### THE IMPORTANCE OF IODINE FOR THE HUMAN ORGANISM

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**Abstract:** Iodine plays a crucial role in human physiology as an essential component of thyroid hormones. This review examines iodine's biological significance, consequences of deficiency, and preventive measures. Current data indicates that iodine deficiency affects nearly 1.9 billion people worldwide, particularly impacting cognitive development in children and pregnant women. The paper analyzes thyroid dysfunction mechanisms (hypothyroidism, hyperthyroidism) and their clinical manifestations. We discuss diagnostic approaches and public health strategies for iodine deficiency disorders (IDDs), emphasizing the importance of universal salt iodization programs. Recent research on iodine's extrathyroidal functions and optimal supplementation guidelines are also presented.

**Keywords**: iodine deficiency, hypothyroidism, hyperthyroidism, thyroid dysfunction, goiter

### 1. Introduction

Iodine is a vital micronutrient required for thyroid hormone synthesis, with recommended daily intake ranging from 90µg (infants) to 200µg (pregnant women) [WHO, 2023]. Approximately 31% of the global population remains at risk of iodine deficiency disorders (IDDs), particularly in mountainous regions and areas with low soil iodine content [1].

The thyroid gland utilizes iodine to produce thyroxine (T4) and triiodothyronine (T3), which regulate:

- Basal metabolic rate
- Neurocognitive development
- Cardiovascular function
- Reproductive health

This paper systematically reviews:

- 1. Iodine's physiological roles
- 2. Pathogenesis of iodine deficiency disorders
- 3. Clinical management strategies
- 4. Current prevention programs

### 2. Materials and Methods

We analyzed 48 peer-reviewed studies (2018-2023) from PubMed, Scopus, and WHO databases using keywords: "iodine deficiency", "thyroid pathophysiology", and "iodine prophylaxis".

### 3. Results and Discussion

### 3.1 Epidemiological Data

- Global prevalence: 1.9 billion at risk, including 350 million Europeans [2]
- Highest risk groups:
- o Pregnant women (increased fetal requirements)
- o Children (neurodevelopmental vulnerability)

### 3.2 Pathophysiological Mechanisms

### **Iodine Deficiency Consequences:**

Severity	Clinical Manifestations	
Mild	Fatigue, weight gain	
Moderate	Goiter, cognitive impairment	
Severe	Cretinism, stillbirth	

### **Thyroid Dysfunction Types:**

- 1. **Hypothyroidism**: Reduced hormone production
- o Symptoms: Bradycardia, cold intolerance
- 2. Hyperthyroidism: Excessive hormone secretionSymptoms: Tachycardia, weight loss
- 3.3 Diagnostic Approaches
- Laboratory tests: TSH, free T4, urinary iodine
- Imaging: Thyroid ultrasound, scintigraphy

### 3.4 Prevention Strategies

- Universal salt iodization (USI) programs
- Dietary sources:
- Seafood (150-1000μg/100g)
- o Iodized salt  $(20-40\mu g/g)$
- o Dairy products (30-50µg/100ml)

The present review synthesizes current evidence on iodine's pivotal role in human physiology and the far-reaching consequences of its deficiency. Our analysis reveals several critical findings that warrant further examination.

### 5.1 Global Health Implications

The persistent burden of iodine deficiency disorders (IDDs) among 1.9 billion individuals underscores a significant public health challenge. While universal salt iodization (USI) programs have reduced goiter prevalence by 40% in endemic regions since 1990 [Zimmermann, 2021], emerging data suggests:

- **Geographic disparities**: Mountainous and inland populations show 3× higher deficiency rates than coastal communities [WHO, 2023]
- **Vulnerability paradox**: Despite global progress, pregnant women in Europe exhibit rising deficiency rates (from 15% to 21% during 2015-2022) due to reduced salt consumption trends [2]

### **5.2 Pathophysiological Insights**

Our examination of thyroid dysfunction mechanisms reveals two key phenomena:

- 1. **The J-shaped risk curve**: Both deficiency (<100 μg/day) and excess (>500 μg/day) iodine intake correlate with thyroid dysfunction [Leung, 2023]
- 2. **Selenium interplay**: The selenoenzyme deiodinase's role explains why concurrent selenium deficiency exacerbates hypothyroidism in iodine-deficient populations [3]

Notably, the transition from subclinical to overt hypothyroidism follows a distinct pattern:

- Stage 1: Increased TSH with normal T4 (compensated)
- Stage 2: Elevated TSH with low T4 (decompensated) This progression highlights the importance of early detection through neonatal screening programs, which prevent 70% of intellectual disability cases in endemic regions [1].

### 5.3 Diagnostic and Therapeutic Challenges

Current approaches present several limitations:

Method	Sensitivity	Specificity
Urinary iodine	82%	75%
Thyroid ultrasound	91%	88%
TSH testing	95%	89%

Key unresolved issues include:

- "Iodine paradox": Some populations develop autoimmune thyroiditis post-
- **Supplementation timing**: Optimal iodine doses for pregnant women remain debated (150-250 µg/day)

### **5.4 Prevention Strategies Re-evaluation**

While USI programs remain cost-effective (\$0.02-0.05 per person annually), our analysis suggests needed improvements:

- 1. **Targeted supplementation:** 
  - Schoolchildren: Biannual iodine capsules
- Pregnant women: Prenatal vitamin formulations 0
- **Monitoring advancements:** 2.
  - Mobile testing units for remote areas
- AI-assisted ultrasound interpretation  $\cap$

### **5.5 Future Directions**

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Emerging research areas demand attention:

- Extra-thyroidal effects: Iodine's potential in:
- Fibrocystic breast disease management 0
- Gastric cancer prevention (through antimicrobial action) 0
- Personalized nutrition: Genetic testing for sodium-iodide symporter polymorphisms

### 4. Conclusion

This comprehensive review elucidates the critical role of iodine in maintaining human health and the profound consequences of its deficiency. The evidence presented demonstrates that despite global efforts, iodine deficiency disorders remain a significant public health challenge, affecting nearly 2 billion people worldwide, with particular vulnerability among pregnant women and children in endemic regions.

Key findings from our analysis include:

- The dual burden of iodine disorders, where both deficiency and excess can lead to thyroid dysfunction, necessitates precise monitoring and tailored interventions
- The crucial interaction between iodine and selenium in thyroid hormone metabolism highlights the need for combined nutritional approaches
- Current prevention strategies, particularly universal salt iodization, have proven effective but require adaptation to address emerging challenges like changing dietary patterns

The clinical implications of this review are substantial:

- Early detection through neonatal screening programs prevents irreversible neurocognitive damage
- Population-level monitoring must be strengthened, especially in high-risk groups
- Healthcare provider education about subtle deficiency symptoms can improve diagnosis rates

Moving forward, three key areas demand attention:

- 1. Development of more accurate biomarkers for iodine status assessment
- 2. Implementation of targeted supplementation programs for vulnerable populations
- 3. Investigation of iodine's potential extra-thyroidal benefits in chronic disease prevention

The economic and social costs of iodine deficiency - including reduced workforce productivity and increased healthcare expenditures - underscore that iodine prophylaxis represents one of the most cost-effective public health interventions available. As dietary patterns continue to evolve globally, maintaining vigilance against iodine deficiency remains paramount.

### Future research should focus on:

- Personalized iodine supplementation based on genetic profiling
- Innovative food fortification technologies
- Long-term outcomes of different prophylaxis strategies

In conclusion, while significant progress has been made in combating iodine deficiency disorders, sustained multidisciplinary efforts are essential to eliminate this preventable cause of global disease burden and ensure optimal thyroid health for all populations.

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