

Pancreatic hormones

Pancreatic cells with endocrine activity are of endoderm origin. They create specific formations in the tissue of the pancreas - **islets of Langerhans**.

Typy buněk

main

- A-cells (25%)
- B-cells (60%)
- D-cells (10%)

PP-Cells (F-cells)

Hormones

Functions of pancreatic hormones

They maintain a constant value of glycemia in the body. During starvation, stress or during physical exertion, they mobilize energy reserves, and in case of increased glycemia, they take care of its removal from the bloodstream. They stimulate growth.

Insulin

It is produced by the **B-cells** of the pancreas. From a chemical point of view, it is a **peptide** made up of 51 AMK.

Scheme of formation: preproinsulin → proinsulin → insulin. Insulin secretion has a **pulsatile character** and its main stimulus is an increase in glycemia. Stimulation of secretion takes place as follows: increase in plasma glucose value → increase in glucose value in the B-cell → its oxidation increases and thus the amount of ATP in the cell → energy-controlled K⁺ channels close (depolarization occurs) → voltage-controlled Ca²⁺ channels open → the increased amount of calcium cations in the cell induces insulin exocytosis and reopening of K⁺ channels. Insulin secretion is stimulated during digestion by numerous factors:

- **cholinergic fibers n. X;**
- **gastrin** – formed in the antrum of the stomach and duodenum; its function is secretion of HCl and growth of gastric mucosa;
- **secretin** – formed mainly in the duodenum; gastrin antagonist, stimulates the flow of bile in the liver;
- **GIP (glucose-dep. insulinotropic peptide)** – formed in the duodenum and jejunum (stimulus for release are the fission products of nutrients – glucose!); stimulates the release of insulin (after glucose administration, more insulin is released!), suppresses acid secretion;
- **GLP1 (enteroglucagon).**



insulin pen

Insulin output is also increased by AMK present in plasma (Arg, Lys), free MK and some pituitary and steroid hormones. On the contrary, they reduce adrenaline and noradrenaline, galanin and somatostatin. A decrease in the level of glucose (starvation, long-term physical exertion) stimulates CNS chemoreceptors and subsequently sympathetic activation occurs. Insulin **lowers glycemia** (sugar concentration in the plasma), promotes the formation of fats and storage of glycogen as a reserve in the liver, where it induces glycolysis and glycogenesis and suppresses gluconeogenesis. Normally, about 2/3 of the amount of glucose is resorbed, which is then used interdigestively (mobilization by glucagon – enough energy independent of food intake). Furthermore, insulin is used to store AMK (in the form of proteins) mainly in skeletal muscles (**anabolism**), stimulates growth and **lipogenesis** and affects potassium distribution.

Hyperinsulinism results in hypoglycemia and can even cause hypoglycemic shock (coma) (see Hypoglycemia). **Hypoinsulinism** leads to hyperglycemia and subsequently to the development of DM (see Diabetes mellitus).

By overeating, the capacity to store glycogen will be exceeded, so the liver turns it into MK and it is stored in adipose tissue in the form of triacylglycerols.

Glucagon

Glucagon is a **peptide hormone** (made up of 29 AMK) produced by **A-cells**. It arises from **proglucagon**, is stored in granules and released exocytotically. The stimulus for secretion is hypoglycemia, the presence of AMK (Ala, Arg) and sympathetic stimulation. Secretion is inhibited by the presence of glucose, somatostatin and increased concentration of MK. It is an **insulin antagonist**. Maintains glucose levels between food intake and high glucose consumption. It supports glycogenolysis in the liver, gluconeogenesis from lactate, AMK and glycerol (lipolysis). An increased concentration of AMK in the plasma promotes insulin secretion → hypoglycemia, therefore, when AMK is administered, glucose must also be given so that AMK is not used as an energy source.

Somatostatin

It is a **peptide** composed of 14 AMK, produced by the **D-cells** of the pancreas, also in the stomach and intestine, especially after a meal (reacts to increased amounts of glucose and Arg). It reduces the production of HCl, slows down the digestion process. Paracrine suppresses insulin production. It is a gastrin antagonist, inhibits glucagon (stops storage and reduces glucogenesis) and the overall exocrine action of the pancreas.

Outside the gastrointestinal tract, somatostatin is produced in the hypothalamus and subsequently suppresses the secretion of somatotropin in the pituitary gland (hence its name). Therefore, agonists on somatostatin receptors are used, for example, in the therapy of acromegaly (octreotide)^[1].

Pancreatic polypeptide

It is formed by 36 AMK mainly in the head of the pancreas by **PP-cells**. Its role is to autoregulate the endo and exocrine function of the pancreas and stimulate the production of gastric juices. Secretion increases during starvation, physical exertion and acute hypoglycemia, on the contrary, it decreases under the influence of somatostatin and after administration of glucose.

Links

Related Articles

- Pancreas
- Diabetes mellitus
- Insulin
- Glucagon

Used literature

- SILBERNAGL, Stefan – DESPOPOULOS, Agamemnon. *Atlas fyziologie člověka : 186 barevných tabulí*. 6. edition. Praha : Grada, 2004. 448 pp. pp. 282,284. ISBN 978-80-247-0630-6.
- WIKIPEDIA EN. *Pancreatic Polypeptide* [online]. ©2006. The last revision 2014-01-08, [cit. 2014-02-16]. <wikipedia:en:Pancreatic polypeptide>.

1. HERDEGEN, Thomas. *Kurzlehrbuch Pharmakologie und Toxikologie : 328 Tabellen*. 2.. edition. Thieme, 2010. 535 pp. pp. 225. ISBN 9783131422927.