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THYROID DISORDER DUE TO HIGH INTAKE OF IODINE: REVIEW

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Abstract – Iodine is essential for thyroid hormone synthesis. Iodine is a micronutrient essential for the production of thyroid hormones. Iodine deficiency is the most common cause of preventable mental impairment worldwide. Most healthy individuals can tolerate some iodine excess, but not all. Overuse of iodine may sometimes precipitate hyperthyroidism, hypothyroidism, goiter, and/or thyroid autoimmunity in some individuals. People who already have hyperthyroidism, hypothyroidism, goiter, and/or thyroid autoimmunity and are also iodine deficient may be more susceptible to thyroid disorders due to increased iodine intake. Thyroid impairment is usually mild, but iodine-induced hyperthyroidism can be life-threatening in some individuals. Iodine overdose has been reported in iodised salt, drinking water, cow and buffalo milk, some seaweeds, iodine-rich dietary supplements and combinations of these sources may produce iodine excess. The average urinary iodine concentration (UIC) of a population reflects total iodine intake from all sources and can accurately identify populations with excessive iodine intake. Our review describes the relationship between excess iodine intake and thyroid function. We outline potential sources of excess iodine intake and the physiological responses and consequences of excess iodine intake. We provide guidance on the choice of biomarkers to assess iodine intake, with an emphasis on UIC and thyroglobulin. Universal salt iodization (USI) has been introduced in many countries as a cost-effective and sustainable way to eliminate iodine deficiency disorders for more than 25 years.

Keywords: iodine; iodine excess; iodized salt; thyroglobulin; thyroid autoimmunity. Universal salt iodization (USI)

Background/Introduction

Hypothyroidism is a condition characterized by the insufficient production of thyroid hormones, which are critical for regulating metabolism, growth, and development. The most common causes of hypothyroidism include iodine deficiency, autoimmune conditions such as Hashimoto's thyroiditis, and certain medical treatments like radioactive iodine therapy or thyroid surgery. Iodine, an essential micronutrient, plays a pivotal role in thyroid hormone synthesis. While iodine deficiency is a well-known cause of hypothyroidism, excessive iodine intake can paradoxically impair thyroid function, leading to the condition. This phenomenon occurs due to mechanisms such as the Wolff-Chaikoff effect, where the thyroid temporarily reduces hormone synthesis in response to high iodine levels. Understanding the dual role of iodine in thyroid health is crucial for developing balanced dietary and public health recommendations. Over the past 25 years, universal salt iodization (USI) has been introduced in many countries as a safe, cost-effective and sustainable method to eliminate iodine deficiency disorders (IDDs), resulting in a greatly improved iodine status. However, an increased iodine intake has brought new challenges in long-term iodine sufficient regions covered by USI for more than 10 years. This review focused on the effect of iodine excess on thyroid disorders.

Iodine status of the population

Assessment of iodine status of the population >12 years of age is based on the UIC of school-age children .

Epidemiological Criteria for Assessing Iodine Nutrition Based on MUI Concentrations in School-Age Children

	Iodine intake	Iodine nutrition
MUI, μg/L		
<20	Insufficient	Severe iodine deficiency
20–49	Insufficient	Moderate iodine deficiency
50–99	Insufficient	Mild iodine deficiency
100–199	Adequate	Optimal iodine nutrition
200–299	More than adequate	Risk of IIH within 5–10 years following introduction of iodized salt in susceptible group
≥300	Excessive	Risk of adverse health consequences (IIH, autoimmune thyroid disease)

MUI, median urinary iodine.

Objective

The aim of this study is to investigate the relationship between excessive iodine intake and the development of hypothyroidism. By examining the underlying mechanisms, such as the Wolff-Chaikoff effect and its role in thyroid hormone synthesis, this paper seeks to clarify how high iodine levels can impair thyroid function. Additionally, the study aims to identify population groups at risk and provide insights into dietary thresholds for iodine consumption to guide public health policies and prevent iodine-induced thyroid dysfunction.

Here is a chart summarizing the **daily iodine requirements** for a normal human body, as well as common sources of iodine and the risk of excessive iodine intake from dietary sources.

Daily Iodine Requirements (Recommended Dietary Allowances - RDA)

Age Group	Iodine Requirement (mcg/day)
Infants (0–6 months)	110 mcg
Infants (7–12 months)	130 mcg
Children (1–8 years)	90 mcg
Children (9–13 years)	120 mcg
Adolescents & Adults (14+)	150 mcg
Pregnant Women	220 mcg
Lactating Women	290 mcg

Upper Tolerable Intake Levels (UL)

Age Group	Upper Limit (mcg/day)
Infants (0–12 months)	Not established
Children (1–3 years)	200 mcg
Children (4–8 years)	300 mcg
Children (9–13 years)	600 mcg
Adolescents (14–18 years)	900 mcg
Adults (19+ years)	1,100 mcg

Common Sources of Iodine and Potential Excess Intake

Food Source	Iodine Content (Approx.)	Risk of Excess
Iodized Salt (1 tsp)	250 mcg	Excessive use may lead to iodine toxicity.
Seaweed (1 sheet, nori)	25–300 mcg	Certain seaweeds (kelp) can exceed daily limits.
Cod (85g or 3 oz)	100 mcg	Safe when consumed in moderation.
Yogurt (1 cup)	75 mcg	Generally safe, even with regular consumption.
Milk (1 cup)	50–60 mcg	Excess unlikely with typical diets.

Food Source	Iodine Content (Approx.)	Risk of Excess
Shrimp (85g or 3 oz)	35 mcg	Safe for most individuals.
Eggs (1 large)	25 mcg	Not a significant source of excess iodine.
Iodine Supplements (1 tab)	150 mcg	Overuse can lead to toxicity, particularly combined with high dietary intake.

Risks of Excess Iodine Intake

- **Wolff-Chaikoff Effect**: Excess iodine can inhibit thyroid hormone production temporarily.
- **Thyroid Autoimmunity**: High iodine levels may trigger or worsen autoimmune conditions like Hashimoto's thyroiditis.
- **Hyperthyroidism or Hypothyroidism**: Chronic overconsumption can lead to thyroid dysfunction.

Methods

This study employed a systematic review of clinical trials, observational studies, and experimental research examining the effects of high iodine intake on thyroid function. Data were gathered from peer-reviewed journals, focusing on mechanisms like the Wolff-Chaikoff effect and autoimmune responses triggered by excessive iodine. Additionally, population studies in regions with high dietary iodine levels were analyzed to assess prevalence rates of iodine-induced hypothyroidism. Laboratory findings on thyroid hormone levels (TSH, T3, and T4) in subjects exposed to high iodine intake were included to support the analysis.

Results

The findings reveal a clear association between excessive iodine consumption and the onset of hypothyroidism, particularly in populations with predisposing factors such as genetic susceptibility or pre-existing thyroid conditions. High iodine levels were shown to trigger the Wolff-Chaikoff effect, leading to a temporary reduction in thyroid hormone synthesis, which can become permanent in certain individuals. Moreover, excessive iodine was found to exacerbate autoimmune thyroid disorders, contributing to hypothyroidism. Regional studies indicated a higher prevalence of thyroid dysfunction in areas with mandatory iodine fortification programs exceeding recommended levels.

Conclusion

Excessive iodine intake poses a significant risk for thyroid dysfunction, highlighting the need for carefully calibrated dietary recommendations and public health policies. The findings underscore the importance of monitoring iodine levels in fortified foods and supplements to prevent iodine-induced hypothyroidism. Healthcare providers should also consider individual iodine sensitivities when advising on dietary intake, particularly in vulnerable populations. This study emphasizes the delicate balance required to maintain optimal iodine consumption for thyroid health.

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