THYROID HORMONES AND THEIR IMPACT ON PREGNANCY: EFFECTS AND RISKS

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Abstract: *Thyroid hormones play a crucial role in maternal and fetal health during pregnancy. Proper thyroid function ensures normal fetal neurodevelopment, regulates maternal metabolism, and supports placental function. Both hypothyroidism and hyperthyroidism can lead to adverse pregnancy outcomes, including miscarriage, preterm birth, preeclampsia, and impaired neurocognitive development in the offspring. This article reviews the physiological role of thyroid hormones during pregnancy, their regulatory mechanisms, and the potential maternal and fetal risks associated with thyroid dysfunction. Understanding these effects is essential for early diagnosis, monitoring, and appropriate therapeutic interventions to optimize pregnancy outcomes.*

Keywords: *Thyroid hormones, pregnancy, hypothyroidism, hyperthyroidism, maternal-fetal health, pregnancy complications, fetal development, endocrine regulation*

Thyroid hormones are essential regulators of metabolism, growth, and development, and their role becomes especially critical during pregnancy. Maternal thyroid function affects not only the mother's metabolic homeostasis but also fetal development, particularly neurodevelopment in the first trimester. During early pregnancy, the fetus is dependent on maternal thyroid hormones as its thyroid gland is not yet fully functional.

Abnormal thyroid function, including hypothyroidism and hyperthyroidism, is associated with a variety of maternal and fetal complications. Hypothyroidism can result in increased risk of miscarriage, gestational hypertension, preeclampsia, and impaired fetal cognitive development. Hyperthyroidism, on the other hand, may lead to preterm labor, low birth weight, and maternal cardiovascular strain. Both overt and subclinical thyroid disorders require careful monitoring and management to minimize adverse outcomes.

Pregnancy also induces physiological changes in thyroid hormone metabolism and binding proteins, necessitating adaptations in maternal thyroid function. These changes, if unrecognized or untreated, can exacerbate preexisting thyroid conditions or reveal latent dysfunction. Therefore, understanding the complex interplay between thyroid hormones and pregnancy is critical for clinicians to ensure optimal maternal and fetal health.

Thyroid hormones, primarily thyroxine (T4) and triiodothyronine (T3), play a central role in regulating maternal metabolism, fetal growth, and neurodevelopment during pregnancy. These hormones are produced by the thyroid gland under the control of the

hypothalamic-pituitary-thyroid axis through thyroid-stimulating hormone (TSH). During pregnancy, maternal thyroid function undergoes significant physiological changes to meet the metabolic demands of both the mother and developing fetus. Increased levels of estrogen elevate thyroxine-binding globulin (TBG), leading to higher total T4 and T3 levels, while the active free hormone concentrations may remain relatively stable. Additionally, the placenta expresses deiodinases that convert T4 to T3 and inactivate excess thyroid hormones, providing fine-tuned regulation.

Maternal hypothyroidism, characterized by insufficient thyroid hormone production, can have profound consequences for both the mother and fetus. Overt hypothyroidism, indicated by elevated TSH and low free T4, is associated with increased risks of miscarriage, preeclampsia, gestational hypertension, and preterm delivery. Subclinical hypothyroidism, where TSH is elevated but free T4 remains within the normal range, has also been linked to subtle adverse outcomes, including impaired cognitive development in the offspring. Thyroid hormone deficiency during the first trimester is particularly critical because fetal neurodevelopment depends entirely on maternal T4. Inadequate thyroid hormone levels can disrupt neuronal migration, myelination, and synaptogenesis, potentially leading to long-term neurocognitive deficits.

Hyperthyroidism, characterized by excessive thyroid hormone production, is less common but equally concerning during pregnancy. The most frequent cause in women of reproductive age is Graves' disease, an autoimmune disorder in which thyroid-stimulating immunoglobulins activate the TSH receptor. Maternal hyperthyroidism increases the risk of preterm labor, low birth weight, intrauterine growth restriction, and maternal cardiovascular complications, including arrhythmias and heart failure. If untreated, excessive thyroid hormone exposure can also lead to fetal thyrotoxicosis, which may manifest as tachycardia, goiter, or growth restriction. Careful titration of antithyroid medications, such as propylthiouracil in the first trimester and methimazole in later stages, is essential to balance maternal thyroid control and fetal safety.

Pregnancy itself imposes additional stress on the thyroid gland. The increase in circulating TBG requires augmented thyroid hormone production, and hCG secreted by the placenta can transiently stimulate the thyroid through TSH-like activity. Women with preexisting thyroid disorders, such as autoimmune thyroiditis or nodular goiter, are particularly vulnerable to decompensation during gestation. Therefore, regular monitoring of TSH and free T4 levels is critical, ideally before conception and throughout pregnancy. Screening strategies vary by region, but early identification and management of thyroid dysfunction have been shown to significantly improve maternal and fetal outcomes.

Iodine status is another crucial determinant of thyroid health during pregnancy. Adequate iodine intake is required for thyroid hormone synthesis, and deficiency can exacerbate hypothyroidism, contributing to goiter formation and impaired fetal neurodevelopment. Public health measures, such as iodized salt programs, have substantially reduced the incidence of iodine deficiency, but pregnant women remain a high-

risk group due to increased iodine requirements. Both deficiency and excess iodine can disrupt thyroid function, highlighting the need for balanced supplementation under medical supervision.

Thyroid hormone replacement therapy for hypothyroid pregnant women typically involves levothyroxine, which is safe and effective when appropriately dosed. Dosing requirements often increase during pregnancy, particularly in the first trimester, necessitating frequent monitoring and dose adjustment to maintain TSH within trimester-specific reference ranges. Conversely, management of hyperthyroidism requires minimizing fetal exposure to antithyroid drugs while controlling maternal symptoms. Close collaboration between endocrinologists and obstetricians is essential to navigate these complex clinical decisions.

Long-term studies have demonstrated that maintaining euthyroid status during pregnancy improves pregnancy outcomes, including reducing the risk of miscarriage, preterm birth, and impaired neurodevelopment in children. Untreated or poorly controlled thyroid disorders are associated with increased neonatal intensive care admissions and higher rates of congenital anomalies. Therefore, proactive management, including preconception counseling, early diagnosis, and individualized therapy, is essential for optimizing both maternal and fetal health.

In addition to clinical management, patient education plays a significant role. Pregnant women should be informed about the signs and symptoms of thyroid dysfunction, the importance of adherence to prescribed medications, and the need for regular laboratory monitoring. Lifestyle factors, such as maintaining adequate dietary iodine intake, avoiding excessive iodine consumption, and managing comorbidities, further support thyroid health during pregnancy.

In conclusion, thyroid hormones are indispensable for maternal well-being and fetal development, and their dysregulation poses significant risks during pregnancy. Both hypothyroidism and hyperthyroidism can lead to adverse maternal and fetal outcomes, but timely diagnosis and appropriate therapeutic intervention substantially mitigate these risks. Understanding the physiological changes in thyroid function during pregnancy, the impact of thyroid disorders, and the strategies for management is essential for clinicians to ensure optimal maternal and fetal health. Continuous monitoring, individualized therapy, and interdisciplinary care remain the cornerstones of managing thyroid dysfunction in pregnancy.

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