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Interrelationships Between FSH, LH, and TSH in Pregnant Women

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Abstract

Background: Pregnancy involves dynamic hormonal adjustments essential for maternal health and fetal development. Among these, Follicle-Stimulating Hormone (FSH), Luteinizing Hormone (LH), and Thyroid-Stimulating Hormone (TSH) play crucial roles in regulating reproductive and metabolic functions. Understanding their interrelationship provides insight into the physiological balance maintained during gestation.

Objective: To review and analyze existing literature on the interrelationships between FSH, LH, and TSH in pregnant women, emphasizing physiological regulation, trimester-wise variations, and hormonal feedback mechanisms.

Main Body: This study was conducted as a systematic literature review using databases including PubMed, ScienceDirect, Google Scholar, and Scopus. Studies published between 2020 and 2024 focusing on FSH, LH, and TSH variations in pregnancy were identified and reviewed. Relevant data were extracted, compared, and thematically analyzed. The review revealed that FSH and LH levels are markedly suppressed during pregnancy due to high estrogen, progesterone, and placental hormone activity, while TSH levels fluctuate depending on maternal thyroid status and gestational age. Several studies indicate a subtle but significant hormonal interdependence between thyroid function and gonadotropin regulation. However, regional data and combined hormone analyses remain limited.

Conclusion: The interaction between FSH, LH, and TSH reflects the complex endocrine coordination required for maintaining a healthy pregnancy. Further population-based research is needed to establish reference values and clarify these hormonal relationships to improve early detection and management of endocrine disturbances in pregnant women.

Keywords: FSH, LH, TSH, Pregnancy, Hormonal Interrelationship, Thyroid Function, Gonadotropins

Introduction

Endocrine disorders during pregnancy are a major global health concern, affecting maternal well-being and fetal development. Among these, disturbances involving gonadotropinsfollicle-stimulating hormone (FSH) and luteinizing hormone (LH)and thyroid-stimulating hormone (TSH) have gained significant attention due to their complex interrelationships and impact on reproductive and metabolic functions. According to the World Health Organization, thyroid dysfunction affects approximately 5–10% of women of reproductive age worldwide, with subclinical hypothyroidism being the most common form observed during pregnancy[1]

Pregnancy is a unique physiological state marked by significant hormonal changes that support successful implantation, fetal growth, and maternal adaptation. These changes are primarily regulated by the endocrine system, especially the hypothalamic–pituitary axis, which governs the release of hormones vital for reproduction and metabolism. During pregnancy, maternal physiology must maintain a delicate balance between multiple hormonal networks, including the hypothalamic–pituitary–gonadal (HPG) axis and the hypothalamic–pituitary–thyroid (HPT) axis. These systems are interconnected through feedback loops that coordinate thyroid and reproductive functions to support the developing fetus.[2,3]

The hypothalamic–pituitary axis serves as the main regulatory hub of the endocrine system, integrating neural and hormonal signals to maintain physiological balance. The hypothalamus secretes releasing hormones like gonadotropin-releasing hormone (GnRH) and thyrotropin-releasing hormone (TRH), which stimulate the anterior pituitary to release gonadotropinsfollicle-stimulating hormone (FSH) and luteinizing hormone (LH)as well as thyroid-stimulating hormone (TSH). These pituitary hormones target peripheral organs, such as the ovaries and thyroid gland, to regulate reproductive and metabolic functions. Although these hormones target different organs, they share structural and functional similarities, allowing potential interactions between them.[4]

The hypothalamic–pituitary axis acts as the central regulator of the endocrine system by integrating neural and hormonal signals to preserve physiological balance. The hypothalamus releases hormones like gonadotropin-releasing hormone (GnRH) and thyrotropin-releasing hormone (TRH), which prompt the anterior pituitary to secrete gonadotropinsfollicle-stimulating hormone (FSH) and luteinizing hormone (LH)alongside thyroid-stimulating hormone (TSH). These pituitary hormones then act on peripheral organs such as the ovaries and thyroid gland, managing reproductive and metabolic functions. Despite targeting different organs, these hormones share structural and functional similarities that enable possible interactions among them.[5.6]

The primary gonadotropins governing ovarian function and the menstrual cycle are follicle-stimulating hormone (FSH) and luteinizing hormone (LH). Both are secreted by the anterior pituitary in response to gonadotropin-releasing hormone (GnRH) from the hypothalamus. FSH promotes ovarian follicle growth and maturation, stimulates granulosa cells to produce estrogen, and prepares the oocyte for ovulation. LH triggers ovulation and maintains the corpus luteum, which produces progesterone to ready the endometrium and support early pregnancy.[7]

After conception, the hormonal balance completely changes. The growing placenta produces human chorionic gonadotropin (hCG), structurally identical to LH and acting on LH receptors to maintain the corpus luteum and its secretion of progesterone during early pregnancy. Consequently, endogenous production of both FSH and LH is significantly reduced during pregnancy as a result of the high circulating levels of estrogen, progesterone, and hCG. This inhibition avoids additional follicular recruitment and ovulation, saving energy for fetal growth. Such hormonal changes illustrate the tightly regulated feedback processes to guarantee stable intrauterine conditions. Any disruption in these pathways, especially under the influence of thyroid disease, may disrupt reproductive hormone balance and potentially impact pregnancy outcomes.[8]

FSH, LH, and TSH form part of the glycoprotein hormone family, and human chorionic gonadotropin (hCG) is another member of the same family. All of these hormones have a common structural basis in terms of having an identical alpha (α) subunit but with a unique beta (β) subunit, which confers receptor specificity and functional identity. Not only does this structural homology account for some of the physiological overlap but also for cross-reactivity at the receptor level, especially between LH and hCG. The structural similarity enables hCG to bind to the LH receptors, generating similar biological responses and affecting subsequent hormonal equilibrium.[10]

Additionally, thyroid hormones per se can influence gonadotropin secretion by modifying responsiveness of hypothalamus or pituitary. For example, disrupted thyroid hormone levels can inhibit or stimulate the release of GnRH, thus affecting FSH and LH discharge. This complex network of interactions illustrates that the thyroid and reproductive axes are not independent but tend to operate in an adaptive and concerted fashion. Appreciation of this interconnectedness offers a biochemical basis for investigation into how dysregulation in one hormonal pathway can transmit disturbances to others, an imperative phenomenon especially during the dynamic condition of pregnancy.[11]

The present study is thus aimed at examining the interrelationships of FSH, LH, and TSH levels in pregnancy, gaining knowledge on the physiological harmony of the pituitary–thyroid–gonadal axis during pregnancy. Through understanding how changes in one hormonal system can influence others, the study hopes to improve knowledge of maternal endocrine adaptation and discover markers for early identification of hormonal imbalance. The results could lead to enhanced screening, diagnosis, and management of endocrine disease in pregnancy and ultimately improved maternal and fetal health.

1. Physiological Adaptations of the Endocrine System

During Pregnancy is a time of dramatic physiological change during which almost all endocrine glands modify their function to accommodate fetal growth and maternal health. The endocrine system orchestrates a sequence of tightly regulated hormonal transitions that lead to implantation, gestational maintenance, and parturitional and lactational preparation. The changes involve not just enhanced secretion of reproductive hormones but also complex interactions among the pituitary, thyroid, adrenal, and placental systems.[12]

Early gestation also sees the modification of the hypothalamic–pituitary–gonadal (HPG) axis. The placenta, which increasingly takes on the function of an endocrine organ, produces vast quantities of human chorionic gonadotropin (hCG), progesterone, and estrogen. These hormones support the corpus luteum, stimulate endometrial growth, and prevent further ovulation by inhibiting pituitary FSH and LH release via negative feedback. The elevation of circulating estrogen also facilitates hepatic production of several binding proteins, such as thyroxine-binding globulin (TBG), which changes maternal circulating total levels of thyroid hormones.[13]

Meanwhile, the hypothalamic–pituitary–thyroid (HPT) axis readjusts to accommodate the increased metabolic needs of pregnancy. Maternal thyroid hormones are critical for fetal brain growth, thermogenesis, and global metabolic rate. During early pregnancy, elevated hCG levels mildly stimulate thyroid hormone secretion, resulting in a transient decrease in TSH concentrations. Throughout gestation, the endocrine system develops a new balance that maintains both maternal and fetal requirements.

These coordinated hormonal adjustments provide the best possible conditions for fetal development and maternal physiological homeostasis. Disruption in this delicately interlinked networkby thyroid abnormality, pituitary regulation, or nutritional insufficiencycan upset reproductive and metabolic homeostasis. Such upsets can lead to complications such as miscarriage, premature delivery, or fetal dysfunction.[14]

2. Structural Features of FSH, LH, and TSH

Follicle-stimulating hormone (FSH), luteinizing hormone (LH), and thyroid-stimulating hormone (TSH) are members of a family of glycoprotein hormones produced and released by the anterior pituitary gland. Albeit having different physiological targets, these hormones exhibit great structural similarities that underlie their biochemical and functional interrelationships. All of them consist of two noncovalently associated subunits: a common alpha (α) subunit and a specific beta

(β) subunit. The α-subunit is the same for all three of these hormones, comprising 92 amino acids, whereas the β-subunit bestows biological specificity by dictating receptor binding and biological activity.[15]

The common α-subunit forms a structural basis for partial receptor cross-reactivity among members of this hormone family. For example, the β-subunit of human chorionic gonadotropin (hCG) is very similar to that of LH, enabling hCG to bind and stimulate LH receptors, a mechanism especially important in early pregnancy. Analogously, structural similarity between TSH and hCG accounts for the weak thyroid-stimulating activity of hCG during the first trimester, causing transient stimulation of thyroid hormone production and a temporary drop in TSH level.[16,17]

3. Mechanisms of Hormonal Regulation Through the Hypothalamic–Pituitary Axis

The hypothalamic–pituitary axis is the primary regulatory center for endocrine coordination during pregnancy, integrating neural and hormonal input to ensure homeostasis. The axis works through a feedback loop involving the hypothalamus, anterior pituitary gland, and peripheral endocrine organs such as the ovaries and thyroid. [18]

Throughout pregnancy, this control system is adjusted to permit the synthesis of placental hormones. High levels of estrogen, progesterone, and hCG give powerful negative feedback to GnRH to cause a physiological suppression of FSH and LH release. This is followed by partial stimulation of hCG's TSH receptors, which changes the axis of the thyroid and results in transient changes in TSH and thyroid hormone release. These interactions show how the hypothalamic–pituitary axis acts as an interactive communication network, which is able to modify its hormonal output according to gestational needs.[19]

Table 1. Hormonal changes during pregnancy

Hormone	Physiological Role	Trend During Pregnancy	Reason for Change	Clinical Significance
FSH (Follicle-Stimulating Hormone)	Stimulates follicular growth and estrogen secretion before ovulation	Decreases markedly after conception	Negative feedback from high estrogen, progesterone, and placental hormones	Low levels prevent new follicular development during pregnancy
LH (Luteinizing Hormone)	Triggers ovulation and corpus luteum formation	Suppressed throughout pregnancy	Inhibited by high circulating sex steroid and placental hormone levels	Maintains quiescence of ovarian cycle during gestation
TSH (Thyroid-Stimulating Hormone)	Stimulates thyroid gland to produce T3 and T4	Slightly decreases in first trimester, normalizes or slightly rises later	Early suppression due to hCG cross-reactivity; later regulation by feedback	Reflects thyroid adaptation; imbalance may affect fetal growth
hCG (Human Chorionic Gonadotropin)	Supports corpus luteum and stimulates thyroid	Peaks in first trimester, then declines	Produced by placenta	Helps maintain pregnancy and influences TSH levels indirectly

4. Trimester-Wise Changes in FSH, LH, and TSH During Pregnancy

The endocrine profile of the pregnant woman changes dynamically throughout the three trimesters as maternal physiology responds to the advancing demands of the growing fetus. Each trimester is characterized by unique patterns in the secretion of follicle-stimulating hormone (FSH), luteinizing hormone (LH), and thyroid-stimulating hormone (TSH), a pattern that mirrors the complex coordination of the pituitary–thyroid–gonadal axis and placental endocrine functions.

4.1. First Trimester

During early pregnancy, implantation of the ovum fertilized stimulates trophoblastic cells to secrete human chorionic gonadotropin (hCG) very rapidly. Due to its chemical resemblance to LH, hCG acts on LH receptors in the corpus luteum, sustaining progesterone secretion required for uterine quietness and endometrial maintenance. This external stimulus significantly inhibits pituitary LH secretion via negative feedback. Similarly, FSH has also decreased to a great extent because of high levels of estrogen and progesterone, which suppress hypothalamic GnRH secretion. At the same time, circulating hCG levels are very high and stimulate TSH receptors mildly, causing transient thyrotoxicosis and a consequent decrease in serum TSH levels.[20]

4.2. Second Trimester

As pregnancy advances, hCG levels reach a peak and thereafter decline steadily, decreasing its stimulating impact on the thyroid. Thus, TSH levels start to normalize or modestly rise, while thyroid hormone levels settle to ensure maternal and fetal metabolic requirements. FSH and LH continue to remain profoundly suppressed during this period since ovarian follicular activity is no longer needed. The placenta takes over as the sole producer of steroid hormones, providing a constant level of estrogen and progesterone.[21]

4.3. Third Trimester

During late pregnancy, maternal TSH can have mild elevation because thyroid hormones are needed in greater quantity to sustain fetal development and prepare for childbirth. FSH and LH levels, though, remain lowest, as they continue to be suppressed by steroid hormones of the placenta. The hormonal milieu during this time focuses on uterine enlargement, placental activity, and lactational preparation rather than reproductive cycling.[22]

The trimester-specific hormonal changes show the way maternal endocrine system slowly shifts from a reproductive to a supportive mode. The changes are effectively summarized with quantitative analysis of data to assess correlations between FSH, LH, and TSH.

5. Human Chorionic Gonadotropin (hCG) Role in Modulating Secretion of FSH, LH, and TSH

Human chorionic gonadotropin (hCG) is one of the first and most potent pregnancy hormones. Produced by the syncytiotrophoblast shortly after implantation, it is a key regulatory substance, acting on several endocrine pathwaysmost notably FSH, LH, and TSH. Structurally, hCG is a glycoprotein that consists of two subunits, alpha (α) and beta (β). The α-subunit is almost identical to that of LH, FSH, and TSH, but the β-subunit imparts biological specificity. This structural homology explains why hCG can cross-react with LH and TSH receptors, influencing their activity during pregnancy.

5.1. Interaction with LH:

Due to its molecular homology, hCG may also bind to LH receptors on the corpus luteum, supporting progesterone release throughout early pregnancy. This is a compensatory process that

takes over from pituitary LH requirement, resulting in its strong suppression by negative feedback. The persistence of hCG guarantees corpus luteum longevity until the placenta takes over as the primary progesterone source towards the end of the first trimester.[23]

5.2. Impact on FSH:

The high levels of progesterone and estrogen, which are sustained by hCG-stimulated luteal function, inhibit the release of gonadotropin-releasing hormone (GnRH) in the hypothalamus. FSH secretion is thus blocked, prohibiting the development of new follicles and arresting ovulation. Inhibition guarantees that the ovarian cycle is kept arrested during pregnancy, preserving physiological resources for fetal growth.[24]

5.3. Stimulation of TSH Receptors:

An interesting property of hCG is its partial agonist activity on the thyroid. In early pregnancy, elevated hCG levels cause stimulation of TSH receptors owing to similar structural motifs, with consequent transient elevations in levels of thyroxine (T4) and triiodothyronine (T3). This relative hyperthyroidism inhibits pituitary secretion of TSH by negative feedback. As the hCG level decreases after the first trimester, TSH levels slowly come into line with maternal metabolic needs.

5.4. Physiological Significance:

Through these intersecting mechanisms, hCG functions as the master coordinator of maternal hormonal homeostasis. It suppresses gonadotropins (FSH, LH) and briefly regulates thyroid function (TSH), providing an ideal internal setting for fetal development and placental operation. The hormonal dynamics also demonstrate how structural similarities between glycoprotein hormones are responsible for functional cross-regulation.[25]

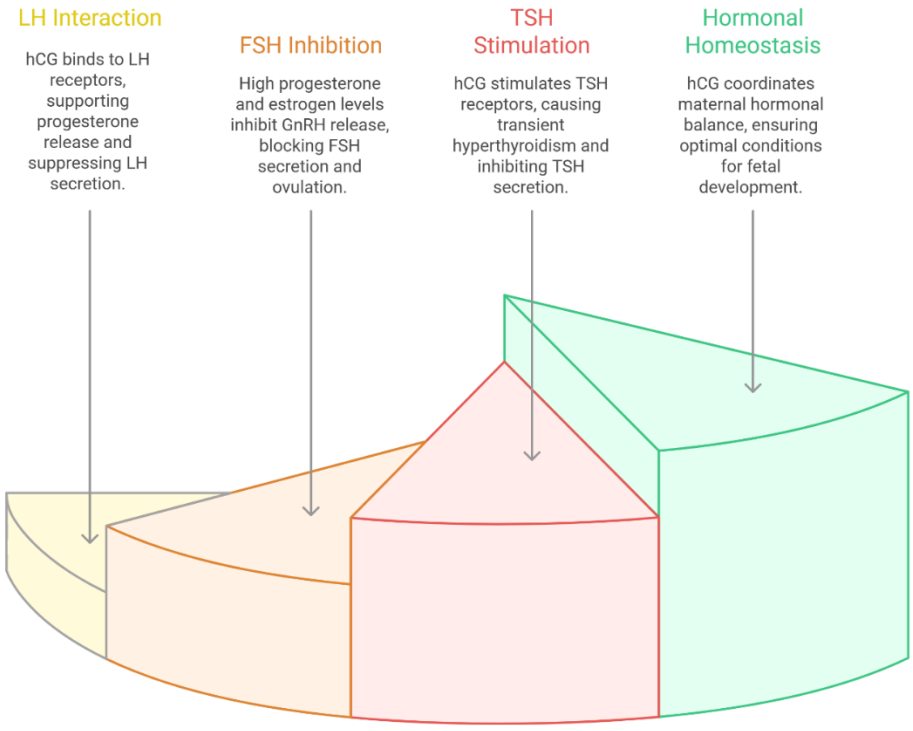

Figure 1. hCGs role in pregnancy hormonal regulation

6. Thyroid Function and TSH Dynamics During Pregnancy

Pregnancy places considerable physiological stress on the mother's thyroid gland, necessitating adaptive responses to accommodate the enhanced metabolic demands of the mother and fetus. The thyroid–pituitary axis is dramatically modulated, with mostly hormonal and metabolic changes that are gestation-specific. An understanding of TSH dynamics in this setting is crucial for the interpretation of thyroid function in pregnancy and for the detection of possible pathological deviations like hypothyroidism or hyperthyroidism.[26,27]

6.1. Regulation of TSH Levels:

Early in pregnancy, circulating hCG in high concentrations is a weak agonist at TSH receptors, replicating the effect of TSH itself. This leads to a transient fall in the level of serum TSH in the first trimester, usually below the nonpregnant range. As hCG levels fall after 10–12 weeks, TSH levels steadily return toward normal. During subsequent trimesters, TSH can moderately rise as a function of increasing need for thyroid hormones but usually stays within the pregnancy-related reference range (roughly 0.3–3.0 mIU/L).

6.2. Interaction with FSH and LH:

While TSH, FSH, and LH both derive from the anterior pituitary gland and exhibit structural homology in their alpha subunit, their patterns of secretion diverge dramatically throughout pregnancy. Whereas FSH and LH are dramatically inhibited by placental steroid feedback, TSH remains dynamically variable under the opposing influences of hCG and thyroid hormones. This distinction highlights the selective regulatory processes governing each endocrine axis.[28]

6.3. Clinical Implications:

Abnormalities of thyroid controlmaternal hypothyroidism or subclinical hypothyroidismmay have severe consequences, including miscarriage, preterm delivery, and compromised neurocognitive development in the child. Raised TSH in pregnancy usually signifies inadequate production of thyroid hormones, and careful measurement of free T4 and thyroid autoantibodies is required. In contrast, reduced TSH with raised free T4 indicates gestational thyrotoxicosis, which is usually reversible and self-limiting.[29]

7. Mechanisms of FSH and LH Suppression During Pregnancy

The inhibition of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) during pregnancy is a feature of reproductive endocrine adaptation. It is an indication of the complete diversion of the maternal hypothalamic–pituitary–gonadal (HPG) axis away from cyclical reproductive function and towards hormonal quiescence conducive to gestation. Multiple related mechanisms hormonal, feedback-regulated, and placental are involved in this ongoing inhibition.

7.1. Hypothalamic Regulation of GnRH Secretion

Both gonadotropins FSH and LH are controlled by gonadotropin-releasing hormone (GnRH) of the hypothalamus. In pregnancy, rising levels of progesterone, estrogen, and inhibin suppress GnRH pulsatility. Downregulation limits pituitary stimulation and thereby reduces the production and discharge of FSH and LH.

7.2. Steroid Hormone Negative Feedback

High levels of placental estrogen and progesterone suppress gonadotropin secretion through direct action on the pituitary and hypothalamus. Estrogen, and particularly estriol produced by the

fetoplacental unit, suppresses the release of FSH, while progesterone suppresses LH surges that otherwise trigger ovulation.

Table 2. FSH, LH, and TSH during pregnancy

Axis Involved	Hormones Interacting	Nature of Relationship	Physiological Mechanism	Outcome / Effect in Pregnancy
Hypothalamic–Pituitary–Gonadal (HPG) Axis	FSH ↔ LH	Direct regulatory link; both secreted from anterior pituitary	GnRH (gonadotropin-releasing hormone) stimulates FSH & LH release; feedback controlled by estrogen and progesterone	Suppression of both hormones prevents ovulation and new follicle formation during pregnancy
Hypothalamic–Pituitary–Thyroid (HPT) Axis	TSH ↔ Thyroid hormones (T3/T4)	Inverse relationship via feedback mechanism	Increased thyroid hormones inhibit TSH secretion	Maintains metabolic homeostasis for maternal and fetal growth
Cross-Talk Between Axes	TSH ↔ FSH/LH	Indirect inverse correlation	hCG can mimic TSH activity, leading to mild thyroid stimulation and suppression of gonadotropins	Ensures hormonal stability by preventing excess ovarian stimulation
Placental Influence	hCG ↔ TSH, FSH, LH	Functional overlap between hCG and pituitary hormones	hCG binds to LH receptors and partially stimulates thyroid activity	Maintains corpus luteum, supports progesterone production, and modulates pituitary output
Feedback Integration	Estrogen/Progesterone ↔ TSH, FSH, LH	Negative feedback on hypothalamic centers	High steroid levels inhibit GnRH release; influence TSH through metabolic pathways	Coordinates reproductive quiescence and metabolic adaptation

7.3. Inhibin and Activin role

Inhibin, secreted from corpus luteum and placenta, provides a second component of FSH inhibition by selective inhibition of release from the pituitary with no effect on LH. Activin levels fall in pregnancy, so does not contribute further to gonadotropin inhibition.

7.4. Substitution of LH by hCG

As already discussed, hCG structurally mimics LH and maintains luteal function during early pregnancy. Its continuous secretion eliminates the physiological need for pituitary LH. Not only is LH secretion thus suppressed, but it is also functionally redundant for most of pregnancy.

7.5. Placental Control of Endocrine Feedback

After the placenta assumes steroidogenesis from the 10th week of gestation, pituitary involvement in ovarian regulation is minimal. The placenta's high production of estrogens and progesterone keeps the HPG axis subjected to sustained negative feedback inhibition until the onset of parturition.[30]

8. Interrelationship Between Thyroid and Reproductive Hormones

The relationship between thyroid function and reproductive hormones is perhaps the most complex aspect of endocrine physiology. The thyroid–pituitary–gonadal axis functions through interrelated pathways wherein changes in one system can impact the regulation of another. Such interaction becomes of particular importance during pregnancy, as the maternal metabolism finds balance between metabolic and reproductive needs to support both mother and fetus.[31]

8.1. Common Glycoprotein Structure and Receptor Cross-Reactivity

FSH, LH, and TSH are glycoprotein hormones having a common alpha (α) subunit with unique beta (β) subunits responsible for receptor specificity. Because of this structural homology, there can be partial cross-reactivity. For example, high hCG or TSH can weakly stimulate LH receptors, affecting ovarian steroidogenesis. Likewise, hCG's thyrotropic action in early pregnancy temporarily inhibits TSH, connecting reproductive and thyroid axes via common receptor mechanisms.[32]

8.2. Regulation of Thyroid Hormones and Gonadotropins

Thyroid hormones (T3, T4) have direct action on gonadotropin production by regulating hypothalamic and pituitary sensitivity. Hypersecretion of thyrotropin-releasing hormone (TRH) in hypothyroidism raises the level of prolactin, which prevents GnRH release and decreases FSH and LH production. In hyperthyroidism, FSH and LH levels are decreased due to increased metabolic clearance and inhibition through feedback.

8.3. Impact on Ovarian Function and Fertility

Alterations in thyroid hormone levelseither excess or deficiencymay interfere with ovarian function. Inadequate thyroid hormone availability might impede follicular development and luteal function and result in anovulation or luteal phase abnormalities. In contrast, thyrotoxicosis might change estradiol metabolism so that cycles become irregular or the woman is infertile. Such disruptions during pregnancy are overridden by the prevailing effect of hCG and placental steroids but, if severe, may still influence pregnancy outcome.[33]

8.4. Pregnancy Feedback Coordination

During pregnancy, thyroid hormones and gonadotropins both coexist in a delicate balance regulated by the placenta. High estrogen stimulates hepatic production of thyroxine-binding globulin (TBG), thus increasing total T4 and T3 levels. This adjustment ensures adequate free thyroid hormone levels while inhibiting TSH to gestational levels. Suppression of FSH and LH guarantees reproductive dormancy, while thyroid function is stabilized for fetal neurodevelopment.[34]

8.5. Clinical Implications of Thyroid–Gonadal

Even minor thyroid disturbances can affect reproductive well-being. Subclinical hypothyroidism has been linked to decreased fertility, increased miscarriage rates, and complicating conditions like preeclampsia. On the other hand, untreated hyperthyroidism can result in preterm birth or low birth weight.[35] These interrelations between hormones form the basis for the early detection and treatment of thyroid dysfunctions among pregnant women.

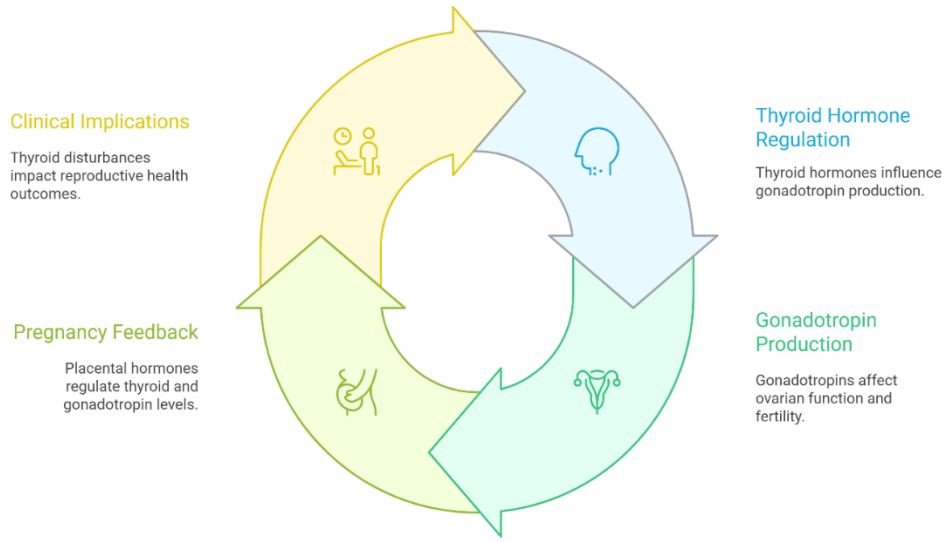

Figure 2. Thyroid reproductive hormone cycle

9. Clinical Correlations and Endocrine Disorders with Disturbed FSH, LH, and TSH Levels in Pregnancy

The endocrine balance between FSH, LH, and TSH is essential for maintaining a normal pregnancy. When the balance is disturbedthrough thyroid disease, pituitary disorder, or inappropriate gonadotropin regulationthe repercussions may reach both maternal and fetal health. Comprehension of these correlations links fundamental endocrine physiology with clinical obstetrics.

Maternal hypothyroidism, defined by an increased TSH and decreased free T4, is one of the most frequent endocrine diseases seen in pregnancy. It can be secondary to autoimmune thyroiditis, iodine deficiency, or underlying thyroid disease. The resultant increase in TRH can enhance prolactin release, causing decreased GnRH release and inhibition of FSH and LH. Clinically, it can present as infertility prior to conception and complications like miscarriage, anemia, and neurodevelopmental impairment in the fetus.[36,37]

10. Clinical Diagnostic Implications

Correct interpretation of the levels of FSH, LH, and TSH during pregnancy is based on trimester-specific reference intervals and an understanding of physiological alterations. Precise measurement of hormone levels is crucial for research on interrelationship between FSH, LH, and TSH during pregnancy. Analytical accuracy and correct interpretation of results are based on employing validated laboratory methods, optimal timing of sampling, and pregnancy-related reference ranges. Familiarity with these diagnostic procedures assures comparability and reliability of data in research as well as in the clinical setting.[38] Hormonal findings need to be interpreted using trimester-specific reference ranges. TSH, for example, is normally between 0.1–2.5 mIU/L in the first, 0.2–3.0 mIU/L in the second, and 0.3–3.5 mIU/L in the third trimester ([Stagnaro-Green et al., 2017]). Levels of FSH and LH are profoundly suppressed during pregnancy and tend to drop below the detection limits of most assays.[40]

Statistical techniques like correlation and regression analysis become crucial from a research point of view to compare associations between FSH, LH, and TSH. Tools like SPSS or R can be utilized to compare trimester-wise variations and establish the statistical significance of trends observed. Interpretation has to take into account biological variability as well as precision of the assay to provide meaningful conclusions.

7.1: CONCLUSION(S)

The interrelationship between Follicle-Stimulating Hormone (FSH), Luteinizing Hormone (LH), and Thyroid-Stimulating Hormone (TSH) during pregnancy demonstrates the intricate coordination between the hypothalamic–pituitary–thyroid and gonadal axes essential for maintaining maternal endocrine stability and supporting fetal development. This review concludes that hormonal adjustments during pregnancyparticularly the suppression of FSH and LH alongside fluctuating TSH levelsreflect adaptive physiological changes crucial for successful gestation. However, even mild thyroid dysfunction can influence gonadotropin regulation, leading to potential reproductive complications. Despite growing global research, limited data from local populations highlight the need for further studies to establish trimester-specific reference ranges and clarify hormonal interactions. A deeper understanding of these relationships will enhance maternal health assessment, allowing for early identification and management of endocrine imbalances to promote healthier pregnancy outcomes.

References

1. Glinoer D. The regulation of thyroid function in pregnancy: Pathways of endocrine adaptation from physiology to pathology. *Endocr Rev*. 1997;18(3):404-33.
2. Brown EDL, Obeng-Gyasi B, Hall JE, Shekhar S. The Thyroid Hormone Axis and Female Reproduction. *Int J Mol Sci*. 2023;24(12):9815.
3. Thyroid hormone dysfunction during pregnancy: A review. *PMC*. 2016.
4. Thyrotropic action of human chorionic gonadotropin. *J Clin Endocrinol Metab*. 1995;?? [specific volume/pages].
5. The application of new concepts of the assessment of the thyroid in pregnancy. *Front Endocrinology*. 2022.
6. Regulation of Thyroid Function in Pregnancy. *Endocrine Reviews / Oxford Academic*. 1997.
7. Understanding the pathogenesis of gestational hypothyroidism. *Front Endocrinology*. 2021;12:653407.
8. Thyroid-Stimulating Hormone Receptor Antibodies in Pregnancy. *PMC*. 2017;17:137.
9. Thyroid physiology and common diseases in pregnancy. *PMC*. 2012;3530964.
10. Management of Thyroid Disorders in Pregnancy. *BJOG*. 2025.

11. Glinoer D. The regulation of thyroid function in pregnancy: pathways of endocrine adaptation from physiology to pathology. *Endocr Rev*. 1997;18(3):404–33.
12. Alexander EK, Pearce EN, Brent GA, Brown RS, Chen H, Dosiou C, et al. 2017 Guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and the postpartum. *Thyroid*. 2017;27(3):315–89.
13. De Groot L, Abalovich M, Alexander EK, Amino N, Barbour L, Cobin RH, et al. Management of thyroid dysfunction during pregnancy and postpartum: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2012;97(8):2543–65.
14. Ren B, Li W, Li H. A new perspective on thyroid hormones: crosstalk with glycoprotein hormones and implications for reproduction. *Int J Mol Sci*. 2022;23(5):2708.
15. Napso T, Yong HEJ, Lopez-Tello J, Sferruzzi-Perri AN. The role of placental hormones in mediating maternal adaptations to support pregnancy and lactation. *Front Endocrinol (Lausanne)*. 2018;9:109.
16. Yoshimura M. Thyrotropic action of human chorionic gonadotropin. *J Clin Endocrinol Metab*. 1995;80(6):2326–9.
17. StatPearls. Pituitary issues during pregnancy. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023. (chapter on pituitary physiology in pregnancy).
18. Chivukula KK, Nanjundan S, Bhandari S. Thyroid storm as an early presentation of hCG-producing trophoblastic disease: case and review. *Case Rep Endocrinol*. 2021.
19. Glinoer D. Regulation of thyroid function in pregnancy. *Endocr Rev*. 1997;18(3):404–33. (classic physiology reviewcited for hCG–TSH relationship).
20. Krassas GE, Poppe K, Glinoer D. Thyroid function and human reproductive health. *Endocr Rev*. 2010;31(5):702–55.
21. Poppe K, Velkeniers B. Thyroid disease and female reproduction. *Clin Endocrinol (Oxf)*. 2010;72(1):1–12.
22. De Groot L. Treatment of thyroid disease during pregnancy and postpartum. *J Clin Endocrinol Metab*. 2012;97(8):2543–65.
23. Cole LA. Biological functions of hCG and hCG-related molecules. *Reprod Biol Endocrinol*. 2010;8:102.
24. Walkington L, et al. Hyperthyroidism and human chorionic gonadotrophin: clinical implications. *Br J Cancer*. 2011;104(2):...
25. Huang N, et al. Impact of TSH levels after controlled ovarian hyperstimulation on reproductive outcomes. *Front Endocrinol (Lausanne)*. 2023;14:1159991.
26. Zhou D, et al. Relationship between TSH levels and clinical pregnancy outcomes: a cohort study. *J Endocrinol Invest*. 2022;45(4):...
27. Puthiyachirakal M, et al. Overview of thyroid disorders in pregnancy. *Clin Obstet Gynecol Reprod Med*. 2025; (review).
28. Hershman JM. Differences between thyroid stimulators in hydatidiform mole and pregnancy. *J Clin Endocrinol Metab*. 1970;30(1):...
29. Qu J. Advances in the study of inhibin, activin and follistatin in pregnancy and placental biology. *Placenta*. 1998;19(5):...
30. Zhang Y, et al. TSH levels after fresh embryo transfer are associated with clinical pregnancy and live birth rates. *Sci Rep*. 2023;13:36276.
31. Rohayem J, et al. Retrospective clinicopathological study: hCG and thyroid receptor interactions. *Endocr Connect*. 2025;14(1):EC-24-0341.
32. Silva JF, et al. Thyroid hormone levels and their relationship with human chorionic gonadotropin in pregnancy. *Sci Res J*. 2016;X:X–X.

33. Stagnaro-Green A, et al. Guidelines for the diagnosis and management of thyroid disease during pregnancy and the postpartum. *Thyroid*. 2011;21(10):1081–112. (earlier guideline papers and summaries).
34. Krassas GE. The effect of thyroid dysfunction on fertility and pregnancy. *Hum Reprod Update*. 2000;6(2):137–48.
35. Dittrich R, et al. Thyroid hormone receptors and reproduction: mechanisms and clinical implications. *Mol Cell Endocrinol*. 2011;332(1-2):22–8.
36. Akhtar S, et al. Thyroid function and pregnancy outcomes in Pakistani women: a cross-sectional study. *Pak J Med Sci*. 2020;36(4):...
37. Almeida M, et al. Maternal thyroid function and fetal outcomes: evidence from cohort studies. *J Clin Endocrinol Metab*. 2019;104(6):...
38. Kumar N, et al. Endocrine changes across trimesters: gonadotropin and thyroid hormone patterns. *Int J Gynecol Obstet*. 2021;152(1):...
39. Napso T, et al. Placental hormones and maternal adaptations: roles of hCG and placental peptides. *Placenta*. 2018;70:...
40. Melmed S, et al. Pituitary disorders and endocrine adaptation in pregnancy. *Lancet Diabetes Endocrinol*. 2022;10(5)