UDC 612.015.3+616.314.18+616-092.9+54615+616-008.9

Structural and metabolic features of rats' dental pulp in cases of diet-related iodine deficiency, insulin resistance, and their combination

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The features of peroxidative oxidation of proteins and lipids in dental pulp homogenate, as well as the histological peculiarities of dental soft tissues under conditions of iodine deficiency, a high-carbohydrate diet, and their combination, were investigated. Limited iodine content in the animals' daily diet is accompanied by activation of peroxidation in the dental pulp (a two- to three-fold increase in pro-oxidant levels compared to indices in intact rats), vascular congestion with stasis, plasma sequestration, and the development of perivascular diapedesis haemorrhages. An excessive intake of carbohydrates leads to a predominant accumulation of protein peroxidation products in the soft tissues of the teeth (an increase in the products of oxidative protein modification by 3.3-7.3 times compared to the control), and a reduction in the area of pulp loose connective tissue compared to baseline data. The associated effect of iodine deficiency and impaired glucose tolerance leads to a significant increase in the intensity of oxygen-dependent reactions in dental pulp (an increase in the content of protein and lipid peroxidation products by 3.6-5.8 times compared with values in animals with isolated iodine deficiency and by 4.3-5.3 times related to the values in rats under a high-carbohydrate diet). The connective tissue of the pulp is characterised by hypercellularity (a reduction of its area per cell by 56.9% compared to the control, and by 49.5% and 54.21% related to the values in animals with isolated iodine deprivation or under conditions of an excessive intake of carbohydrates, respectively). The reaction of the microcirculatory bed is characterized by the accumulation of non-sulphated glycosaminoglycans in the ground substance of the pulp connective tissue. Thus, diet-related disturbances in humoral regulation led to oxidative stress in the dental pulp, which disrupts trophism and increases the risk of pathological tooth loss in the context of changes in the glycaemic profile, particularly against a background of iodine deficiency.

Key words: dental pulp; peroxidation of proteins and lipids; histological changes in dental soft tissues; iodine deficiency; high-carbohydrate diet.

INTRODUCTION

The issue of comorbidity is recognized as a priority in modern medicine [1, 2]. The complex, multifactorial interrelationships among the humoral, nervous, and immune systems play a crucial role in the aetiopathogenesis of many dental diseases [3]. Therefore, the study of functional and structural changes in the hard and soft tissues of the tooth requires an analysis of all aspects of the metabolic imbalance arising against a background of hormonal disturbances. The anatomical and physiological features of the pulp reflect a wide range of pathological changes that may occur not only locally within the dental crown and root canal cavities. The extensive innervation and vascularisation of the soft connective tissue, represented by many cellular elements, ensure the pulp's involvement in the formation of collagen fibers – the main component of connective tissue – and perform dentin-forming, protective, trophic, and sensory functions in relation to all elements of the

tooth [4, 5]. Therefore, the manifestations of pulp damage result from multiple pathogenetic mechanisms.

Given the reduced metabolic activity resulting from limited iodine intake and thyroid hormone deficiency, dystrophic changes may occur in the dental pulp, primarily due to impaired trophism [6, 7]. This leads to reduced blood supply, decreased immunological resistance, and accelerated calcification, sclerosis, and obliteration of the root canals, thereby increasing the risk of pathological tooth loss [8, 9]. At the same time, a high-carbohydrate diet alters the progression of all metabolic pathways, including those in the tooth's soft tissues. Microangiopathy leads to thickening of the pulp capillary walls, narrowing of their lumen, and ischemia. Chronic hypoxia contributes to excessive connective tissue growth and sclerosis of the pulp chamber [10, 11]. Metabolic disturbances, along with neuropathy, increase the area of pulp necrosis, leading to the subsequent generalization of the pathological process [12]. The development of apoptosis and cellular alterations in the dental pulp, occurring against a background of humoral disorders, may be associated with free-radical-mediated oxidation reactions. Disturbances in the dynamic balance of the pro-oxidant-antioxidant system in the context of endocrinopathies lead to the accumulation of reactive oxygen species and metabolites in the soft tissues of the tooth [13, 15, 16]. It is known that free radicals are cytotoxic, altering pulp reactivity and disrupting the mineralization of the hard components of the dentoalveolar complex [4, 5].

The dental manifestations of endocrine diseases have been documented in several fundamental scientific studies [1, 4, 6, 9]; however, the pathogenetic mechanisms underlying the isolated and combined effects of iodine deficiency and a high-carbohydrate diet on the structural and metabolic characteristics of dental soft tissues require further investigation.

Aim - to analyse the intensity of oxidative processes and investigate the histological structure of dental pulp in the case of experimental modelling of iodine deficiency, insulin resistance, and their combination.

METHODS

The study was carried out on sexually mature male rats weighing 150-180 g, which were divided into three experimental groups (30 animals in each): Group 1 - rats in which iodine deficiency was induced; Group 2 - animals that were on a high-carbohydrate diet; Group 3 - rats with combined endocrinopathy (the combined effect of iodine-deficient and high-carbohydrate diets). To induce iodine deficiency, the animals were maintained on a low-iodine diet for 8 weeks [17]. To model insulin resistance, the animals were given a 10% fructose solution instead of drinking water for 8 weeks [17]. Animals of the control group (intact rats, n = 30) were kept on a standard diet and drinking regimen in the vivarium.

The rats were withdrawn from the experiment by decapitation under ketamine anaesthesia (Ketamine, "Farmak JSC", Kyiv, Ukraine, 100 mg/kg body weight intraperitoneally). Thyroid status was determined by the levels of free triiodothyronine (fT_3), thyroxine (fT_4), and thyroid-stimulating hormone (TSH) in blood serum, and the fT_3/fT_4 ratio was calculated. To assess the rats' iodine status, iodine concentration was determined from single urine samples collected using metabolic cages. Carbohydrate metabolism was assessed based on fasting blood glucose level, serum immunoreactive insulin level, and glycated haemoglobin, followed by calculation of the HOMA-IR (Homeostasis Model Assessment of Insulin Resistance) index [18]. Peroxidation processes were investigated in blood serum and dental pulp homogenates by measuring the levels of oxidative protein modification (OPM) products, diene conjugates (DC), and active products reacting with thiobarbituric acid (TBA-AP) [19].

For histological examination, incisors from the upper and lower jaws were extracted,

followed by pulpal extirpation. To obtain a homogenate, the pulp was processed in a porcelain mortar at low temperature with the addition of 0.5 M Tris-HCl buffer solution (pH 7.3). The teeth were fixed in a 10 % neutral formalin solution (pH 7.0). The fixation time was 24 h, after which the teeth underwent acid decalcification for two days. A series of paraffin tissue sections, 4-6 μm thick, was prepared using a sled microtome. The histological sections were stained with haematoxylin and eosin, and alcian blue, by Steedman [20]. Histological examinations were carried out using a Leica DME light microscope (Germany). To ensure the objectivity of the quantitative analyses, computerized morphometry and densitometry of the specimens in the histological slides were performed using a Nikon Coolpix 4500 digital camera. Subsequently, digital copies of the images were analysed using Image Tool 3.0 for Windows. Morphometric analysis of dental pulp was performed, considering the area of loose connective tissue in the dental pulp per cell and the optical density of the ground substance of the loose connective tissue in the dental pulp.

The keeping, feeding, and withdrawal of animals from the experiment were in accordance with the main provisions of the Rules for Carrying Out Work Using Laboratory Animals (1977), the rules of the European Convention for the Protection of Vertebrate Animals Used for Experimental Research and Other Scientific Purposes (1986), EEC Directive No. 609 (1986), Order of the Ministry of Health of Ukraine No. 281 dated November 1, 2000, "On measures to further improve organizational standards for work using experimental animals" and Law of Ukraine No. 3447-IV "About the protection of animals from cruelty", 2006) after the experiment design was approved by the Ethics Committee of Ivano-Frankivsk National Medical University (Protocol No. 121/21 dated 13 May 2021).

Statistical data processing was performed using the Excel computer program of the Microsoft Office 365 ProPlus package. For each sample, we assessed whether the distribution of the index under study was normal using the Shapiro-Wilk test. Given that the obtained data followed a Gaussian distribution, the results were consistent within an interval of $M \pm m$. The Student's t-test was used to assess the reliability of differences between groups. A difference in parameters was considered statistically reliable at $P < 0.05$.

RESULTS AND DISCUSSION

Humoral-associated metabolic disorders altered the pro-oxidant balance in the examined tissues. Thus, under conditions of insufficient iodine intake in animals of the first experimental group, activation of peroxidation processes was observed (Table 1), mainly in the dental pulp homogenate, due to intensified free-radical destruction of protein substrates. This manifested as an increase in the content of most OMP fractions: E_{356} – twice ($P < 0.001$), E_{370} – by 2.75 times ($P < 0.02$), E_{430} – by 2.13 times ($P < 0.001$) compared with the corresponding values in intact rats. However, the opposite changes in blood serum OMP levels in the animals of this group are noteworthy, namely a reliable decrease in OMP content: E_{356} – by 82.52%, E_{370} – by 79.34%, E_{430} – by 77.69% compared to the control.

Analysis of oxygen-dependent destruction of lipid biomolecules in animals with iodine deficiency revealed a reliable activation of lipid peroxidation (LP) only in dental pulp homogenates, where the levels of DC and TBA-AP had doubled ($P < 0.001$) compared with similar indexes in intact animals (Table 2).

The reduction in the intensity of peroxidation processes in the blood serum of animals on a diet with restricted iodine content can be explained by slowed metabolism associated with the development of iodine deficiency [6, 7]. Conversely, the structural features, high baseline metabolic activity, and pronounced hemodynamic circulation of the tooth's soft tissues create conditions for the accumulation of free-radical oxidation products of bio

Table 1. The content of oxidized protein modification products in dental pulp and blood serum of intact rats, animals with iodine deficiency, insulin resistance, and their combination (M ± m)

Research design	E_{356}, nm	E_{370}, nm	E_{430}, nm	E_{530}, nm
		Dental pulp		
Intact animals (control group)	0.100 ± 0.009	0.080 ± 0.040	0.080 ± 0.008	–
Iodine deficiency (Group 1)	0.2000 ± 0.004****	0.220 ± 0.003**	0.170 ± 0.002****	0.150 ± 0.005
Insulin resistance (Group 2)	0.590 ± 0.160* P_{1-2}<0.05	0.580 ± 0.150** P_{1-2}<0.05	0.260 ± 0.060*	0.040 ± 0.010 P_{1-2} < 0.001
Combined endocrinopathy (insulin resistance on the background of iodine deficiency, Group 3)	0.720 ± 0.040**** P_{1-3} < 0.001	0.220 ± 0.040*	0.460 ± 0.030**** P_{1-3} < 0.001 P_{2-3} < 0.05	0.010 ± 0.002 P_{2-3} < 0.05
		Blood serum		
Intact animals (control group)	3.09 ± 0.82	3.05 ± 0.79	1.21 ± 0.47	0.14 ± 0.05
Iodine deficiency (Group 1)	0.54 ± 0.25****	0.63 ± 0.21***	0.27 ± 0.09**	0.05 ± 0.03
Insulin resistance (Group 2)	1.21 ± 0.75	1.18 ± 0.55	0.47 ± 0.18	0.06 ± 0.01
Combined endocrinopathy (insulin resistance on the background of iodine deficiency, Group 3)	0.91 ± 0.35***	0.97 ± 0.12*	0.44 ± 0.05*	0.08 ± 0.02

Note: Here and in the following tables, *P < 0.05; **P < 0.02; ***P < 0.01; ****P < 0.001 - a reliable difference between the indexes for similar values in intact animals.

substrates in the pulp. This intensifies cellular sensitization, promotes the development of inflammatory oedema and vascular constriction, leading to subsequent necrosis of all elements of the pulp chamber.

Manifestations of oxidative stress were also detected in animals maintained on a high-carbohydrate diet. In particular, in rats of the second experimental group, a predominant activation of peroxidative protein degradation in the soft tissues of the teeth was observed (see Table 1): the content of the E_{356} fraction increased by 5.90 times, E_{370} – by 7.25 times, and E_{430} – by 3.25 times (P < 0.05) compared with baseline values.

Changes in lipoperoxidation processes under conditions of a high fructose diet were characterized by polarity (see Table 2). In the dental pulp homogenate of animals with insulin resistance, the DC content decreased by 50.00% against a background of an increase in TBC-AP content by 69.44 % (P < 0.001) compared with the values in intact rats. It is worth noting that in the blood serum of animals in this experimental group, only the final product of LP showed a reliable change, exceeding the baseline index by 72.28%.

It is known that advanced glycation end-products, which are intensively formed and accumulate during persistent hyperglycaemia, disrupt the biological properties of protein molecules [5, 16]. The predominant activation of oxidative protein degradation in dental homogenates from animals with impaired glucose utilization adversely affects pulp homeostasis and exacerbates changes in its structural organization.

The association between iodine deficiency and impaired glucose tolerance often results in a synergistic effect, in which pathogenic

Table 2. Content of diene conjugates and thiobarbituric acid-reactive products in dental pulp and blood serum of intact rats, animals with iodine deficiency, insulin resistance, and their combination (M ± m)

Research design	Diene conjugates, cu/ml (homogenate, blood serum)	
	Dental pulp	Blood serum
Intact animals (control group)	0.060 ± 0.004	0.420 ± 0.130
Iodine deficiency (Group 1)	0.120 ± 0.001****	0.280 ± 0.120
Insulin resistance (Group 2)	0.030 ± 0.002****	0.340 ± 0.140
Combined endocrinopathy (insulin resistance on the background of iodine deficiency, Group 3)	0.130 ± 0.010**** $P_{2-3} < 0.001$	0.540 ± 0.201
Substances reacting with thiobarbituric acid, nmol/ml (homogenate, blood serum)		
Intact animals (control group)	0.36 ± 0.02	3.03 ± 0.71
Iodine deficiency (Group 1)	0.72 ± 0.04****	4.76 ± 0.65
Insulin resistance (Group 2)	0.61 ± 0.02****	5.22 ± 0.16**
Combined endocrinopathy (insulin resistance on the background of iodine deficiency, Group 3)	2.27 ± 0.82*	14.01 ± 0.13**** $P_{1-3} < 0.001$ $P_{2-3} < 0.001$

Note: Here and in the following tables, *P < 0.05; **P < 0.02; ****P < 0.001 – a reliable difference between the indexes for similar values in intact animals.

mechanisms overlap, mask the disease course, and modulate the progression of pathological processes [1, 15]. On the other hand, this combination creates the conditions for the development of a strong reaction, particularly against a background of oxidative stress, which develops in the context of both thyroid and carbohydrate imbalances [3, 16]. Thus, modelling of insulin resistance in combination with iodine deficiency led to a more pronounced intensification of protein peroxidation in dental pulp, accompanied by a simultaneous slowing of this process in blood serum (see Table 1). In particular, in the dental pulp of rats in the third experimental group, a reliable increase in the content of most OMP fractions was observed: E_{356} by 7.20 times, E_{370} by 2.75 times, and E_{430} by 5.75 times, respectively, compared with the values of the control group. Noteworthy are the opposite changes in the content of OMP in the blood serum of animals in this group, which confirms a reliable decrease in the content of E_{356} – by 70.55%, E_{370} – by 68.20%, and E_{430} by 63.64% in relation to the similar indexes in animals that were on a standard diet.

Humoral disorders with a combined aetiology were also accompanied by marked activation of LP across all examined tissues (see Table 2). In particular, in the dental pulp of animals in the third experimental group, the content of DC increased by 2.17 times (P < 0.001) and TBA-AP by 6.31 times (P < 0.05), respectively, compared to the control values. At the same time, in the blood serum of these animals, only an increased content of the end product of lipoperoxidation by 4.62 times (P < 0.001) was observed in relation to baseline values.

A comparative analysis of free-radical oxidation of proteins in the studied tissues of animals with isolated iodine deficiency and impaired glucose tolerance against a background of iodine deprivation revealed a predominant intensification of oxygen-dependent processes under conditions of combined endocrine pathology (see Table 1). Thus, in the dental pulp of rats in the third experimental group, the content of the E_{356} fraction increased by 3.60 times, and the E_{430} fraction by 5.75 times ($P_{1-3} < 0.001$) compared with the corresponding indexes in animals with iodine deficit. However,

analysis of these processes in the tissues of animals in the second and third experimental groups revealed multidirectional changes. In particular, in the dental pulp of rats that were on a diet with excessive carbohydrate content against a background of iodine deficiency, a reliable increase in the content of E_{430} fraction was observed, by 76.92%, with a simultaneous decrease in the content of E_{530} fraction, by 75.00 %, in relation to similar indexes in animals with isolated high-carbohydrate loading.

A comparative study of lipid peroxidation in rats with monoiodine deficiency and combined endocrine pathology revealed increased lipid peroxide formation under conditions of combined hormone-associated disorder (see Table 2). In particular, in the blood serum of rats with isolated iodine deficiency, the intensification of LP occurred mainly due to end products, the content of which in rats with combined endocrine disorder was 2.94 times higher ($P_{1-3} < 0.001$) in relation to the corresponding indices in animals of the first experimental group. At the same time, a comparison of oxidation markers in the tissues of the second and third experimental groups revealed reliable changes not only in blood serum but also in dental pulp homogenate. This is indicated by a 4.33-fold ($P_{2-3} < 0.001$) increase in DC content in the dental pulp of rats with combined endocrinopathy compared with similar indexes in animals maintained on a high-carbohydrate diet. It is worth noting that in the blood serum of animals with impaired glucose tolerance against a background of iodine deficiency, only the TBA-AP content increased, becoming 2.68 times higher ($P_{2-3} < 0.001$) in comparison to the corresponding index in rats of the second experimental group. The intensification of peroxidation processes under these experimental conditions is due to the accumulation of pro-oxidants in the soft tissues of the tooth, their homolytic degradation, and the subsequent destructive effect on the macromolecules of the entire periodontium components.

A morphological light-microscopic examination of the dental pulp was performed to analyse its structural features in animals with endocrine disorders. Modelling of iodine deficiency was accompanied not only by biochemical changes in the pulp homogenate but also manifested as a pronounced reaction of its microcirculatory bed in the form of hyperaemia of the vessels with stasis, plasma sequestration (Fig. 1), and the development of perivascular diapedesis haemorrhages in certain areas. An increase in pulp connective tissue cells was observed, due to an increase in macrophages with isolated, and occasionally moderate, numbers of granulocytes. According to the morphometric examination data (Table 3), the connective tissue area per cell was 358.49 ± 7.85 μm², which was 14.65% ($P < 0.001$) lower than the control value.

In addition, a decrease in the optical density of the ground substance of the pulp connective tissue, stained with alcian blue according to Steedman, was observed, to 163.00 ± 2.08 units, which was by 4.32 % ($P < 0.05$) lower than the corresponding index in animals of the control group.

Keeping animals on a high-carbohydrate diet also led to histological restructuring of the examined tissues. Thus, in the root canal

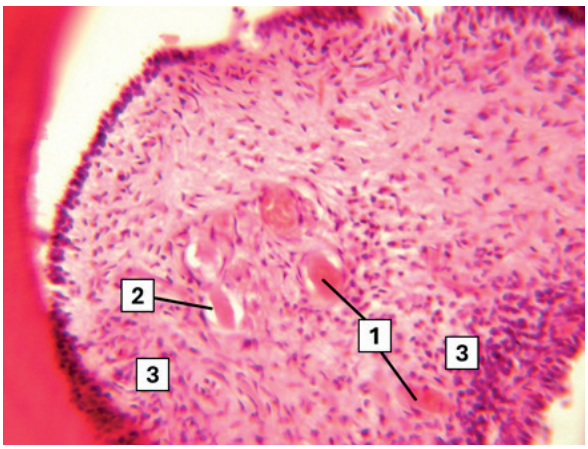

Fig. 1. Radicular pulp of the tooth of a rat under conditions of iodine deficiency. Staining: haematoxylin and eosin. 400×: 1 – stasis in the vessels of the microcirculatory bed, 2 – plasma sequestration, 3 – leukocyte-macrophage focal infiltration

Table 3. Morphometric characteristics of dental pulp of intact rats, animals with iodine deficiency, insulin resistance, and their combination (M ± m)

Research design	Area of loose connective tissue in the pulp per cell, μm²/cell	The optical density of the pulp matrix, units
Intact animals (control group)	420.00 ± 6.29	170.88 ± 2.53
Iodine deficiency (Group 1)	358.49 ± 7.85****	163.00 ± 2.08*
Insulin resistance (Group 2)	395.00 ± 7.11*	170.69 ± 4.33
Combined endocrinopathy (insulin resistance on the background of iodine deficiency, Group 3)	180.89 ± 14.12**** $P_{1-3} < 0.001$ $P_{1-3} < 0.001$	162.88 ± 2.35*

and pulp chamber of the teeth of rats in the second experimental group, the pulp was present, the main part of which consisted of connective tissue with connective tissue fibres, fibroblasts, fibrocytes, isolated macrophages and thin-walled capillary-type vessels filled with erythrocytes (Fig. 2). Reactive changes of the pulp are reflected in a 5.95 % ($P < 0.05$) reduction in the area of pulp loose connective tissue per cell compared with baseline data (see Table 3). At the same time, the optical density of the pulp matrix stained with alcian blue did not differ reliably from that of the control.

A morphological light-microscopic examination of teeth in animals with combined endocrine pathology showed marked changes in the dental pulp. A significant number of macrophages, lymphocytes, fibroblasts, and fibrocytes were observed in the connective tissue of the pulp. There were 180.89 ± 14.12 μm² of connective tissue per cell, which was by 56.93% ($P < 0.001$) less than the corresponding values in the control group. A reaction of the microcirculatory bed was observed in the form of moderately pronounced vascular congestion, as well as a redistribution of non-sulphated glycosaminoglycans towards their accumulation (the optical density of the ground substance was 162.88 ± 2.35 units), which was 4.68% ($P < 0.05$) lower than the similar indexes in animals of the control group (Fig. 3). It is worth noting that the area of loose connective tissue in the pulp

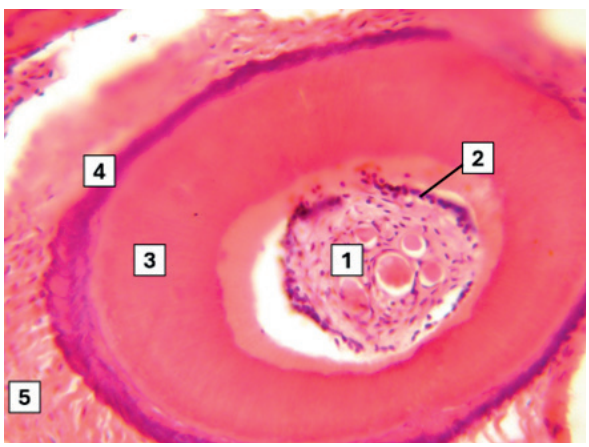

Fig. 2. The root portion of a rat's tooth under conditions of a high-carbohydrate loading. Staining: haematoxylin and eosin. 400×: 1 – pulp with well-vascularised vessels and a small number of cells, 2 – odontoblasts, 3 – dentine, 4 – cementum, 5 – periodontium

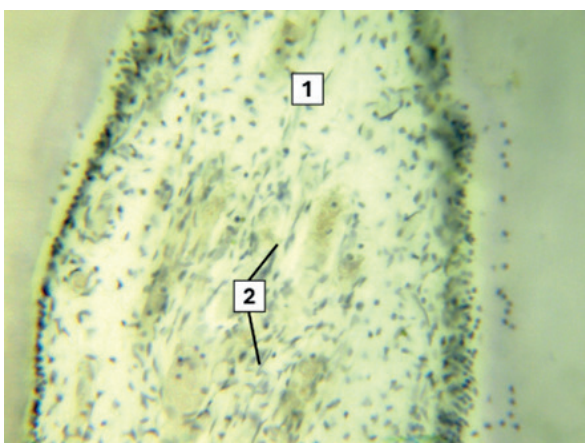

Fig. 3. Dental pulp of a rat under conditions of combined endocrinopathy. Staining: alcian blue according to Steedman. 200×: 1 – glycosaminoglycans in the ground substance of the pulp connective tissue, 2 – vascular congestion of the microcirculatory bed

per cell in rats with insulin resistance against a background of iodine deprivation a reliably decreased by 49.54 and by 54.21%, respectively, in relation to the values in animals with isolated endocrinopathies.

CONCLUSIONS

Disorders of humoral regulation against a background of iodine deficiency and a high-carbohydrate diet led to changes in the dynamic equilibrium of metabolic processes in the soft tissues of the tooth due to excessive generation of free radicals, their accumulation in cellular complexes, and the destabilisation of cell membranes. The development of oxidative stress in the dental pulp of experimental animals has been established, with the most pronounced manifestation of oxidative imbalance occurring in the context of combined endocrinopathy. The accumulation of oxidative protein modifications and lipoperoxidation products leads to changes in pulp architecture, as confirmed by the results of light microscopy. The development of hyperglycaemia promotes the interaction of glycosylated molecules with the receptor apparatus of immunocompetent cells and potentiates immune-inflammatory manifestations in the pulp matrix. The reaction of the microcirculatory bed and the accumulation of non-sulphated glycosaminoglycans in the ground substance of the loose connective tissue of the pulp disrupt the trophism not only of the pulp's own elements but also of the entire tooth. The progression of these disorders may determine the nature and degree of periodontal tissue damage, increasing the risk of pathological tooth loss in the context of an impaired glycaemic profile, particularly against a background of iodine deficiency.

The authors of this study confirm that the research and publication of the results were not associated with any conflicts regarding commercial or financial relations, relations with organizations and/or individuals who may have been related to the study, and interrelations of co-authors of the article.

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СТРУКТУРНО-МЕТАБОЛІЧНІ ОСОБЛИВОСТІ ПУЛЬПИ ЗУБІВ ЩУРІВ ПРИ АЛІМЕНТАРНОЗАЛЕЖНИХ ЙОДОДЕФІЦИТІ, ІНСУЛІНОРЕЗИСТЕНТНОСТІ ТА ЇХ ПОЄДНАННІ

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Досліджували особливості процесів перекисного окиснення білків і ліпідів у гомогенаті пульпи зубів, а також гістологічні особливості м'яких тканин зуба за умов йодної недостатності, високовуглеводної дієти та їх поєднання. Обмежений вміст йоду у добовому раціоні харчування тварин супроводжується активацією пероксидації у пульпі зубів (збільшенням вмісту прооксидантів у два-три рази у порівнянні з показниками інтактних щурів), повнокрів'ям судин зі стазами, секвестрацією плазми, розвитком периваскулярних діапедезних крововиливів. Надмірне поступлення до організму вуглеводів зумовлює переважну кумуляцію продуктів білкової пероксидації у м'яких тканинах зубів (зростання продуктів окисної модифікації білків у 3,3–7,3 раза щодо контролю), зменшення площі пухкої сполучної тканини пульпи у порівнянні з вихідними даними. Асоційований вплив дефіциту йоду та порушеної толерантності до глюкози супроводжується суттєвим зростанням інтенсивності киснезалежних реакцій у пульпі зубів (збільшення вмісту продуктів білкової та ліпідної пероксидації у 3,6–5,8 раза у порівнянні зі значеннями у тварин із ізольованим йоддефіцитом та у 4,3–5,3 раза – щодо показників у щурів на тлі високовуглеводної дієти). Сполучна тканина пульпи характеризується гіперклітинністю (зменшення її площі, що припадає на одну клітину на 56,9% щодо контролю, на 49,5 і 54,21% – щодо значень у тварин із ізольованою йодною депривацією або за умов надмірного споживання фруктози відповідно). Реакція мікроциркуляторного русла характеризується накопиченням несульфатованих глікозаміногліканів в основній речовині сполучної тканини пульпи. Таким чином, аліментарноасоційовані порушення гуморальної регуляції призводять до оксидативного стресу у пульпі зубів, що зумовлює розлади трофіки і збільшує ризик їх патологічної втрати за умов зміни глікемічного профілю, особливо на тлі йодної недостатності.

Ключові слова: пульпа зуба; перекисне окиснення білків і ліпідів; гістологічні зміни м'яких тканин зуба; йодна недостатність; високовуглеводна дієта.

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Recesved 01.04.2026