



## CHANGES IN TOOTH HARD TISSUES AND PERIODONTAL TISSUES DURING ORTHODONTIC TOOTH MOVEMENTS IN RATS WITH EXPERIMENTAL GASTRITIS

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### Abstract

*The purpose of the work is to determine changes in tooth hard tissues, gum tissue, bone tissue of the alveolar ridge of the jaw using a model of orthodontic tooth movement in rats with experimental alimentary gastritis.*

*Studies were performed on 25 white Wistar rats. Assessed the condition of the teeth (the number and depth of the carious cavities), the degree of atrophy of the alveolar ridge of the jaw. Biochemical methods were used to determine elastase activity and total proteolytic activity, alkaline and acid phosphatase in alveolar bone homogenates, calcium and phosphorus concentrations with the calculation of the ratio of calcium to phosphates, malondialdehyde content and catalase activity were determined in gum homogenates, inflammation markers (elastase activity total proteolytic activity).*

*Orthodontic intervention in rats with simulated gastritis contributed to a significant increase in the intensity of caries and the severity of the carious process, an increase in the degree of atrophy of the alveolar ridge of the jaw. A significant decrease in the total proteolytic activity was formed in the jaw bone tissue, an increase in the activity of acid and alkaline phosphatase was noted. In the gum tissue, a sharp intensification of lipid peroxidation (an increase in the content of malondialdehyde) and the simultaneous suppression of antioxidant protection by indicators of catalase activity were determined.*

**KEYWORDS:** orthodontic tooth movement, bone metabolism, markers of inflammation, gums, gastritis.

### INTRODUCTION

In the last decade, there has been a tendency to a decrease in the level of both dental and somatic health in the child population [Polunina N, 2013]. Moreover, according to WHO, the prevalence of dentofacial anomalies is an average of 50%.

A significant part of the anomalies of the dentition is accompanied by local changes in the bone tissue of the alveolar bone and osteoporosis, local circulatory disorders, tissue respiration, traumatic occlusion [Domenyuk DA et al., 2016].

In the practice of modern orthodontics, the use of fixed equipment occupies a leading position. However, orthodontic treatment with the help of

bracket systems, according to many researchers, disrupts the homeostasis of the oral fluid, worsens the hygienic condition of the organs and tissues of the oral cavity [Kalashnikova T., Vasiliev M, 2019], reduces the functional resistance of hard tooth tissues [Serova A, 2017], can contribute to inflammatory changes in periodontal tissues [Manuelli M et al., 2019; Meng S et al., 2019].

And these negative phenomena can be potentiated if the patient has common diseases of the body. In practical health care, the provision of dental care to patients with comorbid pathology is a serious medical and social problem due to the significant frequency of complications and the lack of effectiveness of treatment and preventive measures.

There is evidence that with orthodontic intervention in the presence of background pathology, there is a high risk of complications, such as focal demineralization of enamel, inflammatory, inflam-

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matory-dystrophic changes in periodontal tissues, fenestration and digestion alveolar bone, root resorption. [[Patel A et al., 2009; Van Beek H, 2009; Kolesnik K et al., 2014]

Our attention was attracted by official statistics, which demonstrate that in the structure of somatic pathology in children and adolescents, digestive organs diseases take the second or third place in primary and general morbidity.

Such indicators are due to the action of many factors on the child's body - poor nutrition, an increase in the proportion of low-quality and genetically modified foods in the diet, physical inactivity, stress, bad habits, environmental triggers that deplete compensatory-adaptive systems.

Analysis of literature data indicates an increase in the prevalence and intensity of dental caries, the frequency of inflammatory diseases of periodontal tissues, oral mucosa in children and adolescents with gastroduodenal pathology [Gontarev S et al., 2017; Shtompel A, 2018]. In patients with dentoalveolar anomalies against the background of digestive tract pathology, bone remodeling dysfunction was revealed, which was manifested by a decrease in the formation marker and an increase in bone resorption markers [Kolesnik K et al., 2017]. Therefore, the study of the interference of orthodontic treatment on tooth hard tissues, periodontal tissues, bone modeling of the alveolar ridge of the jaw in adolescents with diseases of the digestive system remains relevant. To address these issues, experimental studies are needed. It was established that in rats, when using highly refined refined products, morphological changes in the main structural components of the gastric mucosa are formed: a decrease in the length of their own glands, thinning of the muscle plate, a decrease in the volume of nuclei and cytoplasm of exocrinocytes [Kalinichenko, Yu et al., 2012].

The aim of our study is to study the effect of orthodontic tooth movement on the state of hard tooth tissues, gum tissue, bone tissue of the alveolar ridge of the jaws

rats with experimental experimental alimentary gastritis.

An experimental study was performed on 25 white Wistar rats (males and females) of herd breeding. Initially, an alimentary model of gastritis was reproduced in 20 rats, which from 21 days of age were on the Stefan diet - a refined high-sugar diet with an excess of refined sugar and fats and a deficiency in calcium, phosphorus and protein.

In 10 rats of 3 months of age (early maturity) with experimental gastritis, orthodontic tooth movement was modeled. Under thiopental anesthesia, using rotary expanders, they provided access to the incisors of the experimental animal, and the incisors of the upper jaw were cleaned using a swab moistened with a 3% hydrogen peroxide solution. The enamel of the upper incisors was etched using the standard method using 37% phosphoric acid for 30 seconds. The etched surface was washed with a stream of water for 30-40 seconds, followed by drying, preventing liquid from entering the respiratory tract. An adhesive system was applied to the vestibular surface of the incisors, followed by photopolymerization for 20 s. A photopolymer material was applied onto the treated surface and a nickel-titanium closing spring was fixed using a steel ligature with a diameter of 0.2 mm [Ren Y et al., 2003].

The following experiment series were identified:

Group 1 (n = 10) - rats with experimental experimental alimentary gastritis;

Group 2 (n = 10) - rats with experimental alimentary gastritis and orthodontic tooth movement.

The control group (n = 5) consisted of intact animals on the standard diet of vivarium. Animals were removed from the experiment under thiopental anesthesia by opening the vessels of the heart. In carrying out the experimental research, they were guided by the requirements of the "European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes", adopted in Strasbourg, 1986.

In rats of the experimental groups, the condition of the teeth (the number and depth of carious cavities), the degree of atrophy of the alveolar process of the lower jaw [Nikolaeva A, Rozovskaya E, 1965] in the bone homogenates were used to determine the activity of proteases - elastase and total proteolytic activity; activity of bone phosphatases



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- alkaline (alkaline phosphatase) and acid phosphatase [Levitsky A et al., 2005]; the concentration of calcium and phosphorus with the calculation of the ratio of the concentration of calcium to phosphates (Ca/P). In gum homogenates, the content was determined malondialdehyde (malondialdehyde) and catalase activity, as well as markers of inflammation (elastase activity and total proteolytic activity) [Levitsky A et al., 2010].

### RESULTS AND DISCUSSION

The results of the study demonstrated the activation of the carious process in animals with experimental alimentary gastritis. An increase in the number of carious cavities by an average of 2 times ( $p < 0.05$ ) and a depth of caries damage by an average of 2.4 times ( $p < 0.001$ ) was observed compared with intact animals. Orthodontic intervention in rats with a simulated gastric pathology significantly increased the intensity of caries from  $2.4 \pm 0.7$  to  $5.7 \pm 0.3$  ( $p < 0.001$ ) and the severity of the carious process ( $p < 0.001$ ). In rats with experimental alimentary gastritis, a significant increase in the degree of atrophy of the alveolar process of the jaw was observed up to  $16.4 \pm 0.3\%$  ( $p < 0.05$ ) compared with the control group ( $14.3 \pm 0.9\%$ ). Under the influence of orthodontic forces, an increase in the processes of resorption in the bone tissue of the jaws was also noted, which was visualized by a decrease in the alveolar bone with its osteometric assessment by  $15.4\%$  ( $p < 0.05$ ) compared with intact animals. The recorded atrophic changes in the tissues of the alveolar ridge of the jaw in rats of the experimental groups prompted us to study the state of protein and mineral metabolism in this structure. In scientific

research Levitsky A.P. and co-authors Proved that the activity of the proteinases involved in the transformation of collagen into collagen correlates with the level of total proteolytic activity, and the activity of collagenolytic enzymes correlates with the level of bone elastase activity [Levitsky A et al., 2006].

In our study, to assess the state of the alveolar ridge of the jawbones of rats, we studied the dynamics of changes in the activity of these protease groups. The results of the study are summarized in table 1.

It was determined that in rats with experimental alimentary gastritis a significant decrease in total proteolytic activity was formed in the bone tissue of the jaws ( $p < 0.05$ ), which indicated inhibition of the synthesis of the organic matrix of animal bone tissue. Under the influence of the power module, against the background of the reproduced pathology of the stomach, a more significant decrease in total proteolytic activity in the bone tissue of the alveolar process of the jaws was observed ( $p < 0.01$ ). experimental alimentary gastritis modeling was accompanied by increased hydrolysis of the protein base of bone tissue, as evidenced by a significant ( $p < 0.01$ ) increase in bone elastase activity by 1.5 times compared with the control group. Moreover, the destruction of bone collagen in experimental animals was enhanced by orthodontic tooth movements and the highest values of elastase activity were recorded in this group of rats -  $8.31 \pm 0.64 \mu\text{at} / \text{kg}$  ( $p < 0.01$ ).

In addition to the revealed abnormalities in protein metabolism, the modeling of pathology also led to disruption of bone mineralization processes, which could be judged by the activity of phosphatases in the bone tissue of the jaws (table 1). Bone tissue acts as a depot of mineral components, their

TABLE 1

The influence of experimental alimentary gastritis and orthodontic intervention on the activity of proteases and phosphatases of rat alveolar bone.

| Groups                                           | Total proteolytic, $\text{nkcat/g}$               | Elastase, $\text{mkcat/kg}$                   | Alkaline phosphatase, $\text{mkkat/g}$          | Acid phosphatase, $\text{mkkat/g}$             |
|--------------------------------------------------|---------------------------------------------------|-----------------------------------------------|-------------------------------------------------|------------------------------------------------|
| Control                                          | $496.03 \pm 26.19$                                | $5.59 \pm 0.57$                               | $84.07 \pm 2.95$                                | $3.53 \pm 0.21$                                |
| Rats with experimental alimentary gastritis      | $407.06 \pm 18.21$<br>$p < 0.02$                  | $8.24 \pm 0.61$<br>$p < 0.01$                 | $112.37 \pm 4.7$<br>$p < 0.001$                 | $5.9 \pm 0.49$<br>$p < 0.001$                  |
| Rats with OTM+ experimental alimentary gastritis | $342.52 \pm 13.08$<br>$p < 0.001$<br>$p_1 < 0.02$ | $8.31 \pm 0.64$<br>$p < 0.01$<br>$p_1 > 0.05$ | $128.3 \pm 6.15$<br>$p < 0.001$<br>$p_1 > 0.05$ | $7.11 \pm 0.25$<br>$p < 0.001$<br>$p_1 < 0.05$ |

NOTES:  $p$  - significance of differences from indicators of intact rats;  $p_1$  - significance of differences from rats with experimental alimentary gastritis

insufficient intake with food provides the release of these elements from bone hydroxyapatite [Merriam *et al.*, 2000; Volkova *L.*, 2015].

The destruction of hydroxyapatite occurs under the influence of CF of osteoclasts. In rats with experimental alimentary gastritis, an increase in acid phosphatase activity was noted by 25.2% ( $p < 0.001$ ), which indicated an intensification of bone resorption. This process was more pronounced in rats with OMPZ against the background of pathology of the digestive system. In this group of animals, CF activity increased by 34.5% ( $p < 0.001$ ) compared with the intact control.

In parallel with the increase in acid phosphatase activity in the bone tissue of the alveolar ridge of the jaw in rats, which modeled gastritis by an average of 1.7 times ( $p < 0.001$ ) compared with the control group of animals increased alkaline phosphatase activity, which is a marker of the functional activity of osteoblasts. Orthodontic intervention in rats with background pathology contributed to a more significant increase in the activity of alkaline phosphatase - an average of 2 times. Since activation of bone alkaline phosphatase is observed during intensification of mineralization processes, in our study, an increase in the activity of this enzyme can be interpreted as a result of a compensatory reaction to the action of pathological factors.

Reproduction of the pathology of the stomach in rats led to a sharp decrease in the concentration of calcium in the bone tissue of the alveolar bone of the jaw from  $5.18 \pm 0.43$  to  $3.21 \pm 0.41$  mmol / kg ( $p < 0.01$ ). This tendency was intensified under the influence of orthodontic forces, the calcium

content in the alveolar bone homogenates decreased on average 1.7 times ( $p < 0.001$ ) compared with the intact control. Modeling of experimental alimentary gastritis did not have a significant effect on the content of inorganic phosphates in the jaw bone. Only a downward trend in this indicator was determined, but not confirmed statistically. During orthodontic tooth movements in rats with background pathology, the phosphorus concentration in bone tissue significantly decreased from  $3.67 \pm 0.31$  to  $3.18 \pm 0.28$  mol/kg ( $p < 0.05$ ). The absolute values of the level of calcium and inorganic phosphates in the bone tissue do not always reflect the quality of hydroxyapatite. This coefficient of the ratio of the concentration of calcium to phosphates (Ca/P) characterizes this property of the main mineral of bone tissue more clearly. In intact rats, this coefficient was 1.41. Reproduction of gastric pathology reduced this ratio to 1.01, which indicates a decrease in the proportion of calcium in hydroxyapatite crystals and is a negative factor, since the resistance of hydroxyapatite crystals and bone tissue as a whole to adverse effects decreases.

Table 2 shows the effect of orthodontic tooth movements on the background of the experimental alimentary gastritis on the state of the antioxidant-prooxidant system and the activity of inflammation markers in gum homogenates.

The data obtained demonstrate a significant increase in total proteolytic activity in the gingival tissues of rats with experimental alimentary gastritis by 23.3% ( $p < 0.05$ ) and a slight increase in acid phosphatase activity by 15.6% ( $p > 0.05$ ). In rats with experi-

TABLE 2

Indicators of the antioxidant-prooxidant system and markers of inflammation in the gums of rats with experimental alimentary gastritis, a model of orthodontic tooth movement

| Groups                              | Total proteolytic activity, mkat / g           | The activity of acid phosphatase, mkkat / g    | Malondialdehyde content, mmol / kg              | Catalase activity, mkat / kg                   |
|-------------------------------------|------------------------------------------------|------------------------------------------------|-------------------------------------------------|------------------------------------------------|
| Control                             | $205.4 \pm 15.7$                               | $27.24 \pm 2.05$                               | $22.31 \pm 0.96$                                | $13.11 \pm 0.87$                               |
| Rats with alimentary gastritis      | $267.8 \pm 17.9$<br>$p < 0.05$                 | $32.27 \pm 1.63$<br>$p > 0.05$                 | $32.28 \pm 0.89$<br>$p < 0.001$                 | $11.34 \pm 1.05$<br>$p > 0.05$                 |
| Rats with OTM+ alimentary gastritis | $269.9 \pm 11.2$<br>$p < 0.01$<br>$p_1 > 0.05$ | $37.85 \pm 1.57$<br>$p < 0.01$<br>$p_1 < 0.05$ | $51.36 \pm 6.43$<br>$p < 0.001$<br>$p_1 < 0.05$ | $8.22 \pm 0.38$<br>$p < 0.001$<br>$p_1 < 0.05$ |

NOTES:  $p$  - significance of differences from indicators of intact rats;  $p_1$  - significance of differences from rats with experimental alimentary gastritis

mental alimentary gastritis and orthodontic tooth movement, a more pronounced increase in inflammation was observed: total proteolytic activity by 23.9% ( $p < 0.01$ ) and acid phosphatase by 28.1% ( $p < 0.01$ ) in gum homogenates. When reproducing the pathology of the stomach in rats, an increase in the processes of lipid peroxidation in the gum tissues was observed, as evidenced by an increase in the content of malondialdehyde by 1.45 times ( $p < 0.001$ ). At the same time, there was a decrease in the activity of the main antioxidant enzyme - catalase by 13.4%, however, this indicator did not significantly differ from that in intact animals. Orthodontic intervention in rats with experimental alimentary gastritis had a destabilizing effect on the gum cell membranes and significantly affected the balance of the pro-antioxidant system. A sharp increase in the LPO intensity in gum homogenates is noteworthy: the malondialdehyde content increased by 2.3 times ( $p < 0.001$ ) and the simultaneous suppression of antioxidant protection in terms of catalase activity - a decrease of 37.3% ( $p < 0.001$ ).

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## CONCLUSION

In experimental experimental alimentary gastritis, negative changes are observed in the quality of the bone tissue of the alveolar process of the jaw, which is manifested in the development of osteodystrophic processes.

In rats with simulated gastritis, changes in the gum tissue are determined: the development of an inflammatory reaction and the intensification of lipid peroxidation against the background of a decrease in antioxidant defense.

Orthodontic movement of teeth with this background pathology enhances these pathological disorders.

The revealed violations indicate the need for correction of adaptive mechanisms, nonspecific resistance and bone metabolism in adolescents with chronic diseases of the upper gastrointestinal tract during orthodontic treatment.

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