International Journal of Nutrition and Biotechnology Advancements
https://journals.univ-tlemcen.dz/JNBA/index.php/JNBA
Online ISSN: 2992-202X

Review Article

Iodine physiology and thyroid homeostasis: environmental sources, metabolism, and adaptive mechanisms

Idris HANNOUS[1*], Mounia ARAB[2,3], Samir AIT ABDERRAHMANE [1], Djamila MESKINE[4]

[1]*Diabetology Department, Central Army Hospital, Algiers, Algeria.*
[2]*Faculty of Biological Sciences, University of Sciences and Technology Houari Boumediene. Algiers, Algeria.*
[3]*Laboratory of Applied Microbiology in Food, Biomedical and Environment, Department of Biology, Faculty of Nature Sciences and Life, University of Tlemcen, Tlemcen 13000, Algeria.*
[4]*Endocrinology Department, EPH Ibn Ziri-Bologhine, Algiers, Algeria.*

Article history: *March 2026*, *Date of acceptance: April 2026*

Abstract

Iodine is an essential trace element required for the synthesis of thyroid hormones and the support of neurological development. In the environment, iodine circulates primarily through the ocean–atmosphere–land biogeochemical cycle, with the oceans constituting its principal natural reservoir. Dietary iodine intake is largely determined by soil iodine content; marine foods represent the richest natural sources, while iodized salt, dairy products, drinking water, certain medications, and antiseptics serve as additional contributors.

The total body iodine content is estimated at 15–20 mg, of which approximately 75–80% is concentrated in the thyroid gland. Physiological requirements vary with age and are markedly increased during pregnancy and lactation; urinary iodine concentration constitutes the gold-standard biomarker for assessing iodine status at the population level.

Within the thyroid gland, iodide is actively transported by the sodium–iodide symporter (NIS), oxidized by thyroid peroxidase, and incorporated into thyroglobulin, leading to the synthesis of thyroxine (T4) and triiodothyronine (T3). Peripheral deiodinases further regulate thyroid hormone bioavailability, thereby highlighting the close functional interaction between iodine and selenium.

In response to iodine deficiency, the thyroid gland increases iodide uptake, adjusts the T3/T4 secretion ratio, and may develop follicular cell hyperplasia, ultimately resulting in goiter. Conversely, acute iodine excess triggers a transient inhibition of organification known as the Wolff–Chaikoff effect, followed by an escape mechanism; failure of this autoregulation may lead to thyroid dysfunction. Despite the widespread implementation of salt iodization programs, iodine imbalances persist in numerous populations worldwide, underscoring the importance of a comprehensive understanding of iodine physiology and its adaptive mechanisms.

Key words: Iodine, Iodine metabolism, Sodium–iodide symporter (NIS), Thyroid homeostasis, Iodine deficiency and excess.

Introduction

Iodine is an essential micronutrient required for the synthesis of the thyroid hormones triiodothyronine (T3) and thyroxine (T4), which play a central role in the regulation of energy metabolism, growth, and neurocognitive development, particularly during fetal life and early childhood (Zimmermann and Andersson 2012a). The human body contains approximately 15–20 mg of iodine, of which about 75–80% is concentrated in the thyroid gland, where it is utilized for hormone production (Zimmermann and Boelaert 2015). Circulating iodide is actively taken up by thyroid follicular cells through the sodium–iodide symporter (NIS), then oxidized and incorporated into thyroglobulin under the catalytic action of thyroid peroxidase to form iodotyrosines, the precursors of thyroid hormones (Ravera et al., 2017).

Environmental iodine availability is largely determined by its global biogeochemical cycle. The oceans constitute the principal natural reservoir of iodine and account for nearly 70% of the iodine inventory at the Earth's surface (Kaiho, 2017). Through volatilization and atmospheric transport, iodine is redistributed to terrestrial ecosystems, where its concentrations in soils and water are generally low and heterogeneous (Johnson, 2003; Kaiho, 2017). This geochemical variability directly influences the iodine content of foods. Marine products including fish, shellfish, and seaweed represent the richest natural dietary sources, whereas milk, dairy

products, and eggs constitute important contributors in many dietary patterns (Pearce et al., 2016; Zimmermann 2009). Nevertheless, salt iodization remains the most widely implemented public health strategy to prevent iodine deficiency disorders in many regions worldwide (Zimmermann, 2011; Zimmermann, 2009).

Despite substantial progress achieved through salt iodization programs, iodine imbalance remains a significant public health concern. Iodine deficiency may lead to chronic stimulation of the thyroid gland and the development of goiter and other iodine deficiency disorders, whereas excessive iodine intake can also precipitate thyroid dysfunction in susceptible individuals (Zimmermann, 2011; Zimmermann, 2009). In this context, an integrated understanding of iodine metabolism, sources of exposure, and thyroid adaptive mechanisms is essential to better elucidate how variations in iodine intake influence thyroid physiology and population health.

Environmental Iodine cycle and sources of exposure

Iodine is an essential trace element present in the environment at relatively low concentrations. Its availability depends on a complex cycle linking the oceans, the atmosphere, and the soil, and ultimately determines dietary iodine intake in human and animal populations. Understanding this cycle is crucial for assessing the risk of iodine deficiency or excess.

Biogeochemical cycle of Iodine

Oceanic reservoir

The oceans account for approximately 70% of the global iodine inventory at the Earth's surface and represent the primary source of iodine for terrestrial ecosystems (Kaiho, 2017). Iodine concentrations in seawater generally range from 45 to 60 µg/L but can reach very high levels in certain marine algae, ranging from 100 to 6,000 µg/g (Sanyaolu et al., 2021).

In alkaline and oxygenated ocean waters, iodate (IO_3^-) is the thermodynamically stable form of inorganic iodine, whereas iodide (I^-) predominates in coastal and surface waters as a result of biological, photochemical, and chemical reduction processes (Espino-Vázquez et al., 2022 ; Carpenter et al., 2021).

Ocean–atmosphere–soil cycle

Oceanic volatilization represents the principal pathway for the transfer of iodine to the atmosphere. Detected iodine species include particulate iodine (mainly organic), inorganic gaseous iodine (I_2, HI, HIO), and a mixture of volatile organic compounds such as CH_3I, CH_2I_2, CH_2ICl, CH_2IBr, C_2H_5I, and C_3H_7I (Saiz-Lopez et al., 2012). These compounds are subsequently deposited onto land through precipitation and contribute to iodine inputs into soils and freshwater systems via runoff and atmospheric deposition (Saiz-Lopez et al., 2014).

Geographical variability

Iodine concentrations in soils and rocks typically range from 0.1 to 3 mg/kg and 1 to 5 mg/kg, respectively, while concentrations in freshwater and rainwater generally vary between 0.5 and 10 µg/L (Kaiho, 2017; Johnson, 2003). This geographical variability directly influences the iodine content of plants and animals, and consequently the dietary iodine intake of humans (Zimmermann, 2009).

Microorganisms play a key role in iodine speciation and contribute to chemical and biological transformations across different marine compartments (Duborská et al., 2023; Moreda-Piñeiro et al., 2011).

Dietary sources of Iodine

Marine products

Marine foods constitute the richest dietary source of iodine. Fish, crustaceans, mollusks, and seaweeds accumulate substantial amounts of iodine, with concentrations varying according to species and marine environmental conditions (Zimmermann, 2009). Consumption of seaweed, particularly common in several Asian countries, may sometimes lead to excessive iodine intake (Pearce et al., 2016; Andersson et al., 2012).

Dairy products

Milk and dairy products represent important sources of iodine in populations from industrialized countries, owing to their moderate but relatively consistent iodine content (Zimmermann, 2009). Agricultural practices and the use of iodine-containing antiseptics in dairy farming can significantly increase the iodine concentration of milk (Bath et al., 2017; Cao et al., 1994).

Iodized salt

Iodized salt remains the principal vehicle for preventing iodine deficiency worldwide. Fortification levels vary across countries but generally range between 5 and 100 mg/kg (Zimmermann 2009).

Influence of soil and agricultural practices

When soil iodine content is low, plants and the animals that depend on them also exhibit low iodine concentrations. Agricultural practices, livestock feed supplementation, and the use of iodine-containing antiseptics can substantially modify iodine availability within the food chain (Zimmermann, 2009).

Non-dietary sources of Iodine exposure

Iodine-based antiseptics

Iodine-containing antiseptics, including iodine tincture, Lugol's solution, povidone-iodine (PVP-I), and iodoform (CHI_3), are widely used in human and veterinary medicine because of their antibacterial and antifungal properties. Repeated transcutaneous or transmucosal absorption may lead to iodine overload and subsequent thyroid dysfunction (Zimmermann, 2009).

Iodine-rich medications

Certain medications contain large amounts of iodine. Amiodarone (Cordarone®) is responsible for the majority of iodine-induced hyperthyroidism cases. A single 200 mg tablet of amiodarone contains approximately 75 mg of iodine, of which about 6 mg is released into the circulation, leading to prolonged iodine exposure (Bath et al., 2017). Iodized oil preparations such as Lipiodol® Ultra-Fluid have also been used in the treatment of severe iodine deficiency (Bogazzi et al., 2012).

Iodinated contrast media

Iodinated contrast agents used in medical imaging represent an acute and sometimes massive source of iodine exposure and may transiently alter thyroid function, particularly in susceptible individuals.

Dietary supplements

Dietary supplements derived from seaweed (e.g., *Fucus vesiculosus*) or phytoplankton constitute additional sources of iodine. Their consumption is often associated with specific dietary practices or nutritional supplementation regimens (Teas et al., 2004).

Human Iodine physiology

Iodine is an essential trace element required for the synthesis of thyroid hormones. Its homeostasis depends on tightly regulated mechanisms of absorption, distribution, storage, and elimination. Disruptions in iodine balance may lead to a wide spectrum of thyroid disorders, ranging from goiter to iodine-induced hypothyroidism or hyperthyroidism (Zimmermann & Boelaert, 2015). This section describes the distribution of iodine within the human body, physiological iodine requirements, and the biomarkers used to assess iodine status.

Body distribution of Iodine

Total body content

The human body contains approximately 15–20 mg of iodine, representing roughly 0.00002% of total body weight (Zimmermann & Boelaert 2015). Iodine is distributed across several compartments, including extracellular fluids, thyroid proteins, and certain organs such as the mammary gland and the kidneys (Thompson et al., 2020).

Thyroidal reservoir

The thyroid gland contains approximately 75–80% of the total body iodine pool, despite weighing only 15–25 g (Zimmermann and Boelaert, 2015). The thyroid actively concentrates plasma iodide via the sodium–iodide symporter (Na^+/I^-, NIS) and incorporates it into thyroglobulin through an organification process mediated by thyroid peroxidase, ultimately leading to the synthesis of thyroid hormones (T3 and T4) (Ravera et al., 2017; Dai et al., 1996).
Additional iodine reservoirs exist in extracellular fluids, including saliva, gastric juice, and plasma, as well as in the form of extrathyroidal organic iodine, notably circulating iodinated proteins (Thompson et al., 2020).

Physiological Iodine requirements

Age-related requirements

Iodine requirements vary throughout life. Recommended daily intakes are summarized in Table 1 (WHO 2007).

Table 1. Recommended iodine intake according to age group (WHO 2007).

Population group	Iodine intake (µg/day)
Infants (1–12 months)	90–100
Children (1–10 years)	90–100
Adolescents	100–150
Adults	100–150
Pregnancy	200–300
Lactation	200–300

These intake levels cover the needs related to the daily turnover of thyroid hormones as well as urinary iodine losses, which are approximately balanced in individuals with adequate iodine intake (Zimmermann, 2013; WHO, 2007).

Pregnancy and lactation

During pregnancy, maternal iodine requirements increase to approximately 200–300 µg/day in order to support fetal thyroid hormone synthesis. Physiological adaptations include increased renal clearance of iodide, enhanced maternal thyroid hormone production, and transplacental transfer of iodine to the fetus (Mégier et al., 2023; Alexander et al., 2017).
Lactation also requires increased iodine intake to ensure adequate iodine concentration in breast milk and optimal iodine supply to the infant.

International recommendations

The World Health Organization (WHO), UNICEF, and the International Council for the Control of Iodine Deficiency Disorders (ICCIDD) recommend universal salt iodization and age-specific daily iodine intake levels, emphasizing the importance of maintaining adequate iodine intake during pregnancy and early childhood (WHO, 2007).

Biomarkers of Iodine status

Assessment of iodine status in a population relies on several biological and morphological indicators that reflect iodine intake and its effects on the thyroid gland. No single biomarker provides a comprehensive evaluation of iodine nutrition. The principal indicators used in monitoring programs include urinary iodine concentration, serum thyroglobulin, neonatal TSH, and the prevalence of goiter or thyroid volume. Each marker reflects a specific aspect of iodine physiology, and their combined use allows a more reliable assessment of population iodine status (Zimmermann &Andersson, 2012b; WHO, 2007).

Urinary Iodine Concentration

Urinary iodine concentration (UIC) is the most widely used indicator for assessing recent iodine intake. Approximately 90% of ingested iodine is excreted in the urine, making urinary iodine a direct reflection of dietary iodine consumption. Because of substantial intra-individual variability, UIC is not reliable for individual assessment but becomes meaningful at the population level. Consequently, the median UIC is used in epidemiological surveys to estimate iodine intake adequacy. A median UIC ≥100 µg/L is considered adequate in school-age children (König et al., 2011; WHO, 2007).

Thyroglobulin

Serum thyroglobulin (Tg) is a sensitive biomarker of thyroid stimulation associated with iodine deficiency. Produced by thyroid follicular cells, Tg serves as the precursor for thyroid hormone synthesis. In the context of iodine deficiency, increased TSH stimulation leads to enhanced production and release of Tg into the circulation. Tg is particularly useful for evaluating the impact of iodine supplementation programs. However, its interpretation may be limited by the presence of anti-thyroglobulin antibodies or by certain thyroid disorders (Zimmermann et al., 2003).

Neonatal TSH

Thyroid-stimulating hormone (TSH) measured in neonatal screening programs for congenital hypothyroidism provides an indirect indicator of maternal iodine status during pregnancy. Maternal iodine deficiency may result in increased stimulation of the hypothalamic–pituitary–thyroid axis in the newborn, leading to elevated TSH levels. Analysis of the distribution of neonatal TSH values in a population can therefore help estimate the adequacy of iodine intake during pregnancy. According to WHO criteria, a proportion exceeding 3% of neonates with TSH values >5 mIU/L suggests population-level iodine deficiency (Zimmermann, 2012).

Goiter and thyroid volume

Goiter is a classic manifestation of chronic iodine deficiency resulting from prolonged TSH-mediated stimulation of the thyroid gland. This stimulation leads to hyperplasia and hypertrophy of thyroid follicular cells, causing an increase in thyroid volume. Thyroid palpation was historically used to estimate goiter prevalence in field surveys; however, ultrasound is now considered the reference method for accurately measuring thyroid volume. This indicator reflects long-term exposure to iodine deficiency and is useful for evaluating the long-term effects of iodine supplementation programs (Pearce et al., 2013; WHO, 2007).

Limitations and complementarity of biomarkers

Each biomarker presents specific limitations in the assessment of iodine status. Urinary iodine reflects recent intake but does not indicate the physiological consequences of deficiency. Thyroglobulin reflects thyroid stimulation but may be influenced by thyroid autoimmunity. Neonatal TSH primarily reflects maternal iodine nutrition, whereas goiter represents a late marker of chronic exposure. The combined use of these indicators is therefore essential for reliable monitoring of iodine nutrition and the effectiveness of prevention programs (WHO, 2007).

Thyroid metabolism of Iodine

The thyroid gland is responsible for the uptake, organification, and incorporation of iodine into thyroid hormones. After intestinal absorption in the form of iodide (I^-), iodine is transported to the thyroid, where it undergoes several sequential steps: uptake by thyrocytes, transport to the follicular lumen, oxidation and incorporation into thyroglobulin, and ultimately the synthesis of thyroid hormones. The hormones produced are then released into the circulation and further metabolized in peripheral tissues to modulate their biological activity. These processes are tightly regulated by thyroid-stimulating hormone (TSH) (Kaiho, 2017).

Iodide transport into thyrocytes

Thyroidal iodide uptake represents the initial step in thyroid hormone synthesis. This process is mediated by the sodium–iodide symporter (NIS), a transmembrane protein located on the basolateral membrane of thyroid follicular cells.

NIS enables the active transport of iodide into thyrocytes by utilizing the sodium electrochemical gradient generated by the Na^+/K^+-ATPase pump. Through this mechanism, the thyroid gland is able to concentrate intracellular iodine to levels far exceeding those in plasma. NIS expression is primarily stimulated by TSH and constitutes a key determinant of the efficiency of thyroid iodide uptake (Ravera et al., 2017; Dai et al., 1996).

Apical transport of iodide

Once inside thyrocytes, iodide is transported to the follicular lumen, where thyroid hormone synthesis takes place. This transport is mainly mediated by pendrin, a protein located at the apical membrane and encoded by the SLC26A4 gene.

Pendrin facilitates the movement of iodide from the cytoplasm into the thyroid colloid. Alterations in this protein can impair iodide transport and are involved in certain inherited disorders, including Pendred syndrome (Everett et al., 1997).

Iodine organification and hormone synthesis

Iodine organification occurs within the follicular colloid and is catalyzed by thyroid peroxidase (TPO). This enzyme oxidizes iodide into its reactive form and catalyzes its incorporation into tyrosine residues of thyroglobulin, a glycoprotein synthesized by thyroid follicular cells.

Iodination of thyroglobulin leads to the formation of monoiodotyrosine (MIT) and diiodotyrosine (DIT). Coupling of these iodotyrosines results in the formation of the thyroid hormones triiodothyronine (T3) and thyroxine (T4).

Under TSH stimulation, iodinated thyroglobulin is internalized by thyrocytes and degraded within lysosomes, releasing T3 and T4, which are subsequently secreted into the circulation (Ravera et al., 2017).

Peripheral metabolism of thyroid hormones

Thyroxine (T4), the principal hormone secreted by the thyroid gland, largely functions as a prohormone. In peripheral tissues, it is converted into triiodothyronine (T3), the biologically active form, by a family of enzymes known as deiodinases.

Three isoforms have been identified: D1, D2, and D3. Deiodinases D1 and D2 catalyze the conversion of T4 into active T3, whereas D3 inactivates thyroid hormones by producing reverse T3 (rT3).

These enzymes are selenoproteins, which explains the close physiological interaction between iodine and selenium in the regulation of thyroid hormone metabolism (Winther et al., 2020).

Thyroid adaptation to variations in Iodine intake

The thyroid gland possesses autoregulatory mechanisms that allow it to maintain relatively stable hormone production despite fluctuations in iodine intake. These adaptive responses help preserve thyroid homeostasis under conditions of either iodine deficiency or iodine excess. However, when these regulatory systems are overwhelmed or impaired, thyroid dysfunction may occur.

Adaptation to Iodine deficiency

In the context of iodine deficiency, the thyroid activates several compensatory mechanisms aimed at maintaining adequate hormone production.

One of the earliest adaptive responses is an increase in thyroidal iodide uptake by thyrocytes. Reduced circulating concentrations of thyroid hormones lead to increased secretion of thyroid-stimulating hormone (TSH), which stimulates the expression of the sodium–iodide symporter (NIS) and enhances intracellular iodide accumulation (Zimmermann & Boelaert 2015).

Concurrently, the thyroid adjusts its hormone production by modifying the T3/T4 secretion ratio. Triiodothyronine (T3), which contains three iodine atoms, is preferentially produced compared with thyroxine (T4), which contains four iodine atoms (Zimmermann & Boelaert, 2015). This adaptation optimizes the utilization of the limited iodine supply while maintaining adequate peripheral thyroid hormone activity.

When iodine deficiency persists, chronic TSH stimulation induces hyperplasia and hypertrophy of thyroid follicular cells, leading to a progressive increase in thyroid volume and the development of goiter (Zimmermann & Boelaert, 2015). This structural adaptation enhances the gland's capacity to capture iodine from the circulation.

Adaptation to Iodine excess

Excessive iodine intake may also disrupt thyroid hormone synthesis. To prevent excessive hormone production, the thyroid gland relies on an autoregulatory mechanism known as the Wolff–Chaikoff effect (Eng et al., 1999).

This phenomenon corresponds to a transient inhibition of iodine organification within the thyroid when intracellular iodide concentrations become elevated. This inhibitory response temporarily reduces thyroid hormone synthesis and protects the organism from iodine-induced hyperthyroidism.

However, this effect is typically transient. After several days, the thyroid regains its capacity for hormone synthesis through an escape phenomenon, mainly associated with reduced iodide uptake by thyrocytes (Eng et al., 1999). This adaptation restores normal thyroid function despite continued exposure to high iodine intake.

Failure of autoregulatory mechanisms

In certain situations, thyroid adaptive mechanisms may become insufficient or fail, resulting in iodine-induced thyroid dysfunction.

Excess iodine exposure can trigger iodine-induced hyperthyroidism, particularly in individuals with autonomous multinodular goiter or latent Graves' disease. Conversely, in some individuals, especially patients with autoimmune thyroiditis or neonates, the inhibitory effect of iodine may persist and lead to iodine-induced hypothyroidism.

Such disturbances may also occur following exposure to large iodine loads, including iodinated contrast media, iodine-rich medications (notably amiodarone), or iodine-containing antiseptics (Bogazzi et al., 2012).

Understanding these adaptive mechanisms and their limitations is essential for anticipating the thyroidal consequences of variations in iodine intake and for guiding clinical monitoring in populations exposed to significant iodine fluctuations.

Interaction between Iodine and Selenium

Iodine and selenium are two essential micronutrients for the optimal functioning of the thyroid gland. Their interaction is particularly close, as selenium is involved in several steps of thyroid hormone metabolism and in protecting the thyroid from oxidative stress generated during iodine organification (Winther et al., 2020).

Role of selenoproteins

Selenium is incorporated in the form of selenocysteine into a family of proteins known as selenoproteins, several of which play key roles in thyroid physiology. The thyroid gland is among the tissues with the highest selenium concentrations in the human body.

Among these proteins, glutathione peroxidases (GPx) and thioredoxin reductases are responsible for detoxifying hydrogen peroxide (H_2O_2) produced during iodide oxidation and the iodination of thyroglobulin. This antioxidant system protects thyrocytes from oxidative damage generated during thyroid hormone synthesis, a process associated with high production of reactive oxygen species (Winther et al., 2020).

Deiodinases

Selenium is also an essential component of deiodinases (D1, D2, and D3), the enzymes responsible for the peripheral metabolism of thyroid hormones.

Type 1 and type 2 deiodinases (D1 and D2) catalyze the conversion of thyroxine (T4) into the biologically active triiodothyronine (T3), whereas D3 catalyzes hormone inactivation through the conversion of T4 into reverse T3 (rT3) and T3 into T2 (Winther et al., 2020).

Consequently, selenium deficiency may impair the peripheral conversion of T4 to T3 and alter thyroid hormone balance independently of iodine status.

Thyroid oxidative stress

Thyroid hormone synthesis relies on the use of hydrogen peroxide, which is required for thyroid peroxidase (TPO) activity during iodine oxidation and organification. This production of H_2O_2 exposes the thyroid gland to a significant risk of oxidative stress.

Antioxidant selenoproteins help limit this stress by neutralizing reactive oxygen species. In the case of combined iodine and selenium deficiency, the thyroid may experience increased oxidative damage, which can promote thyroid hyperplasia, structural alterations, and certain thyroid dysfunctions (Winther et al., 2020).

This interaction explains why assessment of iodine status in a population should ideally take selenium status into account, as both micronutrients act in a complementary manner in thyroid physiology.

Public health implications

Iodine is an essential micronutrient, and ensuring adequate intake remains a major public health priority. Imbalances in iodine status, whether deficiency or excess, can lead to thyroid disorders and significant neurodevelopmental consequences (Zimmermann, 2009). For this reason, numerous international initiatives aim to ensure sufficient iodine intake across populations.

Iodization programs

Salt fortification with iodine represents the most effective and widely implemented strategy for preventing iodine deficiency disorders. Universal Salt Iodization (USI), recommended by the World Health Organization (WHO), UNICEF, and the International Council for the Control of Iodine Deficiency Disorders (ICCIDD), aims to ensure adequate iodine intake across entire populations (WHO, 2007).

This intervention has led to a substantial reduction in the global prevalence of iodine deficiency disorders, particularly endemic goiter and neurocognitive impairment associated with iodine deficiency during pregnancy (WHO, 2007). Nevertheless, the effectiveness of these programs depends on the coverage of iodized salt, quality control of iodization processes, and regular monitoring of population iodine status (Hannous et al., 2025).

Persistent Iodine deficiency

Despite significant progress, iodine deficiency remains a public health problem in several regions of the world. It may result from low iodine content in soils and foods, insufficient coverage of iodization programs, or changes in dietary habits.

Iodine deficiency leads to a spectrum of conditions collectively referred to as iodine deficiency disorders (IDD), including goiter, hypothyroidism, impaired brain development, and cognitive deficits in children. Pregnant women and young children are the most vulnerable groups due to their increased physiological iodine requirements (WHO, 2007).

Emerging Iodine excess

Alongside persistent iodine deficiency in certain regions, excessive iodine intake is increasingly reported in other settings. This excess may result from excessive salt iodization, the use of iodine-rich dietary supplements, or medical and environmental exposures such as iodinated contrast agents or iodine-containing medications (Farebrother et al., 2019; Farebrother et al., 2018).

Excess iodine intake may disrupt thyroid autoregulation and promote thyroid dysfunction, particularly iodine-induced hyperthyroidism or hypothyroidism related to a persistent Wolff–Chaikoff effect. These

observations highlight the importance of maintaining a precise balance between preventing deficiency and avoiding excessive iodine intake.

Population surveillance

Monitoring iodine status at the population level is a central component of public health strategies. It primarily relies on measuring median urinary iodine concentration in representative population groups, particularly school-age children and pregnant women (WHO, 2007).

This monitoring may be complemented by other indicators such as goiter prevalence, serum thyroglobulin levels, and ultrasound assessment of thyroid volume. The combined analysis of these parameters provides a more accurate evaluation of population iodine status and helps guide fortification and prevention policies.

Sustained control of iodine-related disorders therefore requires an integrated approach combining food fortification, continuous epidemiological surveillance, and adaptive public health policies to maintain optimal iodine nutrition in populations.

Conclusion

Iodine is an essential micronutrient required for the proper functioning of the thyroid gland and for the synthesis of thyroid hormones, which play a fundamental role in metabolism, growth, and neurological development. Thyroid iodine metabolism relies on several tightly regulated mechanisms, ranging from iodide uptake by thyrocytes to the production and peripheral metabolism of the hormones T3 and T4. These processes ensure the hormonal balance necessary for normal physiological function.

The thyroid gland also possesses adaptive mechanisms that enable it to respond to variations in iodine intake. In conditions of iodine deficiency, the gland increases iodide uptake and adjusts hormone synthesis in order to preserve thyroid function, sometimes at the cost of an increase in thyroid volume. Conversely, during iodine excess, autoregulatory mechanisms temporarily limit hormone synthesis to prevent excessive thyroid hormone production.

Maintaining adequate iodine balance therefore remains essential for health. Despite the significant progress achieved through salt iodization programs, iodine deficiency persists in certain regions of the world, while excessive iodine intake may also occur in other contexts.

In this context, further research is needed to better understand the interactions between iodine and other micronutrients, particularly selenium, as well as the mechanisms regulating thyroid function. Improving surveillance and prevention strategies will be crucial for protecting populations against disorders associated with iodine imbalance.

Acknowledgement

The authors have no acknowledgements to declare.

Conflict of interest

There is no conflict of interest to be declared.

References

- Alexander, E. K., Pearce, E. N., Brent, G. A., Brown, R. S., Chen, H., Dosiou, C., & Sullivan, S. (2017). 2017 Guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum. *Thyroid*, *27*(3), 315-389.
- Andersson, M., Karumbunathan, V., & Zimmermann, M. B. (2012). Global iodine status in 2011 and trends over the past decade. *The Journal of nutrition*, *142*(4), 744-750.
- Bath, S. C., Hill, S., Infante, H. G., Elghul, S., Nezianya, C. J., & Rayman, M. P. (2017). Iodine concentration of milk-alternative drinks available in the UK in comparison with cows' milk. *British Journal of Nutrition*, *118*(7), 525-532.
- Bogazzi, F., Tomisti, L., Bartalena, L., Aghini-Lombardi, F., & Martino, E. (2012). Amiodarone and the thyroid: a 2012 update. *Journal of endocrinological investigation*, *35*(3), 340-348.
- Cao, X. Y., Jiang, X. M., Dou, Z. H., Rakeman, M. A., Kareem, A., Zang, M. L., & DeLong, G. R. (1994). Iodination of irrigation water as a method of supplying iodine to a severely iodine-deficient population in Xinjiang, China. *The lancet*, *344*(8915), 107-110.

- Carpenter, L. J., Chance, R. J., Sherwen, T., Adams, T. J., Ball, S. M., Evans, M. J., & Wadley, M. R. (2021). Marine iodine emissions in a changing world. *Proceedings of the Royal Society A: Mathematical, Physical and Engineering Sciences*, *477*(2247).
- Dai, G., Levy, O., & Carrasco, N. (1996). Cloning and characterization of the thyroid iodide transporter. *Nature*, *379*(6564), 458-460.
- Duborská, E., Vojtková, H., Matulová, M., Šeda, M., & Matúš, P. (2023). Microbial involvement in iodine cycle: mechanisms and potential applications. *Frontiers in Bioengineering and Biotechnology*, *11*, 1279270.
- Eng, P. H., Cardona, G. R., Fang, S. L., Previti, M., Alex, S., Carrasco, N., & Braverman, L. E. (1999). Escape from the acute Wolff-Chaikoff effect is associated with a decrease in thyroid sodium/iodide symporter messenger ribonucleic acid and protein. *Endocrinology*, *140*(8), 3404-3410.
- Espino-Vázquez, A. N., Rojas-Castro, F. C., & Fajardo-Yamamoto, L. M. (2022). Implications and Practical Applications of the Chemical Speciation of Iodine in the Biological Context. *Future Pharmacology*, *2*(4), 377-414. https://doi.org/10.3390/futurepharmacol2040026
- Everett, L. A., Glaser, B., Beck, J. C., Idol, J. R., Buchs, A., Heyman, M. A., ... & Green, E. D. (1997). Pendred syndrome is caused by mutations in a putative sulphate transporter gene (PDS). *Nature genetics*, *17*(4), 411-422.
- Farebrother, J., Zimmermann, M. B., & Andersson, M. (2019). Excess iodine intake: sources, assessment, and effects on thyroid function. *Annals of the New York Academy of Sciences*, *1446*(1), 44-65.
- Farebrother, J., Zimmermann, M. B., Abdallah, F., Assey, V., Fingerhut, R., Gichohi-Wainaina, W. N., ... & Andersson, M. (2018). Effect of excess iodine intake from iodized salt and/or groundwater iodine on thyroid function in nonpregnant and pregnant women, infants, and children: a multicenter study in East Africa. *Thyroid*, *28*(9), 1198-1210.
- Hannous, I., Arab, M., Ait Abderrahmane, S., & Meskine, D. (2025). Assessment of the iodine content of salt consumed in the town of Bechar. *International Journal of Nutrition and Biotechnology Advancements*, 2(1), 1-8.
- Johnson, C. C. (2003). The geochemistry of iodine and its application to environmental strategies for reducing the risk from iodine deficiency disorders (IDD).
- Kaiho, T. (2017). *Iodine made simple*. CRC Press.
- König, F., Andersson, M., Hotz, K., Aeberli, I., & Zimmermann, M. B. (2011). Ten repeat collections for urinary iodine from spot samples or 24-hour samples are needed to reliably estimate individual iodine status in women. *The Journal of nutrition*, *141*(11), 2049-2054.
- Mégier, C., Dumery, G., & Luton, D. (2023). Iodine and thyroid maternal and fetal metabolism during pregnancy. *Metabolites*, *13*(5), 633.
- Moreda-Pineiro, A., Romaris-Hortas, V., & Bermejo-Barrera, P. (2011). A review on iodine speciation for environmental, biological and nutrition fields. *Journal of Analytical Atomic Spectrometry*, *26*(11), 2107-2152.
- Pearce, E. N., Andersson, M., & Zimmermann, M. B. (2013). Global iodine nutrition: where do we stand in 2013?. *Thyroid*, *23*(5), 523-528.
- Pearce, E. N., Lazarus, J. H., Moreno-Reyes, R., & Zimmermann, M. B. (2016). Consequences of iodine deficiency and excess in pregnant women: an overview of current knowns and unknowns. *The American journal of clinical nutrition*, *104*(suppl_3), 918S-923S.
- Ravera, S., Reyna-Neyra, A., Ferrandino, G., Amzel, L. M., & Carrasco, N. (2017). The sodium/iodide symporter (NIS): molecular physiology and clinical implications. *Annual Review of Physiology*, 79, 261–289. https://doi.org/10.1146/annurev-physiol-022516-034125
- Saiz-Lopez, A., Fernandez, R. P., Ordóñez, C., Kinnison, D. E., Gómez Martín, J. C., Lamarque, J. F., & Tilmes, S. (2014). Iodine chemistry in the troposphere and its effect on ozone. *Atmospheric Chemistry and Physics*, *14*(23), 13119-13143.
- Saiz-Lopez, A., Plane, J. M., Baker, A. R., Carpenter, L. J., Von Glasow, R., Gómez Martín, J. C., ... & Saunders, R. W. (2012). Atmospheric chemistry of iodine. *Chemical reviews*, *112*(3), 1773-1804.
- Sanyaolu, O. M., Mouri, H., Selinus, O., & Odukoya, A. (2021). Sources, pathways, and health effects of iodine in the environment. In *Practical Applications of Medical Geology* (pp. 565-613). Cham: Springer International Publishing.
- Teas, J., Pino, S., Critchley, A., & Braverman, L. E. (2004). Variability of iodine content in common commercially available edible seaweeds. *Thyroid*, *14*(10), 836-841.
- Thompson, C. J., Verbalis, J. G., Melmed, S., Auchus, R. J., Goldfine, A. B., Koenig, R. J., & Rosen, C. J. (2020). Williams textbook of endocrinology. *Posterior Pituitary (14th ed), Elsevier*, 303-330.

- Winther, K. H., Rayman, M. P., Bonnema, S. J., & Hegedüs, L. (2020). Selenium in thyroid disorders—essential knowledge for clinicians. *Nature Reviews Endocrinology*, *16*(3), 165-176.
- World Health Organization. (2007). Assessment of iodine deficiency disorders and monitoring their elimination: a guide for programme managers.
- Zimmermann, M. B. (2009). Iodine deficiency. *Endocrine Reviews*, 30(4), 376–408. https://doi.org/10.1210/er.2009-0011
- Zimmermann, M. B. (2011, August). The role of iodine in human growth and development. In *Seminars in cell & developmental biology* (Vol. 22, No. 6, pp. 645-652). Academic Press.
- Zimmermann, M. B. (2012). The effects of iodine deficiency in pregnancy and infancy. *Paediatric and perinatal epidemiology*, *26*, 108-117.
- Zimmermann, M. B. (2013). Iodine deficiency and excess in children: worldwide status in 2013. *Endocrine practice*, *19*(5), 839-846.
- Zimmermann, M. B., & Andersson, M. (2012a). Assessment of iodine nutrition in populations: past, present, and future. *Nutrition Reviews*, 70(10), 553–570. https://doi.org/10.1111/j.1753-4887.2012.00528.x
- Zimmermann, M. B., & Andersson, M. (2012b). Update on iodine status worldwide. *Current Opinion in Endocrinology, Diabetes and Obesity*, *19*(5), 382-387.
- Zimmermann, M. B., & Boelaert, K. (2015). Iodine deficiency and thyroid disorders. *The Lancet Diabetes & Endocrinology*, 3(4), 286–295. https://doi.org/10.1016/S2213-8587(14)70225-6
- Zimmermann, M. B., Moretti, D., Chaouki, N., & Torresani, T. (2003). Development of a dried whole-blood spot thyroglobulin assay and its evaluation as an indicator of thyroid status in goitrous children receiving iodized salt. *The American journal of clinical nutrition*, *77*(6), 1453-1458.