glycemic antidiabetic factor
ich regulates blood glucose
characted insulin in the
el noble prize in 1923.
alline insuline in 1926.
ly small protein of 51 amino
rallel chains A and B, inter-
ptide bonds A chain has
amino acid units, this
has 51 amino acids in all.
6,000

ectors

sugar in tissues. Insulin
ylation of glucose by the
xokinase Glucose-6-phosphat
atory cycle to liberated

ucose into cell.
n of glycogen from glucose
and muscles
on of glucose from non-
bs protein and fats
e bodies (Antiketogenic effect)
n inadequate fat
, because fat must be
ed for release of energy
n. The large increase in

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 retension, bone formation etc. Increases the incorporation of amino acids into peptides.

6) Decreased lipoemia i.e 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 (increased apetite) 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