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RELATIONSHIP BETWEEN THYROID DYSFUNCTION AND THE CONDITION OF DENTAL HARD TISSUES AND PERIODONTAL TISSUES IN CHILDREN

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Abstract

The dental health of children is determined by the interaction of local oral factors and systemic mechanisms regulating tissue growth, development, mineralization, and remodeling. Endocrine disorders occupy a special place among systemic factors, since hormones are involved in the regulation of cell proliferation and differentiation, mineral metabolism, bone formation, and organogenesis.

Current evidence indicates that thyroid hormone deficiency during critical periods of development may be associated with delayed formation of teeth and craniofacial structures, whereas an excess of thyroid hormones may accelerate certain developmental processes [1–4]. The review by Leitch et al. emphasizes the importance of thyroid hormones for the development of the craniofacial skeleton and teeth and points to insufficient understanding of the mechanisms through which changes in thyroid status affect these structures.

Keywords: Children, thyroid gland, thyroid hormones, hypothyroidism, hyperthyroidism, dental enamel, mineralization, dental caries, saliva, periodontium, gingivitis, periodontitis.

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Introduction

The relevance of this issue is also determined by the fact that tooth formation and mineralization occur during childhood and may be sensitive to systemic metabolic disturbances. The prenatal period and early childhood are particularly important, as this is when the foundations of both primary and permanent teeth are established and their development takes place.

Clinical studies in children with thyroid dysfunction have described disturbances in the timing of tooth eruption, developmental defects of enamel, increased dental plaque accumulation and gingival inflammation, as well as a tendency toward higher caries severity [5–7]. A study involving 200 children aged 2–16 years demonstrated statistically significant increases in dental plaque scores and gingival index values among children with thyroid disorders, as well as higher rates of developmental defects of enamel.

At the same time, a substantial proportion of contemporary studies examining the relationship between thyroid pathology and periodontal diseases have been conducted in adult patients. Recent systematic reviews support the existence of an association but also point to substantial heterogeneity among studies, a high risk of bias, and a lack of high-quality longitudinal research.

Thus, studying the condition of dental hard tissues and periodontal tissues in children with thyroid dysfunction is of both scientific and practical interest.

The aim of this review is to summarize current evidence on the condition of dental hard tissues and periodontal tissues in children with thyroid dysfunction, analyze the possible pathogenetic mechanisms underlying the observed changes, and identify the main directions for future research.

Tooth development is a complex, multistage process involving interactions between epithelial and mesenchymal cells, their proliferation and differentiation, formation of the crown and root, secretion of the enamel and dentin matrices, followed by tissue mineralization and maturation.

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Thyroid hormones are involved in the regulation of tissue growth and differentiation. Their effects are mediated through specific receptors expressed in various cell populations.

The presence of thyroid hormone receptors in bone cells has been demonstrated in experimental and morphological studies, supporting the possibility of a direct influence of the thyroid system on bone remodeling processes [3, 8].

A contemporary review of the role of thyroid hormones in craniofacial development indicates that thyroid hormone deficiency is associated with delayed development of teeth and craniofacial structures, whereas an excess of thyroid hormones may accelerate certain developmental processes [1].

The prenatal period is of particular importance. In the large population-based Generation R study, which included more than 2,000 children, an association was identified between thyroid-stimulating hormone concentrations in umbilical cord blood and in early childhood and subsequent dental development parameters. In addition, children born to mothers who tested positive for thyroid peroxidase antibodies demonstrated lower indicators of dental development. The authors concluded that the thyroid system may be involved in tooth maturation from the earliest stages of life [6].

These findings support considering thyroid hormones as one of the endocrine factors involved in the regulation of odontogenesis.

One of the most characteristic dental manifestations of hypothyroidism is delayed tooth eruption. The literature describes delayed eruption of both primary and permanent teeth, prolonged retention of primary teeth, delayed root formation, and various dentofacial anomalies [2, 9, 10].

Thyroid hormone deficiency may affect not only the timing of tooth eruption but also mineralization processes. A study of the structure of primary teeth in children with congenital hypothyroidism demonstrated an increased number of areas of enamel with increased porosity. The authors suggested that thyroid hormones may be involved in the process of enamel maturation [11].

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Developmental defects of enamel deserve particular attention. In a study of children with thyroid disorders, the prevalence of developmental defects of enamel was statistically significantly higher in the study group than in healthy children [5].

Therefore, the effects of thyroid dysfunction on dental hard tissues may occur at several levels:

- disturbances in the timing of tooth formation and eruption;
- alterations in mineralization processes;
- impaired enamel maturation;
- increased structural heterogeneity of enamel;
- potentially increased susceptibility to dental caries.

However, the presence of thyroid pathology cannot be regarded as an independent or sole cause of dental caries. Dental caries is a multifactorial disease, and its development is determined by the condition of the enamel, the composition and properties of saliva, the oral microbiome, dietary patterns, oral hygiene, and fluoride exposure.

The relationship between thyroid disorders and dental caries remains insufficiently studied.

In a study of children aged 2–16 years, DMFT/dmft scores were higher among children with thyroid disorders than among healthy children; however, the differences in these indices did not reach statistical significance. At the same time, statistically significant differences were observed in developmental defects of enamel [5].

Different findings were obtained in a study of 75 adolescents aged 12–18 years. In the study group, 45 adolescents had thyroid dysfunction, predominantly hypothyroidism. The authors found a higher prevalence and severity of dental caries, reduced salivary flow rate, and increased oral fluid viscosity. Changes in calcium and inorganic phosphate concentrations in oral fluid were also identified [7].

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These findings suggest that the potential association between thyroid dysfunction and dental caries may be mediated not only by structural changes in enamel but also by alterations in the properties of oral fluid.

At the same time, the available findings cannot be regarded as definitive evidence of a direct causal effect of hypothyroidism on dental caries. Oral hygiene, dietary, socioeconomic, and regional factors must also be taken into account.

Saliva is a crucial component of the natural defense system of the oral cavity. It contributes to tooth cleansing, acid buffering, maintenance of mineral equilibrium, and enamel remineralization.

Changes in salivary flow rate and the physicochemical properties of saliva may increase the risk of demineralization and dental caries.

Reduced salivary secretion and increased oral fluid viscosity have been reported in adolescents with thyroid disorders [7].

A study of patients with hypothyroidism also demonstrated reduced salivary calcium concentrations compared with healthy individuals [12]. The authors suggested that altered calcium concentration in oral fluid may be one of the factors contributing to an increased risk of dental caries.

Thus, a promising area of research is the simultaneous assessment in children of:

1. salivary flow rate;
2. salivary viscosity;
3. pH;
4. buffering capacity;
5. calcium concentration;
6. phosphate concentration;
7. other mineralizing components;
8. parameters of local immunity.

The periodontium is a dynamic structure whose condition depends on the microbial biofilm, immune response, microcirculation, metabolism, and bone tissue status.

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Thyroid hormones may potentially affect the periodontium through several mechanisms.

The first mechanism is related to bone metabolism. Thyroid hormones regulate bone formation and resorption. The presence of thyroid hormone receptors in osteoblasts and osteoclasts supports the possibility of a direct hormonal effect on bone tissue [3, 8].

The second mechanism involves alterations in mineral metabolism. In hypothyroidism, metabolic processes associated with calcium and phosphorus may be altered.

The third mechanism is related to immunoinflammatory responses. Contemporary reviews discuss the potential role of pro-inflammatory mediators, including interleukin-6, in the relationship between thyroid pathology and periodontal diseases [13, 14].

The fourth potential mechanism is alteration of the oral microbiome. Current research suggests that disturbances in microbial homeostasis may contribute to the relationship between endocrine and inflammatory diseases [13].

The most important studies relevant to the issue under consideration are presented in Table 1. In 2020, Aldulaijan et al. conducted a scoping review of the relationship between hypothyroidism and periodontitis. Of 847 unique publications identified, 29 studies remained after screening; however, following critical appraisal, only four publications were considered directly suitable for further analysis. The authors concluded that there was insufficient high-quality evidence and that well-controlled prospective clinical and immunological studies were needed [16].

In 2024, Ortarzewska et al. conducted a systematic review and meta-analysis that included 10 studies. Based on the pooled data, periodontal status was worse in patients with thyroid diseases, particularly hypothyroidism, than in control groups. The authors emphasized the need for further investigation of both hypothyroidism and hyperthyroidism [17].

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In the same year, Jakonyte et al. conducted a systematic review of studies published between 2019 and 2024. The authors examined the potential roles of periodontal pocket depth, clinical attachment loss, interleukin-6, calcium-phosphorus metabolism, and the microbiome. In hypothyroidism, disturbances in mineral homeostasis and changes in bone tissue were discussed, whereas in hyperthyroidism, alterations in alveolar bone structure and inflammatory parameters were considered [13].

In 2025, Ni et al. conducted a systematic review and meta-analysis that included eight studies. A statistically significant increase in overall periodontal parameters was found among patients with thyroid dysfunction. The effects were most pronounced for periodontal pocket depth and clinical attachment loss. However, the quality of the included studies was rated as low, which substantially limits interpretation of the results [18].

Thus, current literature supports the existence of an association between thyroid pathology and periodontal status but does not allow a definitive causal relationship to be established.

Based on the analysis of current evidence, several interrelated mechanisms can be identified.

1. Impaired bone remodeling

Thyroid hormones participate in the regulation of bone cell activity. Their receptors have been identified in osteoblasts, osteoclasts, and other cells of the bone microenvironment [3, 8].

Therefore, pronounced or prolonged changes in thyroid status may affect alveolar bone tissue.

2. Disturbances in mineral metabolism

Alterations in calcium-phosphorus homeostasis may be relevant both to the formation of dental hard tissues and to the condition of the alveolar bone.

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Changes in salivary secretion, viscosity, mineral concentrations, and buffering capacity may contribute to impaired remineralization processes.

3. Immunoinflammatory mechanisms

Periodontal inflammation is determined by the interaction between the microbial biofilm and the host immune response. Changes in the systemic inflammatory milieu associated with endocrine diseases may potentially modify the course of periodontal inflammation.

4. Alterations in the microbiome

Current research discusses a possible association between thyroid disorders and changes in the composition of the oral microbiome. However, this area remains insufficiently studied, particularly in children [13].

5. Influence of age at disease onset

For the pediatric population, the age at disease onset is of fundamental importance. Thyroid dysfunction developing during active tooth formation may potentially have different consequences from disease arising after the completion of the main stages of mineralization.

Hyperthyroidism is characterized by excessive exposure of body tissues to thyroid hormones.

With regard to the dentofacial system, earlier tooth eruption and changes in bone tissue have been described. Contemporary literature also considers a possible association between hyperthyroidism and periodontal pathology [1, 13].

However, substantially fewer studies have been conducted in children than in adults. Therefore, findings from adult patients cannot automatically be extrapolated to the pediatric population.

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The degree of hormonal excess, disease duration, the child's age, treatment characteristics, and the presence of an autoimmune disease are of particular importance.

Autoimmune thyroid pathology, particularly autoimmune thyroiditis, also deserves special attention.

Autoimmune diseases may be accompanied by alterations in systemic immune regulation. Since inflammatory periodontal diseases are also characterized by complex interactions between microbial and immune factors, the presence of an autoimmune disease may act as an additional modifying factor.

A 2025 study of adults with autoimmune hypothyroidism demonstrated more pronounced periodontal changes and statistical associations between clinical attachment loss, the area of inflamed periodontal tissue, and thyroid and inflammatory parameters, including thyroid peroxidase antibodies [20].

However, comparable data are currently almost entirely lacking for children. This represents a promising area for future research.

When assessing the dental status of a child with endocrine pathology, it is insufficient to consider the diagnosis alone.

Dental outcomes may potentially be influenced by:

- the child's age;
- age at disease onset;
- disease duration;
- degree of hormonal deficiency or excess;
- thyroid-stimulating hormone level;
- free T4 and T3 levels;
- presence of thyroid peroxidase antibodies;
- disease control;
- duration of replacement therapy;
- dietary patterns;
- level of oral hygiene;

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- fluoride concentration in drinking water;
- socioeconomic conditions;
- presence of concomitant somatic diseases.

Therefore, future studies should employ multivariable statistical analysis.

MAJOR RESEARCH GAPS

Analysis of the current literature reveals several unresolved issues:

There are insufficient studies specifically involving pediatric populations.

Most studies have a cross-sectional design, which does not allow causal relationships to be established.

Different dental indices are used across studies, making direct comparison of results difficult.

There are insufficient studies simultaneously assessing dental hard tissues, periodontal status, and biochemical parameters of oral fluid.

The relationship between dental changes and TSH and free T4 levels has not been adequately studied.

There is insufficient evidence regarding the role of thyroid peroxidase antibodies and the autoimmune component.

It remains unclear to what extent the severity of dental changes decreases after stable control of thyroid disease is achieved.

Longitudinal studies tracking dental changes in the same child over time are virtually absent.

From a scientific perspective, the most informative approach would be a comprehensive model in which several groups of parameters are assessed simultaneously in children with thyroid dysfunction.

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DISCUSSION

The available evidence suggests that thyroid dysfunction may represent a potentially important systemic modifier of dental health in children.

The most consistent evidence concerns disturbances in tooth development and maturation. Studies in children demonstrate alterations in the timing of tooth eruption and enamel structure, as well as a possible increase in caries risk [5–7, 11].

With regard to periodontal tissues, the current evidence base is less specific to children. Recent meta-analyses of adult patients demonstrate an association between thyroid dysfunction and more pronounced periodontal changes [17, 18], but extrapolation of these findings to the pediatric population requires caution.

It is most likely that the dental manifestations of thyroid dysfunction result from a combination of several mechanisms, including alterations in tooth development and mineralization, disturbances in mineral metabolism, changes in salivary properties, bone remodeling, and immunoinflammatory regulation.

At the same time, thyroid pathology cannot be regarded as the sole cause of dental caries or periodontal diseases. A child's dental health is determined by a combination of factors.

Therefore, a promising research model is one in which thyroid status is considered not in isolation but in relation to age, disease duration, disease control, oral hygiene, diet, salivary composition, and clinical indicators of dental and periodontal health.

CONCLUSION

Analysis of the current literature demonstrates an association between thyroid dysfunction and the condition of the dentofacial system.

Children with thyroid pathology have been reported to exhibit disturbances in the timing of tooth eruption, developmental and maturation defects of enamel, alterations in the mineralization of hard dental tissues, increased caries indices in

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some studies, reduced salivary flow rate, and changes in the physicochemical characteristics of oral fluid.

With regard to periodontal tissues, current studies also indicate a possible association between thyroid dysfunction and inflammatory and structural changes. However, most systematic reviews are based predominantly on studies involving adult patients. A 2024 meta-analysis demonstrated poorer periodontal status in patients with thyroid diseases, particularly hypothyroidism, but emphasized the need for further research. A 2025 meta-analysis confirmed a statistical association between thyroid dysfunction and periodontal parameters; however, the quality of the included studies was limited.

Thus, one of the most significant research gaps remains the insufficient study of the comprehensive dental status of children with thyroid dysfunction.

A promising direction is the implementation of comprehensive clinical and laboratory studies combining the assessment of dental hard tissues, periodontal status, oral fluid parameters, mineral metabolism, and objective indicators of thyroid status.

The findings of such studies may provide a basis for developing algorithms for early dental monitoring and prevention of dental diseases in children with endocrine disorders.

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