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CORRECTION OF BIOCHEMICAL PARAMETERS IN THE JAWS OF RATS USING A THERAPEUTIC AND PROPHYLACTIC COMPLEX UNDER CONDITIONS OF A CARIOGENIC DIET AND DIETARY VITAMIN D DEFICIENCY ACCOMPANIED BY JAWBONE DESTRUCTION

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This study assessed the effect of the proposed therapeutic and prophylactic complex on biochemical markers of rat jawbone tissue under conditions of a cariogenic diet and dietary vitamin D deficiency. Experimental studies were conducted on 30 four-month-old male Wistar rats. The animals were divided into three groups of ten: Group 1, intact animals; Group 2, injury to the mandibular region corresponding to the molar roots and modelling of dietary vitamin D deficiency against a background of a high-sucrose cariogenic diet; and Group 3, the pathology model with administration of the therapeutic and prophylactic complex. Markers of bone resorption and formation, namely acid and alkaline phosphatase activities, were determined in alveolar bone homogenates, and mineralisation was assessed by calcium content. The mineralisation index was calculated as the ratio of alkaline to acid phosphatase activity. Modelling of the pathological condition resulted in a 7.5 % increase in the degree of alveolar atrophy, a 34 % increase in acid phosphatase activity, a 31.12 % decrease in alkaline phosphatase activity and a 13.16 % decrease in calcium content. Administration of the proposed therapeutic and prophylactic complex contributed to correction of these disturbances and normalisation of the studied parameters towards the values observed in intact animals, indicating increased mineral content of alveolar bone and enhanced bone turnover.

Key words: bone destruction, rats, vitamin D deficiency, alveolar bone.

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КОРЕКЦІЯ БІОХІМІЧНИХ ПОКАЗНИКІВ ЩЕЛЕП ЩУРІВ ЛІКУВАЛЬНО- ПРОФІЛАКТИЧНИМ КОМПЛЕКСОМ ЗА УМОВ КАРІЄСОГЕННОЇ ДІЄТИ ТА АЛІМЕНТАРНОГО ДЕФІЦИТУ ВІТАМІНУ D НА ТЛІ ДЕСТРУКЦІЇ КІСТКОВОЇ ТКАНИНИ ЩЕЛЕП

Робота була присвячена оцінці впливу запропонованого лікувально-профілактичного комплексу на біохімічні маркери кісткових тканин щелепи щурів за умов карієсогенної дієти та аліментарного дефіциту вітаміну D. Експериментальні дослідження були проведені на 30 чотиримісячних щурах-самцях лінії Wistar. У ході експерименту тварин було розподілено на три групи по 10 щурів у кожній: 1-ша група – інтактні тварини; 2-га група – травма ділянки нижньої щелепи в проекції коренів молярів та моделювання аліментарного дефіциту вітаміну D на тлі високосахарозної карієсогенної дієти; 3-тя група – модель патології та застосування лікувально-профілактичного комплексу. У гомогенатах кісткової тканини альвеолярних відростків визначали маркери резорбції та кісткоутворення – активність кислотної, лужної фосфатази та стан мінералізації – за вмістом кальцію. Розраховували мінералізуючий індекс як співвідношення значень активності лужної та кислотної фосфатази. Моделювання патологічного стану призвело до збільшення показника ступеня атрофії альвеолярного відростка щелепи на 7,5 %, збільшення показника активності кислотної фосфатази на 34 %, зменшення активності лужної фосфатази на 31,12 %, а вмісту кальцію на 13,16 %. Застосування запропонованого лікувально-профілактичного комплексу сприяло корекції виявлених порушень і нормалізації досліджуваних показників у напрямку значень інтактних тварин, що свідчить про збільшення мінеральної насиченості альвеолярної кістки та посилення інтенсивності кісткового обміну.

Ключові слова: деструкція кістки, щури, дефіцит вітаміну D, альвеолярна кістка.

The work is a fragment of the research project "Improving the diagnosis and treatment of major dental diseases in patients with bone metabolism disorders", state registration No. 0125U003929.

Bone tissue is a highly specialised type of connective tissue. Its principal distinction from other tissues is its mineralised nature. This property accounts for many of the biological, physiological, morphological and biomechanical characteristics of bone tissue and skeletal organs, as well as their multiple functions [13].

Bone metabolism comprises two opposing processes: the formation of new bone tissue by osteoblasts and the degradation of existing bone by

osteoclasts. Bone mass depends on the balance between resorption and formation over a given period and on the number of activated remodelling sites. Jawbone remodelling proceeds more rapidly than remodelling in other bones of the human skeleton [9].

The mobility and flexibility of the mandible, which are required to withstand the continuous and multidirectional loads associated with mastication and speech, depend on the characteristics of its collagen framework [7]. The collagen matrix

undergoes substantial pathological changes in destructive periodontitis, representing an important element in the development of destructive lesions in the maxillofacial region. Studies of the bone collagen matrix in destructive periodontitis help elucidate the mechanisms of destruction and restoration of the supporting structures of the tooth [1]. In destructive forms of chronic apical periodontitis, changes in jawbone tissue include local bone resorption, destruction of the alveolar cortical plate and replacement of bone structures by inflammatory or granulation tissue [4].

Bacterial endotoxins promote the release of pro-inflammatory cytokines (IL-1 β , IL-6 and TNF- α) and mediators of the RANK/RANKL/OPG system in the periapical region. These factors stimulate the formation and increased activity of osteoclasts, the cells responsible for dissolution of the mineralised bone matrix. Within the inflammatory focus, calcium and phosphorus concentrations decrease, acid phosphatase activity, a marker of resorption, increases, and alkaline phosphatase activity, a marker of osteoblast function, decreases. Chronic inflammation compresses the microcirculatory bed, leading to local necrosis of individual bone trabeculae and thinning of the jaw cortical plate, potentially resulting in perforations [15].

Bone resorption increases, mineral density decreases and calcium metabolism is disturbed [8]. These changes have been demonstrated in experimental studies involving a high-sucrose diet and experimental periodontitis [5, 6, 14].

Vitamin D, particularly its hormonal forms, plays an important role in the regulation of metabolic processes throughout the body, including bone tissue.

The treatment of destructive forms of chronic apical periodontitis remains an important clinical problem. Elimination of periapical inflammation removes the pathogenic influence of the odontogenic inflammatory focus on the body and allows the tooth to retain its full function, with complete healing of the periapical bone lesion [11].

The purpose of the study was to assess the effect of the proposed therapeutic and prophylactic complex on biochemical markers of rat jawbone tissue under conditions of a cariogenic diet and dietary vitamin D deficiency against a background of jawbone destruction.

Materials and methods. Experimental studies were conducted on 30 colony-bred male Wistar rats aged four months, with a mean body weight of 214.8 $\pm$ 3.2 g. Inclusion criteria were confirmed Wistar strain and male sex; age of 16 $\pm$ 1 weeks, verified from breeding records; baseline body weight within the specified range; absence of visible injury, malocclusion or systemic or oral pathology on veterinary inspection; and no previous exposure to experimental procedures or pharmacological agents. Exclusion criteria comprised clinically detectable disease or behavioural abnormalities, body weight

outside the predefined range, wounds or oral lesions, aggressive behaviour, modelling errors and failure to adapt during the seven-day acclimatisation period. Only male Wistar rats were enrolled to minimise biological variability associated with sex-specific endocrine cycles.

The study was conducted from 1 July to 2 August 2025.

All animals were maintained in the accredited barrier-type vivarium of SE "ISMFS NAMS" under the husbandry conditions specified in the institute's standard operating procedure, which fulfils Ukrainian legislation and ARRIVE 2.0 recommendations. Rats were housed in groups of four or five in individually ventilated Euro-Type IV polycarbonate cages (floor area approximately 1,500 cm²; internal dimensions 425 $\times$ 266 $\times$ 185 mm), fitted with stainless-steel wire lids and placed on ventilated racks providing 12 $\pm$ 2 air changes per hour. Cages contained a 3-cm layer of autoclaved aspen-wood shavings (LIGNOCEL® Hygienic Animal Bedding, JRS GmbH), replaced twice weekly. Environmental enrichment was available ad libitum and included shredded paper nesting material (Nestlets), hardwood gnawing blocks (2 $\times$ 2 $\times$ 6 cm) and one cardboard tunnel (9 cm in diameter and 12 cm long) per cage; these items were refreshed at each cage change.

The room was maintained at 21 $\pm$ 2 °C and 55–75 % relative humidity, with a 12:12 h light/dark cycle and lights on from 07:00 to 19:00. Noise was below 50 dB SPL. Reverse-osmosis-filtered water was supplied in polycarbonate bottles with stainless-steel sipper tubes and replaced three times weekly.

Daily husbandry included visual health checks and removal of soiled enrichment; full cage sanitisation (hot-water wash and autoclave) was performed weekly.

Each rat was weighed, assigned an individual code and allocated to an experimental arm by simple computer-generated randomisation using Microsoft Excel. The animals were assigned to three experimental groups:

1. Intact group: rats undergoing no surgical procedures and receiving no drug treatment, maintained on a complete balanced diet (n=10).

2. Pathology group (TLJ): injury to the mandibular region corresponding to the molar roots and modelling of dietary hypovitaminosis D against a background of a high-sugar cariogenic diet (CD) (n=10).

3. Treatment group (TLJ+TPC): injury to the mandibular region corresponding to the molar roots and administration of the therapeutic and prophylactic complex (TPC) against a background of dietary hypovitaminosis D modelled using a high-sugar cariogenic diet (n=10).

No animal met an exclusion criterion, and no deaths or withdrawals occurred after group allocation. Consequently, the composition of all three groups

remained unchanged throughout the 33-day observation period, and data from every animal were included in the final analysis.

The experimental studies were conducted at the Laboratory of Biochemistry and Vivarium of the State Establishment “The Institute of Stomatology and Maxillofacial Surgery of the National Academy of Medical Sciences of Ukraine” (SE “ISMFS NAMS”). All experiments followed standard operating procedures approved by the institute and developed in accordance with the guidelines of the Pharmacological Committee of the Ministry of Health of Ukraine and the General Ethical Principles of Animal Experiments adopted by the Seventh National Congress on Bioethics (Kyiv, 2019), with reference to the European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes (Strasbourg, 1986). The study was approved by the Bioethics Commission of Odesa National Medical University (protocol No. 26 dated 23 October 2024).

Mandibular injury was induced in the vivarium using a portable dental drill and a sterile carbide bur under thiopental anaesthesia.

The cariogenic diet was based on the formulation of M.S. Bugayova and S.A. Nikitin as modified by I.V. Khodakov et al. [12]. Its composition was refined sugar, 57 %; skimmed-milk curd, 18.5 %; white wheat breadcrumbs, 19 %; unrefined sunflower oil, 4.5 %; and table salt, 1 %. Undevit was omitted to allow separate administration of vitamins A and D₃. Retinol acetate (100,000 IU/mL; PJSC Technolog, Uman, Ukraine) was added at 0.048 mL per 100 g of feed. The reduction in oil content by 0.5 percentage points was compensated for by an equivalent increase in breadcrumbs.

The therapeutic and prophylactic complex comprised Mumie gel, Osteovit, Aquadetrim Vitamin D₃ and Forteza oral spray. Mumie gel contained centaury, echinacea and sage infusions, mumie and 0.05 % chlorhexidine. It was developed by the Laboratory for the Development and Research of Oral Hygiene Products of SE “ISMFS NAMS” (State Sanitary and Epidemiological Expertise Conclusion No. 1378/16 dated 10 December 2019) and administered orally once daily at 0.3 mL per 200 g of body weight. Its intended actions were anti-inflammatory, adaptogenic, regenerative, osteotropic, anticoagulant, antibacterial, haemostatic and immunomodulatory effects, together with increased salivation. Osteovit (Elite-Pharm LLC, Dnipro, Ukraine) was administered at 150 mg/kg of body weight. Its active components were glucosamine sulfate, chondroitin sulfate, Boswellia extract, vitamin D₃ (cholecalciferol, 130 IU) and calcium carbonate (providing 50 mg calcium); excipients included calcium stearate, microcrystalline cellulose, lactose, starch and sorbitol. The preparation was included for its anti-inflammatory effects, support of cartilage formation and regeneration, calcium supply and promotion of calcium and phosphorus absorption. Aquadetrim Vitamin D₃ aqueous solution

(cholecalciferol, 15,000 IU/mL; Medana Pharma S.A., Poland) was given orally at 0.000297 mL per 100 g of body weight, corresponding to 4.45 IU of vitamin D₃, to support calcium and phosphate metabolism, skeletal mineralisation and growth, and immune function. Forteza 0.15 % oral spray (benzylamine hydrochloride, 1.5 mg/mL; Abdi Ibrahim İlaç Sanayi ve Ticaret A.Ş., Istanbul, Türkiye) was administered at 0.3 mL per 200 g of body weight for its analgesic, anti-inflammatory, antieudative and disinfectant properties.

At the end of the experiment, the animals were euthanised on day 33 by total cardiac exsanguination under thiopental anaesthesia (20 mg/kg).

The alveolar processes were removed from the mandibles. Acid and alkaline phosphatase activities were determined in alveolar bone homogenates as markers of resorption and bone formation, and mineralisation was assessed by calcium content using the methods described in [3]. The homogenates contained 75 mg of tissue per mL of 0.1 M citrate buffer, pH 6.1. The mineralisation index (MI) was calculated as the ratio of alkaline phosphatase (ALP) to acid phosphatase (ACP) activity.

The results were processed using Microsoft Excel 2010. Differences between group means were analysed using Student's t-test after checking conformity of the distributions in the groups with a normal distribution. Differences were considered statistically significant at $p < 0.05$ [2, 10].

Results of the study. Figure 1 presents the degree of atrophy of the mandibular alveolar process in rats. Dietary vitamin D deficiency combined with bone destruction caused substantial changes in the periodontal tissues. In Group 2 (pathology + CD), the degree of mandibular alveolar atrophy increased by 7.5 % ($p < 0.05$).

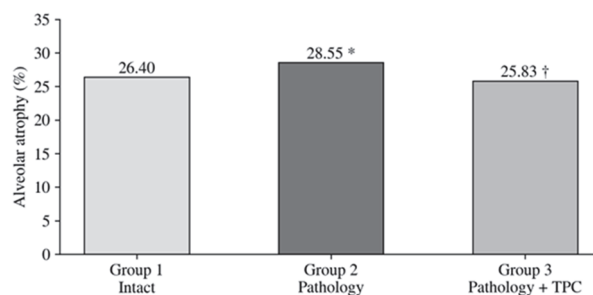


Fig. 1. Effect of the TPC on the degree of mandibular alveolar atrophy in rats under conditions of a cariogenic diet and dietary vitamin D deficiency against a background of bone destruction. * $p < 0.05$ compared with the intact group; † $p < 0.05$ compared with Group 2.

Administration of the TPC significantly reduced the degree of mandibular alveolar atrophy by 9.5 % compared with Group 2 ($p < 0.05$).

The next stage involved determination of markers of bone resorption and remodelling in rat jaws in relation to TPC administration. Dietary vitamin D deficiency was accompanied by an increase in ACP activity, a marker of bone resorption (Table 1).

Table 1

Effect of the therapeutic and prophylactic complex on mineral metabolism in rat bone tissue under conditions of a cariogenic diet and dietary vitamin D deficiency against a background of bone destruction, M±m

Groups	ALP activity, $\mu\text{kat/kg}$	ACP activity, $\mu\text{kat/kg}$	MI ALP/ACP	Calcium, mol/kg
Group 1 Intact	44.82±2.17	6.20±0.30	7.22±0.31	3.04±0.12
Group 2 Pathology + CD	30.87±1.84 $p<0.001$	9.40±0.42 $p<0.001$	3.28±0.19 $p<0.001$	2.64±0.10 $p<0.02$
Group 3 Pathology + CD + TPC	46.90±2.23 $p>0.5$ $p_1<0.001$	6.84±0.28 $p>0.5$ $p_1<0.001$	6.85±0.27 $p>0.4$ $p_1<0.001$	3.30±0.17 $p>0.25$ $p_1<0.002$

Note: p – significance of differences compared with the intact group; p_1 – significance of differences compared with Group 2 (pathology + CD); alveolar process homogenates: 75 mg/mL in 0.1 M citrate buffer, pH 6.1; n=10

ACP activity in the jawbone tissue of Group 2 rats was 34 % higher than in intact animals ($p<0.001$). Administration of the TPC to Group 3 animals reduced the elevated bone ACP activity by 27.23 % ($p_1<0.001$).

The other parameters studied in the jawbone tissue of Group 2 animals decreased: ALP activity by 31.12 % and calcium content by 13.16 %. These differences were statistically significant. The decreases in these bone tissue parameters were associated with greater alveolar atrophy in the rats.

The TPC positively affected mineral metabolism in the alveolar bone under conditions of bone destruction and dietary vitamin D deficiency. ALP activity in Group 3 increased by 34.2 % compared with Group 2 ($p_1<0.001$), and calcium content increased by 20 % ($p_1<0.002$).

The imbalance in mineralisation processes, particularly bone phosphatase activities, under conditions of bone destruction and dietary vitamin D deficiency was reflected by the ALP/ACP ratio. This ratio was 7.22 in intact rats and decreased to approximately 3.3 in Group 2. Analysis of the ratio indicated a predominance of bone resorption over bone formation, with a 54.6 % decrease relative to intact animals.

Discussion. The present findings demonstrate that mandibular injury under conditions of a cariogenic diet and dietary vitamin D restriction is accompanied by disturbances in alveolar bone metabolism. Increased ACP activity, together with reduced ALP activity, calcium content and MI, indicates an unfavourable relationship between tissue resorption and mineralisation. The increase in alveolar atrophy provides a complementary structural measure of these changes. This interpretation agrees with the role of altered bone remodelling in alveolar destruction described by Usui et al. [15], while taking into account the different nature of the experimentally induced injury.

The reduction in tissue calcium is consistent with impaired maintenance of the mineral component of alveolar bone. Vitamin D contributes to calcium–phosphate homeostasis and supports the conditions required for skeletal mineralisation [13]. Its restricted dietary supply may therefore influence the response

to local injury. In the present model, this factor acted together with a cariogenic diet and mandibular trauma, and the observed changes characterise their combined effect.

The direction of the biochemical changes agrees with the experimental observations of Dienha et al. [5], who reported increased ACP activity, reduced ALP activity and lower calcium content in the alveolar processes of rats receiving a high-sugar diet. Studies of osteotropic preparations [6] and biologically active matrices [1] also support the relevance of assessing bone remodelling during the repair of jawbone defects. These findings provide a basis for evaluating approaches that combine correction of metabolic disturbances with support of local tissue recovery.

Administration of the proposed complex improved the biochemical profile of alveolar bone. Compared with untreated pathology, ACP activity decreased by 27.23 %, while ALP activity and calcium content increased by 51.93 % and 25.00 %, respectively; MI increased from 3.28 to 6.85. The concurrent reduction in alveolar atrophy supports a favourable effect on bone preservation. The calcium- and vitamin D-containing components provide a rationale for metabolic support, whereas the locally acting components were included to modify the inflammatory environment. The findings relate to the combined regimen, since its individual components were not assessed separately.

The results support further investigation of metabolic and local measures as complementary approaches to jawbone recovery under unfavourable nutritional conditions. For chronic apical periodontitis, control of the endodontic source of infection remains essential [11]. The experimental findings may inform subsequent studies of supportive treatment, with morphological assessment and longer observation helping to clarify the extent and persistence of bone repair.

Limitations. The study focused on biochemical parameters and alveolar atrophy assessed at the end of the experiment. Further evaluation of the morphological features of bone repair would complement the interpretation of the observed changes.

Conclusions

1. Bone destruction against a background of dietary vitamin D deficiency and a cariogenic diet in laboratory animals resulted in a 31.12 % decrease in ALP activity and a 13.16 % decrease in calcium content, indicating increased resorption and reduced bone formation.

2. Dietary vitamin D deficiency was accompanied by a 1.5-fold increase in ACP activity in the alveolar process, a marker associated with bone resorption.

3. The therapeutic and prophylactic complex helped maintain bone remodelling markers at levels close to those observed in intact animals.

Further research should focus on developing an effective method for preventing complications and accelerating healing in chronic destructive periodontitis according to serum vitamin D levels, with the use of an appropriate therapeutic and prophylactic complex.

References

1. Vyshnevskaya HO, Kakabadze ZSh, Shnaider SA. Vidnovlennia kistkovoho parodontalnoho defektu biolohichno aktyvnym matryksom z hialuronovoiu kyslotoiu ta trombotsytarnymy faktoramy rostu v eksperymenty. Visnyk stomatolohii. 2020;36(2):10–16. DOI: 10.35220/2078-8916-2020-36-2-10-16. [in Ukrainian].
2. Holovanova IA, Bielikova IV, Liakhova NO. Osnovy medychnoi statystyky: navchalnyi posibnyk dlia aspirantiv ta klinichnykh ordynatoriv. Poltava; 2017. 113 p. [in Ukrainian].
3. Hubskey YuI, Nizhenkovska IV, Korda MM, et al. Biolohichna i bioorhanichna khimii: pidruchnyk. Vol. 2, Biolohichna khimii. Hubskey YuI, Nizhenkovska IV, editors. Kyiv: Medytsyna; 2016. p. 497–506. [in Ukrainian].
4. Danylevskiy MF, Borysenko AV. Terapevtychna stomatolohii: pidruchnyk. Vol. 2, Kariies. Pulpit. Periodontyt. Rotovyi sepsys. Borysenko AV, editor. 4th ed., revised and expanded. Kyiv: Medytsyna; 2026. 327 p. [in Ukrainian].
5. Dienha OV, Kozoriz VV, Khodakov IV, Nepriakhina OV, Tsymbaliuk OG. Korektsiia biokhimichnykh pokaznykiv shchelep ta krovi shchuriv likuvalno-profilaktychnym kompleksom za umov modeliuvannia kariiesu zubiv. Visnyk Vinnytskoho natsionalnoho medychnoho universytetu. 2025;29(2):178–182. DOI: 10.31393/reports-vnmedical-2025-29(2)-01. [in Ukrainian].
6. Lysokon YuYu. Dynamika markeriv remodeliuvannia kistkovoї tkanyny u eksperymentalnykh tvaryn pry zastosuvanni osteotropnykh preparativ dlia vidnovlennia kistkovykh defektiv. Visnyk problem biolohii i medytsyny. 2020;(3):336–340. DOI: 10.29254/2077-4214-2020-3-157-336-340. [in Ukrainian].
7. Pykaliuk VS, Mostovyi SO, Lavreniuk VYe. Strukturni osoblyvosti kistkovoї tkanyny nyzhnoi shchelepy. Klinichna anatomiia ta operatyvna khirurhiia. 2023;22(1):76–83. DOI: 10.24061/1727-0847.22.1.2023.11. [in Ukrainian].
8. Ponyrko AO, Riabenko TV. Porivnialna kharakterystyka stanu kistkovoї tkanyny u shchuriv riznykh vikovykh hrup za umov indukovanoi hiperhliemii. Ukrainskyi zhurnal medytsyny, biolohii ta sportu. 2019;4(5):67–71. DOI: 10.26693/jmbs04.05.067. [in Ukrainian].
9. Rybak VA, Kopchak AV, Pavlychuk TO, Shnaider SA. Kliniko-rentgenolohichni osoblyvosti remodeliuvannia autolohichnykh kistkovykh transplantativ ta ksenohennykh kistkovo-zamishchuiuchykh materialiv u patsientiv z defektamy kistok lytsevoho cherepu pry zastosuvanni plazmy zbahachenoi faktoramy rostu. Visnyk stomatolohii. 2018;(3):65–75. [in Ukrainian].
10. Rohach IM, Keretsman AO, Sitkar AD. Pravylny vybranny metod statystychnoho analizu – shlyakh do yakisnoyi interpretatsiyi danykh medychnykh doslidzhen. Naukovy visnyk Uzhhorodskoho universytetu, seriya “Medytsyna”. 2017;2(56):124–128. [in Ukrainian].
11. Fomenko YuV, Shapovalova AS. Suchasnyi pidkhid u likuvanni periodontytu pry povtornomu endodontychnomu vtruchanni. Kryterii uspikhu. Kharkivskyi stomatolohichniy zhurnal. 2024;1(2):197–206. DOI: 10.26565/3083-5607-2024-2-09. [in Ukrainian].
12. Khodakov IV, Khromahina LM, Makarenko OA, Mudryk LM. Modyfikatsiia kazeino-sakharoznoi diiety M.S. Buhaiovoi ta S.A. Nikitina (1954) dlia modeliuvannia kariiesu zubiv u shchuriv. Visnyk stomatolohii. 2023;47(1):71–76. DOI: 10.35220/2078-8916-2023-47-1.12. [in Ukrainian].
13. Bouillon R, Marrocci C, Carmeliet G, Bikle D, White JH, Dawson-Hughes B, Lips P, Munns CF, Lazaretti-Castro M, Giustina A, Bilezikian J. Skeletal and extraskeletal actions of vitamin D: current evidence and outstanding questions. Endocr Rev. 2019;40(4):1109–1151. DOI: 10.1210/er.2018-00126.
14. Morimoto J, Senior A, Ruiz K, Wali JA, Pulpitel T, Solon-Biet SM, Cogger VC, Raubenheimer D, Le Couteur DG, Simpson SJ, Eberhard J. Sucrose and starch intake contribute to reduced alveolar bone height in a rodent model of naturally occurring periodontitis. PLoS One. 2019;14(3):e0212796. DOI: 10.1371/journal.pone.0212796.
15. Usui M, Onizuka S, Sato T, Kokabu S, Ariyoshi W, Nakashima K. Mechanism of alveolar bone destruction in periodontitis-periodontal bacteria and inflammation. Jpn Dent Sci Rev. 2021;57:201–208. DOI: 10.1016/j.jdsr.2021.09.005.

Conflict of interest. The authors have no conflicts of interest to declare.

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Article received: 9.07.2025