Т. 10. №9 2024
https://doi.org/10.33619/2414-2948/106

UDC 614(083)
AGRIS M40
https://doi.org/10.33619/2414-2948/106/29

HORMONAL REGULATION OF THE AMOUNT OF SUGAR IN THE BLOOD CONVERTER

©Seyidova L., *ORCID: 0009-0002-2206-7823, Nakhchivan State University, Nakhchivan, Azerbaijan, leylaseyidova2012@gmail.com*

ГОРМОНАЛЬНАЯ РЕГУЛЯЦИЯ КОЛИЧЕСТВА САХАРА В КРОВИ

©Сеидова Л. М., *ORCID: 0009-0002-2206-7823, Нахичеванский государственный университет, г. Нахичевань, Азербайджан, leylaseyidova2012@gmail.com*

Abstract. Although hormones differ in their place of origin, order of distribution, chemical composition and nature of action, they are united under a very important general biological pattern: they participate in metabolism, morphological differentiation of the organism, growth and development, reproduction and regulation and coordination of its basic functions. The nervous system carries out the connection and influence between different parts of the organism directly through hormones. Hormones are substances of a protein nature or steroids that act in extremely small quantities. Their effect is carried out in 2 ways: through the nervous system and directly through the blood to the organs.

Аннотация. Хотя гормоны различаются по месту их образования, по способу распространения, по химическому составу и характеру действия, они объединены очень важной общей биологической закономерностью: они участвуют в обмене веществ, морфологической дифференцировке организма, его росте и развитии, размножении, а также в регуляции и координации его основных функций. Нервная система осуществляет связь и действие между различными частями тела непосредственно через гормоны. Гормоны — это вещества белковой природы или стероиды, которые действуют в довольно небольших количествах. Их действие осуществляется 2 способами: через нервную систему и непосредственно к органам через кровь.

Keywords: insulin, hormone, diabetes, blood, islets of Langerhans.

Ключевые слова: инсулин, гормон, диабет, кровь, острова Лангерганс.

Hormones of pancreatic tissue, such as insulin and glucagon, form the antagonist system of regulating the amount of glucose in the blood [1]. The concentration of glucose in the blood plasma fluctuates within the limits of 80-120 mg in a healthy person.The intake of foods rich in carbohydrates results in an increase in the concentration of sugar in the blood. Part of the glucose enters the bloodstream, where it is converted into reserve glycogen. A higher sugar content in the blood than normal is perceived by the β cells of the Islets of Langerhans as a signal for the secretion of insulin. Since the main activity of insulin consists in a sharp multiplication of the degree of absorption of glucose by tissues, during the transition of glucose from the blood to the cell of skeletal muscles and other tissues of other activity, the concentration of sugar in the blood decreases, thereby restoring the normal amount of glucose in the blood plasma [2].

Т. 10. №9 2024
https://doi.org/10.33619/2414-2948/106

A decrease in the amount of glucose in the blood results in the activation of α-cells of glucagon-producing islet tissues. The activity of glucagon consists in accelerating the process of converting glycogen into glucose, that is, in a compensatory increase in the amount of sugar in the blood. An increase in the amount of sugar in the blood slows down the secretion of glucagon [3, 4].

Thus, the islet tissue of the pancreas becomes an effective regulator of the amount of sugar in the blood thanks to hormones that act antagonistically, responding to both an increase and a decrease in the amount of sugar in the blood [5].

Discussion and conclusions of the study

The human body takes 70% of the energy to continue their life and activities throughout the day to the fragmentation (oxidation) of carbohydrates. Carbohydrates taken into the body through food are subjected to the following changes: a) polysaccharides are broken down into monosaccharides in the digestive system and are absorbed into the blood from the intestines and transported to organs and tissues; b) in the tissues, monosaccharides burn (oxidize) and generate energy; c) the last products of oxidation are removed from the body [6].

Glucose, which is absorbed into the blood from the intestines, enters the portal vein, is converted into glycogen from one part, and reserves are collected in the liver. Other parts enter the bloodstream and go to the tissues. It should be noted that liver control has an exceptional role in the normal success of blood glucose. The surplus of glucose in the blood is converted into glycogen in the liver and stored as a reserve. Special signals from the brain to a person whose glucose level is falling below the normal level quickly convert the stored glycogen into glucose and raise the high level of glucose in the blood, thus preventing hypoglycemia [7].

This mechanism works more during sleep or when the body is hungry for a long time. There is also glycogen in the muscles, but the experiment proved that the breakdown mechanism of this glycogen starts late and does not prevent hypoglycemia, the glucose entering the liver is converted into glucose-6-phosphate under the action of the enzyme hexokinase and ATF. The activated G6F participates in either glycogen synthesis or glycolytic oxidation, depending on the conditions. Thus, G6F is converted into glucose-1-phosphate G1F by the action of the phosphoglucomutase enzyme, which is directly involved in glycogen synthesis [8].

The split of the glycogen is mainly in phosphorid. At this time, with the help of the fossorilase enzyme in the liver, it is converted to glucose-6-monophosphates, and then glucose-1 monophosphate. This is divided into free glucose and phosphate acid with the participation of the liver phosphathaza. However, in the liver, the brain and the muscles, the glycogenic phosphoridic decomposition, there is a hydrolytic division. But this is a very slow process [9].

In addition to this, the other factor that helps to keep glucose level in the blood in the blood is the hormones of adrenaline and Noradrenaline secreted in the brain article of adrenal glands. The adrenaline liver fossorilinates the activity of the phosphorilaz, and creates a divorce of glycogen and hypergliklemia. The adrenaline, which includes large amounts of ghane in case of irritation or stress in the central nervous system, causes hypergliamia [10].

The breakdown of glycogen becomes mainly by phosphorolytic way. At this time, with the help of the phosphorylase enzyme in the liver, it is converted into glucose-6-monophosphate, and then glucose-1-monophosphate. This is broken down into free glucose and phosphoric acid with the participation of liver phosphatase. However, in addition to the phosphorolytic breakdown of glycogen in the liver, brain, and muscles, there is also a hydrolytic breakdown pathway. But this is a very slow process [11].

Apart from what has been said, another factor that helps to keep the level of glucose in the blood normal is adrenaline and noradrenaline hormones secreted in the brain substance of the

Т. 10. №9 2024
https://doi.org/10.33619/2414-2948/106

adrenal glands. Adrenaline increases the activity of liver phosphorylase, causing breakdown of glycogen and causing hyperglycemia. Adrenaline, which enters the blood in large quantities during the irritation of the central nervous system or during stress, causes hyperglycemia in the above-mentioned way [13].

Very strong positive or negative emotions, as well as arousal that has arisen in the cerebral cortex during stress, awaken the sympathetic nervous system, which, in turn, leads to the release of large amounts of adrenaline and noradrenaline from the adrenal glands and sympathetic ganglia. Such cases increase the activity of phosphorylase in the liver, causing the breakdown of glycogen accumulated in this organ, and thus hyperglycemia occurs. Such emotional hyperglycemia is a biological reaction aimed at ensuring the intensive activity of the brain and other organs during other intensive mental or physical work during stress.

The resulting hyperglycemia, on the other hand, causes an insulin response. And this regulates the absorption of glucose. In diabetes mellitus, not only carbohydrate, but also fat and protein metabolism are very disturbed, which leads to hyperaminoacidemia and hyperlipidemia. The latter, in turn, leads to ketoacidosis. Hyperlipidemia is characterized by an increase in the total amount of cholesterol, triglycerides, free fatty acids. In particular, it is characterized by an increase in low and very low density lipoproteids, and a decrease in the level of high density lipoproteids. And the excessive entry of fats into the liver leads to an increase in ketone corpuscles. The pH of the blood changes towards acidosis. And in conditions of acute insulin deficiency, this circumstance can lead to diabetic ketosis, that is, to ketoacidotic coma [14].

Currently, diabetes is one of the most widespread diseases of the endocrine system. So, after cardiovascular diseases, traumas, malignant tumors, diabetes mellitus is more common. Very important research has been carried out in the pathogenesis and treatment of diabetes mellitus, and although successes have been achieved, this disease is growing rapidly all over the world.

Blindness, which occurs in connection with the development of diabetic retinopartia, is many times more common than blindness caused by other eye diseases. One of the main causes of blindness all over the world is diabetes mellitus.

Diabetes can develop gradually or acutely. Sometimes it proceeds for a long time secretly, without giving any symptoms. But stressful situations in life, mental and physical trauma, pancreatitis, pregnancy, obesity lead to the transition of the disease from a hidden picture to an obvious one. Experts of the World Health Organization recommend using the term "prediabetes" when carrying out research work:

1. Prediabetes refers to persons with potential diabetes or diabetes (according to Anamnesis surveys), women with a child weighing 4.5-5 kg, twins of the same egg, one of whom has diabetes, the other has potential diabetes or prediabetes. In prediabetes, no identifiable laboratory disorders in carbohydrate metabolism are detected. The sugar load test is the same as in healthy individuals [15].

2. Latent diabetes-in this case, the level of sugar (glucose) in the blood is within normal limits. Sugar is never detected in urine if the permeability of the kidneys to sugar has not changed. However, the sugar load test is positive in patients with latent sugar diabetes. WHO experts call this condition symptomatic and subclinical diabetes mellitus.

3. Overt diabetes mellitus - in this case, all the clinical symptoms typical of diabetes – that is, the 3 "p" symptoms, as they are called in Latin - polydipsia (ingestion of too much fluid), polyuria (urinating too much) and polyphagia (binge eating) are more common. In addition, hyperclicemia (i.e., sugar levels above normal) and glucosuria (excretion of sugar in the urine) are always found in the blood.

Type I diabetes (insulin dependent) is a disease in which beta cells of the pancreas are completely destroyed and fail, and the body does not have enough insulin. As a rule, children, and

Т. 10. №9 2024
https://doi.org/10.33619/2414-2948/106

young people get sick with this disease. That is why this diabetes is often referred to by doctors as diabetes of the young elderly or non-insulin dependent diabetes. That is, from the point of view of pathogenesis neutrality, they divide diabetes into two main ones.

Type I diabetes mellitus in most cases occurs as a result of a genetic predisposition, in particular, as a result of a change in the system of HLA (human lejcocyte antigens – antigen of human leukocytes). That is, after that, the sensitivity of beta-cells of the pancreas to viral antigens increases, and this sensitivity leads to the formation of compounds that lead to their (beta-cells) breakdown. This causes absolute-relative insulin deficiency in the body of patients (Type I diabetes mellitus). After that, clinical signs of diabetes mellitus develop.

With Type I diabetes mellitus, the beta cells of the pancreas in the body do not produce insulin in the required amount or completely, resulting in a lack of insulin. The only method of treatment for Type I diabetes mellitus is a daily injection of insulin and, of course, a strict diet and proper nutrition regimen with it.

Type I diabetes mellitus is characterized by the following features:

1. All types of diabetes have the characteristics of genetic predisposition. However, the set of genes that play a role in the development of type I diabetes is not related to the development of type II diabetes.

2. In type I diabetes mellitus, a minimum of 80% of the beta cells of the pancreas are destroyed, which leads to a complete shortage of insulin, making insulin injections inevitable.

3. Type I diabetes develops as a result of an autoimmune process in the body, it is the autoimmune processes that destroy beta cells and the body is deprived of insulin.

4. The autoimmune process in the body and the complete lack of insulin together lead to unstable and severe type I diabetes, which requires constant control of sugar in the blood.

5. Currently, in relation to all 2 types of diabetes, the divisions “thin” and “fat”, “old” and “young” are not relevant. So, all types of diabetes can form in people of any age and any weight. However, unlike type II, the fact that type I diabetes does not depend on nutrition and lifestyle is a fact. In the formation of type II diabetes, however, hypodynamia and overeating are risk factors.

6. The cause of the formation of type I diabetes mellitus is often a viral infection. Insulin treatment of type I diabetes mellitus. Due to the fact that type i diabetes is insulin-dependent, insulin injections in this group of patients are the only means of treatment. Although insulin therapy simulates the physiological effect of insulin, there is currently no alternative treatment to subcutaneous insulin therapy. Under Normal conditions, insulin enters the venous system, from where it passes into the liver, where about half loses its activity, and half appears on the periphery [16].

This happens so quickly that the high level of glycemia is maintained in a very short time, even after eating. The effect of insulin injected subcutaneously is different. It enters the bloodstream late, and later into the liver, and after that, for a long time, the concentration of insulin in the blood remains at a non-physiologically high level. This deficiency of insulinotherapy forces patients to control their own nutrition, physical work, metabolism and other factors affecting the level of glycemia. However, the modern tactics and strategy of insulinotherapy allow the lifestyle of type I diabetes mellitus to be very close to the normal way of life. It is possible to carry it out only by training patients with diabetes mellitus [17].

The need to apply training programs for diabetes has long been recognized. In the early 80s of the last century, diabetologists acquired highly purified insulin preparations, human insulin, improved means for injecting insulin (disposable insulin syringes and syringe pens), methods of Express analysis of glycemia and glucosuria with the help of test papers and individual glucometers. Contrary to expectations, their introduction did not lead to continuous compensation

Т. 10. №9 2024
https://doi.org/10.33619/2414-2948/106

of carbohydrate metabolism and a reduction in delayed complications. Experts came to the conclusion of the need for a new approach that allows you to effectively manage this complex chronic disease by involving the patient himself in active control of diabetes and its treatment. Currently, therapeutic training has been officially adopted by the World Health Organization, which is an important and integral part of the treatment of any type of diabetes.

As for Type I diabetes, this first of all means that the patient must be a very competent insulinotherapist. Insulinotherapy is a generally accepted strategy of patients of the first type.

The generally accepted treatment strategy for patients with Type I diabetes mellitus is intensive insulinotherapy. Intensive insulinotherapy is understood as a mode of multiple insulin injections that simulates the physiological secretion of insulin by beta cells. Obviously, under physiological conditions, the basal secretion of insulin occurs continuously and (regardless of food intake, including at night) makes up one unit every hour.

During physical work, the secretion of insulin is significantly reduced in the norm. To maintain glycemia in the norm at the time of feeding, a significant amount of insulin secretion is additionally required (10-1 units per 2 g of carbohydrates). This complex kinetics of Insulin secretion, relative constant basal and nutritionally varying levels, can be imitated in the following order: before food intake, the patient is given a short-term insulin injection, and basal insulinemia is maintained by long-term insulin injections. Such a method of insulinotherapy is also called basis-bolius insulinotherapy [18].

No matter how intensive insulinotherapy approaches physiological conditions, it should not be forgotten that simple insulin injected subcutaneously does not exactly correspond to the physiological kinetics of insulin alimentary secretion. This insulinotherapy also requires the patient to determine blood glucose one time each day and take this into account when choosing the dose of insulin. As a rule, short-term insulin injections are made 3 times a day before meals, sometimes more. The basal demand for insulin is often provided by two long-term insulin injections. The most common scheme of induced insulinotherapy consists of the following combinations of injections:

Injection of short-and long-acting insulin in the morning (before breakfast);

Afternoon (before lunch) injection of short-acting insulin;

Injection of short-acting insulin in the evening (before dinner);

Long-term insulin injection at night, before sleep.

This scheme is considered a basic scheme, for each patient it may undergo some changes: the amount of short-term insulin may be more or less, depending on any conditions (for example, additional food intake, sudden blood sugar rises, diseases). When we talk about the dose of insulin, it cannot be said that it must be injected in an unchanged order forever. It can be changed frequently depending on the patient's lifestyle. Therefore, theoretically, it makes no sense to calculate the dose for a specific patient in advance (daily dose per 1kg of body weight, day and night requirement, basal and prandial ratio of insulin), these are only average statistical amounts. The only criterion that allows you to correctly determine the dose of insulin is the measurements of glycemia, which are measured by the patient himself [14].

Type II diabetes (non-insulin dependent diabetes mellitus) is a disease characterized by resistance to insulin and accompanied by a relative lack of insulin. During this disease, the amount of insulin in the blood is normal or exceeds the norm. However, the biological activity of insulin becomes weak. If we assume that insulin with an abnormal structure is synthesized. It does not have biological activity, but it is very weak. That is, one or more of the amino acid residues in the A or B-chain of insulin are not located in their usual place in the chain, but in another place [16].

As a result, the biological activity of such insulin changes. As a rule, Type II diabetes mellitus occurs after the age of 40, and insulin is not required to treat patients. Such patients are treated with

Т. 10. №9 2024
https://doi.org/10.33619/2414-2948/106

either diet or sugaring pills. 80% of patients have obesity of varying degrees. Clinical signs develop gradually and are most often aggravated by macroangiopathies (vascular disorders). They are not prone to ketosis. The genetic predisposition in this disease is very great, and it has already been recognized by everyone that this disease is genetic.

In Type II diabetes, the probability that close relatives (blood relatives) will get sick with this disease is 50%, while in Type I diabetes this figure is 10%. In general, 80-90% of people with diabetes all over the world have Type II diabetes, and the remaining percentage is Type I diabetes.

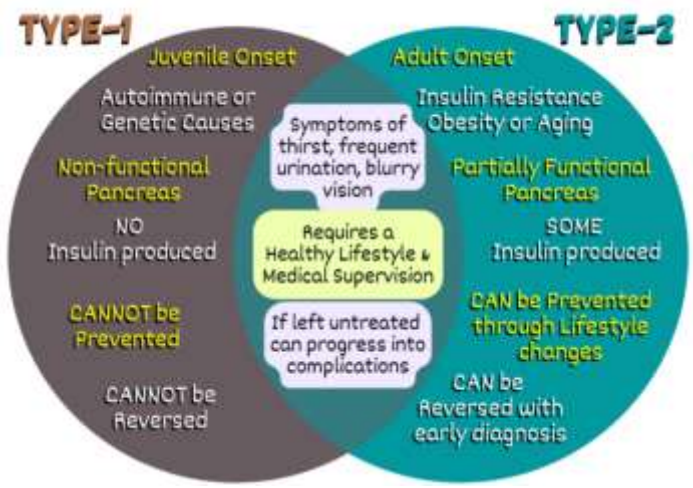

Figure. Differences & Similarities between Types 1 & 2 Diabetes Mellitus

If one of the fraternal twins gets sick with type II diabetes, the probability of the other getting sick is 95-100%.

Type II diabetes is often polyethiologically and polyphogetic. It transmits a genetic tendency to the disease in certain forms of genes. However, obesity, proper nutrition, hypodynamia and stress cause this disease. According to the American scientist L.Salans, the father is sick of diabetes, 50% of the probability of being discouraged, the same is 35%.

In some ethnic groups, Type II diabetes are many and in some cases there is no degree. II diabetes are not found in a number of diabetes in a number of diabetes II of the New Guinea (Papua). These people do not treat it because there are no II diabetes.

In general, there are a number of genetic factors that cause diabetic II diabetes. They are obesity, age, many carbohydrated foods, hypodinamia, pregnancy, protein foods, sufficient foods, stress.

Table

Symptoms	*Type I*	*Type II*
The patient's age when the disease begins	Up to 40	More than 40
Starting	sharp	gradually
Body weight	Less than the norm	normal or more than normal (obesity)
Gender	men get very sick	women get sick more than men
Clinical signs (polyuria, polydipia)	very and clearly	in a moderate degree
Ketoacidosis	Happens	usually not
Acetone (in the blood and urine)	Increases	it does not increase in the blood

Т. 10. №9 2024
https://doi.org/10.33619/2414-2948/106

Symptoms	*Type I*	*Type II*
		and is not determined in the urine
Glucose (in the blood and urine)	Increases	Increases
Relationship with chapters	usually occur in autumn-winter months	Does not happen
The amount of insulin and S-peptide in the blood	The amount of insulin and S-peptide decreases	it becomes the norm or hyperinsulinemia, the amount of S-peptide is in the norm or more than the norm
Signs of inflammation in the pancreas	Happens	Does not happen
Antibodies to the islet apparatus	is found	not detected
Treatment	diet, insulin	diet, sugar-sulfanilamide or biquanid drugs (rarely insulin)
Acute complications (comas)	Happens	Does not happen
Chronic complications	microangiopathies(most commonly retinol and nephropathies)	macroankiopaties

Comparative diagnosis of Type I and II diabetes mellitus

Pathogenesis of type II diabetes II is directly related to insulin. The insulin resistance causes the strengthening of the insulin secretion and the resulting hyperinsulinema. The hyperinsulinemia decreased insulin receptors in the cell membrane, which in turn causes the increase in resistance to insulin. Reduces the insulin synthesis of beta cells. As a result, there is a lack of relative insulin. For this reason, hypocrise causes macroankiopathy [12].

Hyperinulism is a disease that is going on with the hypersecretary of insulin and causes hypoglycemia. The most insulinomas in the etiology of the disease, that is, the benign tumors of beta cells (adenoma) or cancer play a key role. They divide their hyperinsulate and in two places with secondary and secondary. The primary hyperination is associated with tumors (pleasant or malignant) of pancreas. Secondary hyperinationism is connected with external factors (diseases of the nervous system or lack of incidence of insulin) [19].

They also call the primary form hypoglycemic disease. The secondary hyperinsulism also call symptomatic hypoglycemia [1]. Harris and V.Oppel was Harris and V.A.Oppel in 1924, which first described the clinic of hyperinationism. However, the task of the adek apparatus was described in 1902 by Nikols during the first time. Inuloma is usually developing in persons between 35-60 years (equally in women and men). In most cases, it becomes benign tumors (adenoma).

In some cases, the small amount of glucalo in the blood of alpha cell damage causes hyperinates.

Secondary hyperinationism can occur in the diseases of the nervous system (especially in neurosis), as a result of injection of hypothalamus, or after stomach resections.

Relative hyperinationism is hungry for a long time (in stenosis of neurotic anorexia or stomach), can occur as a result of rapidly fragmentation and oxidation of carbohydrates.

Factors outside the pancreat are also caused by hyperination. These include: malignant tumors, hepatitis (acute and chronic), hepatitis, hypotire propagandity, hypotire, hypotire, hypotire, hypotire, hypotire.

Hyperination causes glucose levels in the blood, as a result, the brain cells cannot adopt glucose, and the hypoxia begins. In response to them, hypothalamus is irritated and the hyperinct of the katexolamines from the adrenal glands begin. That is, the body evaluates this situation as stress and the hypothalamo-pituitary and the rigger system is activated. The spasm of peripheral arteries and brain vessels occurs from the impact of cathexolamines, and the brain vessels are changing and

Т. 10. №9 2024
https://doi.org/10.33619/2414-2948/106

the brain's edema occurs. The disease goes with hypoglycemia grips. When the patients begin to blow, the blaze of the body before the patients occurs when the hypoglycemia occurs. Patients are symptoms such as heartbeat, sweating, headache, dizziness. Patients are psychic awakening during grip.

Conclusions

1. Insulin is extracted in beta cells. They are 3.5-4 times more than alpha cells in the gland. Beta cells are easily solved in alcohol. In large-scored cattle, 150 mg of insulin in the pancreas. In a day, the blood secretes about 2 mg of insulin in a day.

2. Increased insulin glucose increases its reputation to cells, its muscular tissues, intensify water stability in tissues, intensifying the synthesis of proteins, weakens the synthesis of oils from proteins and carbohydrates. As a result of insulin's influence, the amount of muscle cells and neurons in the form of a glucose to prevent glucose in the blood.

3. A sharp hypoglycemic view is formed after insulin is injecting a healthy animal body. The amount of glucose in the blood falls, respiratory instances, as a result of waking up the centers of the centers, there are general awakening, to be crumpled. These manifestations of insulin hypoglycemia lose the glucose solution soon. In other words, insulin affects the exchange of the carbohydrate as contrary to the adrenaline. Thus, both of these hormones regulate the relative stability of glucose in the blood. The adrenaline accelerates the secretion of insulin, and its accession to Ghana increases the secretion of adrenaline. The long-term application of insulin weakens the activities of Langerhans, and raises the operation of the brainy brain in the adulthood. As a result of poisoning with insulin, those glands lose their lipids. In this case, the hormone reflects the impact of glucaqon in alpha cells.

References:

1. Karaev,A. (1945). Endokrinologiya. Baku. (in Azerbaijani).
2. Hasanov, R. M. (2008). Endokrinologiya. Baku. (in Azerbaijani).
3. Gaiton, A. K., & Kholl, D. E. (2008). Meditsinskaya fiziologiya. Moscow. (in Russian).
4. Yakovlev, V. N. (2005). Normal'naya fiziologiya. Voronezh. (in Russian).
5. Ammann, R., & Warshaw, A. L. (1985). Acute pancreatitis: Clinical aspects and medical and surgical management. *Bockus gastroenterology*.
6. Barrett, E. J. (2003). Insulin’s effect on glucose production: direct or indirect?. *The Journal of clinical investigation*, *111*(4), 434-435.
7. Barthel, A., & Schmoll, D. (2003). Novel concepts in insulin regulation of hepatic gluconeogenesis. *American Journal of Physiology-Endocrinology and Metabolism*, *285*(4), E685-E692. https://doi.org/10.1152/ajpendo.00253.2003
8. Caumo, A., & Luzi, L. (2004). First-phase insulin secretion: does it exist in real life? Considerations on shape and function. *American Journal of Physiology-Endocrinology and Metabolism*, *287*(3), E371-E385. https://doi.org/10.1152/ajpendo.00139.2003
9. DeWitt, D. E., & Hirsch, I. B. (2003). Outpatient insulin therapy in type 1 and type 2 diabetes mellitus: scientific review. *Jama*, *289*(17), 2254-2264. https://doi.org/10.1001/jama.289.17.2254
10. Hall, J. E., Summers, R. L., Brands, M. W., Keen, H., & Alonso-Galicia, M. (1994). Resistance to metabolic actions of insulin and its role in hypertension. *American journal of hypertension*, *7*(8), 772-778. https://doi.org/10.1093/ajh/7.8.772

11. Hattersley, A. T. (2004). Unlocking the secrets of the pancreatic β cell: man and mouse provide the key. *The Journal of clinical investigation*, *114*(3), 314-316. https://doi.org/10.1172/JCI22506

12. Kowluru, A. (2003). Regulatory roles for small G proteins in the pancreatic β-cell: lessons from models of impaired insulin secretion. *American Journal of Physiology-Endocrinology and Metabolism*, *285*(4), E669-E684. https://doi.org/10.1152/ajpendo.00196.2003

13. List, J. F., & Habener, J. F. (2004). Glucagon-like peptide 1 agonists and the development and growth of pancreatic β-cells. *American Journal of Physiology-Endocrinology and Metabolism*, *286*(6), E875-E881. https://doi.org/10.1152/ajpendo.00007.2004

14. Mann, G. E., Yudilevich, D. L., & Sobrevia, L. (2003). Regulation of amino acid and glucose transporters in endothelial and smooth muscle cells. *Physiological reviews*, *83*(1), 183-252. https://doi.org/10.1152/physrev.00022.2002

15. Pessin, J. E., & Saltiel, A. R. (2000). Signaling pathways in insulin action: molecular targets of insulin resistance. *The Journal of clinical investigation*, *106*(2), 165-169. https://doi.org/10.1172/JCI10582

16. Russell, D. W. (2003). The enzymes, regulation, and genetics of bile acid synthesis. *Annual review of biochemistry*, *72*(1), 137-174. https://doi.org/10.1146/annurev.biochem.72.121801.161712

17. Shi, Y., Taylor, S. I., Tan, S. L., & Sonenberg, N. (2003). When translation meets metabolism: multiple links to diabetes. *Endocrine reviews*, *24*(1), 91-101. https://doi.org/10.1210/er.2002-0018

18. Thomson, S. C. & Townsend, C. M. (2001). Endocrine Pancreas.

19. Seyidova, L. (2024). The Role of Patiral Hormones in Metabolism. *Bulletin of Science and Practice*, *10*(7), 324-333. https://doi.org/10.33619/2414-2948/104/34

Список литературы:

1. Qarayev A. Endokrinologiya. Bakı, 1945. 250 s.

2. Mammadhasanov R. M. Endokrinologiya. Bakı, 2008. 239 c.

3. Гайтон А. К., Холл Д. Э. Медицинская физиология. М.: Логосфера, 2008. 1296 с.

4. Яковлев В. Н. Нормальная физиология. Воронеж, 2005. 522 с.

5. Ammann R., Warshaw A. L. Acute pancreatitis: Clinical aspects and medical and surgical management // Bockus gastroenterology. 1985.

6. Barrett E. J. Insulin’s effect on glucose production: direct or indirect? // The Journal of clinical investigation. 2003. V. 111. №4. P. 434-435.

7. Barthel A., Schmoll D. Novel concepts in insulin regulation of hepatic gluconeogenesis // American Journal of Physiology-Endocrinology and Metabolism. 2003. V. 285. №4. P. E685-E692. https://doi.org/10.1152/ajpendo.00253.2003

8. Caumo A., Luzi L. First-phase insulin secretion: does it exist in real life? Considerations on shape and function // American Journal of Physiology-Endocrinology and Metabolism. 2004. V. 287. №3. P. E371-E385. https://doi.org/10.1152/ajpendo.00139.2003

9. DeWitt D. E., Hirsch I. B. Outpatient insulin therapy in type 1 and type 2 diabetes mellitus: scientific review // Jama. 2003. V. 289. №17. P. 2254-2264. https://doi.org/10.1001/jama.289.17.2254

10. Hall J. E., Summers R. L., Brands M. W., Keen H., Alonso-Galicia M. Resistance to metabolic actions of insulin and its role in hypertension // American journal of hypertension. 1994. V. 7. №8. P. 772-778. https://doi.org/10.1093/ajh/7.8.772

11. Hattersley A. T. Unlocking the secrets of the pancreatic β cell: man and mouse provide the key // The Journal of clinical investigation. 2004. V. 114. №3. P. 314-316. https://doi.org/10.1172/JCI22506

12. Kowluru A. Regulatory roles for small G proteins in the pancreatic β-cell: lessons from models of impaired insulin secretion // American Journal of Physiology-Endocrinology and Metabolism. 2003. V. 285. №4. P. E669-E684. https://doi.org/10.1152/ajpendo.00196.2003

13. List J. F., Habener J. F. Glucagon-like peptide 1 agonists and the development and growth of pancreatic β-cells // American Journal of Physiology-Endocrinology and Metabolism. 2004. V. 286. №6. P. E875-E881. https://doi.org/10.1152/ajpendo.00007.2004

14. Mann G. E., Yudilevich D. L., Sobrevia L. Regulation of amino acid and glucose transporters in endothelial and smooth muscle cells // Physiological reviews. 2003. V. 83. №1. P. 183-252. https://doi.org/10.1152/physrev.00022.2002

15. Pessin J. E., Saltiel A. R. Signaling pathways in insulin action: molecular targets of insulin resistance // The Journal of clinical investigation. 2000. V. 106. №2. P. 165-169. https://doi.org/10.1172/JCI10582

16. Russell D. W. The enzymes, regulation, and genetics of bile acid synthesis // Annual review of biochemistry. 2003. V. 72. №1. P. 137-174. https://doi.org/10.1146/annurev.biochem.72.121801.161712

17. Shi Y., Taylor S. I., Tan,S. L., Sonenberg N. When translation meets metabolism: multiple links to diabetes // Endocrine reviews. 2003. V. 24. №1. P. 91-101. https://doi.org/10.1210/er.2002-0018

18. Thomson S. C. Townsend C. M. Endocrine Pancreas. 2001, p. 646-661.

19. Seyidova L., The Role of Patiral Hormones in Metabolism // Бюллетень науки и практики. 2024. Т. 10. №7. С. 324-333. https://doi.org/10.33619/2414-2948/104/34

Работа поступила
в редакцию 20.08.2024 г.

Принята к публикации
27.08.2024 г.

Ссылка для цитирования:

Seyidova L. Hormonal Regulation of the Amount of Sugar in the Blood Converter // Бюллетень науки и практики. 2024. Т. 10. №9. С. 287-296. https://doi.org/10.33619/2414-2948/106/29

Cite as (APA):

Seyidova, L. (2024). Hormonal Regulation of the Amount of Sugar in the Blood Converter. *Bulletin of Science and Practice, 10*(9), 287-296. https://doi.org/10.33619/2414-2948/106/29