

полости рта для скрининга про- и пребиотиков / [А. П. Левицкий, О. А. Макаренко, И. А. Селиванская и др.]. – К.: ГФЦ, 2007. – 22 с.

19. **Патент** на корисну модель, Україна 43140, МПК (2009) G01N 33/48. Спосіб оцінки ступеня дисбіозу (дисбактеріозу) органів і тканин / [Левицкий А. П., Денга О. В., Селиванська І. О. та ін.]. – Опубл. 10.08.2009, Бюл. № 15.

20. **Експериментальні** методи дослідження стимуляторів остеогенеза: методическі рекомендації / [А. П. Левицкий, О. А. Макаренко, О. В. Денга и др.]. – К.: ГФЦ, 2005. – 50 с.

21. **Ферментативний** метод оцінки стану кісткової тканини / [А. П. Левицкий, О. А. Макаренко, І. В. Ходаков І. В. та ін.] / Одеський медичний журнал. – 2006. – № 3. – С. 17-21.

22. **Лапач О. Н.** Статистические методы в медико-биологических исследованиях с использованием Excel / Лапач О. Н., Чубенко А. В., Бабич П. Н. – К.: Морион, 2000. – 320 с.

REFERENCES

1. **Lopez-Marcos J. F.** Aetiology, Classification and Pathogenesis of Pulp and Periapical Disease. *Med. Oral Pathol.* 2004; 9: 52-62.

2. **Tsarev V. N., Ushakov R. V., Iasnikova E. Ia. [i dr.].** The etipathogenetic factors of development of periodontal inflammation diseases. *Stomatolog.* 2005; 6: 16-23.

3. **Mitronin A. V.** The main principles of complex diagnosis and conservative therapy of the patients with apex periodontitis at presence of the body diseases. *Stomatolog.* 2006; 8: 12-19.

4. **Borisenko A. V., Kodlubovskii Iu. Iu.** The methods of periodontitis curing (review of literature). *Sovremennaiia stomatologiya.* 2010; 1: 15-20.

5. **Tanomaru-Filho M., Polisel-Neto A., Leonardo M. R. [et al.].** Methods of Experimental Induction of Periapical Inflammation. *International Endodontic Journal.* 2005; 38: 477-482.

6. **Levitsky A. P., Denga O. V., Ivanov V. S. [i dr.].** *Eksperymentalnyi karies zubov* [The experimental dental caries]. *Eksperymentalnaia stomatologiya. Ch. I. Eksperymentalnye modeli stomatologicheskikh zabolevanii.* [The experimental stomatology. P. I. The experimental models of stomatological diseases]. Odessa: KP OGT, 2017: 59-67.

7. **Sheshukova O. V.** The role of parodontopathogenic infection in periodontitis development of temporary teeth. *Ukrain's'kyj stomatologichnyj al'manah.* 2006; 3: 66-68.

8. **Chajara A., Rondi M., Delpech B. [et al.].** Increased hyaluronan and hyaluronidase production and hyaluronan degradation in injured aorta of insulin-resistant rats // *Arteriosclerosis, Thrombosis and Vasc. Biol.* – 2000. – v. 20, № 6. – P. 1480-1487.

9. **Levitsky A. P., Makarenko O. A., Selivanskaya I. A. [i dr.].** *Kvertulin. Vitamin P, prebiotik, gepatoprotektor* ["Querthulin", Vitamin P, prebiotic, hepatoprotector]. Odessa, KP OGT, 2012: 20.

10. **Levitsky A. P.** *Lizotsym vmesto antibiotikov* [Lysozyme instead of antibiotics]. Odessa, KP OGT, 2005: 74.

11. **Den'ga O. V., Tsevuikh L. B., Levitsky A. P.** The biochemical indices of periodont tissues at the experimental therapy of periodontitis. *Visnyk stomatologii.* 2007; 4: 40-44.

12. **Zhurochko E. I., Chepurova N. I., Rossakhanova L. N.** Diagnosis and treatment of destructive forms of periodontitis in patients with oral dysbiosis. *Visnyk stomatologii.* 2010; 4: 15-17.

13. **Zhurochko E. I., Chepurova N. I., Makarenko O. A. [i dr.].** The influence of the combined use of «Lysomucoid» and «Bactulinum» on the biochemical indices of saliva in patients with periodontitis. *Visnyk stomatologii.* 2011; 1: 26-29.

14. **Levitsky A. P., Makarenko O. A., Selivanskaya I. A. [i dr.].** *Primeneniye mukoalnykh geley v stomatologii:*

metodicheskie rekomendatsii [The use of mucosal gels in dentistry]. Odessa, KP OGT, 2012:20.

15. **Levitsky A. P., Denga O. V., Makarenko O. A. [i dr.].** *Biokhimicheskie markery vospaleniya tkaney rotovoy polosti: metodicheskie rekomendatsii* [Biochemical markers of inflammation of oral cavity tissue: method guidelines]. Odessa, KP OGT, 2010: 16.

17. **Stalnaya I. D., Garishvili T. G.** *Metod opredeleniya malonovogo dialdegida s pomoshchyu tiobarbiturovoy kisloty* [The method of revelation of malonic dialdehyde with thiobarbituric acid]. Moskva, Meditsina, 1977: 66-68.

18. **Levitsky A. P., Makarenko O. A., Selivanskaya I. A. [i dr.].** *Fermentativnyy metod opredeleniya disbioza polosti rta dlya skringinga pro- i prebiotikov: metodicheskie rekomendatsii* [Enzymatic methods for determination of oral dysbiosis for screening pro- and prebiotics: method guidelines]. Kiev, GFC, 2007: 22.

19. **Levitsky A. P., Denga O. V., Selivanskaya I. A. [ta in.].** The method of estimation of the degree of dysbiosis (dysbacteriosis) of organs and tissues. Patent of Ukraine 43140. IPC (2009) G01N 33/48. Application number u 200815092. Date of filling: 26.12.2008. Publ.: 10.08.2009. Bul. № 15.

20. **Levitsky A. P., Makarenko O. A., Denga O. V. [i dr.].** *Eksperymentalnye metody issledovaniya stimulyatorov osteogeneza: metodicheskie rekomendatsii* [The experimental methods of the study of osteogenesis stimulators]. Kiev, GFK, 2005:50.

21. **Levitsky A. P., Makarenko O. A., Khodakov I. V. [ta in.].** The enzymatic method of the estimation of the state of osseous tissue. *Odeskiy medychnyy zhurnal.* 2006; 3:17-21.

22. **Lapach S. N., Chubenko A. V., Babich P. N.** *Statisticheskiye metody v medico-biologicheskikh issledovaniyakh s ispolzovaniem Excel* [Statistical methods in medical and biological research by using Excel]. Kiev, Morion, 2000: 320.

Поступила 06.11.17



УДК 616.314.17-008.1-084+599.323.4:612.396.32

¹ T.A. Pyndus CM, ² O.V. Denga MD,
³ V.V. Gargin, MD

¹Lviv Medical Institute

² State Establishment "The Institute of Stomatology and Maxillo-Facial Surgery National Academy of Medical Science of Ukraine"

³Kharkiv National Medical University

COMPLEX PROPHYLAXIS OF PATHOLOGICAL CHANGES IN RATS PERIODONTAL TISSUES WITH MODELING OF METABOLIC SYNDROME (MORPHOLOGICAL STUDY)

ABSTRACT

Introduction. *Metabolic syndrome creates prerequisites for the development of parodontitis and between them there is a potentially significant association leading to*

a higher risk of development of pathology in the oral cavity.

Purpose of the study. The aim of the research was to study the effect of the developed therapeutic-prophylactic complex on the parodontal tissues of rats during the modeling of the metabolic syndrome.

Materials and methods. In the experiment, 21 rats were used: an intact group, a standard ration of the vivarium - 7 animals; comparison group, model of metabolic syndrome - 7 animals; the main group, the model of metabolic syndrome plus prevention - 7 animals. The therapeutic and prophylactic complex included anti-inflammatory, lowering cholesterol, regulating lipid metabolism and other drugs. Microscopic preparations were studied on a microscope "Olympus BX-41" with the program "Olympus DP-soft version 3.2".

Results. Conclusions. When modeling the metabolic syndrome in rats, a complex of pathological changes occurred in the periodontal tissues. Application of the developed therapeutic and prophylactic complex led to a decrease in the inflammatory processes of soft tissues, restoration of the integrity of the epithelial cover, the disappearance of inflammatory infiltration.

Key words: rat, metabolic syndrome, morphometry, prophylaxis.

¹ Т. А. Пиндус, ² О. В. Деньга, В. В. Гаргин

¹ Львовский медицинский институт

² Государственное учреждение «Институт стоматологии и челюстно-лицевой хирургии Национальной академии медицинских наук Украины»

³ Харьковский национальный медицинский университет

**КОМПЛЕКСНАЯ ПРОФИЛАКТИКА
ПАТОЛОГИЧЕСКИХ ИЗМЕНЕНИЙ
В ТКАНЯХ ПАРОДОНТА КРЫС
ПРИ МОДЕЛИРОВАНИИ
МЕТАБОЛИЧЕСКОГО СИНДРОМА
(МОРФОЛОГИЧЕСКОЕ ИССЛЕДОВАНИЕ)**

При моделировании метаболического синдрома у крыс происходило формирование комплекса патологических изменений в тканях пародонта с развитием комплекса дистрофически-воспалительных процессов на фоне поражения микроциркуляторного русла. Применение разработанного лечебно - профилактического комплекса привело к уменьшению в ротовой полости крыс воспалительных процессов мягких тканей, сопровождающихся восстановлением целостности эпителиального покрова, исчезновением воспалительной инфильтрации, восстановлением микроциркуляции.

Ключевые слова: крысы, метаболический синдром, морфометрия, профилактика.

¹ Т. О. Пиндус, ² О. В. Деньга, ³ В. В. Гаргин

¹ Львівський медичний інститут

² Державна установа «Інститут стоматології та щелепно-лицевої хірургії Національної академії медичних наук України»

³ Харківський національний медичний університет

**КОМПЛЕКСНА ПРОФІЛАКТИКА
ПАТОЛОГІЧНИХ ЗМІН У ТКАНИНАХ
ПАРОДОНТА ЩУРІВ ПРИ МОДЕЛЮВАННІ
МЕТАБОЛІЧНОГО СИНДРОМУ
(МОРФОЛОГІЧНЕ ДОСЛІДЖЕННЯ)**

При моделюванні метаболического синдрома у щурів відбувалося формування комплексу патологічних змін в тканинах пародонта з розвитком комплексу дистрофічно-запальних процесів на тлі поразки мікроциркуляторного русла. Застосування розробленого лікувально-профілактичного комплексу призвело до зменшення у ротовій порожнині щурів запальних процесів м'яких тканин, що супроводжувались відновленням цілісності епітеліального покриву, зникненням запальної інфільтрації, відновленням мікроциркуляції.

Ключові слова: щури, метаболический синдром, морфометрія, профілактика.

Metabolic syndrome (MS) is accompanied by abdominal obesity, hyperglycemia, high blood pressure and dyslipidemia [1], has a close connection with the development of cardiovascular diseases, type 2 diabetes mellitus [2, 3] and leads to morphofunctional changes arising in this case, observed practically in all organs and tissues of the body.

It is believed that the metabolic syndrome creates the presupposition for the development of periodontitis [4, 5] and that there is a potentially significant association between them, leading to a higher risk of oral cavity pathology development [6, 7]. Considering the increasing prevalence of MS among the world's population at present, the search for ways to correct the periodontal status thus acquire a significant social and general medical significance.

This experimental study purpose was a morphological research of the developed therapeutic and prophylactic complex effect on the rats periodontal tissues during MS modeling.

Materials and methods. In the experiment, 1.5-2 months of age white male rats were used. Intact group consisted of 7 individuals (control group). The animals of this group have a normal diet (mixed fodder and a mixture of barley with wheat) and there was free access for rats to drinking water. The second group of animals consisted of 7 individuals (comparison group), the metabolic syndrome was simulated by introducing 20 % of the internal pig fat into the diet, instead of drinking water was used 10 % fructose ad libitum solution.

The third group (main group) consisted of 7 individuals who were on the background of modeling MS complex therapy, which included daily administration of per os: "Chlorophyllin" – 300 mg / kg

(Pharmaceutical company "Vertex", Ukraine, anti-bacterial, antiseptic and anti-inflammatory), "Laktusan" – 2 ml / kg (Felitsata Ukraina, prebiotic, normalizes metabolism, intestinal microflora, strengthens immunity), "Oxifit MAP" – 1 drop / kg ("Medagroprom", Ukraine, regulates lipid metabolism, provides tissue with oxygen, improves metabolism), "Sera Active" – 70 mg / kg ("Euro Plus", Ukraine, anti-inflammatory action, improve blood circulation).

Daily the "Kvertulin" gel was applied locally on the gums to 0.2 ml / rat ("Odesa Biotechnology", Ukraine, increases local nonspecific resistance). Experiment duration was 70 days. After the withdrawal of the animals from the experiment (according to the European Convention for the Protection of Vertebrates from 18.03.1986), soft tissue fragments of the oral cavity were fixed in 10 % formalin, poured into paraffin and after sections were made slices 5x10-6 m thick, which were stained with hematoxylin and eosin, picrofuxin by van Gieson, according to Mallory, put a PAS-reaction.

Morphometric examination of micropreparations was carried out on the microscope "Olympus BX-41" with subsequent processing of data on the program "Olympus DP-soft version 3.2". Data received were processed statistically.

Results of research and their discussion. During the examination of animals from group where MS was modeled, there was a pronounced paleness of the visible mucosa, a moderately pronounced puffiness (comparison group). In the group of animals receiving complex therapy, the visual pattern was generally similar to that in the group of intact animals.

The study of histological preparations of animals of all groups showed that the oral mucosa coated with multilayer squamous nonkeratinized epithelium excluding the gum surface, where the epithelium was keratinizing. In the group of MS rats (comparison), the epithelium revealed dystrophic changes in epithelial cells, uneven thickness of the epithelium. In the cytoplasm of the epithelial cells of the prickly and basal layers, vacuoles appeared, the dimensions of which often were comparable with the dimensions of the nuclei. The latter were displaced toward the periphery, a tendency was observed to flatten the cells of the spiny layer. The PAS reaction in the epithelium was slightly positive, more pronounced in the upper stratum of the spiny layer, with the presence of zones of weak staining localized mainly in the basal regions. In the underlying connective tissue, edema and the appearance of small foci of lymphocytes and plasmocytes appeared in the perivascular space. In its own plate, acanthotic bands were found, the number of fibroblasts was increased, there were isolated white blood cells, sclerotization of the reticular layer was noted. Reticular fibers

were less convoluted, more thickened and compacted than in the control group, sometimes with partial homogenization. Collagen fibers were collected in bundles, areas of their hyalinization were noted.

The walls of the vessels in the tissues of the oral cavity of animals of the group with MS were somewhat thickened, swollen, with signs of edema. The most pronounced signs of mucoid swelling were observed in the zones of perivascular cell infiltration. In the perivascular space, there were often signs of fragmentation and lysis of collagen fibers. Endothelium was with large nuclei. Endothelial and adventitial cells were slightly enlarged in size, moderate hyperchromatism of the nuclei was noted. Foci desquamation of endothelial cells with bare basal membranes was revealed. The capillary network was more dense at the expense of the appearance of young capillaries. The network of newly formed capillaries was surrounded by individual fibroblasts, elements of lymphocytic, plasmacytic, macrophagal series, mast cells, neutrophilic leukocytes, basal membrane of the vessels were unevenly thickened and intensely PAS-positive.

Three groups of rats typing results of the oral cavity mucosa cellular composition are presented in Table 1.

The morphological changes in periodontal tissue of rats described above are generally considered as manifestations of inflammatory and dystrophic processes in the case of microcirculation disturbances, metabolic disorders characteristic of MS development.

Histological examination of the main group animals periodontal tissue that received complex preventive treatment against the background of MS modeling revealed a clear division of the gingival epithelium into granular, spiny and basal layers, while the stratum corneum was of moderate thickness and layered structure. The cells of the granular layer were spindle-shaped in 3-4 rows, the keratogialin grains in the cytoplasm were small. The cells of the spine layer were large, with a light cytoplasm and a well-expressed cell membrane. Basal epithelial cells were elongated, localized along a well-marked basal membrane with a perpendicular direction of the cell axis toward it. The nuclei of basal epitheliocytes were oval in shape, homogeneous, hyperchromic, the cytoplasm was moderately basophilic. The epithelium in the region of the dentogingival pocket of the rats of the main group was without signs of submerged growth, there was no keratinization from the side of the tooth, the cells of the spiky layer were somewhat flattened. The connective tissue of its own plate was represented by both elastic fibers and collagen fibers, some of which had signs of hyalinization.

The basal membrane separating the epithelium

from the mucosa proper, was thickened, homogeneous, PAS-positive. In the papillary layer of the mucous membrane of the gum proper, along with the elastic fibers, a fairly large amount of collagen fibers was found, in some cases, a tangle of collagen fibers directly under the gingival epithelium was observed. The collagen fibers of the reticular layer were coarse, homogeneous, merging with each other with signs of hyalinization. As the distance from the surface among the connective tissue fibers, thicker fibers prevailed, both singly and with small beams, the border of their transition to periodontium was indistinct.

The collagen fibers in the van Gieson coloration were fuchsinophiles. The PAS response was moderately positive, more pronounced in the surface layers, where more reticulin fibers are located, and less pronounced in deeper areas with a predominance of collagen fibers. Arterioles, capillaries and venules were moderately full-blooded. The picture of the microcirculatory bed was well defined, with the presence of a rounded, oval or elongated shape and branching vessels. The basal membrane of the vessels was thickened, PAS-positive. Endotheliocytes were large cells with large light nuclei (Fig. 1).

Table 1

Relative volumes of cellular elements the actual plate of the mucosa in experimental rats (% , M±m)

Cellular elements	Intact animals (control), n=7	MS (comparison group), n=7	MS + complex therapy (main group), n=7
histiocytes	17,6±1,03	15,37±1,43	18,71 ± 1,48
young fibroblasts	11,36±1,15	8,32±1,37	12,92 ± 1,56
mature fibroblasts	42,2±3,2	29,1±2,03	37,82 ± 3,37*
fibroblasts	19,9±2,53	17,17±2,18	21,30 ± 1,96
lymphocytes	3,9±0,12	11,02±1,29	4,21 ± 0,34*
mast cells	1,9±0,03	3,4±0,16	2,0 ± 0,03*
plasmocytes	1,2±0,03	7,27±1,43	1,1 ± 0,03*
macrophages	1,0±0,02	3,0±0,12	1,1 ± 0,02*
leucocytes	0,6±0,03	5,3±0,16	0,7 ± 0,01*

Note: * p <0,05, the indicator of reliability of differences in the results of the main group from the comparison group

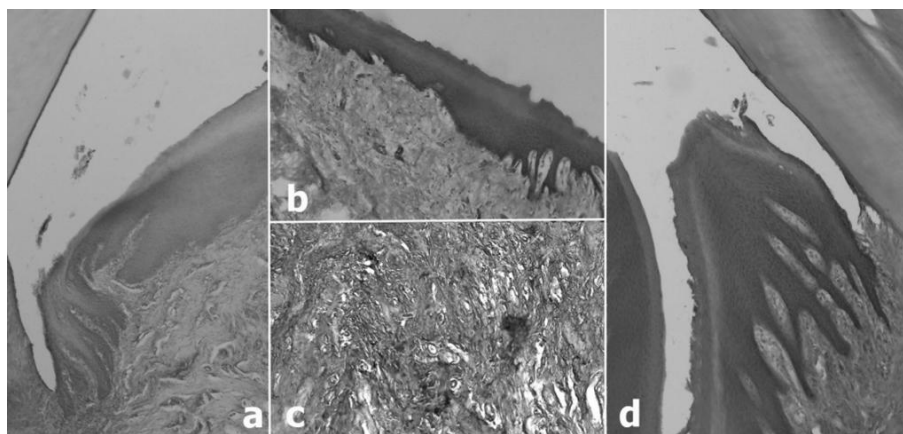


Fig. 1. a – comparison group (formed by an MS). Inflammatory changes in the area of the epithelial-dental connection. Formation of zones of thinning of the epithelial layer against the background of necrobiotic processes, the appearance of newly formed vessels of the microcirculatory bed. Inflammatory infiltration of the lamina propria of the mucosa. Staining with hematoxylin and eosin. x100; b – main group (MS after correction course). Clear division of the gingival epithelium into granular, spiny and basal layers. Appearance of acanthotic strings. Homogeneous basement membrane of the epithelium. Single cellular elements of the lymphoid series. Thickening of the walls of the vessels of the microcirculatory bed. Staining with hematoxylin and eosin. x200; c – main group (MS after correction course). Sclerosis of the lamina propria of the mucosa represented by both elastic fibers and collagen fibers, some of which are hyaline. Coloring by van Gieson. x200; d – main group (MS after correction course). The presence of pronounced acanthotic cords in the zone of epithelial-dental connection. Staining with hematoxylin and eosin. x100.

The observed pathological changes in the tissues of the periodontal and microcirculatory bed of rats during the modeling of the metabolic syndrome can also be interpreted as a detailed picture of perio-

dontitis. As we know, pathological processes in the periodontium can develop not only as a result of exposure to the microbial flora of plaque against the backdrop of the failure of immune systems with the

subsequent development of inflammatory processes, but as a chronic condition (periodontopathy) in which inflammatory processes can be not only non-triggering, to recede into the background [8].

It can be argued that the MS modeling performed by us led to pathological processes in the epithelium and in the propria of the mucosa in accordance with the known markers for the development of the periodontal inflammatory process [9]. So we established hyperkeratosis of the interdental papilla epithelium, gingival groove, epithelium of attachment, which can be considered as one of the elements of the developing protective reaction necessary to protect the foci epithelial layer thinning. The most pronounced changes were observed in the area of the dentogingival pocket. Associated with this process were also observed acanthotic sprouting.

Inflammatory changes in MS of the mucosa propria were characterized by edema, appearance of inflammatory cells which composition was statistically significantly different from that in the control group. There were also signs of an acute inflammatory process, with a relatively significant relative volume of leukocytes (Table 1.). At the same time, it is necessary to note violations in the microcirculatory bed, the consequences of which, of course, have a morphological mapping in both dystrophic and sclerotic processes. At the same time, the damage to the basal membranes was systemic, which is generally combined with the literature data on the development of pathological processes in the oral mucosa [10, 11].

The use of the therapeutic and prophylactic scheme proposed by us, including anti-inflammatory, immunity-enhancing and local non-specific resistance, improving blood circulation and metabolism, aimed at correcting soft tissue damage, resulted in a decrease in inflammatory manifestations in the main group of animals, improvement in trophism of soft tissues. Morphological study of the main group tissues showed the restoration of the structure of both the epithelium and its own plate of the mucosa. The division of the gingival epithelium into granular, spiny and basal layers corresponded to the structure of the epithelium of intact animals. Signs of dystrophic changes in epithelial cells were practically absent. The thickness of the epithelium was uniform, and in the zone of the dentogingival pocket there were no signs of submerged growth. Such characteristics of the epithelial layer are an important sign of the effectiveness of its protective properties. Sclerotic changes in the propria of the mucous membrane are a consequence of the transferred inflammatory process, the absence of which was indicated only by single cells of the lymphoid series. There were also signs of restoration of blood supply, as evidenced by the state of the vessels of

the microcirculatory bed in the main group of animals.

Considering that the disruption of communication between the epithelial cells, the epithelium of the mucous gum and the tooth surface is the basis for the formation of the periodontal pocket (in our case as a consequence of MS), and in periodontitis the most pronounced changes in the epithelium are localized in the areas adjacent to the periodontal pocket, epithelial-dental junction are said to prevent the development of the periodontal pocket in the main group of animals. It should also be noted that the presence of acanthotic cords in the animals of the main group can be considered as a consequence of the elimination mechanism in combination with the leukocyte-destructive effect (the accumulation of inflammatory cells in the inflammatory growth of the epithelium, followed by the death of the epithelium and the surrounding of the immunocompetent tissue), through which they are removed from the gum sites of dying immunocompetent tissue.

Thus, the use of complex therapy based on the use of antibacterial, anti-inflammatory, permeability-reducing capillaries, lowering cholesterol, regulating lipid metabolism, improving metabolism, excreting toxic substances and increasing local non-specific resistance in the oral cavity, against the backdrop of modeling in rats of the metabolic syndrome to a decrease in soft tissues of periodontal inflammation, dystrophic changes in epithelial cells and improvement of trophism, restoration of intact stnosti soft oral tissues animal mouth.

Conclusions. When modeling the metabolic syndrome in rats, a complex of pathological changes occurred in the periodontal tissues with the development of a complex of dystrophic-inflammatory changes against the background of the lesion of the microcirculatory bed. The use of the developed therapeutic and prophylactic complex led to a decrease in the oral cavity of rats inflammatory processes in the soft tissues of periodontal disease, accompanied by restoration of the integrity of the epithelial cover, the disappearance of inflammatory infiltration, the restoration of microcirculation.

REFERENCES

1. **Eckel R. H., Grundy S. M., Zimmet P. Z.** The metabolic syndrome. *Lancet*. 2005;365:1415–28.
2. **Grundy S. M.** Metabolic syndrome scientific statement by the American Heart Association and the National Heart, Lung, and Blood Institute. *Arterioscler Thromb Vasc Biol* 2005;25:2243–2244.
3. **Musskopf M. L., Daudt L. D., Weidlich P., Gerchman F., Gross J. L., Oppermann R. V.** Metabolic syndrome as a risk indicator for periodontal disease and tooth loss. *Clin Oral Investig*. 2017 Mar;21(2):675–683.
4. **Apoorva S. M., Sridhar N., Suchetha A.** Prevalence and severity of periodontal disease in type 2 diabetes mellitus (noninsulin-dependent diabetes mellitus) patients in Bangalore

city: an epidemiological study. J Indian Soc Periodontol 2013;17:25–29.

5. Lee J. B., Yi H. Y., Bae K. H. The association between periodontitis and dyslipidemia based on the Fourth Korea National Health and Nutrition Examination Survey. J Clin Periodontol 2013;40:437–442.

6. Kwon Y. E., Ha J. E., Paik D. I., Jin B. H., Bae K. H. The relationship between periodontitis and metabolic syndrome among a Korean nationally representative sample of adults. J Clin Periodontol 2011;38: 781–786.

7. Fukui N., Shimazaki Y., Shinagawa T., Yamashita Y. Periodontal status and metabolic syndrome in middle-aged Japanese. J Periodontol 2012;83:1363–1371.

8. Gomes-Filho I. S., Freitas Coelho J. M., da Cruz S. S., Passos J. S., Teixeira de Freitas C. O., Aragão Farias N. S., et al. Chronic periodontitis and C-reactive protein levels. J Periodontol. 2011;82:969–978.

9. Loos B. G. Systemic markers of inflammation in periodontitis. J Periodontol. 2005;76(Suppl 11):2106–2115.

10. Nazaryan R., Kryvenko L., Gargin V. The role of nitric oxide synthase in the modulation of the immune response in atopic disease. New Armenian Medical Journal. 2017, 11(2): 52–57.

11. Nazaryan R., Kryvenko L. Salivary oxidative analysis and periodontal status in children with atopy. Interventional Medicine & Applied Science. 2017; 9(3):1–5.

Поступила 02.11.17



УДК 616.153.1:577.152:616.623

**М. О. Остафійчук¹, к. мед. н.,
В. І. Струк¹, к. мед. н.,
А. П. Левицький³, д. биол. н.**

¹Вищий державний навчальний заклад «Буковинський державний медичний університет»

²Державна установа «Інститут стоматології та щелепно-лицьової хірургії Національної академії медичних наук України»

ВПЛИВ «ЛИЗОЦИМА-ФОРТЕ» НА СТАН СЛИЗОВОЇ ОБОЛОНКИ ПОРОЖНИНИ РОТА ЩУРІВ З ЕКСПЕРИМЕНТАЛЬНИМ СТОМАТИТОМ

У щурів відтворювали перекисну модель стоматиту. Встановлено розвиток в слизовій оболонці щоки запалення та дисбіозу при зниженні рівня антиоксидантного захисту та неспецифічного імунітету. Введення препарату лізоцим-форте (лізоцим + кверцетин + інулін + цитрат кальція + желатин) попереджає розвиток запалення і дисбіозу, перевищуючи за терапевтичним ефектом препарат порівняння квертулін.

Ключові слова: експериментальний стоматит, лізоцим-форте, квертулін, дисбіоз.

**М. А. Остафійчук¹, В. І. Струк¹,
А. П. Левицький²**

¹Высшее государственное учебное учреждение «Буковинский государственный медицинский университет»

²Государственное учреждение «Институт стоматологии и челюстно-лицевой хирургии Национальной академии медицинских наук Украины»

ВЛИЯНИЕ «ЛИЗОЦИМА-ФОРТЕ» НА СОСТОЯНИЕ СЛИЗИСТОЙ ОБОЛОЧКИ ПОЛОСТИ РТА КРЫС С ЭКСПЕРИМЕНТАЛЬНЫМ СТОМАТИТОМ

У крыс воспроизводили перекисную модель стоматита. Установлено развитие в слизистой оболочке щеки воспаления и дисбиоза при снижении уровня антиоксидантной защиты и неспецифического иммунитета. Введение препарата лизоцим-форте (лизоцим + кверцетин + инулин + цитрат кальция + желатин) предупреждает развитие воспаления и дисбиоза, превышая по терапевтическому эффекту препарат сравнения квертулин.

Ключевые слова: экспериментальный стоматит, лизоцим-форте, квертулин, дисбиоз.

М. А. Ostafiychuk¹, V. I. Struk¹, A. P. Levitsky²

¹Bukovinian State University

²State Establishment «The Institute of Stomatology and Maxillo-Facial Surgery of the National Academy of Medical Science of Ukraine»

THE EFFECT OF «LYSOZYME-FORTE» ON THE ORAL MUCOSA STATE OF RAT WITH THE EXPERIMENTAL STOMATITIS

ABSTRACT

The aim. To investigate effect of lysozyme-forte on the inflammation and dysbiosis development into cheek mucosa of rat with the experimental stomatitis.

The materials and methods. The peroxide model of stomatitis was reproduced in rats. The state of oral mucosa was determined by the levels of biochemical indices of inflammation and dysbiosis. Lysozyme-forte and quertuline (the preparation of comparison) were introduced per os. The experiment lasted 2,5 months.

The findings. The levels of markers of inflammation (elastase and malonic dialdehyd) and the microbe sowing (urease) were raised into oral mucosa of rats with experimental stomatitis but the levels of non-specific immunity index (lysozyme) and antioxidant defence (catalase) were reduced. The both preparations were reduced the levels of inflammation and urease indices, but were raised the levels of lysozyme and catalase. The lysozyme-forte was more effective.