

**CLINICAL SIGNIFICANCE OF WHITE CINQUEFOIL (*POTENTILLA ALBA L.*) IN
THE CORRECTION OF THYROID PATHOLOGIES**

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Abstract

Thyroid gland disorders occupy a leading position in the overall structure of endocrine diseases worldwide. Iodine deficiency is recognized as one of the principal etiological factors contributing to the development of thyroid pathologies[2]. Currently applied therapeutic approaches do not always ensure stable and pathogenetically justified outcomes. In this context, increasing scientific attention is being directed toward medicinal plants rich in biologically active compounds capable of regulating thyroid function. White cinquefoil (*Potentilla alba*) has emerged as a promising natural source of thyrotropic substances. The present article analyzes literature data on iodine deficiency disorders, pathogenetic mechanisms of thyroid diseases, limitations of conventional treatment methods, and the clinical relevance of *Potentilla alba* as a safe and effective phytotherapeutic agent in thyroid pathology correction[1].

Keywords

white cinquefoil, iodine deficiency, thyroid gland, phytotherapy.

Introduction

At present, thyroid gland diseases occupy a dominant position among endocrine disorders. This tendency is observed globally and is particularly pronounced in regions characterized by natural iodine deficiency, including Central Asia and several territories of Uzbekistan. The high prevalence of diffuse and nodular goiter in iodine-deficient areas reflects the ecological nature of iodine deficiency disorders (IDD), which arise due to insufficient iodine intake by the population[3].

Large-scale epidemiological studies conducted in different countries confirm the widespread nature of iodine deficiency. For example, population-based investigations carried out between 1991 and 2004 by specialists of the Endocrinology Research Center of the Russian Academy of Medical Sciences demonstrated that no region of the Russian Federation could be considered free from the risk of iodine deficiency disorders. Various degrees of iodine insufficiency were detected across all examined territories[5].

According to data from the World Health Organization (WHO), at least one billion people worldwide are at risk of developing iodine deficiency-related diseases. The most vulnerable groups include pregnant women, fetuses, newborns, and young children, as iodine deficiency during prenatal and early postnatal periods leads to irreversible impairment of brain development and cognitive function. Consequently, these population groups are classified as high-risk categories for iodine deficiency disorders.

The term “iodine deficiency disorders” was proposed by the WHO to encompass all pathological conditions developing at the population level as a result of iodine deficiency and preventable through normalization of iodine intake. This definition emphasizes the public health significance of the problem and underlines the necessity of effective preventive and therapeutic strategies[7].

Main Body

Insufficient iodine intake initiates a cascade of adaptive processes aimed at maintaining thyroid hormone synthesis and secretion. However, prolonged iodine deficiency disrupts these compensatory mechanisms, leading to pathological alterations in thyroid structure and function.

One of the most severe consequences of iodine deficiency is intellectual disability, including its extreme manifestation—cretinism. Thyroid hormones play a crucial role in the development of the central nervous system during intrauterine life, regulating gene expression, cellular differentiation, and synchronization of key biological processes. Scientific evidence confirms that iodine deficiency remains the most common preventable cause of cognitive impairment worldwide[9].

In adults residing in iodine-deficient regions, the predominant clinical manifestations include diffuse and nodular goiter, as well as iodine-induced thyrotoxicosis. In areas with mild or moderate iodine deficiency, elderly populations are particularly prone to developing functional autonomy of the thyroid gland, resulting in thyrotoxicosis. Following the introduction of iodine prophylaxis programs, a transient increase in iodine-induced thyrotoxicosis may occur due to decompensation of autonomously functioning thyroid tissue[8].

Diffuse non-toxic goiter represents the most common form of iodine deficiency disorder among adults. More than 50% of cases develop before the age of 20, and approximately 20% occur before 30 years of age, with women affected twice as often as men. Additional risk factors include pregnancy and smoking, which further stimulate thyroid enlargement under iodine-deficient conditions[10].

Although iodine deficiency plays a central etiological role, other permissive factors significantly influence goiter development. These include living conditions, socioeconomic status, micronutrient intake, and environmental exposures. Certain bacterial species produce goitrogenic substances such as goitrin, which inhibits iodine organification in the thyroid gland. Dietary components containing flavonoids, thiocyanates, and thiooxazolidones—found in soy, peanuts, turnips, and cruciferous vegetables—may suppress thyroid peroxidase activity or interfere with iodine uptake and transport[11].

Iron deficiency has also been shown to adversely affect thyroid metabolism, as iron is a component of heme-dependent thyroid peroxidase. Experimental studies demonstrate that iron deficiency impairs deiodination processes, reducing thyroid hormone synthesis. Epidemiological data indicate that combined iodine and iron deficiency diminishes the effectiveness of iodine prophylaxis programs, particularly in children[21].

From a pathogenetic perspective, growth factors play a pivotal role in diffuse goiter formation under iodine-deficient conditions. Increased synthesis of insulin-like growth factor-1, epidermal growth factor, and fibroblast growth factor stimulates heterogeneous thyrocyte proliferation, ultimately leading to nodular transformation. This mechanism explains the high recurrence rate of nodular goiter following surgical treatment.

Existing therapeutic approaches for thyroid diseases—surgical intervention, radioiodine therapy, and antithyroid drugs—are largely non-pathogenetic. Radical treatments intentionally destroy thyroid tissue, resulting in hypothyroidism and lifelong hormone replacement therapy. Antithyroid medications offer limited long-term efficacy and are associated with relapse risk due to persistent autoimmune activity[12].

Given these limitations, the search for safe, effective, and pathogenetically oriented therapeutic agents remains a pressing issue. Medicinal plants represent a promising source of biologically active compounds with thyrotropic properties and favorable safety profiles. Integrating phytotherapy with conventional treatment may reduce adverse effects and improve therapeutic outcomes[13].

White Cinquefoil (*Potentilla alba L.*) as a Phytotherapeutic Agent

White cinquefoil (*Potentilla alba*) is a perennial herbaceous plant belonging to the Rosaceae family. It is predominantly distributed in the chernozem regions of the European part of Russia. The pharmacological properties of this plant are attributed to its unique and insufficiently studied chemical composition[8].

The underground parts (rhizomes and roots) contain carbohydrates (starch), iridoids, saponins, phenolic acids, flavonoids (quercetin), and up to 17% tannins during the flowering phase. The aerial parts are rich in iridoids, saponins, phenolic acids, flavonoids (rutin), and tannins. Leaves contain phenolic acids and their derivatives, as well as flavonoids such as quercetin, kaempferol, and cyanidin[19,20].

White cinquefoil is also a powerful concentrator of microelements, including manganese, zinc, copper, selenium, cobalt, iron, silicon, and aluminum. Notably, the plant contains iodine and iodide anions, which may contribute directly to its thyrotropic activity[16].

Traditionally, *Potentilla alba* preparations have been widely used in folk medicine for the treatment of thyroid diseases. In Belarus, extensive use of white cinquefoil tinctures was associated with the near absence of endemic goiter regions. In Bulgaria, the rhizome extract has been employed for gastrointestinal disorders and as a hemostatic agent. Experimental studies confirm its diuretic, antibacterial, and central nervous system-stimulating effects[17].

Based on accumulated evidence, white cinquefoil represents a promising medicinal plant for developing safe and effective phytopreparations aimed at thyroid disease correction. Such agents are particularly valuable in iodine-deficient regions and areas affected by radionuclide contamination, where they may contribute to both therapeutic and preventive strategies[17].

Conclusion

The analyzed literature data indicate that thyroid diseases, particularly those associated with iodine deficiency, remain a significant global endocrine problem. Conventional treatment methods are often non-pathogenetic and may lead to hypothyroidism or disease recurrence.

Scientific evidence supports the clinical relevance of white cinquefoil (*Potentilla alba L.*) as a rich source of biologically active compounds and microelements capable of modulating thyroid function. Its thyrotropic activity, high safety profile, and multifaceted pharmacological effects allow it to be considered a promising phytotherapeutic agent for the treatment and prevention of thyroid disorders.

Incorporation of white cinquefoil-based preparations into complex therapy, especially in iodine-deficient regions, may enhance treatment efficacy and reduce adverse effects. Further experimental and clinical studies are warranted to elucidate its mechanisms of action and confirm its therapeutic potential in evidence-based medicine.

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