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FACTORS ASSOCIATED WITH PERSISTENT HYPOTHYROIDISM IN UZBEKISTAN: A NARRATIVE REVIEW

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ABSTRACT:

Background: Hypothyroidism remains a common public health challenge in Uzbekistan despite targeted iodine prophylaxis and legislative mandates for universal salt iodization. Iodine deficiency is traditionally regarded as the principal cause of hypothyroidism in endemic regions; however, additional nutritional and clinical factors may also contribute to persistent disease burden.

Objective: This narrative review synthesizes current international evidence and regional empirical data to evaluate the contributing factors of persistent hypothyroidism in Uzbekistan.

Methods: A comprehensive search of PubMed, Google Scholar, and national legislative databases was conducted for peer-reviewed literature and institutional reports published between 2000–2025.

Results: The sustained prevalence of hypothyroidism in Uzbekistan is associated with chronic environmental iodine deficiency, inconsistent quality control in salt fortification, high national rates

of concurrent iron deficiency anemia, which may impair thyroid peroxidase (TPO) activity, and specific gaps in routine prenatal screening. Localized trace element variations, including selenium deficiency, may also affect peripheral hormone metabolism.

Conclusion: Addressing the high prevalence of hypothyroidism requires an integrated micronutrient approach that addresses concurrent iodine and iron deficiencies, alongside optimized quality controls for fortified salt and expanded access to first-trimester screening.

INTRODUCTION

Hypothyroidism is a common endocrine disorder characterized by insufficient thyroid hormone production, which leads to reduced metabolic activity, cardiovascular alterations, and potential neurodevelopmental delays [1, 2]. Globally, the prevalence of overt hypothyroidism is estimated to range between 0.2% and 5.3%, with higher rates documented in regions characterized by environmental micronutrient limitations [2, 3]. Within Central Asia, and specifically in the Republic of Uzbekistan—the most populous country in the region—thyroid dysfunction represents a significant clinical priority for the healthcare infrastructure [4, 5].

The geographic and ecological environment of Uzbekistan plays a distinct role in population health. As a landlocked territory, the local soil and water systems exhibit low baseline levels of essential trace elements, particularly iodine [5, 6]. In response to endemic goiter, the government introduced public health programs, culminating in the 2007 National Law "On Prevention of Iodine Deficiency Disorders," which established mandatory guidelines for universal salt iodization [6]. Early implementation of these measures led to a documented reduction in visible goiter rates compared to historical baselines recorded in the late 1990s [5, 6]. However, contemporary indicators show that subclinical and overt thyroid insufficiency remain common across multiple demographic sectors, particularly among women of reproductive age and pregnant populations [4, 7]. Available nutritional and reproductive health data suggest that thyroid-related micronutrient deficiencies remain clinically relevant among women of reproductive age in Uzbekistan [4, 7]. This pattern

indicates that analyzing thyroid health through a single nutritional variable is insufficient. A broader evaluation is required to understand how various biological, environmental, and clinical management factors interact within the local population.

Iodine deficiency is traditionally regarded as the principal cause of hypothyroidism in endemic regions; however, additional nutritional and clinical factors may also contribute to persistent disease burden. This paper evaluates these contributing factors to support evidence-based clinical and public health strategies.

METHODS

To construct this narrative review, a literature search was conducted across international and regional biomedical databases, including PubMed, Google Scholar, and the national legal database LexUZ. The search strategy utilized combinations of the following keywords: "hypothyroidism", "iodine deficiency", "Uzbekistan", "anemia", "selenium", and "pregnancy". The chronological scope was defined from 2000–2025 to capture both foundational consensus statements and recent epidemiological updates from the region.

The selection criteria focused on peer-reviewed articles, clinical trials, and official institutional reports. Local health metrics were sourced from validated publications, including the Uzbekistan Nutrition Survey and the UNICEF Multiple Indicator Cluster Survey (MICS) for Uzbekistan. Preference was given to peer-reviewed English-language publications and institutional reports with available epidemiological data. Duplicate and non-peer-reviewed sources were excluded. Articles were screened for relevance to micronutrient status, thyroid epidemiology, maternal health, and endocrine public health in Central Asia, leaving verified peer-reviewed studies, clinical guidelines, and institutional reports for qualitative synthesis. The review followed a narrative synthesis approach rather than a formal systematic review methodology.

IODINE DEFICIENCY AND SALT IODIZATION

Environmental iodine deficiency remains a foundational factor for endemic thyroid disorders in Uzbekistan. Iodine is an essential substrate required for the synthesis of thyroxine (T_4) and triiodothyronine (T_3) [1, 8]. Because the physical geography of Central Asia features topsoil depletion, locally grown agricultural crops and natural water sources provide inadequate iodine concentrations [5]. As a result, the population depends on fortification strategies to maintain adequate iodine intake [8]. To address this, the 2007 national legislation required all food-grade salt manufactured or sold in Uzbekistan to be fortified with potassium iodate (KIO_3) at a standard level of 40 ± 15 mg/kg [6]. While this legal framework provides a uniform mandate, field evaluations indicate that variations remain in retail-level execution.

National nutritional surveys suggest that adequately iodized household salt coverage remains suboptimal in several regions of Uzbekistan, and reported coverage rates remain inconsistent across surveys and regions [4, 7].

This distribution variance is more frequent in rural provinces, where smaller local producers sometimes operate outside national quality control mechanisms [5, 7]. Additionally, potassium iodate is susceptible to chemical decomposition when exposed to high humidity, improper storage, or unsealed packaging, which are common conditions within traditional local supply chains [5]. The regular use of inadequately fortified salt contributes to low-grade iodine insufficiency, keeping median urinary iodine concentrations below optimal targets in vulnerable cohorts and contributing to the sustained prevalence of thyroid insufficiency [7, 8].

IRON DEFICIENCY AND THYROID FUNCTION

An important factor influencing public health outcomes in Uzbekistan is the interaction between iodine deficiency and iron deficiency anemia. Iron deficiency acts as a direct biochemical inhibitor of thyroid hormone synthesis [9, 10]. The primary step in thyroid hormone synthesis involves thyroid peroxidase (TPO), a membrane-bound, heme-dependent enzyme that catalyzes the iodination of tyrosine residues on thyroglobulin [10]. The underlying physiological process involves an integrated sequence. Severe iron deficiency reduces cellular ferritin stores, which limits the availability of the heme prosthetic group required for the activation of TPO. Without adequate heme binding, the enzyme's capacity to properly iodinate thyroglobulin decreases. This alteration results in a lower synthesis of circulating thyroid hormones, which can subsequently elevate serum Thyroid Stimulating Hormone (TSH) levels and contribute to signs of subclinical or overt hypothyroidism [9, 10]. Clinical studies led by Hess et al. have demonstrated that iron deficiency can limit the clinical response to iodine supplementation [9]. Anemic individuals show a lower reduction in goiter volume and may maintain elevated TSH levels even after receiving standard iodine doses, indicating that proper iron status is required for optimal iodine utilization [9].

This mechanism is directly relevant to the patient population in Uzbekistan. According to the UNICEF Multiple Indicator Cluster Survey (MICS), the prevalence of anemia among women of reproductive age remains elevated, ranging between 35.0%–49.0% depending on the specific region [4]. Concurrently, national surveys demonstrate that adequate salt iodization coverage is low, leaving a substantial portion of reproductive-age women trapped in concurrent iodine and iron deficiencies [4, 7]. When an individual suffers from concurrent iodine insufficiency and iron deficiency anemia, thyroid hormone synthesis may become

impaired through both reduced iodine availability and decreased TPO activity [9, 10]. Consequently, managing thyroid insufficiency by focusing only on iodine intake without correcting underlying ferritin and hemoglobin levels may lead to poor therapeutic outcomes [9].

SELENIUM AND ENVIRONMENTAL CONTRIBUTORS

Thyroid function is also influenced by other environmental trace elements, particularly selenium. Selenium is an integral component of selenoproteins, which include the iodothyronine deiodinase enzyme family (Dio1, Dio2) and glutathione peroxidases [11, 12]. The deiodinases are responsible for the peripheral conversion of thyroxine (T_4) into the active metabolic form, triiodothyronine (T_3) [11]. Glutathione peroxidases serve a protective function within thyrocytes by neutralizing excess hydrogen peroxide produced during hormone synthesis [12].

Ecological variability in selenium intake has also been discussed in parts of Central Asia [11, 12]. When dietary selenium intake falls below required levels, peripheral deiodination may slow down, leading to lower circulating active T_3 levels even if baseline T_4 production appears stable [11]. Furthermore, concurrent deficiencies in both selenium and iodine can increase cellular stress within the thyroid gland, as the reduction in protective glutathione peroxidases leaves thyrocytes more susceptible to oxidative changes [11, 12].

PREGNANCY AND MATERNAL THYROID HEALTH

Pregnancy requires significant adaptations from the maternal endocrine system. During the first trimester, maternal thyroid hormone production must increase to support both maternal metabolic requirements and early fetal neurological development, as the fetus relies entirely on the transplacental transfer of maternal thyroxine before its own thyroid gland develops at approximately 12–14 weeks of gestation [13, 14]. This adaptation requires an increase in daily iodine intake, driven by enhanced renal clearance and placental enzymatic clearance [1, 15].

In Uzbekistan, this increased demand represents a specific clinical vulnerability. The Uzbekistan Nutrition Survey noted that the median urinary iodine concentration among evaluated pregnant women was 117.3 µg/L, which is below the international adequacy baseline of 150–249 µg/L established for pregnancy [7]. Suboptimal iodine intake during gestation can lead to maternal hypothyroxinemia and an elevation of maternal TSH, increasing the risk of gestational thyroid insufficiency [13, 15]. According to clinical guidelines, unmanaged maternal thyroid insufficiency is associated with a higher risk of adverse obstetric outcomes, including gestational hypertension, preterm delivery, and potential limitations in early neurocognitive development indices [13, 16]. Although these

risks are well-established, access to specialized endocrine screening services may vary between urban and rural healthcare settings in Uzbekistan. Diagnostic testing often occurs reactively after clinical signs appear, which may delay necessary clinical interventions during critical phases of early development [14, 17].

GENETIC AND AUTOIMMUNE FACTORS

While nutritional and environmental factors are primary contributors to endemic thyroid dysfunction in Uzbekistan, genetic traits and autoimmune processes also influence clinical patterns. Autoimmune thyroid disorders have recognized genetic susceptibility patterns, particularly among first-degree relatives [2, 18]. Studies indicate that genetic variations within the human leukocyte antigen (HLA) complex and specific polymorphisms in the TSHR and thyroglobulin genes can influence individual susceptibility to autoimmune thyroiditis [18, 19]. Genetic factors indicate an underlying susceptibility that can be influenced by environmental shifts, such as sudden variations in iodine intake or concurrent micronutrient deficiencies [3, 18]. Additionally, congenital hypothyroidism requires early biochemical identification; while newborn screening systems are established in major urban perinatal centers, ensuring consistent coverage in remote rural areas remains an ongoing operational objective for the local healthcare system [4, 20].

PUBLIC HEALTH CHALLENGES IN UZBEKISTAN

The ongoing prevalence of thyroid insufficiency in Uzbekistan is linked to specific operational challenges within the public health sector. Although the 2007 legislation created an appropriate legal mandate for salt iodization, maintaining uniform quality control across all local retail markets remains difficult [6, 7]. Regulatory oversight is frequently concentrated around large production centers, which can allow lower-quality or unfortified salt from smaller, independent suppliers to reach rural communities [4, 7].

At the primary care level, a common challenge is the separate management of interconnected nutritional deficiencies. Primary care clinics often manage iron deficiency anemia and thyroid disorders under separate protocols [7]. This separated approach can reduce overall treatment efficacy, as uncorrected iron deficiency can limit the physiological response to thyroid therapies [9, 10].

Furthermore, public health education regarding proper micronutrient use requires expansion. Communities in remote regions may lack clear information regarding the correct storage of iodized salt, the loss of iodine content over time, and the specific nutritional requirements of pregnancy [4, 7]. These communication gaps, combined with infrastructure

variations in rural health centers, can limit the early identification of subclinical thyroid disorders [4, 15].

PREVENTION STRATEGIES

Improving thyroid health metrics in Uzbekistan requires a coordinated approach across multiple sectors of the healthcare system. The first priority is to strengthen regulatory compliance regarding salt iodization. Introducing automated testing methods at domestic production sites and increasing quality checks within retail markets can help ensure that commercial food-grade salt consistently meets national fortification standards [6, 7].

In clinical practice, primary care protocols should be updated to encourage integrated nutritional assessments. Clinicians evaluating patients for elevated TSH or subclinical thyroid signs should consider screening for concurrent iron deficiency anemia by checking hemoglobin and serum ferritin levels [9]. If an iron deficiency is identified, an integrated management plan that addresses both iron and thyroid levels should be utilized to improve overall clinical outcomes [9, 10].

For maternal health services, expanding access to first-trimester screening for TSH and free T_4 within primary care clinics is an important objective [13, 16]. These clinical protocols should be supported by targeted public health education campaigns that emphasize balanced nutrition and the correct selection and storage of fortified foods, particularly for families living in rural areas [4, 7].

CONCLUSION

The sustained prevalence of hypothyroidism in Uzbekistan is a public health issue that involves multiple interacting factors. Available evidence indicates an association between persistent thyroid insufficiency in Uzbekistan and chronic environmental iodine deficiency, variations in retail salt fortification quality, high rates of concurrent iron deficiency anemia that can affect TPO enzyme function, and remaining gaps in early prenatal screening.

To support long-term improvements, public health strategies should transition toward an integrated micronutrient approach. This strategy includes strengthening the enforcement of existing salt iodization laws, encouraging concurrent screening for both iron and iodine deficiencies in primary care, and expanding access to first-trimester screening for pregnant women. Addressing these factors may improve long-term maternal and population health outcomes in Uzbekistan.

REFERENCES

1. Chaker L, Razvi S, Bensenor IM, Azizi F, Pearce EN. Hypothyroidism. Nat Rev Dis Primers. 2022;8(1):30. doi:10.1038/s41572-022-00357-7
2. Taylor PN, Albrecht D, Scholz A, Gutierrez-Buey G, Lazarus JH, Dayan CM, Okosieme OE. Global epidemiology of hyperthyroidism and hypothyroidism. Nat Rev Endocrinol. 2018;14(5):301–316. doi:10.1038/nrendo.2018.18
3. Zimmermann MB, Boelaert K. Iodine deficiency and thyroid disorders. Lancet Diabetes Endocrinol. 2015;3(4):286–295. doi:10.1016/S2213-8587(14)70225-6
4. UNICEF. Multiple Indicator Cluster Survey (MICS) for Uzbekistan: Final Report. Tashkent: UNICEF and State Committee of the Republic of Uzbekistan on Statistics; 2023.
5. Gerasimov G. Iodine deficiency disorders in Central Asia: history and current status. Thyroid. 2002;12(10):897–903. doi:10.1089/105072502761016534
6. Republic of Uzbekistan. Law of the Republic of Uzbekistan No. ZRU-97: On Prevention of Iodine Deficiency Disorders. Tashkent: LexUZ; May 3, 2007.
7. Rohner F, Woodruff BA, Aaron GJ, Nizamov F, Ismailov S, et al. Infant and Young Child Feeding Practices and Micronutrient Status in Uzbekistan: Results of a National Survey. Nutrients. 2019;11(11):2642. doi:10.3390/nu11112642
8. World Health Organization, UNICEF, ICCIDD. Assessment of iodine deficiency disorders and monitoring their elimination: a guide for programme managers. 3rd ed. Geneva: World Health Organization; 2007.
9. Hess SY, Zimmermann MB, Adou P, et al. Treatment of iron deficiency anemia with oral iron improves thyroid function and efficacy of iodized salt in anemic, goitrous children. Am J Clin Nutr. 2002;75(5):919–925. doi:10.1093/ajcn/75.5.919
10. Ruf J, Carayon P. Structural and functional aspects of thyroid peroxidase. Arch Biochem Biophys. 2006;445(2):269–277. doi:10.1016/j.abb.2005.08.016
11. Schomburg L. Selenium, selenoproteins and the thyroid gland: interactions, development and clinical relevance. Nat Rev Endocrinol. 2012;8(3):160–171. doi:10.1038/nrendo.2011.174
12. Köhrle J. Selenium in endocrinology — international perspective. Endocr Connect. 2016;5(5):R38–R48. doi:10.1530/EC-16-0036
13. Alexander EK, Pearce EN, Brent GA, et al. 2017 Guidelines of the American Thyroid Association for the Diagnosis and Management of Thyroid Disease During Pregnancy and the Postpartum. Thyroid. 2017;27(3):315–389. doi:10.1089/thy.2016.0457

14. Morreale de Escobar G, Obregon MJ, Escobar del Rey F. Role of thyroid hormone during early brain development. Eur J Endocrinol. 2004;151(Suppl 3):U25–U37. doi:10.1530/eje.0.151u025

15. Korevaar TIM, Medici M, Visser TJ, Peeters RP. Thyroid disease in pregnancy: new insights in pathogenesis and clinical management. Nat Rev Endocrinol. 2017;13(10):610–622. doi:10.1038/nrendo.2017.93

16. Stagnaro-Green A, Abalovich M, Alexander E, et al. Guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and postpartum. Thyroid. 2011;21(10):1081–1125. doi:10.1089/thy.2011.0087

17. Lazarus JH, Bestwick JP, Channon S, et al. Antenatal thyroid screening and childhood cognitive function. N Engl J Med. 2012;366(6):493–501. doi:10.1056/NEJMoa1106104

18. Tomer Y. Mechanisms of autoimmune thyroiditis: from genetics to epigenetics. Trends Endocrinol Metab. 2014;25(11):561–569. doi:10.1016/j.tem.2014.06.009

19. Goldner W, Shostrom V, Yu F, et al. Familial risk of autoimmune thyroid disease in a defined population-based database. Thyroid. 2013;23(8):934–941. doi:10.1089/thy.2012.0435

20. Rastogi MV, LaFranchi SH. Congenital hypothyroidism: Orphanet Journal of Rare Diseases clinical framework. Orphanet J Rare Dis. 2010;5:17. doi:10.1186/1750-1172-5-17