
SPECIAL ARTICLE

Maternal Iodine Requirements in Pregnancy and Lactation: Critical roles and monitoring strategies

Arissara Kuptarak, M.D.*,
Vorapong Phupong, M.D., FRTCOG.*

* Department of Obstetrics and Gynecology, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

ABSTRACT

Iodine plays a vital role in fetal neurodevelopment and is closely linked to thyroid function in both mothers and infants. Iodine deficiency during pregnancy is associated with adverse obstetric outcomes, impaired fetal growth, and in severe cases, can result in neonatal cretinism. An adequate iodine status in pregnancy is defined by a median urinary iodine concentration of 150–249 µg/L. Several methods are available to assess iodine sufficiency in pregnant and lactating women, each with its own strengths and limitations. These include urinary iodine concentration, serum iodine levels, serum thyroglobulin, breastmilk iodine concentration, neonatal thyroid-stimulating hormone levels, and thyroid volume assessment. While iodine supplementation is crucial in deficiency states, excessive iodine intake can also disrupt thyroid function, potentially leading to either hyperthyroidism or hypothyroidism. Therefore, monitoring and maintaining appropriate iodine levels during pregnancy and lactation is essential for maternal and neonatal health.

Keywords: iodine, prenatal, lactation, iodine assessment, urinary iodine, serum iodine, breastmilk iodine.

Correspondence to: Vorapong Phupong, M.D., FRTCOG, Department of Obstetrics and Gynecology, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand. Email: vorapong.p@chula.ac.th

Received: 20 March 2026, **Revised:** 2 April 2026, **Accepted:** 4 April 2026

Introduction

Iodine is one of the minerals that required during pregnancy⁽¹⁾. The recommended dietary allowance (RDA) for iodine is in the range of 0.20 to 0.25 milligrams/day, slightly varying between recommendations⁽¹⁾. Iodine is an essential nutrient for the production of thyroid hormones in pregnant women and infants. It is also important for the development

of the nervous system. A deficiency in prenatal iodine can lead to childhood intellectual disability and autism⁽²⁾. Risks of miscarriage, preterm labor, low birthweight, preeclampsia and stillbirth are increased in iodine deficiency during pregnancy. Moreover, iodine deficiency remains a leading contributor to the development of maternal hypothyroidism⁽³⁾. The increased iodine requirement during pregnancy is

primarily attributed to the need to maintain maternal euthyroid, facilitate transplacental transfer of thyroid hormones to the fetus, and compensate for enhanced renal iodine clearance⁽⁴⁾.

Insufficient iodine intake during lactation can hinder maternal thyroid hormone production, potentially disrupting the infant's physical, neurological, and cognitive development. When maternal iodine levels are inadequate, the iodine content in breast milk decreases, directly limiting the iodine supply available to exclusively breastfed infants. Inadequate iodine during the early postnatal period—especially within the first six months—can lead to significant impairments in cognitive and psychomotor development, which may not be fully reversible even if iodine status is corrected later⁽⁵⁾.

Iodine deficiency has emerged as a major global health concern, affecting approximately 2 billion individuals worldwide⁽⁶⁾. The Iodine Global Network (IGN) 2024 annual report identified 21 countries as having insufficient iodine levels, while Thailand was classified as a country with adequate iodine status⁽⁷⁾. However, a study conducted in northern Thailand revealed that nearly 44% of pregnant women exhibited insufficient iodine levels⁽⁸⁾. Moreover, research from Bangkok reported that 58% of lactating women had breast milk iodine concentrations below 150 µg/L, suggesting inadequate iodine intake to meet the nutritional needs of their infants⁽⁹⁾.

Importantly, iodine deficiency is largely preventable through adequate dietary intake and appropriate supplementation. The Institute of Medicine (IOM) has set the daily recommended dietary allowance (RDA) at 220 micrograms for pregnant women and 290 micrograms for those who are breastfeeding⁽¹⁰⁾. According to the World Health Organization (WHO), a median urinary iodine concentration (UIC) ranging from 150 to 249 µg/L indicates adequate iodine status in pregnant women. However, in lactating women, a lower median UIC of 100 µg/L is considered sufficient, as a significant proportion of iodine is secreted into breast milk to support the infant's nutritional requirements⁽¹¹⁾.

Because individual iodine intake fluctuates significantly throughout the day and between days, median UIC thresholds are not considered reliable indicators of an individual's long-term iodine status⁽¹²⁾. As for the concentration of iodine in breast milk, the adequate level should range between 150 and 180 µg/L⁽¹³⁾. This article provides an overview of the significance of sufficient iodine intake during pregnancy and lactation, methods for assessing prenatal and lactating iodine status, dietary sources of iodine, and the potential consequences of iodine deficiency in pregnancy.

Iodine metabolism during pregnancy and lactation

During pregnancy, maternal iodine requirements increase by approximately 50%, primarily due to several physiological adaptations. These include an elevated renal clearance of iodine resulting from an increased glomerular filtration rate, augmented maternal thyroid hormone synthesis to meet both maternal and fetal metabolic demands, and the transplacental transfer of iodine to the fetus—particularly significant during the second and third trimesters, when fetal thyroid hormone production is initiated. Furthermore, in the first trimester of gestation, the fetus relies entirely on maternal thyroxine (T₄), as the fetal thyroid gland does not attain functional maturity until around the 12th week of pregnancy⁽¹⁴⁾.

In the human gastrointestinal tract, iodine is converted to iodide, with over 90% being efficiently absorbed in healthy adults. After entering the circulation, iodide is primarily taken up by the thyroid gland or eliminated via renal excretion. While renal clearance remains relatively constant, thyroidal uptake varies depending on iodine status and dietary intake. Under conditions of adequate iodine supply, the thyroid absorbs less than 10% of circulating iodide; however, in cases of chronic iodine deficiency, uptake can exceed 80%. With the kidneys excrete over 90% of ingested iodine via urine, urinary iodine concentration serves as a promising biomarker for assessing iodine deficiency⁽¹⁵⁾.

Regarding neonatal iodine metabolism, a marked physiological surge in serum thyroid-

stimulating hormone (TSH) occurs within 30 to 60 minutes after birth, reaching levels of approximately 60–80 mIU/mL as the newborn adapts to life outside the womb. This surge declines rapidly, with TSH concentrations stabilizing within 3 to 5 days postpartum. The transient elevation in TSH stimulates the synthesis of T4 and triiodothyronine (T3), which peak around 24 hours after birth and gradually decrease to more stable levels by days 5 to 7. This downward trend continues throughout infancy and childhood, albeit more slowly. Notably, T4 turnover in infancy is significantly increased, with infants producing roughly three times more T4 per kilogram of body weight than adults. However, neonates are born with minimal iodine stores—approximately 300 µg—which are rapidly depleted within a few days. Consequently, an adequate external supply of iodine, primarily through breast milk or infant formula, is critical to support the elevated thyroid hormone demands during early development⁽⁵⁾. Infants exhibit reduced iodine clearance compared to adults, primarily due to the immaturity of their renal function⁽¹⁶⁾. During lactation, iodine is actively transported into breast milk by the sodium/iodide symporter in the mammary gland, ensuring sufficient iodine availability to maintain healthy thyroid function and support continued neurological development⁽¹⁷⁾.

Excessive iodine intake

Excess iodine intake can trigger the Wolff-Chaikoff effect, which reduces thyroid hormone synthesis by inhibiting thyroid peroxidase⁽¹⁸⁾. In some vulnerable individuals, especially fetuses and newborns, failure to escape the acute Wolff–Chaikoff effect may result in iodine-induced hypothyroidism, which may be either short-term or permanent⁽¹⁴⁾. Conversely, excessive iodine intake may also induce the Jod-Basedow phenomenon, characterized by iodine-induced thyrotoxicosis. This condition is more frequently observed in individuals with preexisting thyroid abnormalities, such as nontoxic nodular goiter or euthyroid Graves’ disease, and in populations residing in iodine-deficient regions⁽¹⁹⁾.

According to the WHO classification, a median UIC between 250 and 499 µg/L indicates more than adequate intake, while a median UIC of 500 µg/L or higher is considered excessive⁽¹¹⁾. A prior systematic review reported that 52% of the 10,736 pregnant women from various regions worldwide had excessive iodine levels. Among these studies, 66.6% reported cases of hypothyroidism in the pregnant women, while hyperthyroidism was reported in 0.6% to 19.7% of the studies⁽²⁰⁾.

Currently, there is evidence from various guidelines regarding iodine intake during pregnancy and lactation, as depicted in Table 1.

Table 1. Daily iodine intakes and tolerable upper intake levels for pregnant and lactating women^(10, 21, 22).

	Daily iodine intake (µg/day)		Tolerable upper intake level (µg/day)	
	IOM	WHO	IOM	EFSA
Pregnant women	220	250	1,100	600
Lactating women	290	250	1,100	600

IOM: the institute of medicine, WHO: world health organization, EFSA: european food safety authority.

Assessment of iodine status

Urinary iodine concentrations

Urinary iodine is a reliable indicator of recent dietary iodine intake, since over 90% of consumed iodine is eliminated through the kidneys within 24 to 48 hours after ingestion⁽²³⁾. Due to daily

fluctuations in urinary iodine excretion, accurately determining an individual’s iodine status with 20% precision requires collecting either 10 spot urine samples or 10 individual 24-hour urine samples from pregnant women⁽²⁴⁾.

Urinary iodine concentration is measured

using either the colorimetric Sandell-Kolthoff method or inductively coupled plasma mass spectrometry (ICP-MS)⁽⁵⁾. Generally, a minimal urine volume (0.5–1.0 mL) is sufficient for UIC analysis. However, UIC reflects recent iodine intake

at the time of collection, whereas thyroid size serves as a longer-term biomarker, indicating iodine status over several months or years⁽²⁵⁾. Table 2 presents the UIC cut-off values that indicate health significance for pregnant and lactating women.

Table 2. Criteria for evaluating iodine status using median urinary iodine concentration (UIC) values⁽¹¹⁾.

Population	Median UIC (µg/L)	Iodine intake status
Pregnant women	< 150	Insufficient
	150 - 249	Adequate
	250 - 499	Above requirements
	≥ 500	Excessive
Lactating women	< 100	Insufficient
	≥ 100	Adequate

In some cases, UIC is adjusted for urinary dilution using creatinine concentration (expressed as

UIC/Cr) to improve assessment reliability. The formula is shown below⁽²⁶⁾.

$$\bullet \text{ UIC/Cr (}\mu\text{g/g creatinine)} = \frac{\text{UIC from spot urine sample}}{\text{Urinary creatinine concentration}}$$

During pregnancy, the glomerular filtration rate increases, leading to a higher urine output over a 24-hour period and resulting in dilution of the urine. Consequently, UIC tends to decrease; however, this reduction does not necessarily reflect a true iodine deficiency. A study conducted in China suggested that the UIC/Cr ratio is a more reliable indicator of iodine status in pregnant women

compared to UIC, as it reduces variability caused by differences in urine volume and dilution. The area under the receiver operating characteristic (ROC) curve for the UIC/Cr ratio was 0.92 for identifying iodine deficiency and 0.75 for detecting excessive iodine levels⁽²⁷⁾. The reference intervals for the UIC/Cr ratio in pregnant women are presented on Table 3.

Table 3. The reference intervals for the urinary iodine-to-creatinine (UIC/Cr) ratio in pregnant women⁽²⁸⁾.

	First trimester	Second trimester	Third trimester
UIC/Cr (µg/g)	38.63 - 489.46	58.48 - 644.03	56.27 - 644.93

Twenty-four-hour urinary iodine excretion (24UIE) is regarded as a more accurate indicator of iodine status compared to spot UIC, and is currently considered the gold standard for assessing iodine nutrition. When direct 24-hour urine collection is not feasible, an estimated 24UIE

(e24UIE) can be derived using an equation that incorporates spot UIC, urinary creatinine concentration, and reference values for expected 24-hour creatinine excretion based on age and sex. For best accuracy, spot urine samples used for this estimation should not be collected from the first

morning void⁽²⁶⁾. The median e24UIE is expected to be greater than 225 µg/day, based on a recommended daily iodine intake of 250 µg, of which approximately 90% is eliminated through urine⁽²⁹⁾. The equation proposed by Knudsen for

$$\text{e24UIE (}\mu\text{g/24 hours)} = \frac{\text{Urinary iodine (}\mu\text{g /L)}}{\text{Urinary creatinine (g/L) / Expected creatinine excretion (g/day)}}$$

Expected 24-hour creatinine excretion for women⁽³¹⁾

- 1.23 g for age 25-49 years old
- 1.15 g for age 50-59 years old
- 1.07 g for age 60-69 years old
- 1.00 g for age ≥ 70 years old

Serum iodine concentration

After being consumed, iodate is converted into iodide within the gastrointestinal tract. Over 90% of this iodide is absorbed primarily in the stomach and duodenum. Iodide is completely bioavailable and fully absorbed into the bloodstream. Once in circulation, inorganic iodide is transported to the thyroid, where anywhere from 5% to 100% of it may be taken up, depending on the body's iodine levels⁽¹⁹⁾. Nonpregnant individuals with normal thyroid function typically have plasma iodine concentrations ranging from 40 to 80 µg /L⁽³²⁾. A study conducted in Tianjin, China—an area with adequate iodine intake—found that pregnant women with serum iodine concentrations (SIC) below 79.9 µg/L had a significantly higher risk of developing hypothyroxinemia compared to those with higher SIC levels (odds ratio [OR] 2.44, 95% confidence interval [CI] 1.31–4.75). Conversely, women with SIC levels above 138.5 µg/L were more likely to experience thyrotoxicosis than those with lower SIC levels (OR 13.52, 95% CI 4.21–43.36). The median UIC observed in the study was 156 µg/L⁽³³⁾. Another study carried out in Hangzhou, China—an area with iodine deficiency—reported a median serum iodine concentration (SIC) of 54.44 µg/L, with a range of 3.57 to 128.68 µg/L. In the same study, the median UIC was found to be 128.07 µg/L⁽³⁴⁾. Additionally, a

estimating 24-hour urinary iodine excretion (e24UIE) is presented below and has demonstrated a Pearson correlation coefficient of 0.62 when compared to directly measured 24-hour urinary iodine values⁽³⁰⁾.

study conducted in Fuzhou, China—an area with mild iodine deficiency and a median UIC of 143.4 µg/L—established a reference range for SIC in healthy pregnant women to be between 45.03 and 112.44 µg/L⁽³⁵⁾. Further research is necessary to determine the normal reference range of serum iodine levels during pregnancy.

Serum thyroglobulin (Tg)

Thyroglobulin (Tg) is a glycoprotein produced by thyrocytes and plays a crucial role in the synthesis of thyroid hormones T3 and T4⁽³⁶⁾. Once synthesized, Tg is transported to and stored in the follicular colloid of the thyrocyte, where it undergoes iodination and coupling reactions to form T3 and T4^(37, 38).

When iodine intake is inadequate, reduced circulating T4 levels trigger the release of TRH from the hypothalamus, leading to increased secretion of TSH from the pituitary. TSH promotes the synthesis and breakdown of Tg and also stimulates the growth and proliferation of follicular cells, resulting in thyroid gland enlargement, known as goiter⁽³⁹⁾.

In cases of iodine deficiency, elevated amounts of Tg are released into the bloodstream, with Tg levels showing a positive correlation with thyroid volume. For instance, the average Tg concentration in healthy adults ranges from 5 to 14 µg/L, while individuals with endemic goiter may have mean Tg levels between 94 and 208 µg/L⁽⁴⁰⁾. Bath et al.'s study found that serum Tg levels were significantly higher in the low iodine (18 vs 16 µg/L, $p < 0.001$), while there was no significant difference in TSH levels between the groups ($p = 0.27$)⁽⁴¹⁾. A meta-analysis revealed that the average Tg concentration in iodine-deficient

pregnant women was higher than in those with sufficient iodine levels—10.73 µg/L (95% CI: 5.65–15.82) compared to 7.34 µg/L (95% CI 2.20–12.47). However, the overlapping 95% confidence intervals indicate that the difference was not statistically significant⁽⁴²⁾. Further research is warranted to better understand the role of thyroglobulin in assessing iodine status during pregnancy.

Breastmilk iodine concentration (BMIC)

Breastfed infants depend on their mother's dietary iodine intake to meet their iodine needs. Iodine is concentrated in breast milk at levels 20–50 times higher than in plasma, due to the upregulation of the sodium-iodide symporter in lactating mammary cells⁽⁴³⁾. Following acute iodine intake via potassium iodide (KI), peak iodine concentrations in breastmilk were observed at approximately six hours post-administration⁽⁴⁴⁾. Like UIC, breastmilk iodine concentration (BMIC) shows significant intraindividual variability due to fluctuations in iodine intake, making it unsuitable as a marker of individual iodine status without multiple repeated measurements. Ideally, BMIC should be assessed using inductively coupled plasma mass spectrometry (ICP-MS) following alkali digestion for sample preparation⁽⁵⁾. While further globally representative studies are necessary, existing data indicate that breastmilk iodine concentrations around 150 µg/L during the first six months of lactation are likely sufficient to achieve or exceed iodine equilibrium in infants and help prevent the developmental effects of iodine deficiency⁽⁴⁵⁾.

Other investigation methods

Iodine deficiency leads to a decrease in circulating T4 levels and an increase in serum TSH levels. The difference in TSH levels among individuals is small, with considerable overlap, making blood TSH concentration an unreliable indicator of iodine deficiency in school-age children and adults. In

contrast, TSH levels in newborns serve as a useful indicator of iodine deficiency. Unlike adults, neonates have lower iodine stores in the thyroid, resulting in a much higher iodine turnover rate. This turnover increases even more when iodine is lacking, leading to greater TSH stimulation. As a result, newborns in iodine-deficient populations often exhibit elevated TSH levels during the first few weeks of life—a condition known as transient hyperthyrotropinemia⁽²⁵⁾. The rise in the number of newborns with moderately elevated TSH levels (above 5 mIU/L in whole blood) corresponds to the severity of iodine deficiency during pregnancy⁽⁴⁶⁾. In regions where iodine deficiency is severe, this percentage can exceed 40%. Using a sensitive TSH test on blood samples taken three to four days after birth, a population is considered iodine sufficient if fewer than 3% of newborns have TSH levels above 5 mIU/L⁽⁴⁷⁾.

In cases of mild to moderate iodine deficiency disorders, palpation of thyroid gland has low sensitivity and specificity. Moreover, Thyroid size in a population may take several months or even years to return to normal after iodine deficiency has been corrected⁽²⁵⁾. Ultrasonography offers a more accurate assessment of thyroid volume than palpation. A study conducted in Egypt observed that the thyroid volume in pregnant women was slightly higher than in non-pregnant control subjects, but the difference was not statistically significant (8.6 ± 2.9 vs 7.9 ± 2.5 mL, p value = 0.126)⁽⁴⁸⁾. Furthermore, research from Spain reported that in women with adequate iodine levels during the first and third trimesters, thyroid volume increased by 17.7% from baseline. In contrast, those with insufficient iodine intake experienced a much greater increase of 42.5%, highlighting the impact of iodine status on thyroid enlargement during pregnancy⁽⁴⁹⁾. The distribution of thyroid volume percentiles by pregnancy trimester, along with the corresponding reference ranges, was analyzed from previous study and presented in Table 4.

Table 4. Thyroid volume percentiles based on the trimester of pregnancy⁽⁵⁰⁾.

Trimester	Thyroid volume (mL) according to their percentile				
	2.5	25	50	75	97.5
First	2.47	4.45	5.28	6.58	9.49
Second	3.17	4.41	5.36	6.58	9.01
Third	3.00	4.77	5.55	6.00	12.38

Sources of iodine

Typical dietary sources of iodine include iodized salt, dairy products (partly due to the use of iodophor sanitizers for milk equipment, iodine-enriched cattle feed, and iodate-based bread conditioners in some breads), seaweed and other seafood, as well as drinking water⁽¹⁹⁾.

To prevent iodine deficiency, salt is commonly fortified with a controlled amount of potassium iodate (KIO₃) or potassium iodide (KI), with potassium iodate being the preferred compound. The WHO recommends that iodized salt at the production stage should contain between 20 and 40 milligrams of iodine per kilogram of salt. This level of fortification is intended to supply approximately 150 micrograms of iodine per person per day. When this guideline is followed, the median urinary iodine concentration in the population is typically expected to range from 100 to 199 micrograms per liter, indicating adequate iodine intake⁽²⁵⁾. Thailand has mandated the iodization of food-grade salt since 1994. In addition, a 2010 regulation issued by the Thai

FDA requires that fish sauce, mixed fish sauce, and seasonings produced through the hydrolysis or fermentation of soybean protein must contain between 2 and 3 milligrams of iodine per liter. As a result, the iodine intake from iodized salt used in processed foods is sufficient to meet nearly 100% of the estimated average requirement (EAR) for non-pregnant adults⁽⁵¹⁾.

Iodine levels in retail milk are not regulated by standardized guidelines, resulting in significant variability—ranging from 91 µg/L to 489 µg/kg. This variation is primarily influenced by the iodine content in the feed given to lactating animals, which has been shown to have a linear relationship with the iodine concentration in the milk⁽⁵²⁾.

The iodine concentration in seaweeds varies significantly and is influenced by several factors, including the species, plant part, growth stage, season, and geographic origin. Typically, brown seaweeds contain higher levels of iodine compared to red or green varieties. This variation is detailed in Table 5⁽⁵³⁾.

Table 5. Iodine concentration range in edible seaweed types (µg/g DW)⁽⁵⁴⁻⁵⁶⁾.

Classification and species	Common names	Iodine content (µg/g DW)	Grams of seaweed required to achieve daily iodine intake of 150 µg/ day
Green algae (Chlorophyta)	Wakame, Sea lettuce	30 - 185	2.0 - 6.0
Red algae (Rhodophyta)	Dulse, dillisk, Nori, Irish wakame, Irish moss	20 - 200	0.3 - 3.0
Brown algae (Phaeophyceae)	Kombu	2,500 - 10,000	0.01 - 0.04

A meta-analysis comprising seven studies from China, two from India, and one from Spain reported a broad range in median iodine concentrations in drinking water, spanning from 2.2 µg/L to 617.8 µg/L⁽⁵⁷⁾. In some regions, including Djibouti, Somalia, and parts of China, water with high

iodine content can be the predominant source of dietary iodine intake⁽¹⁹⁾.

Another source of iodine for pregnant women is multivitamin supplements, which typically contain between 150 and 200 micrograms of iodine per tablet as shown below in Table 6.

Table 6. Iodine content in supplement tablets (µg/tablet)⁽⁵⁸⁻⁶⁰⁾.

Commercial supplement brand	Iodine content (µg/tablet)
Triferdine	150
Nataral-EZ	150
Obimin-AZ	200

Impact of iodine deficiency

Pregnant women

Iodine deficiency could lead to hypothyroidism and goiter. Hypothyroidism presents with a broad spectrum of symptoms, which may be asymptomatic or, in rare cases, become severe and life-threatening. Hallmark symptoms commonly include fatigue, lethargy, unexplained weight gain, and heightened sensitivity to cold⁽⁶¹⁾. Adverse cardiovascular alterations may occur in pregnant individuals, including diastolic hypertension, bradycardia, elevated serum cholesterol levels, low-output congestive heart failure, and the presence of pericardial effusion⁽⁶²⁾.

Overt hypothyroidism during pregnancy has been linked to a higher incidence of complications such as pregnancy-induced hypertension, placental abruption, postpartum hemorrhage, preterm birth, low birthweight, and fetal death^(63, 64). Supporting this, a large U.S. national cohort study conducted in 2013, involving 223,512 pregnant women, found that untreated overt maternal hypothyroidism was significantly associated with increased risks of preeclampsia, gestational diabetes mellitus, preterm delivery, cesarean section, and admission of the newborn to a neonatal intensive care unit⁽⁶⁵⁾. In contrast, the effects of maternal subclinical hypothyroidism remain less clearly defined, with conflicting findings reported across various cohort

studies. These discrepancies may stem from differences in the timing of TSH assessments, the diagnostic thresholds applied for subclinical hypothyroidism, and variability in evaluating thyroid peroxidase (TPO) antibody status. Despite these inconsistencies, accumulating evidence indicates a potential association between subclinical hypothyroidism during pregnancy and adverse outcomes, including an elevated risk of miscarriage and preterm birth⁽⁶⁶⁾.

Lactating women

While a definitive link between iodine status and the incidence of postpartum thyroiditis has not been conclusively demonstrated, certain studies have suggested a potential association between excessive iodine intake and an increased prevalence of TPO antibody positivity, although these findings have not been uniformly replicated across all investigations⁽⁵⁾. Recent research involving women with pregestational Hashimoto's thyroiditis indicates that maternal thyroid function in early pregnancy may serve as a predictor for the development of postpartum thyroiditis. Interestingly, the findings suggest that euthyroid status during early gestation is associated with a greater incidence of postpartum thyroiditis compared to those who were hypothyroid. The authors proposed that the likelihood of developing postpartum thyroiditis may be influenced by the volume of remaining functional

thyroid tissue⁽⁶⁷⁾.

Fetus, neonate and childhood

Thyroid hormones play a crucial role in brain development, including neurogenesis, neuronal migration, synapse formation, and myelination. A deficiency of iodine during pregnancy, particularly in the first trimester, can lead to permanent brain damage⁽⁶⁸⁾.

Severe iodine deficiency, identified by a median UIC of less than 50 µg/L, is linked to negative outcomes such as stillbirth, newborn death, and impaired fetal growth⁽⁶⁹⁻⁷¹⁾. Cretinism is the most severe consequence of iodine deficiency during pregnancy and presents in two distinct forms: neurological and myxedematous. It affects an estimated 4% to 10% of children born to mothers with severe iodine deficiency. The neurological form is more prevalent and is marked by cognitive impairment, speech and hearing problems, strabismus, difficulties with motor control, spastic movements, and ataxia⁽⁷²⁾.

In contrast, myxedematous cretinism results from prolonged or severe hypothyroidism and is characterized by symptoms such as myxedema, stunted growth, delayed physical development, skeletal abnormalities, coarse and dry skin, thinning hair and nails, sluggish tendon reflexes, and constipation. Myxedematous cretinism is more commonly associated with postnatal hypothyroidism. Despite their distinct origins and clinical profiles, both neurological and myxedematous forms of cretinism share several overlapping neurological features. These include neurocognitive delays, hearing impairment, abnormalities in motor control due to pyramidal or extrapyramidal system dysfunction, the persistence of primitive reflexes, and other signs of central nervous system involvement. These shared features highlight the critical role of thyroid hormones not only during fetal brain development but also in the early postnatal period, emphasizing the need for both prenatal and continued postnatal monitoring and management of iodine status^(73, 74).

Mild to moderate iodine deficiency is classified by a median UIC ranging from 50 to 150 µg/L^(69, 75, 76).

Even mild to moderate maternal iodine deficiency has been associated with subtle but significant impairments in offspring's neurocognitive development, including lower intelligence quotient (IQ) scores, delayed language acquisition, reduced executive function, and poorer educational achievement^(77, 78). Findings from the Avon Longitudinal Study of Parents and Children (ALSPAC) revealed that children born to mothers with an iodine-to-creatinine ratio below 150 µg/g were significantly more likely to have lower scores in verbal IQ, reading accuracy, and reading comprehension⁽⁷⁹⁾.

The adverse outcomes identified in both maternal and neonatal health highlight the essential role of adequate iodine intake during pregnancy and lactation. Sufficient iodine status is critical not only for optimal neurodevelopment in the offspring but also for supporting normal fetal growth and minimizing the risk of obstetric complications.

Conclusion

Iodine deficiency continues to represent a major global public health concern, despite being entirely preventable. Its association with increased risks of adverse maternal outcomes, impaired fetal growth, and suboptimal neurocognitive development in offspring underscores the critical importance of ensuring adequate iodine intake during pregnancy and lactation. Owing to increased demands during these periods, the WHO recommends a daily iodine intake of 250 µg for pregnant and lactating women. Although 24UIE remains the gold standard for assessing iodine status, it is often impractical in clinical and population settings. Consequently, median UIC is widely used as a reliable, population-level biomarker, with a range of 150 - 249 µg/L indicating adequacy in pregnancy. The UIC/Cr ratio has been proposed as a more robust indicator in pregnant women, as it accounts for urine dilution and inter-individual variability in hydration status. Alternative biomarkers, including serum iodine concentrations and serum thyroglobulin, require further validation to establish clinical utility and standardized reference ranges. Universal salt iodization has been instrumental in

reducing the global burden of iodine deficiency; however, continued surveillance and region-specific studies are warranted. Further research is needed to refine iodine assessment methodologies, particularly in serum and breast milk, and to evaluate iodine sufficiency across diverse populations and geographical contexts to inform future public health strategies.

Potential conflicts of interest

The authors declare no competing interests.

References

1. Thaisriwong C, Phupong V. Nutrition during pregnancy. *Thai J Obstet Gynaecol* 2023;31:388-98.
2. Adams JB, Sorenson JC, Pollard EL, Kirby JK, Audhya T. Evidence-based recommendations for an optimal prenatal supplement for women in the U.S., Part Two: Minerals. *Nutrients* 2021;13:1849.
3. Lee SY. Editorial: Consequences of iodine deficiency in pregnancy. *Front Endocrinol (Lausanne)* 2021;12:740239.
4. Moleti M, Trimarchi F, Vermiglio F. Thyroid physiology in pregnancy. *Endocr Pract* 2014;20:589-96.
5. Andersson M, Braegger CP. The role of iodine for thyroid function in lactating women and infants. *Endocr Rev* 2022;43:469-506.
6. Baffa LD, Angaw DA, Abriham ZY, Gashaw M, Agimas MC, Sisay M, et al. Prevalence of iodine deficiency and associated factors among school-age children in Ethiopia: a systematic review and meta-analysis. *Syst Rev* 2024;13:142.
7. Iodine Global Network Annual Report 2024 [Internet]. 2024 [cited 3 August 2025]. Available from: <https://ign.org/app/uploads/2025/06/12079-IGN-%E2%80%93Annual-Report-2025-1.6.pdf>.
8. Charoenratana C, Leelapat P, Traisrisilp K, Tongsong T. Maternal iodine insufficiency and adverse pregnancy outcomes. *Matern Child Nutr* 2016;12:680-7.
9. Mekrungharas T, Kasemsup R. Breast milk iodine concentrations in lactating mothers at Queen Sirikit National Institute of Child Health. *J Med Assoc Thai* 2014;97(Suppl 6):S115-9.
10. Institute of Medicine (US) Panel on Micronutrients. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Washington (DC): National Academies Press (US) 2001.
11. World Health Organization: Iodine deficiency [cited 2025 7 July]. Available from: <https://www.who.int/data/nutrition/nlis/info/iodine-deficiency>.
12. Rodriguez-Diaz E, Pearce EN. Iodine status and supplementation before, during, and after pregnancy. *Best Pract Res Clin Endocrinol Metab* 2020;34:101430.
13. Azizi F, Smyth P. Breastfeeding and maternal and infant iodine nutrition. *Clin Endocrinol (Oxf)* 2009;70:803-9.
14. Ma ZF, Brough L. Effect of iodine nutrition during pregnancy and lactation on child cognitive outcomes: A review. *Nutrients* 2025;17:2016.
15. Hatch-McChesney A, Lieberman HR. Iodine and iodine deficiency: A comprehensive review of a Re-emerging issue. *Nutrients* 2022;14:3474.
16. Nishiyama S, Mikeda T, Okada T, Nakamura K, Kotani T, Hishinuma A. Transient hypothyroidism or persistent hyperthyrotropinemia in neonates born to mothers with excessive iodine intake. *Thyroid* 2004;14:1077-83.
17. Bizhanova A, Kopp P. Minireview: The sodium-iodide symporter NIS and pendrin in iodide homeostasis of the thyroid. *Endocrinology* 2009;150:1084-90.
18. Leung AM, Braverman LE. Consequences of excess iodine. *Nat Rev Endocrinol* 2014;10:136-42.
19. Sohn SY, Inoue K, Rhee CM, Leung AM. Risks of Iodine Excess. *Endocr Rev* 2024;45:858-79.
20. Candido AC, Vieira AA, de Souza Ferreira E, Moreira TR, do Carmo Castro Franceschini S, Cotta RMM. Prevalence of excessive iodine intake in pregnancy and its health consequences: Systematic review and meta-analysis. *Biol Trace Elem Res* 2023;201:2784-94.
21. Andersson M, de Benoist B, Delange F, Zupan J. Prevention and control of iodine deficiency in pregnant and lactating women and in children less than 2-years-old: conclusions and recommendations of the technical consultation. *Public Health Nutr* 2007;10(12a):1606-11.
22. EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA). Scientific opinion on dietary reference values for iodine. *EFSA J* 2014;12:3660.
23. Zimmermann MB, Andersson M. Assessment of iodine nutrition in populations: past, present, and future. *Nutr Rev* 2012;70:553-70.
24. König F, Andersson M, Hotz K, Aeberli I, Zimmermann MB. Ten repeat collections for urinary iodine from

- spot samples or 24-hour samples are needed to reliably estimate individual iodine status in women. *J Nutr* 2011;141:2049-54.
25. World Health Organization, Unicef. Assessment of iodine deficiency disorders and monitoring their elimination: a guide for programme managers, 3rd ed 2007.
 26. Eriksson J, Barregard L, Sallsten G, Berlinger B, Weinbruch S, Manousou S, et al. Urinary iodine excretion and optimal time point for sampling when estimating 24-h urinary iodine. *Br J Nutr* 2023;130: 1289-97.
 27. Li C, Peng S, Zhang X, Xie X, Wang D, Mao J, et al. The urine iodine to creatinine as an optimal index of iodine during pregnancy in an iodine adequate area in China. *J Clin Endocrinol Metab* 2016;101:1290-8.
 28. Luo J, Li C, Zhang X, Shan Z, Teng W. Reference intervals of the ratio of urine iodine to creatinine in pregnant women in an iodine-replete area of China. *Biol Trace Elem Res* 2021;199:62-9.
 29. Bath SC, Furmidge-Owen VL, Redman CW, Rayman MP. Gestational changes in iodine status in a cohort study of pregnant women from the United Kingdom: season as an effect modifier. *Am J Clin Nutr* 2015;101:1180-7.
 30. Knudsen N, Christiansen E, Brandt-Christensen M, Nygaard B, Perrild H. Age- and sex-adjusted iodine/creatinine ratio. A new standard in epidemiological surveys? Evaluation of three different estimates of iodine excretion based on casual urine samples and comparison to 24 h values. *Eur J Clin Nutr* 2000;54: 361-3.
 31. Kesteloot H, Joossens JV. On the determinants of the creatinine clearance: a population study. *J Hum Hypertens* 1996;10:245-9.
 32. Allain P, Berre S, Krari N, Lainé-Cessac P, Le Bouil A, Barbot N, et al. Use of plasma iodine assay for diagnosing thyroid disorders. *J Clin Pathol* 1993;46:453-5.
 33. Pan Z, Cui T, Chen W, Gao S, Pearce EN, Wang W, et al. Serum iodine concentration in pregnant women and its association with urinary iodine concentration and thyroid function. *Clin Endocrinol (Oxf)* 2019;90: 711-8.
 34. Yu D, He Y, He H, Hu Z, Huang Y, Tang J. Serum iodine concentration in pregnant women and its association with thyroid function. *J Trace Elem Med Biol* 2025;87:127584.
 35. Ji S, Wu X, Wu J, Chen D, Chen Z. Serum iodine concentration and its associations with thyroid function and dietary iodine in pregnant women in the southeast coast of China: a cross-sectional study. *Front Endocrinol (Lausanne)* 2023;14:1289572.
 36. Lamas L, Anderson PC, Fox JW, Dunn JT. Consensus sequences for early iodination and hormonogenesis in human thyroglobulin. *J Biol Chem* 1989;264: 13541-5.
 37. Torrén JI, Burch HB. Serum thyroglobulin measurement. Utility in clinical practice. *Endocrinol Metab Clin North Am* 2001;30:429-67.
 38. Lamas L, Dorris ML, Taurog A. Evidence for a catalytic role for thyroid peroxidase in the conversion of diiodotyrosine to thyroxine. *Endocrinology* 1972;90:1417-26.
 39. Pezzino V, Vigneri R, Squatrito S, Filetti S, Camus M, Polosa P. Increased serum thyroglobulin levels in patients with nontoxic goiter. *J Clin Endocrinol Metab* 1978;46:653-7.
 40. Ma ZF, Skeaff SA. Thyroglobulin as a biomarker of iodine deficiency: a review. *Thyroid* 2014;24:1195-209.
 41. Bath SC, Pop VJ, Furmidge-Owen VL, Broeren MA, Rayman MP. Thyroglobulin as a functional biomarker of iodine status in a cohort study of pregnant women in the united kingdom. *Thyroid* 2017;27:426-33.
 42. Nazeri P, Shab-Bidar S, Pearce EN, Shariat M. Thyroglobulin concentration and maternal iodine status during pregnancy: A systematic review and meta-analysis. *Thyroid* 2020;30:767-79.
 43. Brown-Grant K. Extrathyroidal iodide concentrating mechanisms. *Physiological Rev* 1961;41:189-213.
 44. Leung AM, Braverman LE, He X, Heeren T, Pearce EN. Breastmilk iodine concentrations following acute dietary iodine intake. *Thyroid* 2012;22:1176-80.
 45. Dror DK, Allen LH. Iodine in human milk: A systematic review. *Adv Nutr* 2018;9(suppl 1):347s-57s.
 46. Sullivan KM, May W, Nordenberg D, Houston R, Maberly GF. Use of thyroid stimulating hormone testing in newborns to identify iodine deficiency. *J Nutr* 1997;127:55-8.
 47. Zimmermann MB, Aeberli I, Torresani T, Bürgi H. Increasing the iodine concentration in the Swiss iodized salt program markedly improved iodine status in pregnant women and children: a 5-y prospective national study. *Am J Clin Nutr* 2005;82:388-92.
 48. Elebrashy I, Kamal Eldein HA, Abd-Elstar H, Ghanem N, Mousa S, Assem M, et al. Assessment of thyroid functions and thyroid volume in normal pregnant Egyptian females. *Gynecol Endocrinol* 2020;36: 122-5.
 49. Vila L, Legaz G, Barrionuevo C, Espinel ML, Casamitjana R, Muñoz J, et al. Iodine status and

- thyroid volume changes during pregnancy: results of a survey in Aran Valley (Catalan Pyrenees). *J Endocrinol Invest* 2008;31:851-5.
50. Curí LL, Quesada M, Despaigne DAN, Savariego EJ, González LC, Konaré DB, et al. Reference values and predictive factors for thyroid volume in pregnant women. *Arch Endocrinol Metab* 2023;67:e000656.
 51. Chotivichien S, Chongchaithet N, Aksornchu P, Boonmongkol N, Duangmusik P, Knowles J, et al. Assessment of the contribution of industrially processed foods to salt and iodine intake in Thailand. *PLoS One* 2021;16:e0253590.
 52. Niero G, Visentin G, Censi S, Righi F, Manuelian CL, Formigoni A, et al. Invited review: Iodine level in dairy products-A feed-to-fork overview. *J Dairy Sci* 2023;106:2213-29.
 53. Smyth PPA. Iodine, Seaweed, and the thyroid. *Eur Thyroid J* 2021;10:101-8.
 54. Yeh TS, Hung NH, Lin TC. Analysis of iodine content in seaweed by GC-ECD and estimation of iodine intake. *J Food Drug Anal* 2014;22:189-96.
 55. Bouga M, Combet E. Emergence of seaweed and seaweed-containing foods in the UK: Focus on labeling, Iodine content, Toxicity *Nutr Foods* 2015;4:240-53.
 56. Eveleigh ER, Coneyworth LJ, Avery A, Welham SJM. vegans, vegetarians, and omnivores: How does dietary choice influence iodine intake? A systematic review. *Nutrients* 2020;12:1606.
 57. Azevedo FM, Machamba AAL, Candido AC, Pinto CA, Lopes SO, Macedo MS, et al. Correlation between drinking water and iodine status: A systematic review and meta-analysis. *Biol Trace Elem Res* 2023;201:129-38.
 58. MIMS. Triferdine[cited 2025 2 August]. Available from: <https://www.mims.com/thailand/drug/info/triferdine>.
 59. MIMS. Nataral EZ[cited 2025 2 August]. Available from: <https://www.mims.com/thailand/drug/info/nataral-ez>.
 60. MIMS. Obimin-AZ[cited 2025 2 August]. Available from: <https://www.mims.com/thailand/drug/info/obimin-az>.
 61. Taylor PN, Medici MM, Hubalewska-Dydejczyk A, Boelaert K. Hypothyroidism. *Lancet* 2024;404:1347-64.
 62. Amy M. Valent LAB. Thyroid disease and pregnancy. In: Charles J. Lockwood JAC, Lorraine Dugoff, Judette Louis, Thomas R. Moore, Robert M. Silver, Robert Resnik, editor. *Creasy & Resnik's Maternal-Fetal Medicine: Principles and Practice*. 9th ed. Philadelphia: Elsevier 2023;1187-213.
 63. Davis LE, Leveno KJ, Cunningham FG. Hypothyroidism complicating pregnancy. *Obstet Gynecol* 1988;72: 108-12.
 64. Leung AS, Millar LK, Koonings PP, Montoro M, Mestman JH. Perinatal outcome in hypothyroid pregnancies. *Obstet Gynecol* 1993;81:349-53.
 65. Männistö T, Mendola P, Grewal J, Xie Y, Chen Z, Laughon SK. Thyroid diseases and adverse pregnancy outcomes in a contemporary US cohort. *J Clin Endocrinol Metab* 2013;98:2725-33.
 66. Lee SY, Pearce EN. Assessment and treatment of thyroid disorders in pregnancy and the postpartum period. *Nat Rev Endocrinol* 2022;18:158-71.
 67. Moleti M, Mauro MD, Alibrandi A, Vita R, Benvenega S, Vermiglio F. Postpartum thyroiditis in women with euthyroid and hypothyroid hashimoto's thyroiditis antedating pregnancy. *J Clin Endocrinol Metab* 2020;105:dga197.
 68. de Escobar GM, Obregón MJ, del Rey FE. Iodine deficiency and brain development in the first half of pregnancy. *Public Health Nutr* 2007;10:1554-70.
 69. Zimmermann MB. The adverse effects of mild-to-moderate iodine deficiency during pregnancy and childhood: a review. *Thyroid* 2007;17:829-35.
 70. Chittimoju SB, Pearce EN. Iodine deficiency and supplementation in pregnancy. *Clin Obstet Gynecol* 2019;62:330-8.
 71. Toloza FJK, Motahari H, Maraka S. Consequences of severe iodine deficiency in pregnancy: Evidence in humans. *Front Endocrinol (Lausanne)* 2020;11:409.
 72. McCarrison R. Observations on endemic cretinism in the Chitral and Gilgit valleys. *Proc R Soc Med* 1909;2(Med Sect):1-36.
 73. Boyages SC, Halpern JP, Maberly GF, Eastman CJ, Morris J, Collins J, et al. A comparative study of neurological and myxedematous endemic cretinism in western China. *J Clin Endocrinol Metab* 1988;67:1262-71.
 74. Halpern JP, Boyages SC, Maberly GF, Collins JK, Eastman CJ, Morris JG. The neurology of endemic cretinism. A study of two endemias. *Brain* 1991;114 (Pt 2):825-41.
 75. Gliouer D. The importance of iodine nutrition during pregnancy. *Public Health Nutr* 2007;10(12a):1542-6.
 76. Delange F. The disorders induced by iodine deficiency. *Thyroid* 1994;4:107-28.
 77. Hay I, Hynes KL, Burgess JR. Mild-to-moderate gestational iodine deficiency processing disorder. *Nutrients* 2019;11:1974.
 78. Caron P. Neurocognitive outcomes of children secondary to mild iodine deficiency in pregnant

women. *Ann Endocrinol (Paris)* 2015;76:248-52.

79. Bath SC, Steer CD, Golding J, Emmett P, Rayman MP. Effect of inadequate iodine status in UK pregnant

women on cognitive outcomes in their children: results from the Avon Longitudinal Study of Parents and Children (ALSPAC). *Lancet* 2013;382:331-7.