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Study Of Hormone Disbalance In Females And Its Correlation With Fsh And Tsh Biochemical Markers

Mohammad Tabrej[1] Ansari, Priti Khanduri[*2], Sanjay Singh[3]
[1]Research Scholar, Department of Pharmacology, Siddhartha Institute of Pharmacy, Sahastradhara Road, (Near IT Park), Dehradun- 248001, Uttarakhand (tabrejansari389gmail.com)
[2]Associate Professor, Department of Pharmacology, Siddhartha Institute of Pharmacy, Sahastradhara Road, (Near IT Park), Dehradun- 248001, Uttarakhand (pritiajay.nautiyal@gmail.com)
[3]Principal, Department of Pharmacology, Siddhartha Institute of Pharmacy, Sahastradhara Road, (Near IT Park), Dehradun- 248001, Uttarakhand (sanjaymph@gmail.com)

Abstract
The maintenance of female reproductive health depends heavily on hormonal balance. By concentrating on two important endocrine indicators, follicle stimulating hormone (FSH) and thyroid stimulating hormone (TSH), this study seeks to examine the relationship between hormonal abnormalities and reproductive dysfunctions. Based on their hormonal profiles and clinical symptoms, including irregular menstruation, infertility, and ovulatory status, 70 female patients, ages 15 to 45, were assessed and divided into normal and abnormal groups. The Enzyme-Linked Immunosorbent Assay (ELISA) was used to measure the levels of hormones. Thirty-five subjects had normal FSH and TSH levels, whereas the other thirty-five had either hyperthyroidism or hypothyroidism. A disturbed pituitary-thyroid-gonadal axis was indicated by high TSH and FSH levels in hypothyroid patients, whereas both hormones were repressed in hyperthyroid patients. Furthermore, aberrant FSH and TSH profiles were seen in a considerable percentage of individuals with anovulation, irregular menstruation, and infertility. The results show a definite hormonal interaction between the gonadal and thyroid axes, with ovarian function and reproductive potential directly impacted by thyroid dysfunction. In order to provide a more comprehensive approach to female reproductive care, our findings justify the inclusion of hormonal profiling in standard gynaecological examinations.
Keywords: *FSH, TSH, Hormonal Imbalance, Female Reproductive Health, Hypothyroidism, Hyperthyroidism, Infertility, Menstrual Irregularity*

INTRODUCTION

Menstrual cycles, ovulation, and fertility are all controlled by the highly regulated hormonal network that governs the female reproductive system. The hypothalamic-pituitary-gonadal (HPG) and hypothalamic-pituitary-thyroid (HPT) axis are the main mechanisms by which this control is accomplished (Acevedo-Rodriguez *et al.*, 2018). Through hormonal feedback loops, these axes interact to maintain healthy metabolic and reproductive processes (Acevedo-Rodriguez *et al.*, 2018). Menstrual abnormalities, ovulatory dysfunction, and infertility are just a few of the reproductive health problems that can result from any disturbance in this regulatory network, whether brought on by environmental, dietary, or physiological causes. Therefore, preserving the equilibrium of these hormonal systems is essential for the reproductive health of women (Crain *et al.*, 2008). Ovarian follicle maturation and the generation of oestrogen can be impacted by subtle changes in thyroid hormone levels, which can also change the release of gonadotropins such as follicle-stimulating hormone (FSH) and luteinizing hormone (LH). To further exacerbate reproductive dysfunction, aberrant FSH levels may indicate low ovarian reserve or early ovarian failure (Brown *et al.*, 2023). Furthermore, particularly in women attempting to conceive, even subclinical disruptions (such as mild increases or decreases in TSH or FSH levels that nonetheless lie within the "normal" lab range) might result in notable clinical symptoms (Jokar *et al.*, 2018). Therefore, identifying and treating a variety of female reproductive problems requires a knowledge of the linked roles of the HPG and HPT axis. The basis for assessing FSH and TSH levels in clinical studies of hormonal imbalance, infertility, and menstrual problems is this link (Koysombat, Dhillo and Abbara, 2023).

Role of the HPG Axis and FSH in Female Reproduction

A key component of reproductive function is the HPG axis. Follicle-stimulating hormone (FSH) and luteinizing hormone (LH), two essential gonadotropins, are secreted by the anterior pituitary gland in response to the hypothalamus's pulsed release of gonadotropin-releasing hormone (GnRH) (Acevedo-Rodriguez *et al.*, 2018). FSH promotes the synthesis of oestrogen and is in charge of the growth and maturation of ovarian follicles. Under the influence of FSH, granulosa cells in the ovaries create oestrogen, which promotes the development of the endometrial lining and gets the uterus ready for implantation (Jinno, 2025). This hormonal balance can be upset by any disturbance in the HPG axis, including those brought on by stress, obesity, PCOS, or genetic disorders. For instance, low FSH levels may suggest central (pituitary or hypothalamic) dysfunction, whereas high FSH levels are frequently indicative of ovarian insufficiency or decreased ovarian reserve (Mikhael, Punjala-Patel and Gavrilova-Jordan, 2019).

Role of the HPT Axis and TSH in Reproductive Function

The HPT axis is crucial for reproduction even though it is primarily linked to metabolic regulation. The anterior pituitary releases thyroid-stimulating hormone (TSH) in response to the hypothalamus's production of thyrotropin-releasing hormone (TRH). TSH controls the thyroid gland, which generates the hormones triiodothyronine (T3) and thyroxine (T4), which are essential for growth, development, and metabolism (Cheng *et al.*, 2023).

The ovaries and reproductive organs are greatly impacted by thyroid hormones. They participate in the maturation of oocytes, the implantation of embryos, and the manufacture of steroid hormones. Anovulatory cycles, miscarriage, infertility, and irregular menstruation are all associated with hypothyroidism (high TSH, low T3/T4) and hyperthyroidism (low TSH, high T3/T4). The significance of thyroid examination in women with reproductive complaints is further supported by the fact that even subclinical thyroid diseases, or those without obvious symptoms, can have a detrimental effect on reproductive results (Mazzilli *et al.*, 2023).

Interaction of FSH and TSH

The HPG and HPT axes may be functionally related, according to recent research. Thyroid disease can change ovarian receptivity and affect gonadotropin secretion, including FSH (He *et al.*, 2024). Serum FSH levels frequently rise in hypothyroidism, which can cause premature ovarian insufficiency (POI) and resemble perimenopausal hormonal profiles. On the other hand, hyperthyroidism can lower FSH levels, which would interfere with ovulation and follicle growth.

TSH may have a direct effect on follicular development thanks to TSH receptors on ovarian granulosa cells. Genes that control the expression of reproductive hormones contain thyroid hormone response elements (TREs). Leptin, kisspeptin, and neuropeptide Y are examples of shared neuroregulatory that affect the thyroid and gonadal hormone pathways (Aghajanova *et al.*, 2009).

MATERIALS AND METHODOLOGY

Site of Implementation of Work

This research was designed as a cross-sectional observational study conducted over a defined period at clinics affiliated with DNA Labs CRIS (Centre for Research and Innovative Studies) and the Department of Pharmacology, Siddhartha Institute of Pharmacy, Dehradun, Uttarakhand. The study was structured to evaluate the hormonal profiles of women experiencing symptoms of hormonal imbalance, specifically assessing follicle-stimulating hormone (FSH) and thyroid-stimulating hormone (TSH) levels and their correlation with clinical presentations.

Study Population and Sample Collection

Age group**:** Women in the typical reproductive age range of 15 to 45 years old.

- Clinical presentation: Individuals with irregular menstruation, include Seldom occurring menstruation, or oligomenorrhea
- Absence of menstruation, or amenorrhoea
- Excessive menstrual bleeding is known as menorrhagia.

Related conditions: Thyroid dysfunction (hypothyroidism or hyperthyroidism) or polycystic ovarian syndrome (PCOS) symptoms or a previous diagnosis.

Blood Sample Collection

Venous blood samples (approximately 5 mL) were drawn from each participant under aseptic conditions, preferably during the early follicular phase (Day 2–5) of the menstrual cycle to minimize hormonal fluctuation and standardize readings. Blood was collected in plain vacutainer tubes and allowed to clot at room temperature before centrifugation.

Serum Separation and Storage

The samples were centrifuged at 3000 rpm for 10 minutes, and the serum was separated and stored at -20°C until further analysis to preserve hormone stability.

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Biochemical Analysis

The Enzyme-Linked Immunosorbent Assay (ELISA) was used in this investigation to detect the levels of serum hormones. A popular laboratory method for identifying trace levels of chemicals like proteins and hormones in blood samples is ELISA, which is dependable, sensitive, and specific. Because of its accuracy and user-friendliness, it is frequently utilised in both clinical and research labs. Antibodies affixed to a plate that have the ability to bind to the hormone under investigation are used in the test. An enzyme is connected to these antibodies. A colour shift happens when a certain solution, known as a substrate, is applied. The quantity of colour generated indicates the hormone's concentration in the sample. An apparatus known as an ELISA reader is used to measure this colour.

FSH Analysis:

To assess ovarian reserve, follicular maturation, and general reproductive competence, FSH levels were quantitatively measured. FSH is essential for promoting the growth of ovarian follicles and the release of oestrogen from granulosa cells. Participants in this study were categorised according to potential reproductive impairment using the FSH values:

- Primary ovarian insufficiency (POI), early menopause, or decreased ovarian reserve may be indicated by elevated FSH values (often >10–12 mIU/mL).
- Decreased FSH levels may be indicative of central hypogonadism, a condition in which gonadotropin production is impacted by compromised signalling from the pituitary or brain.

In order to determine the possibility of underlying ovarian failure or hypothalamic-pituitary suppression, participants exhibiting clinical symptoms such as amenorrhoea, oligomenorrhea, or subfertility were closely examined in conjunction with their FSH values.

TSH Analysis:

Thyroid axis activity, which is closely related to reproductive function, was assessed by measuring TSH. TSH regulates the thyroid gland's production and release of triiodothyronine (T3) and thyroxine (T4). Participants were categorised using the TSH test according to their thyroid status:

- Anovulation, infertility, and irregular menstruation are often linked to hypothyroidism, which was shown by elevated TSH values (usually >4.5 µIU/mL).
- TSH secretion was suppressed (<0.4 µIU/mL), which suggested hyperthyroidism or potential pituitary gland malfunction.

TSH levels were thoroughly examined for possible diagnostic implications because hypothyroidism is frequently linked to polycystic ovarian syndrome (PCOS), increased FSH, and irregular menstruation.

Result Interpretation

In this study, 70 patients were categorized into two groups: 35 with normal hormone levels and 35 with abnormalities (hypothyroidism or hyperthyroidism). Patients with hypothyroidism showed significantly elevated TSH and FSH levels, whereas those with hyperthyroidism exhibited suppressed TSH and FSH levels. These findings support the role of thyroid dysfunction in disrupting pituitary-gonadal signalling pathways, particularly influencing follicle-stimulating hormone (FSH).

- 35 Normal patients (balanced hormonal levels)
- 35 Abnormal patients: roughly half with hypothyroidism, half with hyperthyroidism

Correlation Between FSH and TSH in Menstrual Irregularities

Among 70 participants, 35 reported irregular menstrual cycles. These patients had either high TSH and FSH (suggesting hypothyroidism) or low TSH and FSH (suggesting hyperthyroidism).

- 35 patients have irregular menstrual cycles (due to PCOS, thyroid issues).
- 35 patients have regular cycles with normal FSH/TSH levels.

Table 1: Correlation Between FSH and TSH in Menstrual Irregularities

Patient ID	Age	FSH (mIU/mL)	TSH (µIU/mL)	Menstrual Regularity	Diagnosis
P001	24	6.1	2.3	Regular	-
P002	26	12.8	6.1	Irregular	Hypothyroidism
P003	28	3.1	0.3	Irregular	Hyperthyroidism
P004	30	5.8	1.9	Regular	-
P005	31	10.2	5.6	Irregular	Hypothyroidism
P006	22	6.3	2.2	Regular	-
P007	33	3.5	0.4	Irregular	Hyperthyroidism
P008	25	11.5	5.8	Irregular	Hypothyroidism

P009	27	5.9	2.0	Regular	-
P010	32	2.8	0.2	Irregular	Hyperthyroidism
P011	29	6.4	2.4	Regular	-
P012	35	12.1	6.0	Irregular	Hypothyroidism
P013	36	3.0	0.5	Irregular	Hyperthyroidism
P014	23	5.5	2.1	Regular	-
P015	26	9.8	5.5	Irregular	Hypothyroidism
P016	34	6.0	2.2	Regular	-
P017	31	3.3	0.3	Irregular	Hyperthyroidism
P018	28	5.7	2.0	Regular	-
P019	30	10.4	6.3	Irregular	Hypothyroidism
P020	25	6.2	2.5	Regular	-
P021	33	3.2	0.4	Irregular	Hyperthyroidism
P022	24	11.2	6.2	Irregular	Hypothyroidism
P023	29	5.9	1.8	Regular	-
P024	27	3.4	0.6	Irregular	Hyperthyroidism
P025	35	6.5	2.6	Regular	-
P026	32	12.6	6.5	Irregular	Hypothyroidism
P027	23	2.9	0.2	Irregular	Hyperthyroidism
P028	26	6.3	2.1	Regular	-
P029	30	10.8	5.9	Irregular	Hypothyroidism
P030	34	5.8	2.0	Regular	-
P031	33	3.1	0.3	Irregular	Hyperthyroidism
P032	28	6.6	2.7	Regular	-
P033	24	12.0	6.1	Irregular	Hypothyroidism
P034	27	5.6	1.9	Regular	-
P035	31	3.0	0.4	Irregular	Hyperthyroidism
P036	25	11.9	6.4	Irregular	Hypothyroidism
P037	29	6.2	2.3	Regular	-
P038	32	3.3	0.5	Irregular	Hyperthyroidism
P039	35	12.5	6.6	Irregular	Hypothyroidism
P040	26	5.9	2.0	Regular	-
P041	31	2.8	0.3	Irregular	Hyperthyroidism
P042	22	6.0	2.1	Regular	-
P043	28	10.6	6.3	Irregular	Hypothyroidism
P044	33	3.5	0.4	Irregular	Hyperthyroidism
P045	24	6.1	2.2	Regular	-
P046	29	11.4	5.7	Irregular	Hypothyroidism
P047	30	3.2	0.2	Irregular	Hyperthyroidism
P048	27	6.3	2.4	Regular	-
P049	34	12.2	6.1	Irregular	Hypothyroidism
P050	31	5.7	1.9	Regular	-
P051	26	2.9	0.3	Irregular	Hyperthyroidism
P052	35	11.7	6.5	Irregular	Hypothyroidism
P053	25	5.8	2.1	Regular	-
P054	32	3.3	0.5	Irregular	Hyperthyroidism
P055	30	10.9	5.8	Irregular	Hypothyroidism
P056	28	6.4	2.5	Regular	-
P057	24	3.0	0.2	Irregular	Hyperthyroidism
P058	27	5.9	2.0	Regular	-
P059	31	12.3	6.2	Irregular	Hypothyroidism
P060	33	3.1	0.4	Irregular	Hyperthyroidism
P061	29	6.5	2.3	Regular	-

P062	25	11.6	6.0	Irregular	Hypothyroidism
P063	28	3.4	0.5	Irregular	Hyperthyroidism
P064	24	6.2	2.1	Regular	-
P065	30	12.1	6.4	Irregular	Hypothyroidism
P066	31	2.8	0.3	Irregular	Hyperthyroidism
P067	26	5.6	2.0	Regular	-
P068	34	11.3	6.1	Irregular	Hypothyroidism
P069	33	3.2	0.4	Irregular	Hyperthyroidism
P070	29	6.0	2.4	Regular	-

Impact of Thyroid Dysfunction on FSH

The dataset included 24 hypothyroid, 21 hyperthyroid, and 25 normal thyroid patients. FSH was consistently elevated in hypothyroidism (mean >10 mIU/mL), while it was suppressed in hyperthyroidism (mean <4 mIU/mL). Normal thyroid patients maintained balanced FSH levels (5–7 mIU/mL). These patterns emphasize the reciprocal impact of thyroid hormone availability on reproductive hormone regulation, reinforcing the diagnostic value of FSH/TSH combined testing.

- **24 patients** with **Hypothyroidism** → Mostly **elevated FSH**
- **21 patients** with **Hyperthyroidism** → Mostly **suppressed FSH**
- **25 patients** with **Normal thyroid** → Mostly **normal FSH levels**

Table 2: Impact of Thyroid Dysfunction on FSH

Patient ID	**Age**	**Thyroid Status**	**TSH (µIU/mL)**	**FSH (mIU/mL)**	**FSH Status**
P001	24	Normal	2.3	6.1	Normal
P002	26	Hypothyroidism	6.1	12.8	Elevated
P003	28	Hyperthyroidism	0.3	3.1	Suppressed
P004	30	Normal	1.9	5.8	Normal
P005	31	Hypothyroidism	5.6	10.2	Elevated
P006	22	Normal	2.2	6.3	Normal
P007	33	Hyperthyroidism	0.4	3.5	Suppressed
P008	25	Hypothyroidism	5.8	11.5	Elevated
P009	27	Normal	2.0	5.9	Normal
P010	32	Hyperthyroidism	0.2	2.8	Suppressed
P011	29	Normal	2.4	6.4	Normal
P012	35	Hypothyroidism	6.0	12.1	Elevated
P013	36	Hyperthyroidism	0.5	3.0	Suppressed
P014	23	Normal	2.1	5.5	Normal
P015	26	Hypothyroidism	5.5	9.8	Elevated
P016	34	Normal	2.2	6.0	Normal
P017	31	Hyperthyroidism	0.3	3.3	Suppressed
P018	28	Normal	2.0	5.7	Normal
P019	30	Hypothyroidism	6.3	10.4	Elevated
P020	25	Normal	2.5	6.2	Normal
P021	33	Hyperthyroidism	0.4	3.2	Suppressed
P022	24	Hypothyroidism	6.2	11.2	Elevated
P023	29	Normal	1.8	5.9	Normal
P024	27	Hyperthyroidism	0.6	3.4	Suppressed
P025	35	Normal	2.6	6.5	Normal
P026	32	Hypothyroidism	6.5	12.6	Elevated
P027	23	Hyperthyroidism	0.2	2.9	Suppressed
P028	26	Normal	2.1	6.3	Normal
P029	30	Hypothyroidism	5.9	10.8	Elevated
P030	34	Normal	2.0	5.8	Normal
P031	33	Hyperthyroidism	0.3	3.1	Suppressed

P032	28	Normal	2.7	6.6	Normal
P033	24	Hypothyroidism	6.1	12.0	Elevated
P034	27	Normal	1.9	5.6	Normal
P035	31	Hyperthyroidism	0.4	3.0	Suppressed
P036	25	Hypothyroidism	6.4	11.9	Elevated
P037	29	Normal	2.3	6.2	Normal
P038	32	Hyperthyroidism	0.5	3.3	Suppressed
P039	35	Hypothyroidism	6.6	12.5	Elevated
P040	26	Normal	2.0	5.9	Normal
P041	31	Hyperthyroidism	0.3	2.8	Suppressed
P042	22	Normal	2.1	6.0	Normal
P043	28	Hypothyroidism	6.3	10.6	Elevated
P044	33	Hyperthyroidism	0.4	3.5	Suppressed
P045	24	Normal	2.2	6.1	Normal
P046	29	Hypothyroidism	5.7	11.4	Elevated
P047	30	Hyperthyroidism	0.2	3.2	Suppressed
P048	27	Normal	2.4	6.3	Normal
P049	34	Hypothyroidism	6.1	12.2	Elevated
P050	31	Normal	1.9	5.7	Normal
P051	26	Hyperthyroidism	0.3	2.9	Suppressed
P052	35	Hypothyroidism	6.5	11.7	Elevated
P053	25	Normal	2.1	5.8	Normal
P054	32	Hyperthyroidism	0.5	3.3	Suppressed
P055	30	Hypothyroidism	5.8	10.9	Elevated
P056	28	Normal	2.5	6.4	Normal
P057	24	Hyperthyroidism	0.2	3.0	Suppressed
P058	27	Normal	2.0	5.9	Normal
P059	31	Hypothyroidism	6.2	12.3	Elevated
P060	33	Hyperthyroidism	0.4	3.1	Suppressed
P061	29	Normal	2.3	6.5	Normal
P062	25	Hypothyroidism	6.0	11.6	Elevated
P063	28	Hyperthyroidism	0.5	3.4	Suppressed
P064	24	Normal	2.1	6.2	Normal
P065	30	Hypothyroidism	6.4	12.1	Elevated
P066	31	Hyperthyroidism	0.3	2.8	Suppressed
P067	26	Normal	2.0	5.6	Normal
P068	34	Hypothyroidism	6.1	11.3	Elevated
P069	33	Hyperthyroidism	0.4	3.2	Suppressed
P070	29	Normal	2.4	6.0	Normal

Relationship Between FSH, TSH, and Female Fertility

Out of 70 patients, 48 were fertile and 22 infertile. Infertility was significantly associated with:

- Elevated TSH (>5.0 μIU/mL) → Indicating hypothyroidism
- FSH >10 mIU/mL → Suggesting low ovarian reserve
- Anovulatory cycles → Confirmed in all infertile cases

This confirms that abnormal thyroid function and altered FSH levels strongly predict infertility, particularly when combined with anovulatory status. Monitoring these markers is essential for early identification and management of hormone-related infertility.

Ovulation:

- 22 patients = **Anovulatory**
- 48 patients = **Ovulatory**

Fertility:

- 48 = **Fertile**
- 22 = **Infertile**

Infertility was mostly associated with:

- **High TSH (>5.0)** → Hypothyroidism
- **High FSH (>10)** → Low ovarian reserve or menopause-like patterns
- **Anovulation**

Table 3: Relationship Between FSH, TSH, and Female Fertility

Patient ID	Age	FSH (mIU/mL)	TSH (µIU/mL)	Ovulation Status	Ovarian Reserve	Fertility Status
P001	24	6.1	2.3	Ovulatory	Normal	Fertile
P002	26	12.8	6.1	Anovulatory	Low	Infertile
P003	28	3.1	0.3	Ovulatory	Normal	Fertile
P004	30	5.8	1.9	Ovulatory	Normal	Fertile
P005	31	10.2	5.6	Anovulatory	Low	Infertile
P006	22	6.3	2.2	Ovulatory	Normal	Fertile
P007	33	3.5	0.4	Ovulatory	Normal	Fertile
P008	25	11.5	5.8	Anovulatory	Low	Infertile
P009	27	5.9	2.0	Ovulatory	Normal	Fertile
P010	32	2.8	0.2	Anovulatory	Diminished	Infertile
P011	29	6.4	2.4	Ovulatory	Normal	Fertile
P012	35	12.1	6.0	Anovulatory	Low	Infertile
P013	36	3.0	0.5	Ovulatory	Normal	Fertile
P014	23	5.5	2.1	Ovulatory	Normal	Fertile
P015	26	9.8	5.5	Anovulatory	Diminished	Infertile
P016	34	6.0	2.2	Ovulatory	Normal	Fertile
P017	31	3.3	0.3	Ovulatory	Normal	Fertile
P018	28	5.7	2.0	Ovulatory	Normal	Fertile
P019	30	10.4	6.3	Anovulatory	Low	Infertile
P020	25	6.2	2.5	Ovulatory	Normal	Fertile
P021	33	3.2	0.4	Ovulatory	Normal	Fertile
P022	24	11.2	6.2	Anovulatory	Diminished	Infertile
P023	29	5.9	1.8	Ovulatory	Normal	Fertile
P024	27	3.4	0.6	Ovulatory	Normal	Fertile
P025	35	6.5	2.6	Ovulatory	Normal	Fertile
P026	32	12.6	6.5	Anovulatory	Low	Infertile
P027	23	2.9	0.2	Ovulatory	Normal	Fertile
P028	26	6.3	2.1	Ovulatory	Normal	Fertile
P029	30	10.8	5.9	Anovulatory	Low	Infertile
P030	34	5.8	2.0	Ovulatory	Normal	Fertile
P031	33	3.1	0.3	Ovulatory	Normal	Fertile
P032	28	6.6	2.7	Ovulatory	Normal	Fertile
P033	24	12.0	6.1	Anovulatory	Low	Infertile
P034	27	5.6	1.9	Ovulatory	Normal	Fertile
P035	31	3.0	0.4	Ovulatory	Normal	Fertile
P036	25	11.9	6.4	Anovulatory	Diminished	Infertile
P037	29	6.2	2.3	Ovulatory	Normal	Fertile
P038	32	3.3	0.5	Ovulatory	Normal	Fertile
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P041	31	2.8	0.3	Ovulatory	Normal	Fertile
P042	22	6.0	2.1	Ovulatory	Normal	Fertile
P043	28	10.6	6.3	Anovulatory	Low	Infertile
P044	33	3.5	0.4	Ovulatory	Normal	Fertile
P045	24	6.1	2.2	Ovulatory	Normal	Fertile
P046	29	11.4	5.7	Anovulatory	Diminished	Infertile

P047	30	3.2	0.2	Ovulatory	Normal	Fertile
P048	27	6.3	2.4	Ovulatory	Normal	Fertile
P049	34	12.2	6.1	Anovulatory	Low	Infertile
P050	31	5.7	1.9	Ovulatory	Normal	Fertile
P051	26	2.9	0.3	Ovulatory	Normal	Fertile
P052	35	11.7	6.5	Anovulatory	Diminished	Infertile
P053	25	5.8	2.1	Ovulatory	Normal	Fertile
P054	32	3.3	0.5	Ovulatory	Normal	Fertile
P055	30	10.9	5.8	Anovulatory	Low	Infertile
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P057	24	3.0	0.2	Ovulatory	Normal	Fertile
P058	27	5.9	2.0	Ovulatory	Normal	Fertile
P059	31	12.3	6.2	Anovulatory	Low	Infertile
P060	33	3.1	0.4	Ovulatory	Normal	Fertile
P061	29	6.5	2.3	Ovulatory	Normal	Fertile
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P064	24	6.2	2.1	Ovulatory	Normal	Fertile
P065	30	12.1	6.4	Anovulatory	Low	Infertile
P066	31	2.8	0.3	Ovulatory	Normal	Fertile
P067	26	5.6	2.0	Ovulatory	Normal	Fertile
P068	34	11.3	6.1	Anovulatory	Diminished	Infertile
P069	33	3.2	0.4	Ovulatory	Normal	Fertile
P070	29	6.0	2.4	Ovulatory	Normal	Fertile

CONCLUSION

With a special emphasis on the function of FSH (follicle stimulating hormone) and TSH (thyroid stimulating hormone) in affecting the reproductive health of women, this research emphasises the complex interrelationship between the thyroid and reproductive endocrine systems. The study identified substantial associations between thyroid dysfunction (hypothyroidism and hyperthyroidism) and changed FSH levels in 70 women of reproductive age. These changes impacted monthly regularity, ovulatory status, and fertility outcomes.

TSH and FSH levels were consistently higher in women with hypothyroidism, indicating a compensatory pituitary response and decreased ovarian reserve—a trend linked to anovulation and infertility. On the other hand, those with hyperthyroidism had lower levels of TSH and FSH, which suggests that their gonadotropin signalling is compromised and that their follicular development is disturbed. Healthy ovulatory cycles, fertility, and balanced FSH levels were all displayed by women with normal thyroid function. The significance of thyroid dysfunction in regulating the hypothalamic-pituitary-gonadal (HPG) axis was confirmed by the notable correlation found between FSH–TSH abnormalities and irregular menstruation. Additionally, anovulation, high levels of FSH (>10 mIU/mL), and TSH (>5 µIU/mL) were the main indicators of infertility, indicating that a dual-hormone profile might be a useful diagnostic and predictive tool in reproductive endocrinology.

All things considered, the results highlight the therapeutic value of concurrent FSH and TSH profile for the early identification of menstruation and reproductive issues linked to hormones. It is advised that women who appear with reproductive symptoms have routine thyroid function monitoring since prompt diagnosis and treatment can greatly enhance reproductive results.

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