Commentary

iMedPub Journals
www.imedpub.com

Journal of Diabetes Research and Endocrinology

2021
Vol.5.No.S1:001

Role of Insulin in Diabetic Patients

Adina Bernice*

Department of Endocrinology, The University of Tennessee Health Science, Tennessee, USA

***Corresponding author:** Adina Bernice, Department of Endocrinology, The University of Tennessee Health Science, Tennessee, USA, E-mail: Bernice.adina@gmail.com

Citation: Bernice A (2021) Role of Insulin in Diabetic Patients. J Diabetes Res Endocrinol Vol.5 No.S1:001.

Received date: September 21, 2021; **Accepted date:** October 05, 2021; **Published date:** October 12, 2021

Description

Pancreas the source of insulin production is an essential organ responsible for both digestion and glucose homeostasis Historically snub, is associated with blood sugar and true enough insulin as profound effects on carbohydrate metabolism besides it also plays a very vital role In fat and protein metabolism absolute or relative insulin deficiency causes diabetes mellitus which a characterized by abnormalities in carbohydrate, protein and fat metabolism The hormones of particular importance in glycaemic regulation are insulin glucagon and more recently glucagon like Peptide.

The adult human Pancreas is made up of numerous collections of cells called islets of Langerhans. There are about 1-2 million islets and it makes, up only about 2% volume of pancreas. While the remaining consists of blood vessels, ducts and the larger exocrine portion of the pancreas which secrete digestive juice and it is made up of aicini.

There are 4 major cells in the islets of Langerhans based on staining characteristics and appearance These are as follows:

Alpha cells

Produce glucagon It increases plasma glucose by increasing hepatic glcongenolysis and glucogenesis; increases lipolysis.

Beta cells

The majority of cells in the islets of Langerhans are beta cells, i.e. about 60-70%. These cells release insulin, which is anabolic in nature.

Delta cells

These cells produce somatostatin, which inhibits secretion of insulin, pancreatic polypeptide, and glucagon by acts locally in a paracrine manner.

F (or PP) cells

Produce pancreatic polypeptide, wh ich slows absorption of food, but its physiological significance, is uncertain.

The amino acid sequence of insulin molecule varies from species to species (pigs, cows, etc). These differences w ill not affect the biological activity if insulin from one species is given to another species, but they are certainly antigenic and induce antibody formation against the injected insulin when given over a long period of time. Human insulin is now used to avoid this problem.

Synthesis of Insulin Insulin is synthesized in the rough endoplasmic reticulum of the cells and it is packed into secretory granules in the Golgi apparatus and released by an exocytosis process. Insulin is synthesized from of amino acids which is a single long chain called preproinsulin.This chain gets splited, i.e. 23 amino acid signal peptide is removed from it and the remaining portion folds on itself with the formation of disulphide bonds, to form proinsulin. The C peptide or connecting peptide helps in the folding and connects the A and B chains. The C peptide gets detached and insulin is formed. C peptide level is an indicator of R cell function in patients who receive exogenous insulin.

The alpha subunit which binds insulin is extracellular, while the beta subunit extends from the cell membrane into the cell. The part of the beta subunit which extends into the cell has tyrosine kinase activity. Various factors like insulin levels, exercise and food effects the number and affinity of insulin receptors. A rise in insulin concentration will decreases the number of insulin receptors called down-regulation and drop down in insulin concentrations will increases the affinity of the insulin receptors. The number of insulin receptors is decreased in case of obesity and incontrast it will increased in times of starvation.

Mechanism of action of insulin

Insulin binds to the alpha subunit of its receptor. This binding triggers tyrosine kinase activity in the II subunit and causes auto phosphorylation of the beta subunit. This in turn causes either phosphorylation or dephosphorylation of certain enzymes and proteins in the cytoplasm. Activating some and inactivating some, thus bringing about the actions of insulin. One of the cytoplasmic substrates for insulin action is the insulin receptor substrate or IRS-I. Protein synthesis and growth promoting actions of insulin are mediated through Phosphoinositol 3-Kinase (PI3K) pathway.