to the combination of capture antibody-Glucagon in sample. Following a wash to remove any unbound combination, and enzyme conjugate is added to the wells. Following incubation and wash steps a substrate is added. A coloured product is formed in proportion to the amount of Glucagon present in the sample. The reaction is terminated by addition of acid and absorbance is measured at 450nm. A standard curve is prepared from seven Glucagon standard dilutions and Glucagon sample concentration determined!!Background/Introduction: Glucagon is a 29 amino acid (aa) peptide produced by the pancreas that plays a critical role in glucose metabolism and homeostasis (1-4). The Glucagon precursor mRNA is expressed by alpha cells (α-cells) of the pancreas, L cells of the intestine, and in the brain (1, 2). Only the pancreatic α-cells express the prohormone convertase PC2, also called PCSK2, which is required to produce Glucagon (2). Intestinal L cells instead express the prohormone convertase PC1, which processes the precursor to the Glucagon-overlapping peptides glicentin and oxyntomodulin. L cells also produce two Glucagon-like peptides, GLP-1 and GLP-2 that are derived from the same Glucagon precursor and influence glucose metabolism, but do not share any common sequence with Glucagon (1, 2). The aa sequence of the mature Glucagon peptide is identical in human, mouse, rat, pig, dog, horse, cow, sheep, and Xenopus. In normal metabolism, Glucagon is secreted in response to low blood glucose (hypoglycemia) and downregulated in response to high blood glucose (hyperglycemia). Although Glucagon binding sites are found in liver, brain, pancreas, kidney, intestine, and adipose tissue, the main activity of Glucagon receptors occurs in the liver, where Glucagon stimulates gluconeogenesis and glycogenolysis, thereby increasing blood glucose (1-4). It is particularly important that the brain receive sufficient glucose, since it is unable to store more than a minute quantity. Therefore the release of Glucagon from α-cells is under control by both hormones and neurotransmitters, and is very responsive to circulating glucose concentration. Insulin, and/or the zinc that islet β cells secrete with it, downregulates Glucagon secretion in intact islets (5, 6). Glucagon secretion is also downregulated by the neurotransmitter γ-aminobutyric acid (GABA), somatostatin produced by islet δ-cells, and GLP-1, but is enhanced by the neurotransmitter L-glutamate, amino acids (especially arginine), and Glucagon itself (2-4, 7). Through receptors on the α-cells, these substances affect potassium, sodium, and calcium channel activity and alter intracellular calcium concentration (2-4). Glucose suppression of Glucagon secretion is probably indirect, acting through paracrine signals from other islet cells (8). Like insulin, Glucagon is dysregulated in type 2 diabetes (T2D) and contributes to its pathology (2-4). Glucagon secretion is less responsive to insulin-mediated suppression in times of high circulating glucose, causing glucagonemia, and increasing the risk of hyperglycemia. Glucagon is also regulated by some of the same messengers that regulate insulin (10-12). Leptin inhibits α-cell glucagon secretion and stimulates β-cell insulin secretion, but glucagon blunts the leptin-mediated insulin secretion (10). Islet α-cells express ghrelin receptors and respond to ghrelin by increasing Glucagon secretion (11). Glucocorticoids, activated by 11β-HSD1, depress Glucagon secretion in hypoglycemia and insulin secretion in hyperglycemia (12). Although genetic polymorphisms of the Glucagon receptor are associated with T2D, downregulation of Glucagon secretion or deletion of the Glucagon receptor in mice that are susceptible to T2D actually improves glycemic control (13, 14).
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NCBI AND UNIPROT INFORMATION

NCBI GI #	4504603
NCBI GeneID	3456
NCBI Accession #	NP_002167.1
NCBI GenBank Nucleotide #	NM_002176.4
UniProt Accession #	P01574; Q5VWC9
Molecular Weight	22,294 Da
NCBI Official Full Name	interferon beta
NCBI Official Synonym Full Names	interferon beta 1
NCBI Official Symbol	IFNB1
NCBI Official Synonym Symbols	IFB; IFF; IFNB; IFN-beta
NCBI Protein Information	interferon beta

UniProt Protein Name	Interferon beta
UniProt Gene Name	IFNB1
UniProt Synonym Gene Names	IFB; IFNB; IFN-beta

For Research Use Only. Not for use in diagnostic or therapeutic procedures. Small volumes of product may become entrapped in the seal of the vial during shipment and storage; if necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the cap.

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Online datasheet: <https://www.aaabiotech.com/human-elisa-kits/interferon-beta-ifnb/281417>