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Morphofunctional transformation of pancreatic islets in immature rats on the 70-th day of experimental diabetes

Miskiv Vasyl[1], Kogut Anna[2], Zhurakivska Oksana[1], Kulinich-Miskiv Mariana[3], Antimis Olga[1], Dutchak Ulyana[1], Hrechyn Andriy[1], Pertsovich Vasyl[1]

[1] Department of Anatomy, Faculty of Medicine, Ivano-Frankivsk National Medical University, Ivano-Frankivsk, Ukraine.

[2] Student of the 2nd year of «Medicine», Ivano-Frankivsk National Medical University, Ivano-Frankivsk, Ukraine.

[3] Department of Phthisiology and Pulmonology with a Course of Occupational Diseases, Ivano-Frankivsk National Medical University, Ivano-Frankivsk, Ukraine.

Address for correspondence:

Miskiv Vasyl

E-mail: miskivv@gmail.com
+380979217969

Annotation: *in studies conducted on animals using histological and electron-microscopic methods of study, analysis of the peculiarities of the reconstruction of pancreatic islets in 3-month-old rats on the 70-th day of the course of experimental diabetes was carried out. Because this period of diabetes in rats is a critical time to study morphofunctional changes, especially in immature rats, where the endocrine and metabolic systems are still developing and interacting with diabetes-induced changes. Ultrastructural features of the pancreas were studied under a PEM-125 K electron microscope at an accelerating voltage of 75 kV. Microphotography of the sample was performed using a Leica DM 750 light microscope and photographed using a digital SSD camera (Industrial digital camera UHCCD05100KPA-U-NA-N-C-SQ-NA). Morphometry was performed on micropreparations using the Bio Vision 4.1 program in automatic or manual mode, taking into account the magnification of objects. Structural changes at each stage of the study were analyzed in 50 fields of view on an area of 0.1 mm² of the studied sample. The obtained data were evaluated using parametric and nonparametric statistical methods. It was established that on the 70th day of experimental streptozotocin-induced diabetes, the average amount of pancreatic islets per 1 mm2 probably decreases compared to control indicators and is (4.62±0.19) ($p<0.05$). The area of islets decreases to (5956.84±547.35) μm2 ($p<0.001$), the same trend is characteristic of B-cells, the number of which decreased to (19.7±0.41) per 0.1 mm². It is experimentally proven that under conditions of diabetes mellitus there is a morphofunctional transformation of the parenchyma of the islets of the pancreas, manifested by the ultrastructural restructuring of the existing B cells, in which the nuclei change: they acquire small size, blurred contours due to the thinning up to the disappearance of the perinuclear space, a large amount of condensed chromatin in which a significant number of small vesicles is detected. The amount of condensed chromatin directly depends on the number of secretory granules in the cytoplasm – the more granules, the more condensed chromatin and the smaller the size of the nucleus. Thus, the introduction of streptozocin causes irreversible changes in the islets of the pancreas, namely their*

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cellular composition decreases by 21%, which is mainly due to a decrease in the number of Insulin cells. Along with this, existing cells are reconstructed in the direction of increasing their functional activity.

Keywords: Pancreas; Insulin-Secreting Cells; Islets of Langerhans; Blood Vessels; Diabetes Mellitus.

Introduction

According to the International Diabetic Federation (IDF) by 2022, 537 million people in the world suffered from this pathology, of which more than 90% (more than 483 million patients) had type 2 diabetes mellitus (DM). According to IDF forecasts, by 2030 up to 643 million people, and by 2045 every eighth adult (about 783 million people) will live with DM, that is, a 46% increase in incidence is predicted [1]. In Ukraine there are about 1 million patients with DM, so the current situation with this pathology in the world and in Ukraine calls attention to the current state of the problem [2]. Analyzing data from the Center for Medical Statistics of the Ministry of Health of Ukraine, at 2019 in Ukraine registered 207 383 patients with diabetes mellitus (data provided without taking into account the statistics of Crimea and the occupied territories of Donetsk and Luhansk regions), taking insulin preparations (58 954 – type 1 diabetes, 138 563 – type 2 diabetes, 9 886 – pediatric patients) [3]. As a result of the findings of the International Diabetic Federation, the prevalence of diabetes in Ukraine is 7.3%. The percentage of undiagnosed diabetes is 65% [4]. Diabetes is ranked 3rd in prevalence after cardiovascular diseases and cancer. According to World Health Organization, diabetes increases mortality by 2–3 times and shortens life expectancy [5]. The number of people with diabetes (20–79 years of age) in Ukraine is 2 million 325 thousand, of which 1,23 million have been diagnosed. These are the data of the 10th edition of the Diabetes Atlas of the International Diabetic Federation for 2021. It is worth noting that 90% have a type 2 DM. Diabetes is the cause of 6.7 million deaths in 2021, one person every 5 seconds. Approximately 44% of the adult population with diabetes (240 million people) are undiagnosed, almost 90% of them living in low- and middle-income countries. The prevalence of diabetes in the population, on average, is 1-8.6%, and among children and adolescents about 0.1–0.3%, and taking into account the undiagnosed cases, in some countries the prevalence can reach 6% [6]. In Ukraine, more than 1 million patients with DM have been registered, another 2–3 million already have diabetes in the developmental stage, but do not suspect the presence of this disease [7].

Experimental models of diabetes in animals, especially rodents, are widely used to study the pathophysiology of diabetes. For example, a study [8] highlighted that STZ-induced diabetes in rats leads to selective beta-cell damage with subsequent islet atrophy and a marked reduction in islet mass. Day 70 of experimental diabetes in rats is a critical time point for studying morphofunctional changes, especially in sexually mature rats where the endocrine and metabolic systems are still developing. Puberty plays a critical role in how the pancreas and islets respond to diabetic conditions. Sexually mature rats develop an endocrine system that interacts with the changes caused by diabetes. Hormonal fluctuations, particularly those related to growth factors, estrogen, and testosterone, are thought to modulate the progression of diabetes and its effects on the pancreatic islets [9]. found that insulin-like growth factors (IGFs) play an important role in beta-cell survival and proliferation. In sexually mature rats, the absence of a fully developed IGF system can accelerate islet destruction in diabetic patients. Furthermore, [10] observed that sexually mature animals show a delayed compensatory response to insulin deficiency compared to sexually mature animals, suggesting that the hormonal environment affects the rate of islet degeneration.

Analyzing the literature available to us, we can comfirm that the age characteristics of rats obviously affect the degree of islet transformation, probably due to the interaction

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between hormonal development and the diabetic state. Future studies aimed at the role of puberty in the progression of diabetes may help to improve the understanding and strategies for the treatment of juvenile diabetes, and the issue of the course of experimental diabetes, in particular, the state of the endocrine part of the pancreas of immature rats, is poorly studied [11, 12].

The aim of the work was to establish the features of the structure of the pancreatic islets (PI) of the pancreas of 3-month-old rats, and their morphological transformation on the 70-th day of the development of experimental diabetes.

Material and methods

The work was carried out on 25 white nonlinear male rats weighing 40–80g. 3 months of age, kept in compliance with all ethical requirements and in accordance with the provisions of the European Convention on the protection of vertebrate animals used for research and other scientific purposes (Strasbourg, 1986), the Law of Ukraine «On the protection of animals from cruel treatment» of December 15, 2009, Directive of the Council of Europe 86/609/EEC (1986). For the experiment animals were divided into two groups: 1 – intact (8 animals), 2 – experimental (17 animals), in which simulated experimental DM by a single intraperitoneal injection of streptozotocin (SIGMA, USA) in 0.1 M citrate buffer pH 4.5 at a dose of 7 mg per 100 g of body weight [13] with the study of the structure of PI on the 70-th day of the experiment, of which 5 animals served as control. Ultrastructural features of the software were studied under the electron microscope PEM – 125 K with an accelerating voltage of 75 kV. Microphotography of the sample was carried out with the help of the Leica DM 750 light microscope and photographed using the digital SSD – camera (Industrial digital camera UHCCD05100KPA-U-NA-N-C-SQ-NA). Morphometry was performed on micropreparates using the program «Bio Vision 4.1» in automatic or manual mode, taking into account the enlargement of objects. The structural changes at each stage of the study were analysed in 50 fields of vision on an area of 0.1 mm² of PI. The data obtained were evaluated using parametric and non-parametric statistical methods.

Results and discussion

Beginning of hyperglycemia leads to extensive damage to the islets, primarily through the destruction of beta cells. Studies have consistently demonstrated a reduction in the size and number of pancreatic islets in diabetic rats. For example, a study by authors [8] emphasized that STZ-induced diabetes in rats results in selective damage to beta cells, with subsequent islet atrophy and a marked reduction in islet mass.

It was found that the endocrine part of the pancreas of 3-month-old nonlinear rats male is represented by pancreatic islets, formed by accumulation of cells surrounded by thin layers of connective tissue, separating them from the exocrine part of the gland. Such islets have predominantly oval or round shape and uneven contours, the average number of pancreatic islets per 1 mm² of parenchyma is (13,62±0,43), and their diameter in animals of this age group is (39,6±0,48) μm, the area is (6754,05±566,31) μm². PI consist of endocrine cells, which on histological preparations look light against the background of a dark exocrine parenchyma. In the cytoplasm of endocrine cells there is a well-developed protein-synthesizing apparatus which includes: Golgi complex, granular endoplasmic reticulum and secretory granules. According to the properties, endocrine cells are divided into four main types: B cell, A cell, D cell, PP cell (Fig. 1).

By the 70th day, morphological changes in the islets of Langerhans are evident, with loss of beta cells leading to decreased islet size and a reduction in their cellular density. Microscopic analyses [14, 15] demonstrated increased fibrosis and disorganization within the islets, along with infiltration by immune cells, indicating ongoing autoimmune responses and chronic inflammation.

The main mass of PI in rats of this age are Insulin cells, which is 27.4±0.57 per 0.1 mm² (69.4%). They are mostly located in the center of PI, surrounding the course of blood vessels. These cells have a polygonal shape and are darker compared to other endocrinocytes (fig. 1).

At the ultrastructural level, the cytoplasm of B cells contains numerous secretory granules (SGs), which consist of a moderate electron-optic

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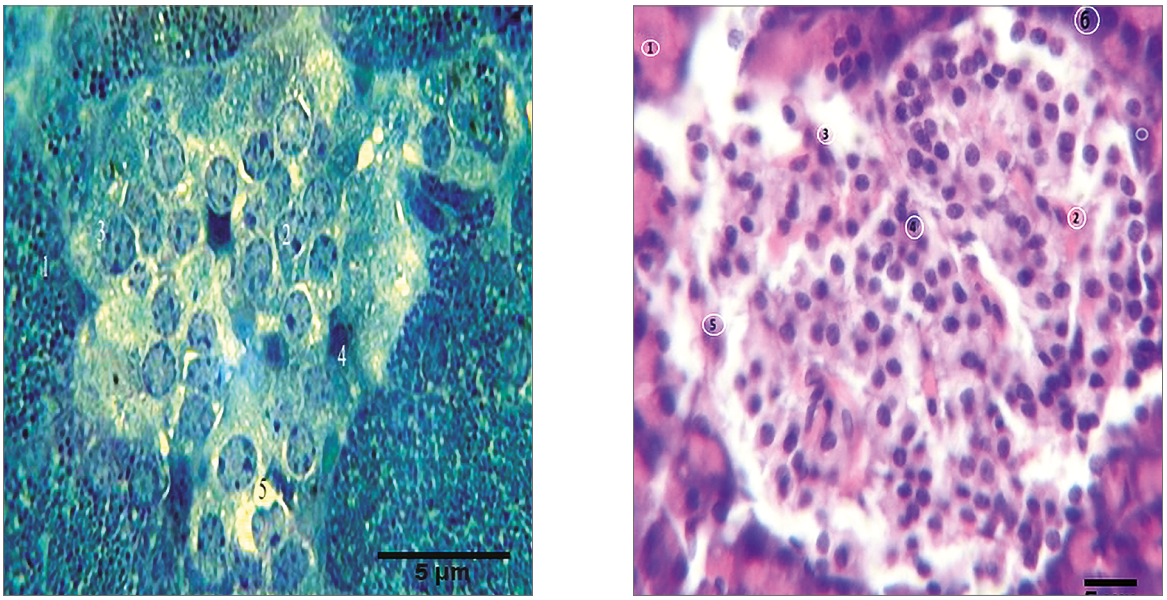

Fig. 1. Cellular composition of the pancreatic islet of a 3-month-old rat. A) Semi-thin cut, stained with toluidine blue. B) Hematoxylin-eosin staining.
1 – exocrinocytes; 2 – B cell; 3 – A cell; 4 – D cell; 5 – capillary of visceral type.

density of the matrix, a membrane and a wide light submembrane curve. In addition, the matrix of the granules is placed somewhat eccentrically. Such granules by the peculiarity we will call in the future secretory granules of the B- type.

Functional changes in insulin secretion The functional capacity of pancreatic islets in diabetic rats is severely impaired. Insulin secretion, the primary function of beta cells, is greatly reduced or completely absent by day 70 of diabetes in many experimental models. Some researchers [16] observed that the remaining beta cells displayed altered insulin granules and impaired insulin secretion in response to glucose stimulation. This finding is supported by other studies [17] who showed that a prolonged hyperglycemic state results in beta-cell depletion with reduced responsiveness to glucose and other insulinotropic stimuli. Glucagon cells make up 25% of the total number of endocrine cells (9.9±0.37) per 0.1 mm² and are localized, as a rule, on the periphery (Fig. 1). Their sizes are smaller than those of insulin cells, and there is less heterochromatin in the nuclei. An imbalance between insulin and glucagon leads to increased hyperglycemia. Some researchers [18] highlighted the role of glucagon in worsening hyperglycemia by promoting hepatic glucose production, thus exacerbating the diabetic state in rats. At day 70 of diabetes, sexually mature rats show a marked increase in the number and size of alpha cells compared to beta cells, indicating a change in islet cell composition. Studies [19] have confirmed alpha cell proliferation in diabetic rat models with increased glucagon secretion, contributing to dysregulation of glucose metabolism. Somatostain cells in young rat islanders are (0,26±0,15) at 0.1 mm², they are star-shaped and are located, mostly, on the periphery of the island, although they can also be found between B cells. Pancreatic polypeptide cells have a polygonal shape, located, mainly, on the periphery of the island between A cells. These cells are characterized by a well-developed granular endoplasmic reticulum and small SGs located along the cytoplasmic membrane. The average number of these cells in PI is (2.1±0.16) per 0.1 mm², that is, 5.2% of the total number of islet cells. On the 70-th day after the simulation of streptozotocin DM in each PI of 3-month-old rats, the decrease in the total number of cells is massive. The average amount of PI per 1 mm² significantly decreases compared to the control indicators and is (4.62±0.19) ($p<0.05$). The area of the islets decreases to (5956,84±547,35) µm² ($p<0,001$), the same trend is typical for B cells, the number of which decreased to (19,7±0,41) on 0.1 mm² of the area of pancreatic islets, number

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of A and PP cells decreasing respectively to (9.3±0,12) and to (3.8±0,11) on 0.1 mm² area of pancreas islands, and the amount of D cells is practically unchanged. Ultrastructurally in existing Insulin cells, the nuclei change: they have small sizes, blurred contours due to the disappearance of the perinuclear space, a large amount of condensed chromatin, in which a significant number of small vesicles are revealed (Figure 2). The content of condensed chromatin directly depends on the number of secretory granules in the cytoplasm – the more granules, the more condensed chromatin and the smaller the size of the nucleus. The organelles in the cytoplasm of such cells are not detected, except for certain fragments of the granular endoplasmic reticulum. Along with this, there are B cells with less SGs and «dark» cytoplasm. In these cells between secretory granules, mitochondria with vague crystals can be detected. The nuclei of such cells are larger, but in the areas of the location of heterochromatin, vesicles are determined, which are primarily detected in the peripheral condensed chromatin. Secretory granules differ from each other in both size and content – there are almost normal structure, separate with a pale core, some empty, there are reduced, there is – enlarged, somewhere defined A type SGs.

On the periphery of the islands in the Glucagon cells are first of all changes in the nuclei. The

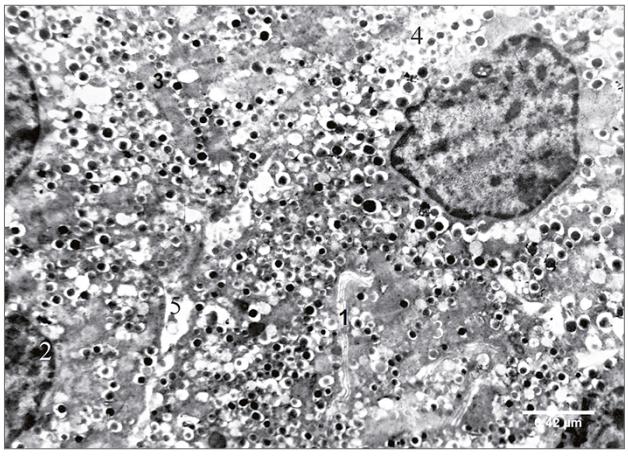

Fig. 2. Ultrastructure of the islet of a 3-month-old rat on the 10-th week of modeling streptozotocin DM
1 – granular endoplasmic reticulum;
2 – B cell nucleus; 3 – B cell cytoplasm; 4 – SGs;
5 – expanded intercellular gap.

structure of which does not correspond to the normal: they lose a rounded shape and acquire an irregular. In a condensed chromatin, the boundaries of which are blurred, vesicles are determined. The secretory granules do not have a clear lining, the illumination zone around the matrix of the secretory material granules is thin, the internal contents have different electron-optic density. SGs are absent around the nucleus and are located mainly on the periphery of the cytoplasm. Intercellular gaps sharply expand with the formation of uneven contours. In the cytoplasm mitochondria with preserved structure are determined.

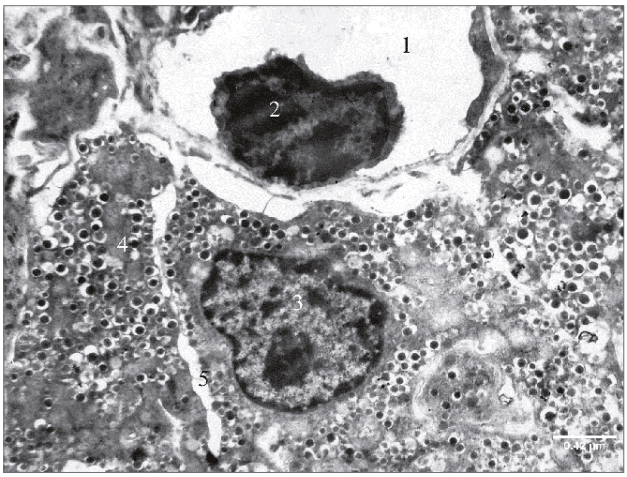

Fig. 3. Ultrastructure of the PI of a non-mature rat on the 70-th day after simulation of streptozotocin DM.
1 – capillary lumen; 2 – endotheliocyte nucleus;
3 – nucleus of A cell; 4 – secretory granules of the B-type; 5 – intercellular gap.

The cells located on the periphery of the islands contain secretory granules of both A-type and B-types, intercellular gaps- expanded.

The extrusion of the content of secretory granules occurs not only in the expanded pericapillary space, but also in locally expanded intercellular gaps (Figure 3).

Conclusion

Therefore, the introduction of streptozotocin causes irreversible changes in the pancreatic islets of the drug, namely their cellular composition decreases by 21%, which is mainly due to a decrease in the number of B cells. Along with this, existing cells are reconstructed in the direction of increasing their functional activity.

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Conflict of interests

The authors declare no conflict of interest in this publication.

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All authors gave their consent for the publication of this manuscript.

ORCID ID and authors' contribution

0000-0002-3924-1544 (A, B, C, D) Miskiv Vasyl

(A, F) Kogut Anna

0000-0002-1041-4237 (B, C, E, F) Oksana Zhurakivska

0000-0001-6091-5438 (B, F) Kulinich-Miskiv Mariana

0000-0001-6040-8137 (D, E, F) Antimis Olga

0000-0002-3715-5650 (E, F) Dutchak Ulyana

0009-0003-8865-135X (C, D, E) Hrechyn Andriy

0000-0002-9491-6292 (C, E, F) Pertsovych Vasyl

A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation, D – Writing the article, E – Critical revision of the article, F – Final approval of article.

REFERENCES

1. Bondareva OO. Clinical and pathogenetic characteristics of acute coronary syndrome in type 2 diabetes: Optimization of prognostic-modifying therapy [dissertation]. Ivano-Frankivsk: Ivano-Frankivsk National Medical University; 2022. Available from: https://www.uacademic.info/ua/document/0824U001338

2. Karabut LV, Matviichuk OP. Diabetes mellitus as a disease of civilization. In: Modern achievements in pharmaceutical technology. Conference materials. Kharkiv: National University of Pharmacy; 2023. p. 115. Available from: https://dspace.nuph.edu.ua/bitstream/123456789/30817/1/115.pdf

3. Aliyev RB. Epidemiology of metabolic syndrome and concepts of its development. Ukr J Med Biol Sports. 2022;7(5):39. https://doi.org/10.26693/jmbs07.05.008

4. Koltunova MO, Podkolzina NV. Organizational approaches to drug provision for patients with type I and II diabetes under the «Available Medicines» program. In: II International Scientific and Practical Distance Conference «Modern Aspects of Drug Development»; 2021. p. 132–133.

5. Burlaka IS, Kudriavtseva TO. Type II diabetes mellitus and dietary fats. In: Modern trends aimed at preserving human health: Collection of scientific papers. Vol. 4. Kharkiv: National University of Pharmacy; 2023. p. 130–134. Available from: https://dspace.nuph.edu.ua/bitstream/123456789/30319/1/Збірник%20статей%20Сучасні%20тенденції%20спрямовані%20на%20збереження%20здоров%27я%20людини%2020-21.04.2023.pdf

6. Lugova D. Modern methods for glucose determination in endocrinological patients [thesis]. Kharkiv: National University of Pharmacy; 2023. Available from: https://dspace.nuph.edu.ua/handle/123456789/29746

7. Savka II, Savka TB. Mechanisms of structural organization changes in organs and their vessels under diabetes mellitus conditions. Ukr J Med Biol Sports. 2020;5(2):36–42. https://doi.org/10.26693/jmbs05.02.036

8. Lenzen S. The mechanisms of alloxan- and streptozotocin-induced diabetes. Diabetologia. 2008;51(2):216–226.

9. Lingohr MK, Buettner R, Rhodes CJ, White MF. The role of insulin-like growth factor I signaling in beta-cell survival and regeneration. J Mol Endocrinol. 2002;29(3):117–131.

10. Hughes SD. Influence of sexual maturity on pancreatic islet response in diabetes. Horm Metab Res. 2000;32(12):528–533.

11. Vodolozhskyi ML, Sydorenko TP, Fomina T-V, Koshman T-V. Current issues and trends in scientific research on «Childhood Diabetes». Endocrinology. 2019;24(2):153–158. https://doi.org/10.31793/1680-1466.2019.24-2.153

12. Nazari A, Mohammadi F, Shafiei E, Najafi R, Abdal K. Cross-sectional study of DMFT index in children and adolescents (9-18 years old) with type 1 diabetes, compared with healthy children in Ilam in 1399. Iran J Pediatr Dent. 2022;17(2):47–54. Available from: http://jiapd.ir/article-1-351-en.html

13. Zhurakivska OYa, Bodnarchuk YuV, Kostiitska IO, Kindrativ YeO, Andriiv AV, Zhurakivskyi VM, et al. Morphofunctional characteristics of the liver in rats at the early stages of streptozotocin diabetes development using cluster analysis. Probl Endocr Pathol. 2021;75(1):84–96. https://doi.org/10.21856/j-PEP.2021.1.11

14. Guvener N. Changes in pancreatic tissue in experimental diabetes mellitus. J Pancreas. 1999;4(3):108–114.

15. Sandikci M, Karagenc L, Yildiz M. Changes in the pancreas in experimental diabetes and the effect of lycopene on these changes: Pdx-1, Ngn-3, and Nestin expressions. Anat Rec (Hoboken). 2017;300(12):2200–2207. https://doi.org/10.1002/ar.23687

ISSN 2786-6661
eISSN 2786-667X

16. Katsumata K, Katsumata Y, Yamada M. Insulin secretion and islet morphology in diabetic rats. Endocrinology. 1992;131(4):1687–1694.

17. Halban PA, Polonsky KS, Bowden DW, Hawkins MA, Ling C, Mather KJ, et al. β-cell failure in type 2 diabetes: Postulated mechanisms and prospects for prevention and treatment. Diabetes Care. 2014;37(6):1751–1758. https://doi.org/10.2337/dc14-0396

18. Prentki M, Nolan CJ. Islet beta cell failure in type 2 diabetes. J Clin Invest. 2006;116(7):1802–1812. https://doi.org/10.1172/JCI29103

19 Kaido T, Yamaoka M, Kanatani Y, Fukuda Y, Kondo T. Alpha-cell hyperplasia in pancreatic islets of diabetic rats. Pancreas. 2004;29(1):30–36.

Морфофункціональна трансформація панкреатичних острівців у нестатевозрілих щурів на 70 добу експериментального цукрового діабету

Міськів Василь[1], Когут Анна[2], Жураківська Оксана[1], Кулинич-Міськів Мар'яна[3], Антимис Ольга[1], Дутчак Уляна[1], Гречин Андрій[1], Перцович Василь[1]

[1] Кафедра анатомії людини, Івано-Франківський національний медичний університет, м. Івано-Франківськ, Україна.

[2] Студент 2 курсу «Лікувальна справа», Івано-Франківський національний медичний університет, м. Івано-Франківськ, Україна.

[3] Кафедра фтизіатрії і пульмонології з курсом професійних хвороб, Івано-Франківський національний медичний університет, м. Івано-Франківськ, Україна.

Address for correspondence:

Miskiv Vasyl

E-mail: miskivv@gmail.com
+380979217969

Анотація: *У дослідженнях проведених на тваринах з використанням гістологічних та електронно-мікроскопічного методів дослідження проведено аналіз особливостей перебудови панкреатичних острівців у щурів 3-х місячного віку на 70 добу перебігу експериментального цукрового діабету. Оскільки саме даний період діабету у щурів є критичним моментом для вивчення морфофункціональних змін, особливо у нестатевозрілих щурів, де ендокринна та метаболічна системи все ще розвивається та взаємодіє зі змінами, викликаними діабетом.*

Ультраструктурні особливості підшлункової залози вивчали під електронним мікроскопом ПЕМ – 125 К при прискорювальній напрузі 75 кВ. Мікрофотографію зразка проводили за допомогою світлового мікроскопа Leica DM 750 і фотографували за допомогою цифрової SSD – камери (Industrial digital camera UHCCD05100KPA-U-NA-N-C-SQ-NA). Морфометрію проводили на мікропрепаратах за програмою «Bio Vision 4.1» в автоматичному або ручному режимі з урахуванням збільшення об'єктів. Структурні зміни на кожному етапі дослідження аналізували в 50 полях зору на площі 0,1 мм² досліджуваного зразка. Отримані дані оцінювали за допомогою параметричних і непараметричних статистичних методів.

Встановлено, що на 70 добу експериментального стрептозотоциніндукованого цукрового діабету середня кількість ПО на 1 мм² вірогідно знижується порівняно з контрольними показниками і становить (4,62±0,19) (p<0,05). Площа острівців зменшується

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до (5956,84±547,35) мкм² (p<0,001), така ж тенденція характерна для В-клітин, кількість яких зменшилася до (19,7±0,41) на 0,1 мм².

Експериментально доведено, що за умов цукрового діабету спостерігається морфо-функціональна трансформація паренхіми острівців підшлункової залози, що проявляється ультраструктурною перебудовою існуючих В-клітин, у яких змінюються ядра: вони набувають невеликих розмірів, нечітких контурів за рахунок витоншування аж до зникнення перинуклеарного простору, великої кількості конденсованого хроматину, в якому виявляється значна кількість дрібних везикул. Кількість коденсованого хроматину прямо залежить від кількості секреторних гранул в цитоплазмі – чим більше гранул, тим більше конденсованого хроматину і тим менші розміри ядра.

Отже, введення стрептозотоцину викликає незворотні зміни в панкреатичних острівцях підшлункової залози, а саме: на 21% зменшується їх клітинний склад, що в основному обумовлено зменшенням числа В-клітин. Поряд з цим існуючі клітини перебудовуються в напрямку підвищення їх функціональної активності.

Ключові слова: підшлункова залоза, В-клітини, острівці підшлункової залози, кровоносні судини, цукровий діабет.