

The Association of Thyroid Disorders with Infertility and Polycystic Ovary Syndrome

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ABSTRACT: Fertility is the inherent capacity of a couple to carry a child via a complicated biological mechanism. The release of thyroid hormones, which are essential to development, growth, and metabolism, is required for the efficacy of this process. A study of the literature concerning thyroid control of female fertility and polycystic ovarian syndrome (PCOS) was conducted utilizing several search engines. Numerous studies have demonstrated significant correlations among thyroid function, female fertility, and polycystic ovarian syndrome (PCOS). Hypothyroidism and hyperthyroidism are thyroid diseases frequently associated with infertility. Research has established a significant correlation between normal thyroid function and reproductive capability by observing the start and resolution of infertility symptoms in relation to thyroid dysfunction and the normalization of circulating T3 and T4 levels. Identifying the underlying processes may facilitate early prevention strategies, precise infertility diagnosis, and the efficacy of treatment interventions for aiding infertile women in conception. PCOS is the predominant metabolic and endocrine disorder in women who are who are fertile, leading to metabolic abnormalities and infertility. PCOS is a heterogeneous clinical defect with an ambiguous etiology, complicating its treatment. The clinical signs of PCOS resemble those of several thyroid disorders. Moreover, many thyroid problems, including subclinical hypothyroidism, not only elevate the prevalence of PCOS but also make its symptoms worse. However, it likewise intensifies related metabolic and reproductive complications.



Keywords: Fertility, Hypothyroidism, Hyperthyroidism, Polycystic ovarian syndrome, Thyroid hormones

1. INTRODUCTION

Thyroid hormones (THs) are essential to both human and animal reproduction. Through certain nuclear receptors, L-thyroxine (T4) and L-triiodothyronine (T3) impact uterine, placental, ovarian tissues, modifying their growth and metabolism [1]. It has been shown that thyroid diseases are frequently encountered. About 4.6% to 9.5% of people have hypothyroidism, and 1.3% to 2.2% of people are at risk for hyperthyroidism. While thyroid cancer can be detected in over 13% of patients, multinodular goiter is a nontumorous disease [2].

It is one of the conditions associated with widespread endocrinopathies for women of reproductive age. Thyroid diseases impact around 14% of adult women [3]. With women being three to five times more likely to need treatment for thyroid diseases compared to men [4].

Hypothyroidism and hyperthyroidism are diseases of the thyroid gland that affect the secretion of thyroxine and triiodothyronine, sometimes leading to infertility. Research has demonstrated this by the observation of the active form and a bound inactive form [5]. Being infertile is described as the failure to become pregnant after a minimum of about 15% of couples experience 12 months of unprotected intercourse. And is especially prevalent in developing nations. [6, 7] Infertility rates for women with thyroid disorders, autoimmune diseases such as Hashimoto's thyroiditis and Graves',

can exceed fifty percent. They have an increased chance of developing early ovarian failure additionally, they are more likely to experience early ovarian failure [8]. A strong correlation is present between female fertility and thyroid function; physically, pregnancy significantly influences the thyroid gland, and thyroid disease has been historically linked to impaired fertility in women [9]. In women, menstruation and ovulation vary due to changes in the release of thyrotropin releasing hormone (TRH) from the pituitary gland, which is linked to the activity of the thyroid gland and ovaries [10]. Thyroid hormones (THs) consider an important role in enhancing the expression of ovarian matrix metalloproteinases, key enzymes that facilitate the breakdown of the extracellular matrix. This process is essential for the timely release of oocytes and the formation of the corpus luteum, both of which are vital for successful reproduction [4]. Subclinical hypothyroidism is associated with the company of PCOS, which is considered as a reproductive condition described by ovulatory failure and androgen excess [11]. Subclinical hypothyroidism in women with PCOS is linked to other health problems such as dyslipidemia and insulin resistance [12]. (TG) Thyroglobulin and (TPO) Thyroid peroxidase antibodies are two examples of specific antibodies that are present in Hashimoto's thyroiditis (HT), an autoimmune disease. In response to these antibodies, an autoimmune reaction causes lymphocyte infiltration, especially involving T cells. Over time, the thyroid gland gradually deteriorates and becomes fibrotic as a result of this immune-mediated process [13]. Consequently, the manufacture of triiodothyronine and thyroxine, which are markers for hypothyroidism, decreases, and thyroid-stimulating hormone (TSH) levels increase [14]. One autoimmune disorder is Graves' disease (GD), which leads to hyperthyroidism [15]. Thyroid conditions have a complex impact on female reproduction, affecting multiple aspects of the women's reproductive axis both directly and indirectly [16]. Thyrotropin is another name for TSH, that is synthesized by the pituitary gland in the brain by releasing TRH (thyrotropin-releasing hormone) [17].

The excretion of kisspeptin and GnRH (gonadotropin releasing hormone) is influenced by thyroid hormones (THs) at the hypothalamic-pituitary level, which is affected directly by metabolic indicators like prolactin and leptin [18, 19]. Elevated TRH secretion indirectly affects GnRH secretion, similar to TRH, is a powerful stimulant of prolactin. Prolactin is recognized for its function in mammary growth and lactation. However, GnRH secretion from the brain [4].

Normal sexual function requires optimal thyroid function via specific nuclear receptors, which control the metabolism of these tissues and growth. Thyroid hormones directly affect the ovaries, testes, and corpora cavernosa, as shown in Figure 1 below [20].

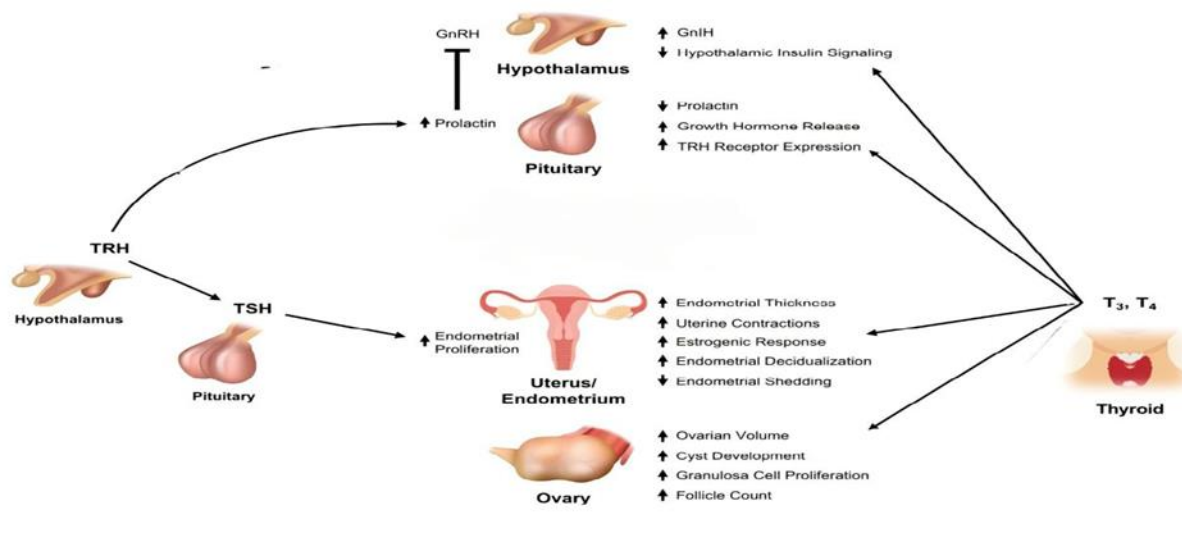


FIGURE 1. - Results of disorders in the secretion of thyroid hormones on the functions of the tissues [4]

Primarily, thyroid hormones serve as key in regulating various processes. Additionally, hereditary factors, insulin resistance, and glucose metabolism also contribute significantly [21].

(THs) Thyroid hormones influence the biological availability of sexual hormones by intensifying the levels of HNF4 α (hepatic nuclear factor), that subsequently enhances the transcription of SHBG (sex hormone binding globulin), resulting in increased SHBG concentrations [22]. Because of SHBG's greater affinity for testosterone compared to estradiol, thyroid failure may result in an imbalance of bioavailable sex hormone concentrations, potentially causing failure of ovulation and erection dysfunction. Conversely, treatment of estrogen works for elevated levels of thyroxine binding globulin [23]. Epidemiologically, hypothyroidism and hyperthyroidism are linked to alterations in sex hormone concentrations such as testosterone, SHBG, and estradiol in both genders, as well as menstrual abnormalities,

menarche, and menopause in females [24, 25]. TSH is believed to assume a major role in various pathways contributing to the pathogenesis and clinical evolution of PCOS [26]. The intricate relationship between thyroid function and female reproduction warrants a thorough examination to identify the subtleties influencing the clinical therapy of thyroid and fertility-related problems [27, 28].

2. THE IMPORTANCE OF THE PITUITARY GLAND IN THE PHYSIOLOGY OF THE FEMALE BODY

The hypothalamic pituitary thyroid (HPT) axis has a multilayered system to balance metabolic processes, where it releases thyrotropin-releasing hormone (TRH) [29].

To stimulate the release of TSH, resulting in stimulates thyroid cells, the pituitary gland, specifically its anterior lobe, releases TRH. TSH then signals the thyroid gland to release T4 and T3. Highly specialized plasma proteins, for instance thyroxine-binding globulin (TBG) and transuterine, transport these thyroid hormones, along with albumin [30]. The thyroid gland regulates the body's physiological functions by converting T4 to T3, which is then converted to FT3. These hormones influence thyroid activity by inhibiting the secretion of TRH and TSH throughout a negative feedback mechanism, as illustrated in the figure [4].

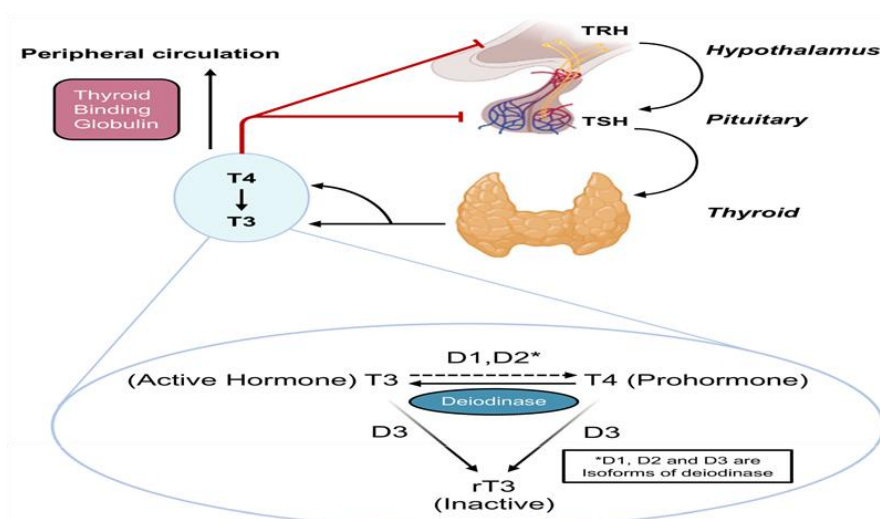


FIGURE 2. - The pituitary-thyroid axis in the hypothalamus. Including the interactions of TRH, TSH, T3, and T4 along the hypothalamus pituitary thyroid axis [31]

The graphic depicts the correlation between thyrotropin releasing hormone "TRH" and thyroid stimulating hormone "TSH", which activates the thyroid gland [31].

Gonadotropin-releasing hormone (GnRH) increases the anterior pituitary gland's gonadotropic cells secrete luteinizing hormone (LH) and follicle-stimulating hormone (FSH). by acting on the exterior layer and granulosa cells of the ovary. This promotes the synthesis of hormones and reproductive peptides, along with the development of ovarian follicles [32]. Factors that can prevent ovulation include defects in the secretion of FSH, sex hormone-binding globulin (SHBG), and imbalances in androgen and estrogen levels [33].

One of the causes of irregular menstruation in women is a thyroid hormone imbalance [34]. When there is hypothyroidism, there is an rise in prolactin secretion and, consequently, an elevate in dopamine, which works to reduce prolactin in the body and thus inhibits GnRH secretion [35].

Ultimately, prolactin affects the corpus luteum, stimulating it to release progesterone, which helps maintain the corpus luteum for as long as possible [36].

Alterations in ovulation and menstruation are primarily caused by abnormal thyroid levels. An imbalance in androgens, estrogen, follicle-stimulating hormone (FSH), and sex hormone-binding globulin (SHBG) may obstruct ovulation [33].

3. THYROID HORMONES (TH) AND THEIR EFFECTS ON THE UTERINE TUBE WITH UTERUS

Thyroid hormones (TH) control the uterine tubes' and uterus's susceptibility to estrogen by interacting with intracellular receptors [37]. The significance of thyroid hormones goes beyond just regulating metabolism; it also plays a vital function in fertility wellness. Both thyroid hormone receptors and thyroid-stimulating hormone receptors are

present in the ovaries and uterus. Additionally, these receptors exhibit high expression levels in the maternal-fetal unit during the implantation of a fertilized egg [38]. Progesterone levels are negatively correlated with deiodinase expression, which decreases throughout the secretory phase, while the uterine epithelium's T3 with T4 receptor declaration peaks during the mid-secretory phase [39].

Changes in serum concentrations of T3 and T4 affect the uterus, fallopian tubes, and reproductive system physiologically through their receptors become active throughout the menstrual period, which is achieved by changing the levels of sex steroids in the plasma [40]. For the ovaries to function normally and for eggs to mature properly, there must be a balance between thyroid hormones, reproductive hormones, and the hormones progesterone and estrogen [40]. Thyroid and TSH receptors are distinctly and abundantly articulated during the crucial phase of implantation within the fetomaternal unit, as well as in the ovary and uterus, highlighting their vital role in reproductive success [38]. Coordinated hormonal modulation of ovarian function is vital for the intricate processes of folliculogenesis and the production of gonadal steroids. The presence of thyroid hormone receptors in cumulus cells, human oocytes, and granulosa cells, combined with the detection of thyroid hormones (TH) in follicular fluid, strongly indicates that thyroid hormones hold a direct and significant influence over ovarian function [41].

Androgen overproduction is a hallmark of PCOS, significantly impacting hormonal balance. Additionally, TSH plays an essential role in ovarian function and follicular maturation, often contributing to ovulation dysfunction and the formation of cysts. To unravel the intricate molecular processes linking TSH to PCOS, further research is essential. Understanding these connections could illuminate new pathways for effective treatments and greater insights into this complex condition [42].

4. THE RELATIONSHIP BETWEEN INFERTILITY AND HYPERTHYROIDISM

1.3% of hyperthyroidism cases in women of childbearing age are caused by Graves' disease, which results from elevated antibodies to the thyroid-stimulating hormone receptor [1]. Women who have hyperthyroidism are at risk of infertility because thyroid hormone controls growth and metabolism of ovarian placental, and uterine tissues, the optimal operation of the female reproductive system [43].

Increased synthesis of (SHBG) affects ovulation and menstrual irregularity (heavy or absent menstrual periods). In rare cases, thyroid problems can lead to premature menopause. Changes in the menstrual cycle, predominately linked to triggered or compromised ovulation, are a type of fertility problem [39].

Hyperthyroidism is characterized by elevated levels of androstenedione, testosterone, total estradiol, and SHBG because of a high rate of testosterone to estradiol and androstenedione to estrone conversion. Despite having higher LH levels, the majority of hyperthyroid women continue to ovulate [44].

The most often seen menstrual alterations are polymenorrhea and oligomenorrhea (22% versus 8%) in comparison to euthyroid women. The incidence of hyperthyroidism among infertile women is 2.1%, whereas the high burden of infertility among adult females with hyperthyroidism scales from 5.8% to 50% [45].

Quinto-Moro et al. studied 193 women between the ages of 18 and 50 who had infertility diagnosed with Graves' disease. The findings indicated that infertility prevalence in Graves' disease was 52.3%. 36.5 years was the average age at diagnosis. Women diagnosed before the age of 35 had fewer pregnancies on average, and this decline continued after diagnosis [46].

The pathophysiological pathways connecting female infertility and hyperthyroidism involve an elevation in serum SHBG, which is a crucial factor leading to increased androgens, their conversion rate, and estradiol levels. The second factor contributing to infertility in women with Graves' illness is thyroid autoimmunity [47].

5. HYPOTHYROIDISM WITH INFERTILITY

Hypothyroidism is distinguished by abnormal levels of TSH and T4 (tetraiodothyronine), characterized by elevated TSH and low T4 concentrations in the bloodstream. Multiple causes contribute to hypothyroidism, with Hashimoto's disease being the primary cause, affecting the thyroid peroxidase enzyme [48]. Thyroid peroxidase stimulates the oxidation of iodine, the first step in thyroid hormone synthesis [49].

Elevated levels of thyroglobulin or peroxidase antibodies in serum may contribute to delayed puberty in females. They also significantly contribute to decreased fertility, menstrual cycle disturbances, and ovulation disorders, ultimately leading to polycystic [50]. Hypothyroidism contributes significantly to some reproductive problems such as ovulation disorders, infertility, menstrual irregularities, amenorrhea, hyperprolactinemia, and spontaneous abortion [51].

The underlying mechanism of menstrual irregularities caused by hypothyroidism involves disturbance pertaining to the hypothalamic-pituitary-ovarian axis. Changes in TSH are the ultimate result of this disturbance response and altered thyroid-releasing hormone (TRH) levels. The altered GnRH response results from increased prolactin production resulting from TRH modification. Ovulation dysfunction and luteal phase irregularities are due to impaired LH response, which is influenced by all of these factors [49].

Fertility issues linked to hypothyroidism throughout the procreative phase encompass recurrent miscarriages, menstrual cycle irregularities, and delayed puberty onset. Research indicates several endocrine variables contributing to infertility, including Cushing's disease, PCOS (polycystic ovary syndrome), diabetes mellitus, and thyroid gland diseases [52],[39].

Research indicates that high levels of TRH encourage the pituitary gland to secrete TSH and estrogen, which inhibits the secretion of FSH and LH, leading to ovarian dysfunction [49]. Hypothyroidism negatively affects pregnancy outcomes, including stillbirth; decreased fertility; placental abruption; recurrent miscarriage, with decreased cognitive, developmental, and learning capabilities [53]. In hypothyroidism, hyperprolactinemia is a key element in the emergence of infertility [54].

6. POLYCYSTIC OVARY SYNDROME WITH DYSFUNCTION

PCOS is the most frequent metabolic and endocrine condition impacting females of fertile stage, impacting 3–15% of the women's population globally [55]. PCOS (Polycystic Ovary Syndrome) induces reproductive difficulties and metabolic dysfunctions, which significantly impact the psychological and cerebral health of women [56]. Some thyroid diseases exhibit clinical manifestations that are comparable to those of PCOS. A growing amount of data points to a connection among PCOS and thyroid conditions [57].

The secretion of thyroid hormones may be influenced by PCOS-related genes that have been demonstrated to control ovarian function and secretion of hormonal [58]. A 2019 study in Denmark observed that women with PCOS are 2.5 times more probable to develop thyroid dysfunction than healthy women [59].

New evidence indicates that hereditary and genetic variables perform essential functions in the growth of these diseases, despite their distinctive clinical indicators [60].

To imperatively understand the genetic substratum and epigenetic modification associated with PCOS and hypothyroidism in order to comprehend the complex etiology of these conditions and to develop targeted therapy strategies. The genetic component of PCOS is widely recognized. In comparison to non-identical twin studies, similar S-twin studies have demonstrated a higher congruence rate for PCOS, indicating a substantial genetic contribution [61].

Genome-Wide Association Studies (GWAS) have recognized a lot of genetic loci related with polycystic ovarian syndrome, including genes that participate in ovarian function, signaling, and hormonal regulation [62]. These shared genetic susceptibilities may be the cause of the co-occurrence of polycystic ovarian syndrome and hypothyroidism in evident individuals [60]

In a study of Pakistani women between the ages of 18 and 30, Raj et al. intended to assess the pervasiveness of SCH in patients with PCOS. It was discovered that SCH demonstrated higher efficiency in PCOS patients (43.5%) than in participants without PCOS (20.5%) when 200 PCOS patients were compared with 200 controls. They also found that PCOS patients' BMI and weight gain were much higher than those of control people [63].

A patient with PCOS had a more than double risk of thyroid diseases than the healthy individuals (HR 2.5, 95% CI 2.3–2.7), according to data from a large Danish cohort study. Patients with PCOS had an incidence rate of 1.4 per 1000 patients annually, whereas those without PCOS had an incidence rate of 0.5 per 1000 patients annually [59].

The prevalence of PCOS in conjunction with GD is currently unknown due to the absence of extensive epidemiological data and the present limitation of information to case reports. Additionally, the PCOS prevalence in conjunction with GD may vary based on race or ethnicity [64].

Studies investigating the relationship between Graves' disease and PCOS in women have been few. A large cohort study (16,197 patients, including 5,399 women with Graves' disease and 10,798 without Graves' disease) revealed [65].

Patients with Graves' disorder had a considerably greater cumulative incidence of PCOS than those without it because Graves' disease is a risk factor for PCOS (annual risk ratio 1.47, 95% confidence interval 1.09–1.98) [65].

7. CONCLUSION

This article aims to investigate the fundamental function of the thyroid gland in female infertility and poly cystic ovarian syndrome by examining hormonal changes in thyroid patients and their relationship to these two conditions. PCOS is a major cause of infertility, and a literature review has been conducted on the disruption caused by the thyroid gland on female reproductive capacity and PCOS, particularly in cases of hypothyroidism. In hypothyroidism, TSH levels perform a vital function in regulating the reproductive system of women, leading to ovulation and menstrual cycle irregularities and potentially infertility. Numerous studies have shown high levels of thyroid antibodies, such as anti-thyroglobulin and anti-TPO, in PCOS patients. It is worth noting that PCOS increases the prevalence of certain thyroid disorders. The precise function of the thyroid gland in PCOS stays ambiguous. The next step in this study is to monitor insulin resistance in polycystic ovary syndrome (PCOS) patients and its effect on the thyroid gland, in addition to comparing oxidative stress in healthy patients with PCOS patients and its effect on infertility.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest

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