abcam

Product datasheet

Human C Peptide peptide ab93903

Description

Product name	Human C Peptide peptide
Purity	92 % HPLC.
Animal free	No
Nature	Synthetic
Species	Human
Sequence	EAEDLQVGQVELGGGPGAGSLQPLALEGSLQ
Amino acids	57 to 87

Specifications

Our **Abpromise guarantee** covers the use of **ab93903** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications	Blocking - Blocking peptide for Anti-C Peptide antibody (**ab14181**)
Form	Lyophilized
Additional notes	C Peptide is part of the molecule of Proinsulin, that consists of three parts: C Peptide and two long strands of amino acids (called the alpha and beta chains) that later become linked together to form the insulin molecule. From every molecule of proinsulin, one molecule of insulin plus one molecule of C Peptide are produced. C peptide is released into the blood stream in equal amounts to insulin. A test of C peptide levels will show how much insulin the body is making. Insulin decreases blood glucose concentration. It increases cell permeability to monosaccharides, amino acids and fatty acids. It accelerates glycolysis, the pentose phosphate cycle, and glycogen synthesis in liver. This peptide can be used with studies using **ab14181**.

Preparation and Storage

Stability and Storage	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Reconstitution	Resuspend in 0.1% acetic acid for required concentration

General Info

Function	Insulin decreases blood glucose concentration. It increases cell permeability to monosaccharides, amino acids and fatty acids. It accelerates glycolysis, the pentose phosphate cycle, and glycogen synthesis in liver.
Involvement in disease	Defects in INS are the cause of familial hyperproinsulinemia (FHPRI) [MIM:176730]. Defects in INS are a cause of diabetes mellitus insulin-dependent type 2 (IDDM2) [MIM:125852]. IDDM2 is a multifactorial disorder of glucose homeostasis that is characterized by susceptibility to ketoacidosis in the absence of insulin therapy. Clinical fetaures are polydipsia, polyphagia and polyuria which result from hyperglycemia-induced osmotic diuresis and secondary thirst. These derangements result in long-term complications that affect the eyes, kidneys, nerves, and blood vessels. Defects in INS are a cause of diabetes mellitus permanent neonatal (PNDM) [MIM:606176]. PNDM is a rare form of diabetes distinct from childhood-onset autoimmune diabetes mellitus type 1. It is characterized by insulin-requiring hyperglycemia that is diagnosed within the first months of life. Permanent neonatal diabetes requires lifelong therapy. Defects in INS are a cause of maturity-onset diabetes of the young type 10 (MODY10) [MIM:613370]. MODY10 is a form of diabetes that is characterized by an autosomal dominant mode of inheritance, onset in childhood or early adulthood (usually before 25 years of age), a primary defect in insulin secretion and frequent insulin-independence at the beginning of the disease.
Sequence similarities	Belongs to the insulin family.
Cellular localization	Secreted.