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GISTO-ULTRASTRUCTURAL CHANGES IN THE MUSCLES OF THE ABDOMINAL IN VENTRAL HERNIA AND AFTER PHYSICAL REHABILITATION IN THE POSTOPERATIVE PERIOD AFTER ALLOPLASTY

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The study of histo-ultrastruction changes of different structural components of direct muscle of abdominal of sexually matured rats – males was a research purpose in the remote terms of ventral hernia and subsequent application of the physical loading of middle aerobic power. Light opticaly, electronical microscopical (for the exposure of structural transformations of muscle components) and histochemical (for determination of activity of succinate dehydrogenase after Nahlas – to identify muscular fibres with different phenotypes) methods at 55 sexually matured rats were studied structural track of adaptation of muscle compartmently at prolonged (240 days) ventral hernia and after 15-30 multiple physical loading of middle aerobic power in an ambassador post-operative period at alloplastic. It is shown that above all things, there are the changes of intramuscular network and morfometric changes blood vessels. These information closely correlate with the making progress changes of subcellular components of muscle fibers accountable for power and plastic balance. It is set that fast oxygen-glycolytics muscle fibers and their peripheral nervous vehicle are most sensible to the terms of prolonged ventral hernia. As a result of application of the physical loading of middle aerobic power the reparative regeneration is intensifyed, that substantially abbreviates the terms of renewal of structurally-functional properties of skeletal muscles in the conditions of ventral hernia illness. Thus, at ventral hernia the primary changes touch the sources of blood supply of abdominal muscles, with the secondary paranecrosis processes in muscle fibers and peripheral nervous vehicle with certain morfometric signs.

Key words: ventral hernia, muscle fibers, blood vessels, neuromuscular endings, physical loading.

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ГІСТО-УЛЬТРАСТРУКТУРНІ ЗМІНИ М'ЯЗІВ ЧЕРЕВОЇ СТІНКИ ПРИ ВЕНТРАЛЬНІЙ ГРИЖІ І ПІСЛЯ ФІЗИЧНОЇ РЕАБІЛІТАЦІЇ В ПОСТОПЕРАТИВНИЙ ПЕРІОД ПІСЛЯ АЛОПЛАСТИКИ

Дослідження гісто-ультраструктурних змін різних структурних компонентів прямого м’яза передньої черевної стінки статевозрілих щурів-самців у віддалені терміни після моделювання вентральної грижі та подальшого застосування фізичного навантаження середньої аеробної потужності було метою даної роботи. Використали світлооптичні, електронномікроскопічні (для вивчення структурних перетворень м’язових компонентів) та гістохімічні (для визначення активності сукцинатдегідрогенази за Нахласом - для ідентифікації м’язових волокон різних фенотипів) методи. У 55 статевозрілих щурів вивчали структурний слід адаптації м'язових волокон при тривалій (240 днів) вентральній грижі та після багаторазових (15-30сеансів) фізичних навантажень середньої аеробної потужності в післяопераційному періоді після алопластики. Показано, що перш за все відбуваються зміни внутрішньом’язової кровоносної сітки та морфометричні зміни нервових елементів. Така структурна перебудова тісно корелює з прогресуючими змінами субклітинних компонентів м’язових волокон, що відповідають за енергетичний та пластичний баланс. Встановлено, що швидкі киснево-гліколітичні м'язові волокна та їх периферичний нервовий апарат найбільш чутливі до умов існування тривалої вентральної грижі. В результаті застосування фізичного навантаження середньої аеробної потужності посилюється репаративна регенерація, що істотно скорочує терміни відновлення структурно-функціональних властивостей скелетних м'язів в ділянці вентральної грижі. Таким чином, при вентральній грижі первинні зміни стосуються джерел кровопостачання м’язів передньої черевної стінки з вторинними процесами паранекрозу в м’язових волокнах та периферичному нервовому апараті з певними морфометричними ознаками.

Ключові слова: вентральна грижа, м’язові волокна, судини, нервово-м’язові закінчення, фізичне навантаження

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The problem of surgical treatment of inguinal hernia still remains very relevant and far from the final decision [5]. This is evidenced by the existence of more than four hundred operational techniques. However, none of them preclude the occurrence of a hernia recurrence in the postoperative period [15]. At the present stage of development of herniology, an active search for ways to increase the effectiveness of various methodological approaches to the plasticity of ventral hernias in both experimental and clinical areas. However, the structural and functional changes of neuromuscular endings (NME) in the area of scar tissue formation, especially during its reorganization, as well as the condition of microvascular, connective tissue components and muscle fibers (MF) in the postoperative period remain insufficiently studied [1, 2].

In our opinion, such a number of methods is due to insufficient knowledge of the processes of restructuring of the peripheral nervous apparatus of the abdominal muscles during inguinal hernias. Destructive processes in the abdominal muscles often occur on the basis of previously arisen and long-existing ventral hernia.

The latter is due to the living conditions, characteristics of the profession, age, various diseases, hypokinesia, obesity [1, 2, 9, 12]. It is known that under conditions of a long-term ventral hernia, not only the metabolism of the muscles [3, 4, 9] changes, but also their structure [10, 13]. The search for factors that enhance the physiological regeneration and resumption of muscle fiber function after alloplasty allowed us to establish a positive influence on these processes of metered exercise [6, 12].

The purpose of the study was to establish the nature of the structural rearrangement of the rectus abdominis muscle, which occurs under the influence of the dosed physical load of average aerobic capacity after the long-term existence of experimentally simulated ventral hernia rats.

Material and methods. The experiment was carried out on 55 adult Wistar male rats. In the first series of the experiment, a ventral hernia was simulated (240 days) [15]. The structure of the rectus abdominis muscle was investigated. Material sampling was performed after 90, 180 (5 animals in each study period) and 240 days (15 animals). The control group consisted of 15 intact animals, which were kept in standard vivarium conditions and 5 animals were removed from the experiment at the times indicated above to maintain the purity of the experiment with respect to age-related changes.

In the second series of the experiment, 10 animals that had previously withstood the conditions of an experimental ventral hernia for 240 days exercised the moderate aerobic power exercise in a treadmill (daily workouts for 15 minutes at a running speed of 20 m/min. After which they were removed from the experiment after 15 and 30 days (5 animals in each term).

The control was carried out with the results obtained in 5 animals of the previous group, i.e. after 240 days of existence of an experimentally simulated ventral hernia. All studies were carried out in accordance with the "Rules of work with the use of laboratory animals."

The material for histological examination was treated with hematoxylin and eosin according to the standard technique. For the study of muscle tissue according to Bilshovsky-Gross, pieces of 0.5x0.5x0.5 cm in size were fixed in a liquid consisting of equal parts of 96.0 % alcohol, neutral formalin and a saturated solution of arsenic for 1 hour.

Then it was transferred without washing to 12.0 % neutral formalin for 10 days. After this, the pieces were rinsed in distilled water and cut in a freezing microtome (MCP-4, PO "HZMT", Kharkov, Ukraine).

Slices of 25 μm thick were transferred to a 20.0 % silver nitrate solution, where their residence time was established empirically and the procedure of impregnation was continued on separate sections. Then the sections were quickly carried out through 4–5 trays filled with 20 % formalin, prepared on tap water, and placed into ammonia silver solution.

A small part (of a 20.0 % silver nitrate solution) was titrated in a watch glass with a drop of 25.0 % ammonia solution until the precipitate disappears. In this solution, sections were impregnated under the control of a microscope. At the end of the impregnation, they were placed for 10–15 minutes into ammonia water, and then for several hours into distilled water for washing. The resulting sections were mounted on slides and covered with polystyrene.

For electron microscopic examination, the material was prepared by the conventional method [8]. Ultrathin sections were obtained on a UMTP-6M ultramicrotome (SEELMI Ukraine software), contrasted using the Reynolds technique and viewed with a PEM-125 K electron microscope (PO SELMI Ukraine software) with an accelerating voltage of 75 kV followed by photography at magnifications from 1200 to 20000 times.

Histological slides were studied under a MS-300 light microscope (TXR, Austria) and photographed using Digital camera for microscope DCM 900 (TXR, Austria).

Morphometry was performed in all animals on 20 histological slides for each study period using the NIH USA "Image J" software in automatic or manual mode with magnification resolution. Structural changes at a certain stage of the study were analyzed in 50 fields of view and the total number of muscle fibers per 1 mm^2 of cross-sectional area, the thickness of muscle fibers, the absolute and relative number of hemocapillaries, as well as the diameter of their lumen, the (fraction of connective and adipose tissue, the number of fibroblasts and macrophages.

Statistical data processing was performed using the software package "Statistica 6". Probability was determined by the criterion for assessing the significance of the difference using the probability factor (Student's criterion). The difference was considered statistically significant at $p<0.05$.

Results of the study and their discussion. Data on changes in the morphometric parameters of muscle fibers in different periods in the simulation of ventral hernia and after exercise of the average aerobic capacity are presented in table 1.

90 days after the start of modeling of the ventral hernia in the endo- and perimisation, the phenomena of intense edema are observed, a pronounced proliferative cellular reaction in the tissues that

surround the neurovascular bundles. In comparison with the animals of the control group, the (fraction of stromal components increases by 1.5 times ($p<0.05$). The nuclei of muscle fibers are localized near the central part of the fiber, the expansion of cisterns of the sarcoplasmic reticulum is noticeable, individual cristae in mitochondria are reduced. In myofibrils, the correct location of the Z-lines is violated.

Table 1

Dynamics of changes in the morphometric parameters of the rectus abdominis muscle with prolonged ventral hernia and after dosed physical exertion (M±m, n=55)

Indices	Control group 1 n=15	When modeling a ventral hernia, days			When modeling a ventral hernia + exercise stress, times	
		90 n=5	180 n=5	240 n=5	15 n=5	30 n=5
The number of muscle fibers per 1 mm^2 cross-sectional area	997.0 ±36.73	345.0 ±23.12*	141.0 ±31.24*	97.0 ±10.33*	374.0 ±14.46♦	466.0 ±21.37♦
The absolute number of hemocapillaries per 1 mm^2 cross-sectional area	82.0 ±4.52	42.0 ±3.26*	21.0 ±2.51*	16.0 ±1.42*	45.0 ±3.15♦	52.0 ±4.67
The relative number of hemocapillaries	12.1 ±0.22	8.2 ±0.46	6.7 ±0.33*	6.1 ±0,25*	8,3 ±1.07♦	9.0 ±1.98
Fraction of connective tissue, %	8.1 ±0.15	14.2 ±0.84*	18.3 ±1.17*	25.2 ±2.74*	194 ±0.72	8.1 ±0.25♦
The number of fibroblasts per 1 mm^2 cross-sectional area	5.2 ±0.14	10.1 ±0.67*	17.4 ±0.72*	26.3 ±3.11*	12.5 ±1.41♦	12.3 ±0.87
The number of macrophages per 1 mm^2 cross-sectional area	2.1 ±0.17	5,1 ±0.33*	6.4 ±1,02*	5.2 ±1.66*	15.3 ±1.81♦	7.2 ±0.64♦
Fraction of adipose tissue, %	4.4 ±0.08	15.2 ±0.54*	19.6 ±1.74*	24.1 ±1.53*	12.2 ±1.04♦	4.1 ±0.17♦

Notes: * – $p<0.05$ – significant difference with the control group; ♦ – $p<0.05$ – significant difference between the indices of the previous observation group.

In comparison with animals of CG, the sarcoplasm of most muscle fibers has a low electron-optical density, contains an increased number of pinocytotic vesicles, an increase in the number of secondary lysosomes, residual bodies and autophagosomes (fig. 1).

An analysis of electron micrographs revealed that 180 days after the start of modeling of a ventral hernia compared with the control and the previous group of animals, intracellular edema in the muscle fibers increased, leading to myofibril fibrillation (fig. 2).

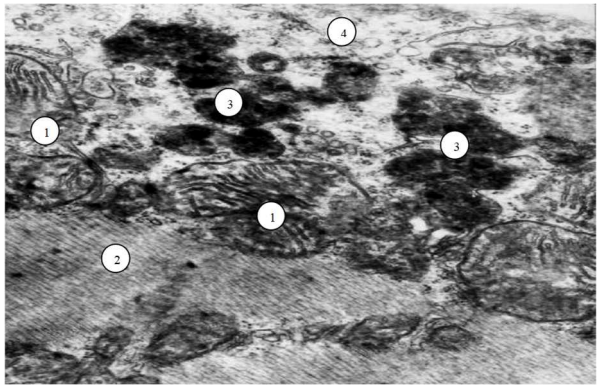

Fig. 1. Concentration of autophagosomes and residual bodies in the area of local destruction of muscle fiber 90 days after the start of modeling of the ventral hernia: 1 – mitochondria, 2 – myofibrils, 3 – residual bodies, 4 – sarcoplasma. Magnification: x 12,000.

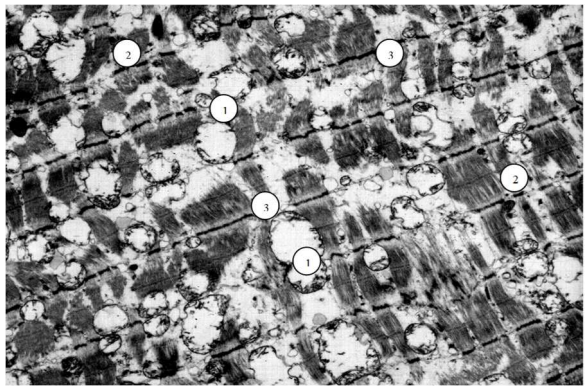

Fig. 2. Ultrastructural organization of muscle fiber 180 days after the start of modeling of the ventral hernia. There is edema of most mitochondria with shortening and fragmentation of the cristae, which indicates a deep disruption of the processes of intracellular metabolism: 1 – mitochondria, 2 – myofibrils, 3 – Z – line. Magnification: x 6000.

A typical phenomenon characteristic of a ventral hernia is the formation of myelin-like bodies and the vacuolization of sarcoplasm. Mitochondria increase in size, their matrix has a low electron – optical density, the cristae are disoriented, shortened, fragmented. Lysosomes are concentrated mainly in the areas of destruction of myofibrils. An increased number of inclusions of various electron-optical density is observed in the sarcoplasm.

240 days after the start of modeling of the ventral hernia, there is a decrease in edema and an increase in the specific proportion of the stromal component of the muscles. At the same time, an increased number of macrophages, fat cells and fibroblasts with a corresponding increase in the number of coarse collagen fibers, which are detected in the form of large bundles parallel to the muscle fibers and the

longitudinal axis of the blood vessels, is detected in the connective tissue skeleton of the rectus abdominis muscle. For most muscle fibers, destructive – atrophic and paranecrotic phenomena are characteristic. Muscle fibers become thinner, lose cross striation, sometimes form local thickenings. During electron microscopic examination during this period of the experiment, destructive lesions have a diffuse character. Muscle fibers decrease in diameter, the phenomena of their lysis are often observed. In such zones, the number of autophagos and residual bodies increases.

The nuclei of muscle fibers have irregular contours, an illuminated nucleoplasm with chromatin marginalization phenomena. Mitochondria with an enlightened matrix, fragmented and sharply reduced cristae, sometimes there is a destruction of the outer membrane, which leads to a decrease in the activity of succinate dehydrogenase in fast oxidative – glycolytic fibers.

Physical exercise of average aerobic capacity after a long – term existing ventral hernia gives a pronounced and fast recovery effect. So, after 15-fold exercise, the number of loci of muscle tissue destruction significantly decreases. The degree of manifestation of this destruction also changes: paranecrotic areas practically do not occur, only refined, spiral – shaped, curved, without transversely striated fiber are observed.

Compared to the results of the first series of studies, the phenomenon of edema was less pronounced. At the same time (table 1), the number of macrophages increased by a factor of 3.0, the number of fibroblasts decreased by a factor of 2.0, and fat inclusions decreased by a factor of 2.2 ($p<0.05$).

However, the above processes occurred in limited sections of the cross – section of muscle tissue, did not have a tendency to generalization, and were eliminated in the first 15 days after exercise of moderate aeroby power. It is noteworthy that an increase in the absolute number of hemocapillaries, which were detected in an area of 1 mm2 in the cross section of the rectus muscle (table 1). This caused an increase in the area of mutual overlap of the blood supply zones between adjacent hemocapillaries.

An increase in the multiplicity of the action of physical activity up to 30 times led to the appearance of a significant amount of glycogen in the sarcoplasm, the channels of the sarcoplasmic reticulum expanded. At the same time, the relative proportion of adipose tissue in the endomization significantly reduceded.

In endothelial cells of intramuscular hemocapillaries, the microrelief of the luminale surface was smoothed, the number and diameter of micropinocytosis vesicles increased.

The study of the abdominal muscles at different times after the modeling of the ventral hernia confirmed the opinion of some researchers [2] about the rather high sensitivity of their composite components to the conditions of modeling the ventral hernia [5].

The revealed changes in muscle fibers already on the 90th day after the start of modeling of the ventral hernia indicate their non-specificity, since they are often found in some myopathies [3, 4], and also as a compensatory-adaptive response of muscle fibers to metabolism under anaerobic conditions [11].

It is known that in the conditions of a ventral hernia a narrowing of the hemocapillary lumen occurs, a delay in metabolic products, oxygen delivery is blocked, leading to tissue hypoxia [6]. It, in turn, is the cause of the local hydration of cellular and non-cellular components of skeletal muscles.

Such structural changes cause a decrease in the active working surface of mitochondria and create prerequisites for the occurrence of ATP deficiency [1, 4]. Tanks of the sarcoplasmic reticulum are narrowed, which indicates the inhibition of synthetic processes on the membranes of these organelles. The end sections of T-tubules are reduced, the number of autophagosomes and residual organs increase, which indicates a profound structural reorganization of skeletal muscles in the simulation of ventral hernia [4].

A decrease in the absolute and relative amount of hemocapillaries in modeling a ventral hernia causes ischemia, as a result of which an increase in the amount of adipose tissue and fibroblasts occurs, which causes an increase in the (fraction of connective tissue in general).

The combination of morphological changes in the rectus abdominis when modeling a ventral hernia can be qualified as a paranecrotic process [15]. Since any stimulus at a certain duration of exposure, total intensity or dose can cause cell death, conditions for modeling a ventral hernia should be considered as a certain step towards the destruction of muscle fibers in response to functional deprivation.

It is the lack of function that is indicated as the most frequent cause of morpho-functional disorders of somatic cells in different tissues of animals and humans [4, 5, 6]. From this point of view, paranecrosis can very quickly turn into necrosis, the morphological signs of which we observed in individual muscle fibers in the long-term period after the modeling of a ventral hernia.

However, it is known that paranecrosis differs from true necrosis by the reversibility of destructive processes. Therefore, in some foreign publications, this term is replaced by the concept of “reversible damage”. Differences in the course of this process depending on the nature or dose of the acting factor have been studied by many scientists [14, 15].

They note that reversible damage is characterized by its non – specificity, since the action of various factors causes a homogeneous complex of structural changes. However, along with such nonspecificity, a number of specific features are observed. This partial specificity is observed in many pathological processes [1, 7, 10]. It should be noted that reversible damage after modeling a ventral hernia occurs on the background of the reduction of intramuscular microcirculation.

The fact of the rapid recovery and blood microcirculation activation during exercise [7] is proved. However, the question of the energy orientation of such a load remains outside the researchers' attention. In the scientific literature, especially in the field of physical culture and sports, there are sometimes diametrically opposite views on the use of physical activity of anaerobic or aerobic nature in various rehabilitation schemes after injuries, which are accompanied by a forced long bed rest [11, 12, 13].

The abdominal muscles are of separate interest because the further fate of the patient often depends on the speed of their morpho – functional recovery. Therefore, morphological studies of the patterns of their regeneration with average aerobic exercise are necessary for the development of modern rehabilitation techniques. The positive dynamics revealed by us in restoring the structure of microvessels after a 15-fold exercise of average aerobic power reflects the close nature of the mutual reorganization of the hemomicrocirculatory network and the components of the abdominal muscles [13, 15].

Being a non-centropic factor, exercise of moderate aerobic power leads to a more rapid reverse flow of destructive processes after modeling a ventral hernia [5]. This is evidenced by the restoration of not only the majority of intracellular organelles, but also the number of hemocapillaries, a decrease in the fraction of connective tissue and fibroblasts. At the same time, there is an increase in the number of macrophages that accelerate the utilization of the breakdown products of muscle fibers [1, 4].

Thus, the accelerated recovery of skeletal muscle tissue under the influence of physical activity indicates the stimulation of metabolic processes that are implemented by increasing the number of hemocapillaries and mitochondria, which provides muscle fiber with energy and plastic material [3, 7, 9]. The more powerful regeneration effect, which is observed under the action of physical exercise of average aerobic power, can be explained by the effect of active mechanical stretching of dystrophically altered muscle fibers, against the background of activated blood circulation in muscles during running of animals in the treadmill, which maintains a high level of metabolic processes [11, 13, 15].

Conclusion

In the field of experimental hernia, the ultrastructural organization of the hemomicrocirculatory bed of the muscles of the anterior abdominal wall undergoes structural restructuring. This is manifested in a significant rarefaction of the bloodstream, the desolation of individual hemocapillaries, the unevenness of their contours, and rupture of capillary loops. Data on a decrease in the total number of hemocapillaries are confirmed by morphometric indices. They indicate a decrease in energy supply and protein synthesizing function in muscles with this pathology, which underlies a decrease in the intensity of their blood supply and reflects a profound transformation of metabolic processes.

Dosed physical exertion of an aerobic nature enhances the reparative regeneration of muscle fibers after experimental ventral hernia.

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BIOMECHANICAL PROPERTIES AND MACROELEMENT ELEMENT COMPOSITION OF LONG TUBULAR BONE OF RATS UNDER EXPERIMENTAL HYPERGLYCAEMIA

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The pathomorphological basis for the development of diabetic disorders in various bones is a relevant subject of modern experimental research on the modeling of diabetes–associated destructive processes in the skeletal system. They are characterized by reduced bone density, which leads to an increased risk of fractures. Bone strength mainly depends on the interaction and concentration of chemical elements such as Ca, P, Mg, and K. Therefore, the purpose of our study was to determine the interdependence of changes in biomechanical parameters depending on the concentration of chemical elements in long tubular bones of rats under chronic hyperglycemia. The study was performed on 72 adult white laboratory rats lasting 180 days. It was found that starting from 30 days of uncontrolled chronic hyperglycemia, the bone mineral density in rats of the experimental group gradually decreased in comparison with the dynamics of the corresponding indexs in animals of the control group. These changes were accompanied by pronounced demineralization of both bones, as evidenced by significant losses of macronutrients (Ca, P, Mg and K). As a result, in chronic hyperglycemia gradually formed a complex violation of the structure and biomechanical properties of long tubular bones, which is a pathomorphological basis in the corresponding loci of the skeletal system.

Key words: rats, hyperglycemia, tubular bones, bone mineral density, macronutrient bone composition.

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БІОМЕХАНІЧНІ ВЛАСТИВОСТІ ТА МАКРОЕЛЕМЕНТНИЙ СКЛАД ДОВГИХ ТРУБЧАСТИХ КІСТОК ЩУРІВ ЗА УМОВ ЕКСПЕРИМЕНТАЛЬНОЇ ГІПЕРГЛІКЕМІЇ

Патоморфологічне підґрунтя розвитку діабетичних порушень у різних кістках є актуальним предметом сучасних експериментальних досліджень з моделювання діабет-асоційованих деструктивних процесів у системі скелету. Для них характерна знижена щільність кісток, що призводить до підвищення ризику переломів. Міцність кісток переважно залежить від взаємодії та концентрації хімічних елементів, таких як Ca, P, Mg та K. Тому метою нашого дослідження було визначення взаємозалежності зміни біомеханічних показників в залежності від рівня концентрації хімічних елементів у довгих трубчастих кістках щурів за умов хронічної гіперглікемії. Дослідження було проведено на 72 статевозрілих білих лабораторних щурах тривалістю 180 діб. Встановлено, що, починаючи з 30 доби неконтрольованої хронічної гіперглікемії мінеральна щільність кісток щурів експериментальної групи поступово зменшувалася у порівнянні з динамікою відповідних показників у тварин контрольної групи. Зазначені зміни супроводжвались вираженою демінералізацією обох кісток, про що свідчать значні втрати макроелементів: Ca, P, Mg та K. У підсумку, в умовах хронічної гіперглікемії поступово формується комплексне порушення структури та біомеханічних властивостей довгих трубчастих кісток, яке являє собою патоморфологічну основу розвитку діабетичних змін у відповідних локусах системи скелету.

Ключові слова: щури, гіперглікемія, трубчасті кістки, мінеральна щільність кістки, макроелементний склад кісток.

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Hyperglycemia is a pathognomonic symptom of diabetes mellitus (DM) – a disease that occurs due to insulin deficiency or insulin resistance and is accompanied by metabolic disorders and the development of pathological changes in bone tissue, resulting in an increased risk of fractures and inhibition of healing [4, 5, 12].

Morphological basis and pathological mechanisms of relationship between diabetes and skeletal health is the subject of debate for a long time, since it is known that glucose metabolism is closely associated with bone metabolism through regulated insulin secretion by the pancreas and adipose tissue [9, 14].