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Morphological features of the prostate gland under conditions of iodine deficiency and iodine deficiency combined with the goitrogenic substances during postnatal ontogenesis

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Abstract. According to data from the World Health Organization, pathological conditions caused by iodine deficiency rank third place of the most common non-infectious human diseases. The aim of the study was to determine the morphofunctional changes in the prostate gland over time at 60 and 90 days of iodine deficiency combined with the consumption of goitrogenic substances during postnatal ontogenesis. The experiment was conducted on 78 white inbred male rats of both prepubertal and pubertal ages. Eleven animals from each age group constituted Group 1 (control), while 14 animals were included in experimental groups 2 and 3 with simulated iodine deficiency. Experimental Groups 4 and 5 experimental groups consisted of animals from both age groups in which combined iodine deficiency with goitrogen consumption was simulated. Material for light and electron microscopy were collected on days 60 and 90 of the experiment. From a morphological view, the structural changes observed in the prostate gland involve several levels. First, due to reduced thyroid stimulation, changes in the metabolic activity of the epithelial cells of the gland's acini and ducts were noted. Second, endocrine imbalance altered the ratio of epithelial to stromal components. Third, since the prostate gland is sensitive to the interaction of androgens, estrogens, thyroid hormones, and pro-inflammatory mediators, the combined effect of iodine deficiency and goitrogenic agents manifested as inflammatory and dystrophic changes, specifically alterations in epithelial cell height, acinar lumen width, the stroma-to-parenchyma ratio, the presence of edema, and cellular infiltration. Thus, iodine deficiency and goitrogenic agents can alter thyroid homeostasis, and the thyroid axis is associated with the function and pathological conditions of the prostate gland. Thus, tth key components of thyroid signaling are detected in the prostate gland, indicating a possible direct effect of thyroid hormones on this organ. Edematous changes in the epithelium of the terminal secretory units result from ischemia caused by edematous processes in the walls of blood vessels and connective tissue elements of the prostate.

Keywords: laboratory animals; experiment; prostate; morphology; morphometry; age; ontogenesis.

INTRODUCTION

According to the World Health Organization, iodine deficiency pathologic conditions take the third place in the list of the none infectious human diseases [1]. Iodine deficiency states are considered the most widespread syndromic lesions of a non-infectious nature throughout the world [2–4]. A third of the Earth's population belongs to the group of risk and is a real target for the iodine deficiency diseases development. Insufficient intake of iodine, which acts as the main component for the formation of thyroid hormones, leads to hypothyroxinemia, which leads to a whole spectrum of disorders of the organs and systems of the body [5–7]. Iodine deficiency is a frequent environmental phenomenon that occurs as a result of a lack of iodine in the soil, food products of plant and animal origin, produced in the region of residence [8]. The problem of iodine deficiency is related not only to its tun away

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by water carried by streams of mountain rivers into the ocean [9], but also to improper soil treatment, application of herbicides, pesticides, overloading with salts of heavy metals, and other pollutants of industrial production [10]. A study of the thyroid gland of people from endemic areas shows that there are changes in the gland tissue that indicate the influence of other stromogenic factors and the body's adaptation to their influence, and some age-related features are also characteristic of the second type of changes [11].

Traditionally, iodine deficiency is studied exclusively through the prism of pathologies of the thyroid gland (goiter, hypothyroidism). However, recent decades prove that the prostate gland is an active extrathyroid store of iodine. It has its own sodium iodide symporters and uses molecular iodine as a powerful antioxidant and cell cycle regulator [12, 13]. Without proper antioxidant support of iodine in prostate tissue, lipid peroxidation is triggered, leading to chronic aseptic inflammation.

The research is very actual, as it solves an important medical and biological task: it reveals deep, previously unstudied morphological mechanisms of prostate gland damage under the conditions of the combined effect of environmental and endocrine factors (iodine deficiency and the influence of goiterogens), which will allow optimizing strategies for preserving male reproductive health.

The aim of the study: to determine the morphologica; and functional changes in the prostate gland in the dynamics from 60 to 90 days of iodine deficiency combined with the consumption of stromogens in postnatal ontogeny, taking into account age characteristics.

MATERIALS AND METHODS

In the experiment took part 78 white outbred male rats, 39 of which were sexually immature animals (3–5 months) and sexually mature rats (6–8 months). 11 animals of both age groups coplete 1 control group control, 14 animals in experimental 2nd group and experimental 3d group were with simulated iodine deficiency. The 4th and 5th experimental groups included sex mature and sex immature rats of both age groups. Animals of these groups were with simulated iodine deficiency and with the consumption of stromogens [14]. All manipulations with rats were carried out in compliance with the humane care rules with animals. The material of the prostate gland for light and electron microscopic examinations was carried out on the 60th and 90th day of the experiment. Morphometrical investigation, biochemical study and statistical data processing was also used [15].

RESULTS AND DISCUSSION

Thyroid status of prepubertal animals in Group 1: Thyrotropin (0.10±0.01) mIU/mL (p<0.01), Triiodothyronine (3.63±0.12) nmol/L (p<0.01), Thyroxine (75.44±4.01) nmol/L (p<0.01); in sexually mature animals – Thyrotropin (0.08±0.00) μIU/mL (p<0.01), Triiodothyronine (2.79±0.15) nmol/L (p<0.01), Thyroxine (55.18±2.72) nmol/L (p<0.01), respectively. Cholesterol levels within the age-normal range are: in immature animals (1.60±0.05) mmol/L (p<0.01); in mature rats (1.40±0.09) mmol/L (p<0.01). Ioduria in sexually immature rats of control group was (97.13±5.40) μg/L, and in mature rats it was (101.06±3.44) μg/L (p<0.01).

Thyroid status of prepubertal animals in Group 2: Thyrotropin (0.17±0.02) mIU/mL (p<0.01), Triiodothyronine (3.46±0.36) nmol/L (p<0.01), Thyroxine (75.49±9.60) nmol/L (p<0.01); in mature rats – Thyrotropin (0.12±0.01) μIU/mL (p<0.01), Triiodothyronine (2.97±0.37) nmol/L (p<0.01), Thyroxine (67.49±8.25) nmol/L (p<0.01), respectively. Cholesterol levels on 60th day of the experiment were: in immature sexually animals (1.63±0.17) mmol/L (p<0.01); in sexually mature animals (1.39±0.12) mmol/L (p<0.01). Ioduria in immature rats of second group was (2.73±0.29) μg/L, and in mature ratsit was (3.77±0.34) μg/L (p<0.01).

At this stage of the experiment, edema is observed in the connective tissue elements of the stroma and in the walls of blood vessels in all lobes of the prostate gland (Fig. 1). The epithelial cells of the terminal secretory portions have vacuolated cytoplasm in the apical pole and basophilia of the basal poles. Blood vessels are surrounded by edematous connective tissue fibers and amorphous stromal material.

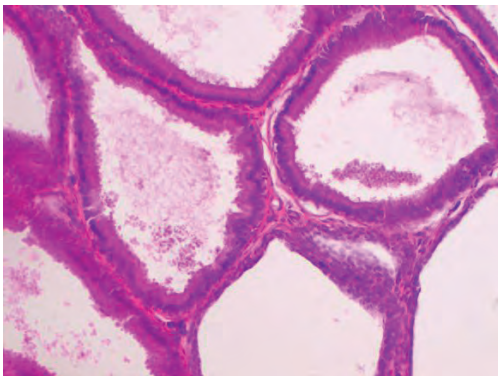

Fig. 1. Structural organization of the prostate gland of a sexually mature animal on the 60th day of iodine deficiency. Staining with hematoxylin and eosin. x200.

Electron microscopy reveals euchromatin and a thin line of heterochromatin in the nuclei. The canals of the granular endoplasmic reticulum are dilated, and the cisternae of the Golgi apparatus are also dilated and fill the cytoplasm unevenly. The mitochondrial matrix is of low electron density, and the cristae are disorganized. Secretory granules, which are localized in the cytoplasm, are of various shapes and sizes.

The height of the epithelium in the terminal secretory sections of the ventral, dorsal and coagulated lobes of the gland in immature animals increased by 0.3 % ($p<0.05$), 0.7 % ($p<0.05$), and 0.9 % ($p<0.05$), respectively. In these lobes of the prostate gland of sexually mature animals, the height of the epithelium increased by 0.2 % ($p<0.05$), 0.4 % ($p<0.05$), and 0.9 % ($p<0.05$), respectively.

Thyroid status of immature animals in group 3: Thyrotropin (0.16±0.02) mIU/mL ($p<0.01$), Triiodothyronine (3.47±0.39) nmol/L ($p<0.01$), Thyroxine (51.31±6.53) nmol/L ($p<0.01$); in sexually mature rats – Thyrotropin (0.23±0.03) μIU/mL ($p<0.01$), Triiodothyronine (2.43±0.29) nmol/L ($p<0.01$), Thyroxine (54.91±7.05) nmol/L ($p<0.01$), respectively. Cholesterol levels on 90th day of the experiment were: in immature sexually animals (1.66±0.20) mmol/L ($p<0.01$); in mature animals (1.44±0.16) mmol/L ($p<0.01$). Ioduria in sexually immature rats of third group was (1.89±0.24) μg/L, and in mature rats it was (2.29±0.21) μg/L ($p<0.01$).

The epithelium of the secretory portions of all lobules of the prostate gland is edematous. The nuclei have indistinct boundaries. The cytoplasm is stained eosinophilic. Edematous connective tissue fibers, amorphous substance, and smooth muscle cells of the gland stroma are visible. Similar edematous changes occur in the walls of blood vessels.

Submicroscopically, light colour cytoplasm of epithelial cells is observed. The tubules of the granular endoplasmic reticulum are dilated, and the Golgi apparatus is represented by deformed vesicles and cisterns. Mitochondria are visible with disorganized cristae and a light dense matrix (Fig. 2).

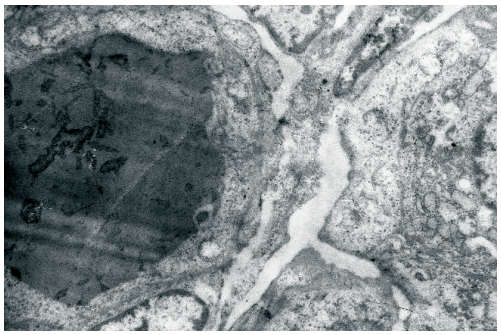

Fig. 2. Structure of the prostate gland of a sexually immature animal on the 90th day of iodine deficiency. x4800.

The height of the epithelium in the terminal secretory portions of the ventral and dorsal lobes and the coagulation gland in immature animals increased by 3.6 % ($p<0.05$). In the mentioned lobes of the prostate gland of sexually mature animals, the height of the epithelium increased by 1.1 % ($p<0.05$), 0.9 % ($p<0.05$), and 1.4 % ($p<0.05$), respectively.

Thyroid status of immature animals in group 4: Thyrotropin (0.18±0.02) mIU/mL ($p<0.01$), Triiodothyronine (3.44±0.36) nmol/L ($p<0.01$), Thyroxine (74.40±7.10) nmol/L ($p<0.01$); in sexually mature rats – Thyrotropin (0.14±0.01) μIU/mL ($p<0.01$), Triiodothyronine (2.39±0.24) nmol/L ($p<0.01$), Thyroxine (63.60±7.46) nmol/L ($p<0.01$), respectively. Cholesterol levels on 60th day of the experiment were: in sexually immature rats (1.66±0.19) mmol/L ($p<0.01$); in mature animals (1.44±0.15) mmol/L ($p<0.01$). Ioduria insexually immature rats of second group was (1.42±0.16) μg/L, and in sexually mature rats it was (1.92±0.21) μg/L ($p<0.01$).

The lumen of the terminal secretory compartments of the prostate lobules contains a homogeneous substance. The epithelial cells have basally located nuclei, the aggregate of which forms a basophilic band. The cytoplasm is eosinophilic with marked clearing at the apical pole. Blood vessels are surrounded by swollen connective tissue fibers and amorphous stromal material. The height of the epithelium in the terminal secretory sections of the ventral and dorsal lobes and the coagulation gland in sexually immature animals increased by 0.5 % ($p<0.05$), 0.9 % ($p<0.05$), and 1.1 % ($p<0.05$), respectively. In the specified regions of the prostate gland of sexually mature animals, the height of the epithelium increased by 0.3 % ($p<0.05$), 0.5 % ($p<0.05$), and 1.0 % ($p<0.05$), respectively.

Ultrastructural examination revealed the presence of euchromatin and heterochromatin in the nuclei of epithelial cells, with heterochromatin concentrated beneath the nucleolus. The tubules and canals of the granular endoplasmic reticulum, as well as the vesicles and vacuoles of the endoplasmic reticulum, are dilated. Myelin-like bodies are visible. The sarcoplasm of smooth muscle cells has a fine-grained structure, with prominent rounded mitochondria having a clear matrix (Fig. 3).

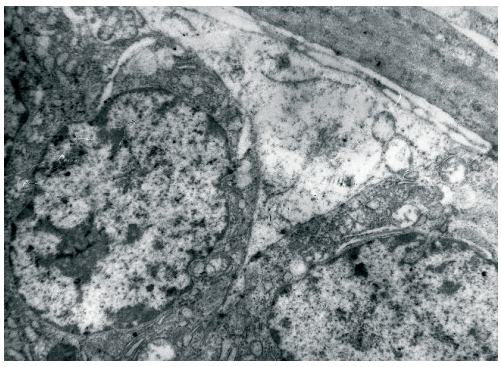

Fig. 3. Ultrastructure of the prostate gland of a sexually mature animal on the 90th day of iodine deficiency with stromogens. x4800.

Thyroid status of prepubertal animals in group 5: Thyrotropin (0.14±0.02) mIU/mL ($p<0.01$), Triiodothyronine (3.36±0.33) nmol/L ($p<0.01$), Thyroxine (50.14±5.73) nmol/L ($p<0.01$); in sexually mature rats – Thyrotropin (0.26±0.02) μIU/mL

(p<0.01), Triiodothyronine (2.06±0.18) nmol/L (p<0.01), Thyroxine (50.88±5.69) nmol/L (p<0.01), respectively. Cholesterol levels on 90th day of the experiment were: in sexually immature rats, (1.68±0.15) mmol/L (p<0.01); in mature animals (1.49±0.14) mmol/L (p<0.01). Ioduria in sexually immature animals of third group was (0.92±0.08) μg/L, and in sexually mature rats it was (1.54±0.140 μg/L (p<0.01).

Edematous changes are observed in the prostate lobes on day 90 of the experiment. Connective tissue fibers and smooth stromal myocytes are edematous, and lymphocytic infiltration is present. The walls of blood vessels are thickened, and stasis is observed in the lumen. Epithelial cells of the terminal secretory units show edema. There is a noticeable lightening of the basal poles of the cytoplasm with nuclear displacement. The apically lightened cytoplasm forms a veil-like contour of the lumen of the terminal secretory units (Fig. 4).

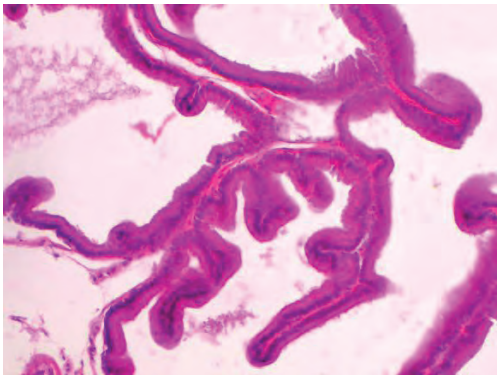

Fig. 4. Structural organization of the prostate gland of a sexually immature animal on the 90th day of iodine deficiency with stromogens. Staining with hematoxylin and eosin. x200.

The height of the epithelium in the terminal secretory sections of the ventral and dorsal lobes and the coagulation gland in immature animals increased by 3.7 % (p<0.05), 4.3 % (p<0.05), and 3.9 % (p<0.05), respectively. In the mentioned lobes of the prostate gland of sexually mature animals, the height of the epithelium increased by 1.2 % (p<0.05), 1.1 % (p<0.05), and 1.6 % (p<0.05), respectively.

Ultrastructural examination reveals deformed nuclei in the epithelial cells due to invagination of their nuclear envelope. Membrane organelles are enlarged and vacuolated. Mitochondria are visible with a lightened matrix and deformed cristae. There is stasis in the lumen of blood vessels. The cytoplasm of endothelial cells is vacuolated. Paravascular edema is evident. The sarcoplasm of smooth muscle cells has a homogenized-granular appearance.

Iodine is an essential trace element required for the synthesis of thyroid hormones, and disruptions in its intake are considered one of the causes of changes in thyroid function [16, 17]. In the current literature, iodine deficiency is described not only as a problem of endemic goiter but as a systemic factor capable of altering metabolic, endocrine, and regulatory processes in the body [5]. For experimental studies of the prostate gland, it is particularly important that thyroid status influences growth, differentiation, metabolism, and cellular interaction with the hormonal microenvironment. Thus, iodine deficiency can be considered a primary factor that, through changes in thyroid hormone levels, potentially affects the organs of the male reproductive system.

The second key aspect of the issue is foods with goitrogenic properties. These include, in particular, plants of the Brassicaceae family, which contain glucosinolates and their derivatives. Certain metabolites of these compounds, particularly thiocyanates and goitrin, may reduce iodine uptake by the thyroid gland or interfere with the synthesis of thyroid hormones [14]. A 2024 systematic review summarizes data on the effect of Brassica vegetables on thyroid function and emphasizes that the effect depends on the type of product, quantity, method of preparation, baseline iodine status, and individual physiological characteristics [18]. Therefore, in the context of adequate iodine intake, moderate consumption of such foods is not necessarily harmful, but in the event of iodine deficiency, their effects may take on greater clinical significance.

A review of the literature published after 2016 reveals that there are very few, if any, studies that simultaneously examine iodine deficiency, the consumption of iodine-rich foods, and the morphofunctional state of the prostate gland. Therefore, the available sources should be interpreted with caution: they do not prove a direct combined effect of these two factors on the prostate, but they do provide a scientific basis for designing such an experiment. The logic of the study can be based on three links: iodine deficiency and goitrogens alter thyroid status; thyroid hormones are involved in the regulation of the prostate; the prostate is a hormone-dependent organ sensitive to changes in the endocrine and inflammatory microenvironment.

A recent review by Anguiano and colleagues supports the concept of the prostate as a target organ for thyroid hormones [19]. The authors emphasize that thyroid hormones may be associated with the growth, differentiation, and metabolic activity of prostate tissue, and the consequences of hypo- or hyperthyroidism depend on age, duration of exposure, and the experimental model. This is important for planning studies in white rats, as the prostate's response may differ in cases of acute, chronic, postnatal, or adult thyroid dysfunction. In this context, iodine deficiency

should not be considered in isolation but rather as a model that alters the body's hormonal background.

Further support for this concept comes from a randomization study by Huang et al., which analyzed genetically predicted indicators of thyroid function and the risk of benign prostate diseases [21]. The authors reported that TSH levels and hypothyroid states were associated with the risk of benign prostatic hyperplasia and prostatitis. This is not experimental evidence of the effect of iodine deficiency, but it shows that thyroid regulation may be linked to pathological processes in the prostate. For morphological studies, this provides grounds for evaluating not only the organ's mass but also the condition of the epithelium, stroma, vascular bed, signs of inflammatory infiltration, and secretory activity.

Data on the extrathyroidal effects of iodine should be considered separately. In a 2023 study by Anguiano et al., molecular iodine exhibited anti-inflammatory effects in a rat model of sex hormone-induced prostate inflammation and also influenced the viability of DU145 cells and IL-6 production [6]. These results cannot be directly extrapolated to a model of iodine deficiency, but they suggest that iodine may be involved in the regulation of oxidative stress, the inflammatory response, and proliferative activity in prostate tissue. Therefore, insufficient iodine intake could theoretically alter not only the thyroid axis but also local mechanisms of tissue reactivity.

New experimental data from 2025 also show that molecular iodine is capable of modulating the viability, invasive properties, and response of prostate cancer cells depending on androgen status and model characteristics [21, 22]. For the study of the prostate gland in white rats, these data are of secondary importance: they do not describe dietary iodine deficiency [23] and do not directly concern goitrogenic foods; however, they confirm that iodine-dependent processes may be biologically significant for prostate tissue [24]. This allows us to formulate the hypothesis that prolonged iodine deficiency, especially in combination with substances that further impair thyroid hormone synthesis, may affect the structural organization of the prostate.

From a morphological view, the expected changes may involve several levels [12, 13, 25, 26]. First, due to reduced thyroid stimulation, changes in the metabolic activity of acinar and tubular epithelial cells are possible. Second, endocrine imbalance may alter the ratio of epithelial to stromal components. Third, since the prostate gland is sensitive to the interaction of androgens, estrogens, thyroid hormones, and pro-inflammatory mediators, the combined effect of iodine deficiency and goitrogens may manifest as inflammatory or dystrophic changes. However, these statements should be presented precisely as a working hypothesis, rather than as a proven fact, since there is insufficient direct contemporary evidence for such a combination of factors.

The article, based on an experiment with white rats, emphasizes that the chosen model is a suitable experimental study. Appropriate indicators include the animal's body weight and prostate weight, the morphological state of the glandular lobes, the height of the epithelium, the lumen of the acini, the ratio of stroma to parenchyma, the condition of the blood vessels, and the presence of edema or cellular infiltration. To strengthen the evidence, a morphological analysis determining thyroid status is presented, as it is the key intermediate link between iodine deficiency, goitrogenic substances, and potential changes in the prostate.

Thus, iodine deficiency and goitrogenic products can alter thyroid homeostasis, and the thyroid axis, according to current data, is associated with the function and pathological conditions of the prostate gland. This is precisely why the experimental study of morphological changes in the prostate under conditions of isolated iodine deficiency and its combination with goitrogenic dietary factors is scientifically justified and complements current understanding of the endocrine regulation of the male reproductive system.

CONCLUSION

Main components of thyroid signaling have been identified in the prostate gland, suggesting a direct effect of thyroid hormones on this organ. Edematous changes in the epithelium of the terminal secretory units result from ischemia caused by edematous processes in the walls of blood vessels and connective tissue elements of the prostate.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Морфологічні особливості передміхурової залози в умовах змодельованого йододефіциту та йододефіциту, поєднаного зі споживанням струмогенів у постнатальному онтогенезі

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Анотація. За даними ВООЗ, патологічні стани, що викликані дефіцитом йоду, займають третє місце у списку найпоширеніших неінфекційних захворювань людини. Метою дослідження було визначити морфофункціональні зміни в передміхуровій залозі у динаміці на 60 та 90 доби йододефіциту, поєднаного зі споживанням струмогенів у постнатальному онтогенезі з урахуванням вікових особливостей. Експеримент виконано на 78 білих безпородних щурах-самцях статевонезрілого та статевозрілого віку. По 11 тварин обидвох вікових груп становили 1 групу (контроль), по 14 тварин увійшли у 2 та 3 дослідні групи зі змодельованим йододефіцитом. 4 та 5 дослідні групи становили щури обидвох вікових груп, яким моделювали поєднаний йододефіцит зі споживанням струмогенів. Забір матеріалу для світлооптичного та електронно-мікроскопічного досліджень проводили на 60 та 90 доби експерименту. З морфологічної точки зору отримані структурні зміни тканин предміхурової залози стосуються кількох рівнів. По-перше, через зниження тиреоїдної стимуляції відзначались зміни метаболічної активності епітеліальних клітин ацинусів і проток залози. По-друге, ендокринний дисбаланс змінював співвідношення епітеліального та стромального компонентів. По-третє, оскільки передміхурова залоза є чутливою до взаємодії андрогенів, естрогенів, тиреоїдних гормонів і прозапальних медіаторів, комбінований вплив йододефіциту та струмогенів проявлявся через запальні та дистрофічні зміни, зокрема зміна висоти епітеліальних клітин, просвіту ацинусів, співвідношення строми й паренхіми, наявність набряку та клітинної інфільтрації. Отже, йододефіцит і струмогенні продукти можуть змінювати тиреоїдний гомеостаз, а тиреоїдна вісь пов'язана з функціонуванням і патологічними станами передміхурової залози. Отже, у передміхуровій залозі виявляються ключові компоненти тиреоїдної сигналізації, що свідчить про можливу пряму дію гормонів щитоподібної залози на цей орган. Набрякові зміни в епітелії кінцевих секреторних відділів є наслідком ішемії через набрякові процеси у стінці кровоносних судин та сполучнотканинних елементах простати.

Ключові слова: лабораторні тварини; експеримент; передміхурова залоза; морфологія; морфометрія; вік; онтогенез.