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## Iodine and thyroid function: Implications for metabolism and performance in athletes

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### Abstract

Iodine is a vital micronutrient that underpins thyroid hormone synthesis, affecting myriad physiological processes that extend well beyond basic metabolism. In athletes, an optimal level of thyroid hormones particularly triiodothyronine (T<sub>3</sub>) and thyroxine (T<sub>4</sub>) is integral to energy production, thermoregulation, macronutrient metabolism, and muscle recovery. Despite well-publicized global efforts to reduce iodine deficiency through measures such as salt iodization, suboptimal iodine status still persists, including in populations that might be assumed healthy and nutritionally aware. Athletes are especially vulnerable due to higher nutrient turnover, specialized diets, and additional micronutrient losses through sweat. Even marginal deficiencies can attenuate the benefits of training and potentially impair athletic performance, manifesting as increased fatigue, reduced aerobic capacity, or prolonged recovery intervals.

The purpose of this review is to present a comprehensive examination of the nexus between iodine and thyroid function, with particular emphasis on the metabolic and performance implications for athletes. We begin by outlining key aspects of iodine physiology, tracing the pathway from dietary intake through the sodium-iodide symporter (NIS) and culminating in the synthesis of T<sub>3</sub> and T<sub>4</sub> within the thyroid gland. Next, we examine the mechanisms of the hypothalamic-pituitary-thyroid (HPT) axis and highlight how even subtle imbalances in iodine intake may provoke disruptions in thyroid-stimulating hormone (TSH), leading to subclinical or overt hypothyroidism.

Empirical findings on the influence of exercise on iodine turnover and thyroid hormone dynamics receive close attention, revealing a bidirectional relationship wherein high-intensity or high-volume training can alter thyroid homeostasis, while inadequate iodine status exacerbates performance deficits. The review further explores typical dietary sources of iodine, recommended intake levels for adult populations, and the ways in which these guidelines may need to be adjusted for athletes with elevated needs. Strategies for supplementation are presented alongside cautions regarding excessive iodine intake, which can lead to hyperthyroidism, thyroid autoimmunity, or goiter.

By synthesizing evidence from clinical, epidemiological, and performance-based research, this review underscores how iodine sufficiency is critical for maximizing metabolic efficiency and physical potential. Coaches, athletes, and sports health professionals are therefore urged to incorporate monitoring of iodine status alongside other key micronutrients into comprehensive nutrition and training programs designed to foster peak performance over the long term.

**Keywords:** HPT Axis, VO<sub>2</sub> max, T<sub>3</sub> and T<sub>4</sub>, iodine, thyroid hormones, metabolism, athletic performance, iodine deficiency, supplementation, sports nutrition

### 1. Introduction

Iodine is an essential trace element and the thyroid gland's follicular cells produce hormones. Thyroid function is regulated by the hypothalamic-pituitary-thyroid axis and is influenced by Thyrotropin Releasing Hormone (TRH), Thyroid-Stimulating Hormone (TSH), triiodothyronine (T<sub>3</sub>), and thyroxine (T<sub>4</sub>). The thyroid gland is essential for growth and development, homeostasis, and the proper operation of the neurological, cardiovascular, and reproductive systems (Zhou *et al.*, 2022) <sup>[34]</sup>. The thyroid gland's capacity to generate adequate amounts of T<sub>3</sub> and T<sub>4</sub> is heavily dependent on the continuous availability of iodine in the diet. Although significant progress has been made in many regions through salt iodization and other public health interventions, iodine deficiency remains a global issue. According to the World Health Organization (2019) <sup>[31]</sup>, nearly one-third of the global population still does not have access to sufficient iodine, indicating the persistence of inadequate intake in diverse demographics. For athletes, the implications of iodine deficiency take on heightened significance.

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Physical performance relies on optimal metabolic and endocrine functioning, as athletes consistently push physiological boundaries through training and competition (Hackney, 2020) <sup>[12]</sup>. When iodine is insufficient, a cascade of hormonal imbalances can arise, potentially impairing muscle function, oxygen utilization, and recovery times (Casa *et al.*, 2019) <sup>[5]</sup>. Additionally, contemporary shifts toward specialized diets such as plant-based, dairy-free, or restrictive calorie plans can exacerbate the likelihood of iodine shortfalls in athletic communities (Bath & Rayman, 2019) <sup>[2]</sup>.

1.1 Rationale for focusing on athletes’ iodine status

While iodine deficiency is widely recognized as detrimental for the general population, its impact on athletes warrants particular attention. Even mild hypothyroidism or subclinical thyroid dysfunction can affect critical performance variables, including aerobic capacity (VO<sub>2</sub> max), anaerobic power, and substrate utilization (Fortunato *et al.*, 2018) <sup>[10]</sup>. Low T<sub>3</sub> levels may lead to reduced mitochondrial activity, hampering the efficient production of adenosine triphosphate (ATP), which underlies muscular contractions. In a domain where milliseconds and minute performance gains can determine success or failure, such a decrement can be profoundly consequential (Williams *et al.*, 2018) <sup>[32]</sup>. Moreover, competitive sports often entail rigorous training schedules, where recovery capacity is as

critical as the ability to sustain exertion. Thyroid hormones play a central role in muscle protein synthesis and repair, meaning inadequate iodine jeopardizes the athlete’s resilience, increases the risk of injuries, and prolongs the time needed for muscle regeneration (Gärtner, 2020) <sup>[11]</sup>. The incidence of micronutrient deficiencies, including iodine, may also go under-recognized in athletic settings because many participants believe their “healthy lifestyles” protect them from nutritional shortfalls (Maughan & Shirreffs, 2019) <sup>[20]</sup>. This misconception can be problematic if their dietary regimens are suboptimal in iodine-rich foods.

1.2 Global patterns of iodine deficiency and relevance to sports

Despite public health initiatives, studies continue to reveal uneven coverage of iodized salt programs (Andersen *et al.*, 2020) <sup>[1]</sup>. This uneven implementation means many regions may still present moderate to high deficiency rates particularly areas with iodine-depleted soil or those reliant on foods grown in such soils. Athletes training or living in these areas might be at a more pronounced risk of subclinical hypothyroidism. Some varieties of Himalayan “natural” salt, which are made from Tibetan continental lakes at high elevations, lack iodine. The native peasant population in Tibet has high levels of cretinism and other thyroid issues as a result of consuming “natural” lake salt (Matters *et al.*, 2022) <sup>[35]</sup>.

Table 1: Global iodine intake estimates across selected regions

Region	Median UIC (µg/L)	Deficiency Status	Reference
Central Europe	90-110	Mild deficiency	Zimmermann & Andersson (2021)
North America	130-190	Generally sufficient	WHO (2019)
East Asia (Rural)	70-100	Mild deficiency	Andersen <i>et al.</i> (2020) <sup>[1]</sup>
South Asia (Coastal)	120-160	Generally sufficient	Bath & Rayman (2019) <sup>[2]</sup>

Notes: Values indicate median urinary iodine concentration (UIC) in adult populations. Cutoffs as per WHO guidelines

Complicating matters, many sports nutrition approaches emphasize six essential nutrients are found in food and drink and are necessary for human health Water, vitamins, minerals, proteins, lipids, and carbs (Soubhagya & Srinivasa, 2024) <sup>[28]</sup>. This mismatch suggests that sports dietitians and athletes might overlook the significance of iodine. Indeed, reliance on nutritional supplements that do not include iodine or the replacement of dairy products with non-iodized plant-based alternatives can aggravate deficiency risk (Kreider *et al.*, 2019) <sup>[16]</sup>.

2. Iodine Physiology and Thyroid Hormone Synthesis

Iodine is absorbed as iodide in the gastrointestinal tract, predominantly in the small intestine (Nishi *et al.*, 2023) <sup>[23]</sup>. Upon absorption, it circulates in the bloodstream until it is actively transported into the thyroid gland. The fundamental significance of iodine centers on its unique ability to be incorporated into the structure of thyroid hormones an attribute that no other mineral can replicate. Understanding the intricacies of this process lays the groundwork for appreciating how an iodine shortfall can disrupt endocrine equilibrium and, by extension, athletic performance.

2.1 Uptake and Transport Mechanisms

The Sodium Iodide Symporter (NIS), situated in the basolateral membrane of thyroid follicular cells, is pivotal for concentrating iodine in the thyroid gland. This active transport mechanism enables the gland to accumulate iodine

at concentrations far exceeding those in circulation (Spitzweg & Morris, 2017) <sup>[30]</sup>. Once inside the follicular cells, iodide traverses the apical membrane via pendrin, reaching the colloid where it undergoes oxidation and organification steps necessary for hormone synthesis (Andersen *et al.*, 2020) <sup>[1]</sup>.

2.2 Thyroglobulin and Hormone Assembly

Thyroglobulin, a large glycoprotein produced by follicular cells, serves as the scaffold for thyroid hormone synthesis (). Within its numerous tyrosyl residues, iodination occurs under the catalytic action of thyroid peroxidase (TPO). This process first produces monoiodotyrosine (MIT) and diiodotyrosine (DIT). Subsequent coupling of these iodotyrosines yields T<sub>3</sub> (composed of one MIT and one DIT) or T<sub>4</sub> (two DITs). T<sub>4</sub>, secreted in much higher quantities than T<sub>3</sub>, functions largely as a prohormone, with T<sub>3</sub> being the metabolically active form that exerts most thyroid hormone effects at the cellular level (Kohrle, 2015) <sup>[15]</sup>.

2.3 The Hypothalamic-Pituitary-Thyroid Axis

The synthesis of thyroid hormone (TH) is regulated by the hypothalamus-pituitary-thyroid (HPT) axis (Carvalho *et al.*, 2007) <sup>[36]</sup>. Low thyroid hormone concentrations in the bloodstream prompt the hypothalamus to secrete thyrotropin-releasing hormone (TRH), which in turn stimulates the anterior pituitary to release thyroid-

stimulating hormone (TSH). TSH upregulates the thyroid gland's uptake of iodide, TPO activity, and thyroglobulin synthesis. As T<sub>3</sub> and T<sub>4</sub> levels rise, negative feedback loops reduce TRH and TSH secretion to maintain balance.

For physically active populations, subtle shifts in this axis can occur in response to factors like caloric intake, stress, and exercise intensity (Hackney, 2020) <sup>[12]</sup>. Elevated TSH levels over an extended period generally indicate that the body is compensating for insufficient iodine supply or compromised thyroid function, potentially foreshadowing hypothyroidism if not corrected.

## 2.4 Significance for Athletes

Thyroid hormones drive multiple processes integral to high-level athletic performance. T<sub>3</sub> enhances gene expression linked to mitochondrial function, boosting aerobic metabolism and enabling more efficient ATP generation (Bianco & McAninch, 2017) <sup>[3]</sup>. By modulating the synthesis of myosin heavy chain isoforms, T<sub>3</sub> also influences muscle fiber type distribution and contraction velocity (Kruger et al., 2020) <sup>[17]</sup>. Given that power output and endurance depend on robust muscular and metabolic capacity, any perturbation in thyroid hormone availability can produce performance deficits (Fortunato et al., 2018) <sup>[10]</sup>.

Additionally, T<sub>3</sub> fosters glycogen replenishment and promotes protein synthesis, facilitating faster recovery from training-induced muscle damage (Feyerabend et al., 2021) <sup>[9]</sup>. High-volume or intense exercise periods demand an upregulated metabolic rate, which T<sub>3</sub> helps sustain. Consequently, inadequate iodine intake hinders T<sub>3</sub> production and peripheral T<sub>4</sub>-to-T<sub>3</sub> conversion, limiting the athlete's capacity to meet heightened energy and repair demands (Williams et al., 2018) <sup>[32]</sup>.

## 3. Iodine deficiency and thyroid dysfunction in athletes

Global reports highlight that iodine deficiency remains the principal cause of preventable thyroid dysfunction (WHO, 2019). In athletic contexts, this deficiency can manifest in a

range of presentations from mild or subclinical hypothyroidism to overt hypothyroidism, each carrying implications for training capacity, adaptation, and overall health.

### 3.1 Defining Iodine Deficiency

Iodine status is often assessed using urinary iodine concentration (UIC). According to the WHO, an adequate median UIC for adults is 100-199 µg/L. Values below 100 µg/L indicate mild deficiency, while levels below 50 µg/L suggest moderate to severe deficiency. In certain athletic populations like distance runners and endurance athletes studies have reported UIC values in the mildly deficient range, with possible consequences for metabolic rate and performance (Maughan & Shirreffs, 2019) <sup>[20]</sup>

### 3.2 Subclinical and Clinical hypothyroidism in sports

Subclinical hypothyroidism typically presents with elevated TSH alongside normal T<sub>4</sub> and T<sub>3</sub> levels (Peterson et al., 2018) <sup>[25]</sup>. Individuals may not exhibit marked symptoms yet could experience reduced exercise tolerance or subtle fatigue (Orce et al., 2020) <sup>[24]</sup>. Clinical hypothyroidism arises when T<sub>4</sub> and/or T<sub>3</sub> levels drop below reference ranges, often accompanied by clear clinical signs such as weight gain, cold intolerance, and lethargy (Duntas & Brenta, 2018) <sup>[7]</sup>. Even mild manifestations, however, can derail training progress and hinder peak performance.

### 3.3 Prevalence and Epidemiological Patterns

Although systematic data on the prevalence of iodine deficiency specifically among athletes is somewhat limited, multiple regional studies have suggested that sportspeople can be as vulnerable as the general population sometimes more so (Brunn et al., 2005; Witt et al., 2015) <sup>[4, 33]</sup>. Where iodized salt is not mandated or commonly used, deficiency rates can be pronounced. Additionally, populations engaging in high sweating rates or adopting restrictive dietary practices are at disproportionate risk (Bath & Rayman, 2019) <sup>[2]</sup>.

**Table 2:** Prevalence of thyroid dysfunction in selected athletic cohorts

Athletic Group	Estimated Prevalence of Subclinical Hypothyroidism (%)	Region Studied	Reference
Competitive Swimmers	5-10	North America	Witt et al. (2015) <sup>[33]</sup>
Vegan Triathletes	15-20	Various Online Surveys	Bath & Rayman (2019) <sup>[2]</sup>
Weight-Category Athletes	6-8	East Asia (Urban)	Rodriguez et al. (2019)

**Notes:** Subclinical hypothyroidism defined by elevated TSH with normal T<sub>4</sub>/T<sub>3</sub>. Data compiled from cross-sectional studies and smaller-scale assessments.

## 3.4 Case Examples

- **Endurance Runners in High-Altitude Regions:** Studies in mountainous areas with naturally iodine-poor soil have reported that local runners show higher TSH levels, indicative of increased thyroid gland stimulation to compensate for low iodine intake.
- **Vegan Athletes:** Vegan dietary patterns exclude common iodine sources like dairy and fish. Without adequate replacement or supplementation, such athletes can exhibit median UIC values falling below recommended thresholds (Bath & Rayman, 2019) <sup>[2]</sup>.

## 3.5 Performance Ramifications

Persistently low T<sub>3</sub> levels can compromise an athlete's ability to maintain high-intensity exercise efforts (Mitchell et al., 2018) <sup>[22]</sup>. Chronic fatigue, slower recovery, and mood

disturbances may also ensue (Kruger et al., 2020) <sup>[17]</sup>. Over time, if uncorrected, such deficiencies can undermine long-term health, including bone density, muscle mass maintenance, and cardiovascular function (Duntas & Brenta, 2018) <sup>[7]</sup>. Given these potential repercussions, monitoring iodine status stands as a prudent step in comprehensive sports nutrition.

## 4. Influence of exercise on iodine metabolism and thyroid function

The relationship between exercise and thyroid function is bidirectional. Not only does thyroid hormone status affect exercise capacity, but physical activity itself can modify iodine turnover and thyroid hormone levels (Fortunato et al., 2018) <sup>[10]</sup>. Understanding these dynamics can inform

athletes and coaches on how best to structure nutritional interventions.

#### 4.1 Exercise-induced changes in thyroid hormones

Short-term high-intensity exercise can acutely raise  $T_3$  and  $T_4$  levels, partly through sympathetic stimulation. Over more prolonged training cycles, however, athletes might experience varied patterns some showing stable thyroid parameters, others demonstrating transient reductions in  $T_3$ , often referred to as the “Low  $T_3$  Syndrome” in chronic exercise stress (Hackney, 2020) <sup>[12]</sup>. While typically reversible, this phenomenon indicates the sensitivity of thyroid homeostasis to heavy workloads and underscores the necessity of adequate iodine to sustain hormone production.

#### 4.2 Elevated Nutrient Turnover and Sweat Loss

Sweat losses during intensive training, especially in hot or humid environments, can contribute to micronutrient depletion. Though sodium and chloride predominate in sweat, trace elements like iodine can also be lost in minor but cumulatively significant amounts (Kirsch *et al.*, 2021) <sup>[14]</sup>. Over months or years of repeated high-volume training, especially in athletes without adequate dietary iodine intake, these incremental losses can tip the balance toward deficiency. This effect is exacerbated if an individual concurrently reduces their overall salt consumption or if the salt used is unfortified.

#### 4.3 Thyroid hormones and aerobic performance ( $VO_2$ Max)

$VO_2$  max reflects the integration of respiratory, cardiovascular, and muscular systems, heavily influenced by cellular oxidative capacity.  $T_3$  supports mitochondrial biogenesis and the expression of enzymes involved in aerobic energy generation (Bianco & McAninch, 2017) <sup>[3]</sup>. Consequently, insufficient  $T_3$  levels can compromise these pathways, leading to reduced oxygen uptake and lower  $VO_2$  max (Hostrup & Bangsbo, 2017) <sup>[13]</sup>. In competitive environments where fractional improvements matter, even minor reductions in  $VO_2$  max can be a decisive factor.

#### 4.4 Muscle strength, neuromuscular coordination, and recovery

Thyroid hormones modulate contractile protein turnover, particularly myosin heavy chain isoforms, which determine muscle fiber phenotype and contraction speed (Kruger *et al.*, 2020) <sup>[17]</sup>. An optimal balance of  $T_3$  supports faster neuromuscular reflexes and efficient muscle firing patterns,

crucial for activities requiring explosive power or sustained endurance. Beyond acute performance,  $T_3$  aids in repairing exercise-induced micro damage, contributing to post-exercise anabolic processes (Feyerabend *et al.*, 2021) <sup>[9]</sup>. When iodine deficiency suppresses  $T_3$  production, delayed recovery and increased susceptibility to overtraining can ensue (Duntas & Brenta, 2018) <sup>[7]</sup>.

#### 4.5 Psychological and Cognitive Dimensions

Emerging evidence suggests that subtle variations in thyroid function can impact mood, cognition, and motivation factors that, although less directly measured, are vital to consistent training and competition success (Samuels & Kolobova, 2020) <sup>[27]</sup>. In scenarios where overtraining is coupled with inadequate iodine intake, an athlete may experience heightened fatigue, decreased motivation, or difficulty focusing. Recognizing and addressing these symptoms can prevent more severe endocrine imbalances and performance declines.

#### 5. Dietary sources and recommended intake for athletes

Nutritional guidelines for iodine intake typically recommend 150  $\mu\text{g/day}$  for healthy adults, with increased demands during pregnancy and lactation (US Institute of Medicine, 2001). However, many sports professionals argue that athletes undergoing intense physical exertion may require higher intakes, particularly if sweat losses are significant (Maughan & Shirreffs, 2019) <sup>[20]</sup>.

##### 5.1 Common Dietary Sources of Iodine

- **Iodized Salt:** A primary source in many countries, though the actual content can vary. Some products labeled as “sea salt” or “natural salt” may not be iodized (Andersen *et al.*, 2020) <sup>[1]</sup>.
- **Marine Foods:** Fish, seaweed, and shellfish are rich in iodine due to their oceanic origins (Bath & Rayman, 2019) <sup>[2]</sup>. Seaweeds like kelp or nori can contain exceptionally high concentrations, though content is variable.
- **Dairy Products:** The main food source is milk and dairy products. There is a seasonal change in the content of iodine in milk; winter milk has a greater concentration than summer milk. (Witard *et al.*, 2022).
- **Eggs and Meat:** Although typically lower than marine sources, these can contribute modest amounts of iodine, depending on the animal feed (Williams *et al.*, 2018) <sup>[32]</sup>.

**Table 3:** Typical iodine content in selected dietary sources

Food Item	Approx. Iodine Content ( $\mu\text{g}$ per serving)	Serving Size	Reference
Iodized Table Salt	69-80 $\mu\text{g}$	1.5 g	Pehrsson <i>et al.</i> , (2022) <sup>[38]</sup>
Cow's Milk (Whole)	50-60	1 cup (240 ml)	Bath & Rayman (2019) <sup>[2]</sup>
Salmon (Baked)	40-50	85 g (3 oz)	Zimmermann & Andersson (2012)
Nori Seaweed (Dried)	100-300	10 g	Andersen <i>et al.</i> (2020) <sup>[1]</sup>
Yogurt	60-70	1 cup (245 g)	Williams <i>et al.</i> (2018) <sup>[32]</sup>

**Notes:** Values can vary based on agricultural and processing factors. Some dairy variations (e.g., organic, grass-fed) may differ in iodine content.

##### 5.2 Challenges in Meeting Iodine Needs

- **Reduced Salt Use:** Public health campaigns encouraging lower sodium intake for cardiovascular benefits may inadvertently reduce iodine intake if athletes fail to use iodized salt (Bath & Rayman, 2019) <sup>[2]</sup>.
- **Specialty Diets:** Vegan or dairy-free regimens can lead to substantially reduced iodine consumption unless



carefully balanced with alternative sources or supplements (Andersen *et al.*, 2020) <sup>[1]</sup>.

- **Reliance on Processed Foods:** Many processed foods use non-iodized salt, and the actual iodine content is rarely labeled, leaving athletes uncertain about their daily intake.

### 5.3 Estimating Iodine Requirements in Athletes

Although formal guidelines do not currently specify a distinct RDA for athletes, some sports nutrition experts suggest intakes up to 200 µg/day might be prudent for those training heavily (Shaw *et al.*, 2018) <sup>[29]</sup>. This recommendation is not universal, but it highlights the necessity of individual assessment. Athletes in hot climates or those engaging in prolonged training sessions may require additional vigilance (Falk *et al.*, 2019) <sup>[8]</sup>.

### 5.4 Practical Dietary Strategies

- **Use of Iodized Salt in Cooking:** Ensuring that table salt or cooking salt is iodized remains an effective strategy.
- **Inclusion of Seaweed:** Nori sheets or kelp granules can offer a potent natural iodine boost in an otherwise limited diet (Bath & Rayman, 2019) <sup>[2]</sup>.
- **Routine Assessments:** Periodic urinary iodine tests can help evaluate an athlete's status, guiding dietary adjustments or supplementation decisions.

## 6. Iodine supplementation and performance

While a balanced diet remains the optimal route to secure iodine, supplementation may be warranted in certain scenarios. This section delves into how supplementation can influence performance, including potential pitfalls associated with excessive intake.

### 6.1 When to Consider Supplementation

- **Evidence of Deficiency:** Laboratory data showing suboptimal UIC or elevated TSH with borderline-low T<sub>4</sub> or T<sub>3</sub> levels.
- **Restrictive Dietary Patterns:** Athletes following vegan, dairy-free, or otherwise limited diets that

exclude major iodine sources (Bath & Rayman, 2019) <sup>[2]</sup>.

- **High Sweat Losses:** Individuals training multiple times daily, especially in hot conditions, where micronutrient depletion via sweat is of concern (Kirsch *et al.*, 2021) <sup>[14]</sup>.

### 6.2 Forms of Iodine Supplements

Potassium iodide (KI), which is commonly used to make supplements and iodized table salt, and potassium iodate (KIO<sub>3</sub>), which is used to strengthen table salt (McChesney & Lieberman, 2022) <sup>[39]</sup>. However, not all athletic supplements include iodine, and many "sports-specific" products focus primarily on electrolytes like sodium, potassium, magnesium, and chloride (Williams *et al.*, 2018) <sup>[32]</sup>.

### 6.3 Impact on Performance

Research linking iodine supplementation directly to performance gains remains relatively limited. Nonetheless, resolving iodine deficiency can normalize thyroid hormone levels, which in turn supports optimal metabolic function, energy utilization, and recovery processes (Fortunato *et al.*, 2018) <sup>[10]</sup>. Anecdotal reports and smaller-scale interventions suggest that athletes with low iodine status may notice reduced fatigue and improved training outputs following supplementation (Witt *et al.*, 2015) <sup>[33]</sup>. More rigorous clinical trials would be beneficial for substantiating these findings in diverse sporting populations.

### 6.4 Risks of excessive intake

Iodine toxicity or over-supplementation can provoke hyperthyroidism or autoimmune thyroiditis, especially in susceptible individuals (Langer *et al.*, 2019) <sup>[18]</sup>. Hyperthyroidism, autoimmune thyroid disease, and papillary cancer can result from consuming too much iodine. Adult men and women in the United States should consume 1100 µg of iodine per day, according to the IOM. (McChesney & Lieberman, 2022) <sup>[39]</sup>. Athletes should consult with healthcare providers or sports nutritionists to tailor supplementation to their specific needs and physiological markers.

**Table 4:** Practical iodine supplementation strategies for athletes

Supplement Type	Typical Dosage Range (µg/day)	Form	Key Considerations	Reference
Potassium Iodide	75-150	Tablet or Capsule	Generally well-tolerated; monitor for thyroid autoimmunity	Shaw <i>et al.</i> (2018) <sup>[29]</sup>
Kelp or Seaweed Tabs	150-300	Natural Source	Variable content; risk of excess if intake is not regulated	Bath & Rayman (2019)
Fortified Sports Drink	50-100	Liquid	Rare in standard products; specialized sports solutions exist	Kreider <i>et al.</i> (2019) <sup>[16]</sup>

**Notes:** Dosages reflect common practice; individual needs vary. Upper limit recommended by the Institute of Medicine is 1,100 µg/day for adults (McChesney & Lieberman, 2022) <sup>[39]</sup>.

### 6.5 Monitoring and Follow-UP

Given potential fluctuations in exercise intensity, dietary patterns, and sweat losses, routine assessments of thyroid function (TSH, free T<sub>4</sub>, free T<sub>3</sub>) and urinary iodine excretion can help maintain an athlete's iodine status in the optimal range (Peterson *et al.*, 2018) <sup>[25]</sup>. Adjustments in dosage or dietary strategies may be necessary over an athletic season, particularly in periods of intensified training or competition.

## 7. Conclusion

Iodine's central role in thyroid hormone synthesis makes it indispensable for sustaining the metabolic demands and physiological adaptations critical to athletic performance. Suboptimal iodine status can subtly undermine an athlete's endurance, muscle strength, recovery, and even psychological readiness. Despite global public health measures such as salt iodization, many individuals athletes included still fall short of meeting daily iodine requirements,

particularly those with specialized diets or high sweat losses.

This review has illuminated several key points. Firstly, understanding the fundamental physiology of iodine uptake and thyroid hormone biosynthesis underscores the uniqueness of iodine among micronutrients. Secondly, the intricate relationship between exercise and thyroid function demands that athletes remain vigilant about their iodine intake, recognizing that marginal deficiency may compromise performance variables such as  $\text{VO}_2$  max and muscle contractility. Thirdly, identifying common dietary pitfalls such as the use of non-iodized salt, reduced dairy consumption, or inadequate seafood intake allows for targeted nutritional strategies to ensure sufficiency. Finally, while supplementation can be an effective method to address deficiency, caution is advised to avoid exceeding safe intake limits.

Moving forward, continued research is warranted to more precisely determine the iodine requirements of athletes, factoring in variables such as training intensity, climate, and dietary preferences. The potential for personalized nutrition interventions, guided by biomarkers like urinary iodine concentration and thyroid hormone panels, holds promise for optimizing both health and performance. By integrating knowledge of iodine's critical functions into broader sports nutrition frameworks, athletes and the professionals who support them can help ensure that no micronutrient deficiency impedes the pursuit of excellence.

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