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Advances in the Management of Thyroid Disease During Preconception, Pregnancy, and Postpartum: A Critical Review of the American Thyroid Association 2026 Guidelines

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Abstract: *Thyroid disorders are among the most prevalent endocrine conditions affecting women of reproductive age and are associated with significant reproductive, obstetric, and neonatal complications. In 2026, the American Thyroid Association (ATA) published updated evidence-based guidelines for the diagnosis and management of thyroid disease during preconception, pregnancy, and the postpartum period. These guidelines represent a substantial revision of the 2017 recommendations and incorporate emerging evidence regarding gestational thyroid physiology, thyroid function testing, iodine nutrition, thyroid autoimmunity, hypothyroidism, hyperthyroidism, Graves' disease, thyroid nodules, thyroid cancer, and postpartum thyroid dysfunction. This review critically examines the methodological framework, key recommendations, strengths, limitations, and clinical implications of the updated ATA guidelines. Comparisons with the 2017 guidelines are presented to highlight major changes in clinical practice. The review further discusses future research priorities necessary to strengthen the evidence base for thyroid disease management in reproductive medicine.*

Keywords: thyroid disease, pregnancy, preconception, postpartum thyroiditis, hypothyroidism, hyperthyroidism, graves' disease, thyroid function testing, clinical guidelines.

INTRODUCTION

Thyroid dysfunction remains one of the most frequently encountered endocrine disorders among women of reproductive age. Both overt and subclinical thyroid abnormalities have been associated with infertility, miscarriage, preterm delivery, hypertensive disorders of pregnancy, impaired fetal neurodevelopment, and postpartum complications (Alexander et al., 2017; Stagnaro-Green, 2011; Korevaar et al., 2019). Consequently, optimal thyroid assessment and management throughout the reproductive continuum has become an increasingly important component of maternal healthcare.

The American Thyroid Association (ATA) 2026 Guidelines represent a comprehensive update of the landmark 2017 recommendations (Alexander et al., 2017). Since publication of the earlier guideline, substantial advances have emerged regarding physiological adaptations of the thyroid gland during pregnancy, interpretation of thyroid function tests, iodine nutrition, thyroid autoimmunity, risk stratification of thyroid disorders, and therapeutic interventions before conception, during pregnancy, and after delivery (Taylor et al., 2024; Wei et al., 2024).

A notable feature of the 2026 revision is its multidisciplinary and international scope. The guideline development process involved collaboration among endocrinologists, obstetricians, reproductive medicine specialists, endocrine surgeons, public health experts, epidemiologists, and patient representatives. This broad representation was intended to enhance clinical applicability and facilitate implementation across diverse healthcare settings.

The updated guidelines were developed around three principal objectives:

1. To improve clinical relevance through multidisciplinary stakeholder engagement.
2. To modernize guideline presentation using practical algorithms, concise recommendations, and enhanced visual aids.
3. To strengthen methodological rigor through comprehensive literature review and adherence to evidence-based medicine principles.

These objectives distinguish the 2026 guidelines from earlier versions and reflect contemporary expectations for clinical practice recommendations (Alexander et al., 2017).

Structured Literature Review

Thyroid Physiology During Pregnancy

Pregnancy induces profound physiological changes in thyroid function. Increased concentrations of human chorionic gonadotropin (hCG) stimulate thyroid hormone production, whereas elevated estrogen concentrations increase thyroxine-binding globulin (TBG) levels, resulting in dynamic alterations in thyroid hormone availability throughout gestation (Glinoer, 2007; Alexander et al., 2017).

Consequently, trimester-specific changes occur in thyroid-stimulating hormone (TSH) and free thyroxine (FT4) concentrations. Recent investigations have demonstrated substantial variation in thyroid hormone measurements according to assay methodology, population characteristics, ethnicity, iodine status, and gestational age (Wei et al., 2024; Taylor et al., 2024). These findings challenge the universal applicability of fixed reference intervals and support the use of population-specific trimester-based reference ranges.

Thyroid Function Testing

Thyroid function testing remains one of the most frequently requested laboratory investigations in women planning pregnancy and during gestation (Alexander et al., 2017). Physiological changes associated with pregnancy, assisted reproductive technologies, and postpartum hormonal fluctuations complicate the interpretation of laboratory values.

Recent evidence has demonstrated that laboratory-specific reference intervals and assay-dependent differences significantly affect the diagnosis and classification of thyroid dysfunction (Taylor et al., 2024; Wei et al., 2024). Therefore, the updated ATA guidelines recommend greater emphasis on individualized interpretation and laboratory-specific standards.

Hypothyroidism and Pregnancy Outcomes

Maternal hypothyroidism is consistently associated with adverse pregnancy outcomes, including miscarriage, preterm birth, placental complications, gestational hypertension, preeclampsia, and impaired neurodevelopment in offspring (Alexander et al., 2017; Korevaar et al., 2019; Toloza et al., 2022).

Evidence supporting levothyroxine (LT4) treatment in overt hypothyroidism remains strong (Alexander et al., 2017; Taylor et al., 2024). However, uncertainty persists regarding treatment thresholds for subclinical hypothyroidism and isolated hypothyroxinemia. Several meta-analyses and randomized trials published since 2017 have yielded conflicting results regarding improvements in fertility and obstetric outcomes following treatment of mild thyroid dysfunction (Korevaar et al., 2019; Negro et al., 2005).

Hyperthyroidism and Graves' Disease

Hyperthyroidism during pregnancy presents unique management challenges because of potential maternal and fetal complications, including miscarriage, fetal growth restriction, preterm delivery, and neonatal thyrotoxicosis (Ross et al., 2016; Kahaly, 2020).

Graves' disease remains the most common cause of hyperthyroidism in women of reproductive age (Ross et al., 2016; Kalra et al., 2024). Recent evidence has clarified the importance of thyroid receptor antibodies (TRAb) in predicting fetal risk and guiding prenatal monitoring (Kalra et al., 2024).

Postpartum Thyroid Dysfunction

Postpartum thyroiditis affects a substantial proportion of women and may progress to permanent hypothyroidism (Alexander et al., 2017). Women with thyroid autoimmunity are at particularly

high risk for postpartum thyroid dysfunction and long-term thyroid disease (Stagnaro-Green, 2011).

The updated guidelines place greater emphasis on postpartum monitoring, risk stratification, and long-term follow-up, particularly among women with positive thyroid autoantibodies and those who experienced thyroid dysfunction during pregnancy (Alexander et al., 2017).

Methodology of the ATA 2026 Guidelines

The ATA 2026 guideline development committee consisted of endocrinologists, obstetricians, reproductive medicine specialists, endocrine surgeons, epidemiologists, a professional methodologist, and patient representatives. This multidisciplinary structure reflects contemporary standards for evidence-based guideline development.

A global needs assessment involving 388 healthcare professionals from Europe, North America, Asia, South America, Africa, and Australia was conducted to identify priority clinical questions and areas of uncertainty. Participants represented multiple disciplines, including endocrinology, obstetrics and gynecology, reproductive medicine, surgery, and primary care.

Systematic literature reviews were conducted with support from biomedical information specialists and evaluated using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) methodology, which has become the international standard for clinical practice guideline development (Alexander et al., 2017).

Evidence quality was categorized as:

- High certainty
- Moderate certainty
- Low certainty
- Very low certainty

Recommendations were classified as:

- Strong recommendations
- Conditional recommendations
- Good Practice Statements (GPS)

The incorporation of GPS recommendations represents an important methodological advancement, allowing clinically important guidance to be provided even when direct evidence remains limited.

RESULTS AND DISCUSSIONS

Table 1. Comparison Between the ATA 2017 and ATA 2026 Guidelines

Domain	ATA 2017	ATA 2026
Guideline Structure	Question-based format	Integrated clinical pathways and flowcharts
Evidence Review	Focus on recent evidence	Comprehensive literature reassessment
Methodology	GRADE framework	Expanded GRADE with GPS statements
Thyroid Function Testing	Primarily TSH-centered	Multiple assessment strategies
Risk Factor Screening	Broad screening criteria	Refined evidence-based risk factors
Assisted Reproduction	Limited discussion	Expanded recommendations
Postpartum Care	Brief recommendations	Detailed follow-up protocols
Clinical Decision Tools	Limited	Extensive algorithms and practical boxes
Patient Involvement	Minimal	Formal patient representation

Definition and Interpretation of Thyroid Function Tests During Pregnancy

Accurate interpretation of thyroid function tests (TFTs) during pregnancy remains a major clinical challenge because physiological adaptations of gestation significantly alter thyroid hormone concentrations and thyroid-stimulating hormone (TSH) dynamics (Glinoer, 2007; Alexander et al., 2017). The 2026 American Thyroid Association (ATA) Guidelines emphasize that the definition of normal and abnormal thyroid function during pregnancy should differ from that applied to non-pregnant women and should ideally be based on laboratory-specific and trimester-specific reference intervals (Alexander et al., 2017; Taylor et al., 2024).

For women planning conception and those in the postpartum period, thyroid dysfunction should continue to be diagnosed according to locally established reference intervals used for the general adult population. During pregnancy, however, substantial physiological changes—including increased human chorionic gonadotropin (hCG) concentrations, elevated estrogen-mediated thyroxine-binding globulin levels, increased plasma volume, and altered thyroid hormone metabolism—necessitate pregnancy-specific interpretation of laboratory results (Glinoer, 2007; Alexander et al., 2017; Taylor et al., 2024).

The ATA identifies laboratory- and trimester-specific reference intervals for both TSH and free thyroxine (FT4) as the preferred diagnostic standard. Such an approach accounts for physiological variations occurring throughout gestation and minimizes the risk of diagnostic misclassification (Alexander et al., 2017; Wei et al., 2024). Nevertheless, the guideline acknowledges that

pregnancy-specific reference ranges remain unavailable in many healthcare institutions worldwide. Consequently, alternative approaches are recommended when local gestational reference intervals cannot be established.

In the absence of trimester-specific reference intervals, the ATA recommends a first-trimester TSH reference range of approximately 0.1–4.0 mU/L (Alexander et al., 2017). This recommendation reflects accumulated evidence demonstrating a physiological reduction in maternal TSH concentrations during early pregnancy due to the thyrotropic effects of hCG (Glinoer, 2007). The proposed upper limit represents a reduction of approximately 0.5 mU/L from conventional non-pregnant reference ranges and is considered appropriate for most populations. In settings where the upper limit of the non-pregnant TSH range exceeds 4.5 mU/L, clinicians may consider adjusting the threshold downward by 0.5 mU/L rather than adopting a universal cutoff (Alexander et al., 2017).

Interpretation of FT4 concentrations presents additional difficulties because of substantial variability among laboratory assays. Analytical differences between immunoassays and measurement platforms can produce significant discrepancies in FT4 concentrations, particularly during the second and third trimesters when changes in binding proteins become more pronounced (Alexander et al., 2017; Taylor et al., 2024). For this reason, the ATA does not endorse a universal gestational FT4 reference range. Instead, when trimester-specific FT4 reference intervals are unavailable, clinicians may utilize alternative indicators of thyroid hormone status, including total thyroxine (TT4) adjusted for gestational age or the free thyroxine index (FT4I) (Alexander et al., 2017).

The guidelines further emphasize that serum TSH remains the most reliable biochemical marker of maternal thyroid status in most clinical situations and should serve as the primary parameter guiding diagnosis and therapeutic monitoring (Alexander et al., 2017; Taylor et al., 2024). Consistency in laboratory methodology throughout pregnancy is also strongly recommended, as changing assay platforms during patient follow-up may introduce analytical variability that complicates longitudinal assessment and clinical decision-making (Wei et al., 2024).

One of the principal strengths of the ATA 2026 recommendations is the recognition that pregnancy-specific physiological changes necessitate a more individualized interpretation of thyroid function tests. Compared with earlier guidance, the updated recommendations provide greater flexibility and acknowledge practical limitations encountered in routine clinical practice, particularly in healthcare settings lacking trimester-specific reference intervals (Alexander et al., 2017).

However, important challenges remain. Many recommendations continue to rely on low-certainty evidence, reflecting the scarcity of large prospective studies evaluating optimal gestational thyroid thresholds. Furthermore, substantial inter-assay variability continues to limit the reliability and comparability of FT4 measurements across laboratories, thereby creating potential inconsistencies

in diagnosis and management (Taylor et al., 2024; Wei et al., 2024). The absence of internationally standardized pregnancy-specific reference intervals also restricts universal implementation of guideline recommendations.

Future research should focus on establishing globally validated trimester-specific reference ranges, improving assay standardization, and developing outcome-based diagnostic thresholds that incorporate both biochemical abnormalities and clinically relevant maternal-fetal outcomes. Such advances may facilitate a more precise and personalized approach to thyroid disease diagnosis and management during pregnancy (Taylor et al., 2024; Wei et al., 2024).

Table 2. ATA 2026 Recommendations for Defining Abnormal Thyroid Function During Pregnancy

Clinical Setting	Recommendation	Strength of Recommendation	Quality of Evidence
Preconception and fertility treatment	Use standard non-pregnant reference intervals	Good Practice Statement	Expert Consensus
Pregnancy	Use laboratory- and trimester-specific TSH and FT4 reference intervals whenever available	Conditional	Low
Pregnancy without local TSH reference intervals	Use TSH range of 0.1–4.0 mU/L during the first trimester	Conditional	Low
Pregnancy without local FT4 reference intervals	Use adjusted TT4 or FT4 index as surrogate markers	Conditional	Low
Clinical assessment	Prioritize TSH as the primary indicator of maternal thyroid status	Good Practice Statement	Expert Consensus
Longitudinal monitoring	Maintain the same assay methodology throughout pregnancy	Good Practice Statement	Expert Consensus

One of the major strengths of the 2026 ATA recommendations is the recognition that pregnancy-specific physiological changes require a more individualized interpretation of thyroid function tests. Compared with the 2017 guidelines, the updated recommendations provide greater flexibility and acknowledge practical limitations encountered in routine clinical practice, particularly in regions lacking trimester-specific reference intervals.

However, several challenges remain. The majority of recommendations continue to be supported by low-certainty evidence, reflecting the scarcity of large prospective studies evaluating optimal gestational thyroid thresholds. Furthermore, substantial inter-assay variability continues to limit

the reliability of FT4 measurements, creating potential inconsistencies in diagnosis across laboratories and healthcare systems. The absence of internationally standardized pregnancy-specific reference intervals also restricts universal implementation of guideline recommendations.

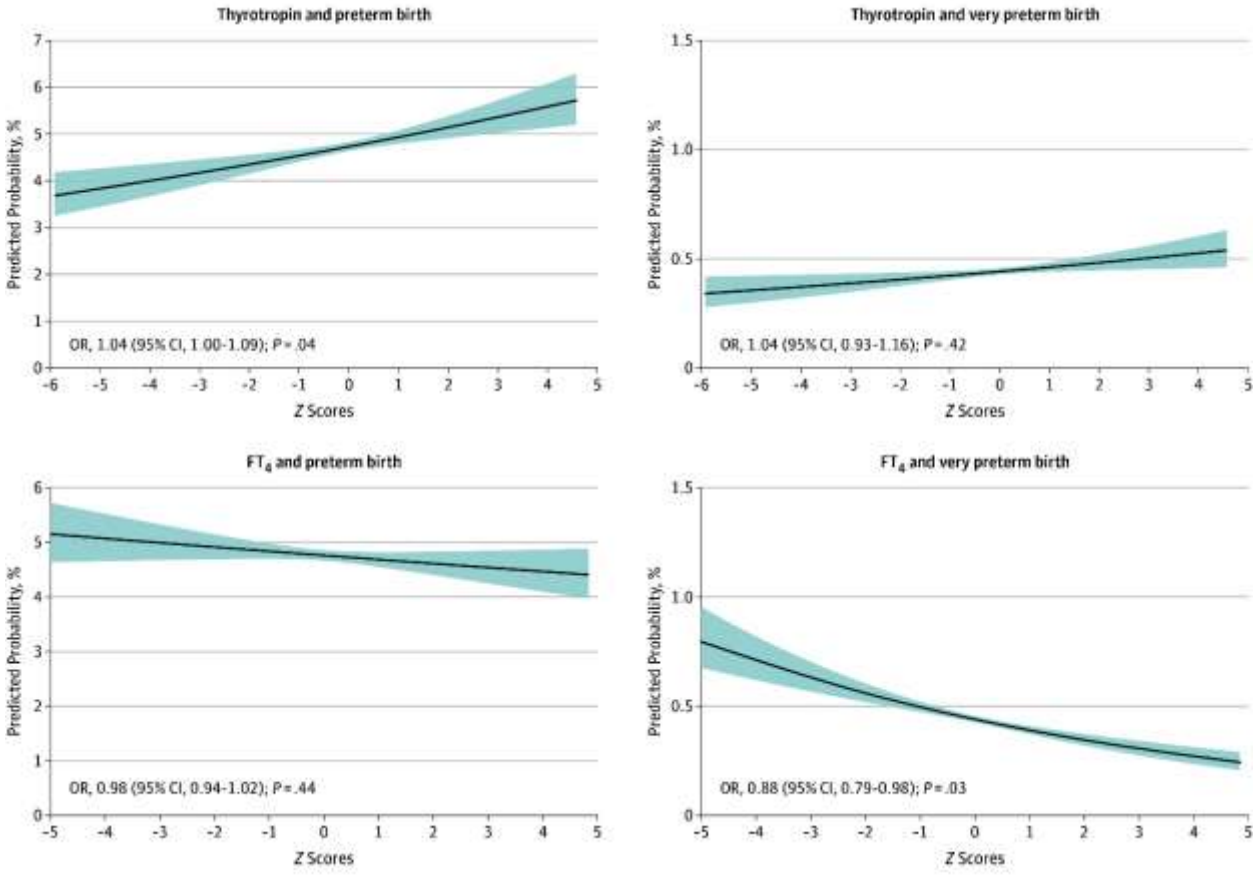

Figure 1. Association of Thyrotropin and Free Thyroxine (FT4) Concentrations With Preterm Birth.

Preterm birth was defined as less than 37 weeks' gestational age and very preterm birth was defined as less than 32 weeks' gestational age. The thyrotropin and FT_4 concentrations for all cohorts were log transformed and then standardized to population-specific standard deviation scores after removal of outliers (±4 SD from the mean) to enable comparison between different cohorts and assays. All analyses were adjusted for maternal age, body mass index, ethnicity, smoking, parity, gestational age at blood sampling, and fetal sex. OR indicates odds ratio (Korevaar et al., 2019).

Future research should focus on establishing globally validated trimester-specific reference ranges, improving assay standardization, and developing risk-based diagnostic thresholds that incorporate both biochemical abnormalities and clinically relevant maternal-fetal outcomes. Such advances

may enable a more precise and outcome-oriented approach to thyroid disease diagnosis during pregnancy.

The 2026 American Thyroid Association (ATA) Guidelines emphasize that accurate diagnosis of thyroid dysfunction during pregnancy requires the application of pregnancy-specific biochemical criteria rather than reference intervals derived from non-pregnant populations (Korevaar et al., 2026; Alexander et al., 2017). Physiological adaptations during gestation—including increased human chorionic gonadotropin (hCG) stimulation of the thyroid gland, estrogen-induced elevations in thyroxine-binding globulin (TBG), and changes in thyroid hormone metabolism—substantially influence circulating thyroid hormone concentrations and thyroid-stimulating hormone (TSH) levels (Glinoer, 2007; Alexander et al., 2017).

The ATA recommends the use of laboratory-specific and trimester-specific reference intervals established from healthy pregnant populations representative of the local demographic characteristics whenever possible (Korevaar et al., 2026). Ideally, these reference populations should consist of women with singleton pregnancies who are free from known thyroid disease, thyroid autoimmunity, and significant iodine deficiency (Alexander et al., 2017; Korevaar et al., 2026). Population-specific gestational reference ranges provide a more accurate representation of physiological thyroid function during pregnancy and reduce the risk of misclassification associated with fixed universal thresholds.

Importantly, the guideline notes that exclusion of additional variables, such as conception through assisted reproductive technology, the presence of non-thyroid medical disorders, or isolated thyroglobulin antibody positivity, does not appear to substantially alter gestational thyroid reference intervals (Korevaar et al., 2026). Excessive exclusion criteria may therefore limit sample size and compromise the statistical reliability of locally derived reference ranges.

Based on the relationship between TSH concentrations and circulating thyroid hormone levels, thyroid dysfunction during pregnancy can be classified into several clinically distinct entities (Korevaar et al., 2026; Alexander et al., 2017). Accurate classification is essential because it directly influences treatment decisions, maternal monitoring strategies, risk assessment, and fetal surveillance throughout gestation.

Table 3. Biochemical Classification of Thyroid Dysfunction During Pregnancy According to the ATA 2026 Guidelines

Thyroid Disorder	**TSH Concentration**	**FT4 Concentration**	**FT3/T3 Concentration**	**Clinical Interpretation**
Overt Primary Hypothyroidism	Elevated (>97.5th percentile)	Reduced (<2.5th percentile)	Usually normal or reduced	Established thyroid hormone deficiency requiring treatment
Subclinical Hypothyroidism	Elevated	Normal	Normal	Early or mild thyroid failure with preserved circulating hormone levels
Isolated Maternal Hypothyroxinemia	Normal	Reduced	Usually normal	Reduced thyroid hormone availability despite normal pituitary response
Overt Hyperthyroidism	Suppressed	Elevated	Elevated and/or elevated FT3	Excess thyroid hormone production with clinical significance
Subclinical Hyperthyroidism	Suppressed	Normal	Normal	Biochemical thyroid overactivity without elevated circulating hormone levels

Clinical Significance

Among the recognized categories of thyroid dysfunction during pregnancy, overt hypothyroidism and overt hyperthyroidism are most consistently associated with adverse maternal and fetal outcomes, including miscarriage, preterm birth, hypertensive disorders of pregnancy, fetal growth abnormalities, and neonatal complications (Alexander et al., 2017; Korevaar et al., 2019; Toloza et al., 2022). Consequently, early identification and appropriate treatment of overt thyroid disease remain fundamental components of antenatal care and maternal risk reduction strategies (Alexander et al., 2017).

Subclinical hypothyroidism (SCH) continues to represent one of the most controversial areas in reproductive endocrinology. Numerous observational studies have reported associations between SCH and adverse reproductive outcomes, including infertility, recurrent pregnancy loss, preterm delivery, and hypertensive disorders of pregnancy (Korevaar et al., 2019; Unuane et al., 2026). However, randomized controlled trials evaluating levothyroxine therapy have produced inconsistent findings regarding improvements in fertility outcomes, miscarriage rates, and live birth rates. Consequently, the ATA 2026 Guidelines advocate a more individualized, risk-based management strategy that incorporates thyroid peroxidase antibody (TPOAb) status, degree of TSH elevation, and reproductive context rather than universal treatment of all women with SCH (Korevaar et al., 2026).

Similarly, isolated maternal hypothyroxinemia remains a controversial diagnostic entity. Several observational studies have suggested associations between reduced maternal free thyroxine (FT4) concentrations and impaired neurocognitive development in offspring, particularly when thyroid hormone deficiency occurs during early gestation (Bath et al., 2013; Stagnaro-Green, 2011). Nevertheless, current evidence remains insufficient to support universal screening or routine levothyroxine treatment in women with isolated hypothyroxinemia. The ATA 2026 Guidelines acknowledge that substantial variability among FT4 assay methodologies contributes significantly to diagnostic uncertainty and complicates interpretation of existing evidence (Korevaar et al., 2026; Alexander et al., 2017).

Critical Appraisal of the ATA 2026 Classification System

The ATA 2026 classification framework represents an important advancement compared with previous approaches because it explicitly incorporates gestational physiology and recognizes the limitations of fixed biochemical thresholds during pregnancy (Korevaar et al., 2026). By prioritizing laboratory-specific and trimester-specific reference intervals, the guidelines promote a more individualized and biologically appropriate interpretation of thyroid function, thereby reducing the risk of overdiagnosis or underdiagnosis associated with universal cut-off values (Alexander et al., 2017; Glinoer, 2007).

Despite these improvements, several challenges remain. First, many healthcare institutions lack access to locally validated gestational reference intervals, necessitating reliance on surrogate thresholds that may not accurately reflect population-specific characteristics (Alexander et al., 2017). Second, substantial inter-assay variability continues to affect FT4 measurements, limiting diagnostic consistency across laboratories and healthcare systems (Korevaar et al., 2026). Third, uncertainty persists regarding the clinical significance and optimal management of SCH and isolated hypothyroxinemia because much of the available evidence originates from observational studies rather than adequately powered randomized controlled trials (Korevaar et al., 2019; Taylor et al., 2024).

Future research should prioritize international standardization of thyroid hormone assays, development of universally applicable pregnancy-specific reference intervals, and large prospective clinical trials evaluating the impact of treatment across the spectrum of maternal thyroid dysfunction on both short- and long-term maternal and offspring outcomes (Taylor et al., 2024; Wei et al., 2024).

Comparison with ATA 2017 Guidelines

Compared with the 2017 ATA Guidelines, the 2026 recommendations place substantially greater emphasis on laboratory-specific and trimester-specific reference intervals rather than generalized diagnostic thresholds (Alexander et al., 2017; Korevaar et al., 2026). The updated guideline also provides a clearer distinction between overt and subclinical disease states, acknowledges the analytical limitations of FT4 assays during pregnancy, and incorporates a more individualized risk-based approach to patient management (Korevaar et al., 2026). These modifications reflect advances in understanding gestational thyroid physiology, improvements in laboratory medicine, and increasing recognition of precision medicine principles in maternal endocrinology (Glinoer, 2007; Taylor et al., 2024).

Iodine Nutrition During Preconception, Pregnancy, and Lactation

Physiological Importance of Iodine in Pregnancy

Iodine is an essential micronutrient required for the synthesis of thyroxine (T4) and triiodothyronine (T3), hormones that play critical roles in maternal metabolism, placental function, fetal growth, and neurological development (Zimmermann, 2012; Andersen et al., 2014). Under normal physiological conditions, the adult human body contains approximately 15–20 mg of iodine, with more than 70% stored within the thyroid gland (Zimmermann, 2012).

Pregnancy substantially increases iodine requirements owing to enhanced maternal thyroid hormone production, increased renal iodine clearance, placental iodine transfer, and the progressive demands of the developing fetal thyroid gland (Glinoer, 2007; Taylor & Vaidya, 2016). The ATA 2026 Guidelines emphasize that adequate iodine intake should ideally be achieved before conception because maternal thyroid hormone production during the first trimester represents the primary source of thyroid hormone available to the fetus before maturation of the fetal hypothalamic-pituitary-thyroid axis (Korevaar et al., 2026). Consequently, inadequate iodine intake during early gestation may adversely affect fetal neurodevelopment before pregnancy is clinically recognized (Bath et al., 2013).

Global Epidemiology of Iodine Deficiency

Despite major public health advances through universal salt iodization programs, iodine deficiency remains an important global health concern (Zimmermann, 2012). Current international estimates

suggest that approximately one-third of the world's population resides in regions where iodine intake remains inadequate, with pregnant women representing one of the most vulnerable groups because of increased physiological requirements (Zimmermann, 2012; Andersen et al., 2014).

Recent epidemiological studies have demonstrated declining urinary iodine concentrations among pregnant women in several developed countries, including regions previously considered iodine sufficient (Andersen et al., 2014; Taylor & Vaidya, 2016). These observations suggest that mild-to-moderate iodine deficiency remains prevalent even in high-income settings.

The ATA 2026 Guidelines highlight a major limitation in iodine assessment: no currently available biomarker can reliably determine long-term iodine status at the individual patient level (Korevaar et al., 2026). Although urinary iodine concentration (UIC) remains the preferred method for population-based surveillance, it exhibits considerable day-to-day variability and should not be used in isolation to assess iodine sufficiency in individual women (Zimmermann, 2012). Therefore, clinical assessment should also consider dietary habits, geographical risk factors, vegetarian or vegan diets, and conditions associated with gastrointestinal malabsorption (Taylor & Vaidya, 2016).

Maternal and Fetal Consequences of Iodine Deficiency

The adverse effects of severe iodine deficiency are well established and represent one of the most preventable causes of intellectual disability worldwide (Zimmermann, 2012). Numerous observational studies have demonstrated associations between severe maternal iodine deficiency and maternal hypothyroidism, fetal hypothyroidism, miscarriage, stillbirth, perinatal mortality, impaired fetal growth, and long-term neurodevelopmental impairment (Zimmermann, 2012; Stagnaro-Green, 2011).

Particularly concerning is the impact of iodine deficiency on fetal brain development. Thyroid hormones are essential for neuronal migration, myelination, synaptogenesis, and cortical maturation during critical stages of fetal neurodevelopment (Bath et al., 2013). Severe maternal iodine deficiency has been associated with reduced cognitive performance, lower educational attainment, and significant intellectual impairment in offspring (Bath et al., 2013; Zimmermann, 2012).

The effects of mild-to-moderate iodine deficiency remain less clearly defined. Although evidence regarding adverse obstetric outcomes is inconsistent, several prospective cohort studies have reported associations between lower maternal iodine status during early pregnancy and reduced cognitive performance, language development, and intelligence quotient (IQ) scores in childhood (Bath et al., 2013; Andersen et al., 2014). However, definitive conclusions remain limited by methodological heterogeneity and the scarcity of large randomized controlled trials (Taylor & Vaidya, 2016).

ATA 2026 Recommendations for Iodine Supplementation

The ATA 2026 Guidelines recommend that pregnant and lactating women achieve a total daily iodine intake of approximately 250 µg through dietary sources and supplementation when necessary (Korevaar et al., 2026). Women at increased risk of iodine deficiency—including those living in iodine-deficient regions, consuming restrictive diets, or experiencing gastrointestinal malabsorption—should ideally initiate iodine supplementation before conception (Korevaar et al., 2026; Taylor & Vaidya, 2016).

Specifically, the guideline recommends supplementation with approximately 150 µg of iodine daily beginning at least three months before planned conception and continuing throughout pregnancy and lactation (Korevaar et al., 2026). In settings where universal salt iodization programs are unavailable or ineffective, annual administration of iodized oil may represent an effective public health strategy for preventing iodine deficiency disorders (Zimmermann, 2012).

Importantly, the ATA also recommends similar iodine supplementation practices for women receiving levothyroxine therapy for hypothyroidism or antithyroid medications for Graves' disease, provided no contraindications exist (Korevaar et al., 2026; Kahaly, 2020). Conversely, excessive iodine intake should be avoided because iodine excess may paradoxically impair thyroid function and contribute to maternal or fetal thyroid dysfunction (Taylor & Vaidya, 2016; Korevaar et al., 2026).

Table 4. ATA 2026 Recommendations for Iodine Nutrition During Pregnancy and Lactation

Recommendation	Strength	Evidence Quality
Achieve total iodine intake of 250 µg/day during pregnancy and lactation	Strong	Moderate
Initiate 150 µg/day iodine supplementation before conception in women at risk of deficiency	Conditional	Moderate
Continue iodine supplementation throughout lactation	Conditional	Moderate
Consider annual iodized oil administration in severely iodine-deficient low-resource regions	Conditional	Moderate
Apply similar supplementation principles in women receiving levothyroxine or antithyroid therapy	Conditional	Low
Avoid excessive iodine exposure except for specific medical indications	Strong	Moderate
Avoid sustained iodine intake exceeding 500 µg/day during pregnancy	Strong	Moderate

Risks of Excessive Iodine Intake

Although iodine deficiency remains a major concern, excessive iodine intake may also adversely affect maternal and fetal thyroid function. The fetal thyroid gland is particularly sensitive to iodine overload, and excessive exposure may induce transient or persistent thyroid dysfunction through autoregulatory mechanisms such as the Wolff–Chaikoff effect.

The ATA therefore recommends avoiding sustained iodine intake exceeding 500 μg/day during pregnancy. Exceptions include specific medical situations requiring iodinated contrast agents or potassium iodide preparations. Even in these circumstances, careful monitoring of maternal and fetal thyroid function may be warranted.

Critical Appraisal of the ATA 2026 Recommendations on Iodine

A major strength of the updated guidelines is their recognition that iodine deficiency remains a global public health challenge despite substantial progress in iodine supplementation programs. The recommendations appropriately balance evidence from epidemiological studies, clinical trials, and biological plausibility while acknowledging the limitations of currently available biomarkers.

Another notable advancement is the emphasis on preconception iodine optimization. This reflects increasing recognition that fetal neurological development begins before many women become aware of pregnancy, making early intervention essential.

Nevertheless, important evidence gaps remain. Most recommendations are supported by low-to-moderate certainty evidence, and high-quality randomized controlled trials evaluating neurodevelopmental outcomes in mildly iodine-deficient populations are lacking. Furthermore, the absence of reliable individual-level biomarkers complicates clinical decision-making and limits the effectiveness of targeted screening strategies.

Future research should focus on developing more accurate measures of individual iodine status, evaluating long-term neurodevelopmental outcomes following supplementation, and establishing region-specific supplementation strategies that account for differences in dietary patterns, iodine fortification programs, and population risk profiles.

Comparison with the ATA 2017 Guidelines

Compared with the 2017 recommendations, the ATA 2026 Guidelines place greater emphasis on the limitations of urinary iodine concentration as an individual diagnostic tool and highlight the continued importance of assessing personal risk factors for iodine deficiency. The updated document also provides more explicit guidance regarding supplementation in resource-limited settings and emphasizes both the risks of iodine deficiency and the potential consequences of excessive iodine exposure.

These modifications reflect advances in understanding iodine physiology during pregnancy and underscore the importance of individualized nutritional assessment within modern maternal healthcare.

for precision-based approaches in maternal endocrine care.

Strengths of the 2026 Guidelines

Several important strengths distinguish the updated guidelines:

International Collaboration

The inclusion of representatives from multiple endocrine and reproductive medicine societies enhances external validity and global applicability.

Robust Evidence Assessment

The extensive systematic review process and adherence to GRADE methodology strengthen methodological transparency.

Patient-Centered Approach

Inclusion of patient representatives acknowledges patient values and shared decision-making.

Practical Clinical Utility

Flowcharts, algorithms, and concise recommendations improve usability in busy clinical environments.

Expanded Focus on Reproductive Medicine

The guideline provides more comprehensive recommendations for infertility, assisted reproductive technologies, and preconception care.

Limitations of the 2026 Guidelines

Despite significant advances, several limitations remain.

Limited High-Quality Evidence

The majority of recommendations continue to rely on low- or moderate-certainty evidence.

Heterogeneity of Studies

Variations in laboratory assays, iodine status, ethnicity, and healthcare systems limit comparability across studies.

Lack of Randomized Controlled Trials

Many clinically important questions remain unsupported by adequately powered randomized trials.

Resource Constraints

Some recommendations may be difficult to implement in low-resource healthcare settings.

Clinical Implications

The 2026 ATA guidelines emphasize individualized care and risk-based management throughout preconception, pregnancy, and postpartum periods. Clinicians should recognize physiological changes affecting thyroid function testing and apply trimester-specific interpretation strategies whenever possible.

The updated recommendations support earlier recognition of thyroid dysfunction, more precise treatment decisions, and improved maternal-fetal outcomes.

Future Research Priorities

Future investigations should focus on:

1. Randomized trials evaluating treatment of subclinical hypothyroidism.
2. Standardization of pregnancy-specific thyroid function testing.
3. Long-term neurodevelopmental outcomes in offspring.
4. Personalized treatment approaches based on thyroid autoimmunity.
5. Cost-effectiveness of universal versus targeted screening strategies.

CONCLUSION

The American Thyroid Association 2026 Guidelines constitute a major advancement in the management of thyroid disease during preconception, pregnancy, and the postpartum period. The updated recommendations incorporate new evidence regarding thyroid physiology, thyroid function testing, iodine nutrition, and reproductive endocrinology while emphasizing individualized patient care and evidence-based decision-making. Nevertheless, important

knowledge gaps remain, particularly concerning subclinical thyroid dysfunction, assay standardization, and long-term maternal and offspring outcomes. Continued high-quality research is essential to further refine future recommendations and optimize maternal-fetal health.

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