ISLETS OF LANGERHANGS

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The Pancreas is a rather diffused gland which is a mixture of two histologically and functionally separate tissues. The bulk of the gland is exocrine in function and forms clusters of glandular cells called acini or lobules lined with cuboidal epithelium. Scattered among the exocrine cells are patches of tissues named Islets of Langerhangs after their discoverer in 1869, and these consists of endocrine cells that discharge their secretion directly in the blood. These cells are small prism shaped, closely packed together and without a lumen, they collectively formed an endocrine gland.

Four kinds of cells have been identified in the islets:

(i) Alpha (α) cells (60% do 70%) which contain alcohol-soluble granules, produce glucagon. Alpha (α) cells are also called A-cells

(ii) Beta (β) Cells (32 to 38%) whose granules are not soluble in alcohol. Beta cell also called B-cells (produce insulin)

(iii) Delta cells (D-cells) produce somatostanin.

(iv) F-cells :- produce Pancreatic polypeptid.

Delta cells and F-cells constitute 2%-8% of the islets of Langerhangs. Beta cells usually found toward the centre of islet, the alpha cells towards the periphery of the islets and D (delta) and F-cells found scattered.

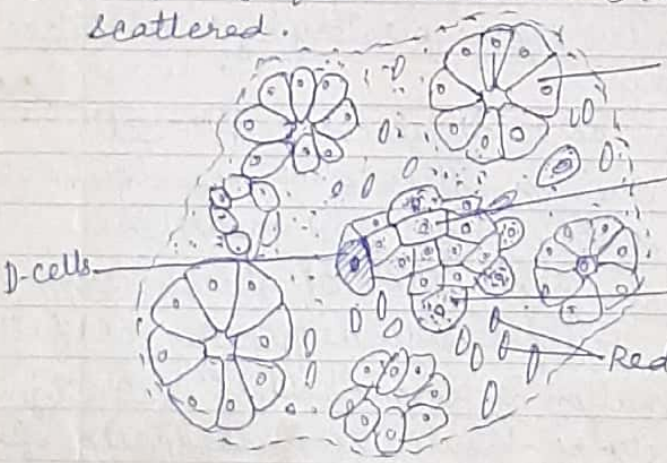

Fig Physiological anatomy of Pancreas.

Insulin :-

Insulin is the hypoglycemic antiketogenic factor and the protein hormone which regulates blood glucose.

Banting and Best extracted insulin in the year 1921 for which they get Noble prize in 1923. Abert prepare pure crystalline insulin in 1926.

Insulin is a relatively small protein of 51 amino acids disposed in two parallel chains A and B, interconnected by two disulphide bonds. A chain has 21, and the B chain 30 amino acid units, thus one molecule of insulin has 51 amino acids in all. Its molecular weight is 6,000.

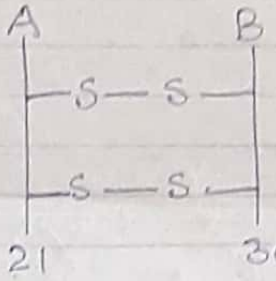

Fig: The insulin structure.

Functions :-

1. Increase combustion of sugar in tissues. Insulin accelerates the phosphorylation of glucose by the enzyme glucokinase or hexokinase. Glucose-6-phosphate thus formed enters the respiratory cycle to liberated chemical energy.
2. It helps transport of glucose into cell.
3. It promotes the formation of glycogen from glucose (glycogenesis) in the liver and muscles.
4. It reduces the production of glucose from non-carbohydrate sources such as protein and fate
5. Prevents formation of Ketone bodies (Antiketogenic effect) When the supply of insulin is inadequate fat metabolism is also affected, because fat must be used instead of carbohydrate for release of energy through cellular oxidation. The large increase in

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the amount of $CH_3CO \cdot COA$ which accumulates from the breakdown of fat is too great to be dealt with by the cellular TCA cycle. Instead of being combined with oxaloacetic acid in the first stage of that cycle, it passes to the liver where it condensed to form acetoacetic acid. The liver is unable to utilize it and therefore, the acid delivered to the body tissue by circulation. In blood, the acetoacetic acid undergoes further dissimilation forming β-hydroxybutyric acid and acetone. These degradation products are known as Ketone bodies.

5) It stimulates protein synthesis and growth e.g., nitrogen retention, bone formation etc. Increases the incorporation of amino acids into peptides.

7) Decreases lipoemia ie accumulation of excess fat in blood and liver.

Deficiency :-

Insulin deficiency causes diabetes mellitus. This is a disorder of carbohydrate metabolism characterised by :

1) Hyperglycemia :- It is a condition in which blood sugar increases above the mammal level (80-120 mg/100 ml)

2) Glycosauria : It is a condition in which glucose is appear in urine in such quantities as it will reduce Benedicts or Fehling's reagent. It occurs when sugar level in blood exceeds more than 180 mg.

3) Polyuria :- The high glucose level in the nephric filtrate increased osmotic pressure, sharply reducing the reabsorption of water back in blood, hence victim of the disease urinate large volume of urine frequently.

4) Dehydration :- The continued loss of water and electrolytes in the urine leads to increasing dehydration

5) Polydipsia :- Because of polyuria, the patient is thirsty inspite of drinking large amount of water

6) Weight loss → Instead of polyphagia (increase appetite) the patient loss weight because excessive breakdown of protein.

7) Abnormal metabolism of fat:-

Muscles are unable to utilize glucose in the absence of insulin, therefore the diabetic feel weak and tired. Since the carbohydrate cannot be used as fuel, fat is used instead to provide energy.

(i) Lipaemia :- Fat are mobilized from the body stores and transferred to the liver, hence fat content of the liver and blood rises.

(ii) Ketosis :- The disproportionate metabolism of fat in diabetes result in the over production of ketone bodies.

(iii) Acidaemia or Acidosis :-

As acetoacetic acid and β-hydroxybutyric acids are produced faster than they can metabolised.

8) Diabetic Coma - The ketoacidiosis is associated with increasing drowsiness and untreated, the patient may become unconscious (diabetic coma).

(B) GLUCAGON :-

It is a polypeptide consisting of 29 amino acids arranged in a single chain. The effect of glucagon is to cause a rise in the blood glucose level by mobilizing the glycogen in the liver, which is broken down to glucose.

The pancreatic hormones are secreted into the hepatic portal vein, so that the liver forms a barrier between the hormones and body cells. The liver destroys glucagon before it can get into the body circulation and its metabolic action is exerted solely upon the liver cells. Therefore the ability of the liver to destroy the hormones has resulted in failure of its effect on muscle glycogen.

(C) SOMATOSTAMIN :-

The same substance are growth inhibiting hormone from the hypothalmus is