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Original Paper

Biochemical and Hormonal Study Associated with Obesity among Women in Samarra City

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ABSTRACT
Thyroid hormones play a fundamental regulatory role in cellular metabolic pathways, particularly governing lipid metabolism. Thyroid dysfunction triggers profound alterations in the plasma lipid profile biochemical perturbations that are highly amplified in obese phenotypes. This study aimed to evaluate the biochemical correlation between thyroid hormones (TSH, T3, and T4), iron biomarkers, and circulating lipid markers in obese women in Samarra, Iraq. A total of 100 women aged 20 to 40 years were included and divided into two groups: 70 obese women (patient group) and 30 healthy women (control group). Thyroid hormones were measured using a chemiluminescence immunoassay analyzer, iron indices (ferritin and hemoglobin) were evaluated using standard laboratory methods, and lipid profile parameters were assessed using the enzymatic colorimetric method. Body mass index (BMI) was also recorded. Statistical analysis was performed using SPSS at a significance level of $P \leq 0.05$. The results showed a marked increase in BMI and TSH levels in obese women compared with the control group, together with a marked decrease in T3 and T4 levels. In addition, total cholesterol, triglycerides, LDL-C, and VLDL-C levels were significantly higher, while HDL-C levels were significantly lower. Ferritin and hemoglobin levels were also significantly decreased in the obese women, indicating iron-deficiency anemia. These findings suggest a strong association between obesity, thyroid dysfunction, dyslipidemia, and impaired iron status.

KEYWORDS: Obesity, BMI, thyroid hormones, ferritin, hemoglobin, lipid profile.

1. INTRODUCTION

The thyroid gland is one of the most important endocrine glands in the body, owing to the vital role of its hormones in regulating metabolic processes and their effects on the nervous system, the circulatory system, the liver, and the rest of the body [1]. The gland secretes two main iodine-containing hormones, triiodothyronine (T3) and thyroxine (T4, also known as tetraiodothyronine), which are essential for normal growth and development. A third hormone, calcitonin (thyrocalcitonin), is a non-iodinated peptide secreted by the parafollicular (C) cells and plays an important role in regulating the concentration of calcium in the blood [2–4]. Thyroid hormones exert their effects on almost all tissues of the body and contribute to the maintenance of many vital functions in adults [5]. Thyroid disorders, whether hyperthyroidism or hypothyroidism, are common worldwide as well as in Iraq, with a marked increase in their diagnosis rates. Women are more likely than men to develop these disorders, which are classified as non-communicable chronic diseases; thyroid disorders affect approximately 14% of adult women and are among the most common endocrine diseases, particularly in women of childbearing age [6–8]. An early sign of thyroid disease may be the appearance of a lump in the neck, sometimes accompanied by pain or difficulty in swallowing [9, 10]. Thyroid disorders also extensively affect many physiological systems, including the cardiovascular system, the central nervous system, and the blood [11], and both hyperthyroidism and hypothyroidism produce metabolic changes that alter the blood lipid profile, body weight, and insulin sensitivity [12].

Obesity is an epidemic disease that affects large numbers of people, particularly in developed countries, and its prevalence is increasing globally. In Turkey, the TURDEP-II study reported that about 36 out of every 100 adults were obese in 2010 [13]. Excess body weight can itself lead to thyroid dysfunction, and many overweight

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individuals develop hypothyroidism; in recent years, interest in the relationship between the thyroid gland and obesity has grown considerably. Recent data indicate that leptin, a hormone secreted by adipose tissue, is one of the important factors that may explain thyroid dysfunction in overweight individuals. A population-based study in Spain found that approximately 10% of the population suffers from thyroid dysfunction [14], and thyroid disorders are also common among patients with diabetes and other metabolic diseases [15]. Thyroid dysfunction has been associated with elevated blood cholesterol and triglycerides, which increases the risk of cardiovascular disease in these patients. Health professionals should therefore be aware of the possibility of thyroid disorders in markedly overweight patients. A particular challenge is the identification of overweight individuals with only a slight imbalance in thyroid hormones; in some cases, a raised thyroid-stimulating hormone (TSH) level may simply be a consequence of excess weight. Such individuals often gain weight and show disturbances in blood lipids, reinforcing the link between thyroid disorders and obesity [16]. Overweight and obesity are important risk factors for disability-adjusted life years (DALYs) and for mortality, and although obesity-related hypothyroidism is a common problem, the number of studies addressing this topic in Iraq remains limited. Therefore, the present study was designed to evaluate thyroid hormones (TSH, T3, and T4), iron indices (ferritin and hemoglobin), and the lipid profile in obese women in Samarra city and to compare them with those of normal-weight women.

2. MATERIALS AND METHODS

Study Design and Sample Collection

This study was conducted in Samarra city, Salah al-Din Governorate, Iraq, during the period from October 2025 to January 2026. It included 100 women aged 20–40 years, divided into two groups: 70 obese women (patient group) and 30 apparently healthy normal-weight women (control group). Body weight and height were measured for each participant, and body mass index (BMI) was calculated by dividing the weight (kg) by the square of the height (m^2) according to the following equation [17]:

$$BMI = \text{Weight (kg)} / \text{Height}^2 \ (m^2)$$

Participants with a BMI of 18.5–24.9 kg/m^2 were considered to be of normal weight, whereas those with a BMI $\geq$ 30 kg/m^2 were considered obese.

Biochemical Analysis

Venous blood samples were collected from all participants for biochemical analysis. Serum levels of thyroid-stimulating hormone (TSH), triiodothyronine (T3), and thyroxine (T4) were measured using a Mindray CL-900i analyzer (Mindray, China) based on chemiluminescence immunoassay (CLIA) technology. Serum ferritin was measured using the same analyzer by CLIA, and hemoglobin (Hb) was determined using an automated hematology (CBC) analyzer according to the manufacturer's approved procedures. Serum lipid indices, including total cholesterol (TC), triglycerides (TGs), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL-C), were measured using ready-made kits (BIOLABO, France) based on the enzymatic colorimetric method, with absorbance read by a spectrophotometer.

Statistical Analysis

Data were analysed using the SPSS software package. Results are expressed as mean ± standard deviation (SD), and the differences in biochemical parameters between the obese women and the normal-weight women were assessed using Student's t-test at a significance level of $P \leq 0.05$.

3. RESULTS

There was a significant increase in BMI in the obese women (37.932 ± 1.784 kg/m^2) compared with the control group (22.447 ± 0.812 kg/m^2), whereas the two groups did not differ significantly in age, as shown in Table (1). The thyroid results showed that the level of TSH was significantly higher in the obese women (7.451 ± 1.617 mIU/L) than in the control group (2.928 ± 0.704 mIU/L), while the levels of T3 and T4 were significantly lower in the obese women (0.391 ± 0.097 ng/mL and 3.681 ± 0.859 µg/dL, respectively) compared with the control group (1.000 ± 0.189 ng/mL and 9.307 ± 1.222 µg/dL, respectively), as shown in Table (2). The blood lipid indices showed significant increases in TC, TGs, LDL-C, and VLDL-C in the obese women compared with the control group, while HDL-C decreased significantly, as shown in Table (3). The iron indices showed a significant decrease in both

ferritin and hemoglobin in the obese women (8.497 ± 2.091 ng/mL and 11.079 ± 1.160 g/dL, respectively) compared with the control group (44.679 ± 6.711 ng/mL and 13.200 ± 0.614 g/dL, respectively), as shown in Table (4).

Table (1): Comparison of age and body mass index (BMI) between obese and normal-weight women.

Groups	Mean ± SD		P-value
Parameters	Normal weight (n = 30)	Obese women (n = 70)	
Age	29.63±5.17	30.43±5.28	> 0.05 (NS)
BMI (kg/m^2)	22.447±0.812	37.932±1.784	< 0.001*

** Significant at P ≤ 0.05; NS = not significant.*

Table (2): Comparison of thyroid hormone levels (TSH, T3, and T4) between obese and normal-weight women.

Groups	Mean ± SD		P-value
Parameters	Normal weight (n = 30)	Obese women (n = 70)	
TSH (mIU/L)	2.928±0.704	7.451±1.617	< 0.001*
T3 (ng/mL)	1.000±0.189	0.391±0.097	< 0.001*
T4 (µg/dL)	9.307±1.222	3.681±0.859	< 0.001*

** Significant at P ≤ 0.05.*

Table (3): Comparison of blood lipid indices between obese and normal-weight women.

Groups	Mean ± SD		P-value
Parameters	Normal weight (n = 30)	Obese women (n = 70)	
TC (mg/dL)	170.967±13.163	263.814±47.472	< 0.001*
TGs (mg/dL)	98.433±16.383	215.114±56.479	< 0.001*
HDL-C (mg/dL)	53.133±5.758	33.414±10.511	< 0.001*
LDL-C (mg/dL)	88.933±13.696	181.843±47.970	< 0.001*
VLDL-C (mg/dL)	23.767±7.403	41.929±11.839	< 0.001*

** Significant at P ≤ 0.05; NS = not significant.*

** Significant at P ≤ 0.05.*

Table (4): Comparison of iron indices between obese and normal-weight women.

Groups	Mean ± SD		P-value
Parameters	Normal weight (n = 30)	Obese women (n = 70)	
Hb (g/dL)	13.200±0.614	11.079±1.160	< 0.001*
Ferritin (ng/mL)	44.679±6.711	8.497±2.091	< 0.001*

** Significant at P ≤ 0.05.*

4. DISCUSSION

The results of the present study showed significant changes in thyroid function, iron and blood indices, and blood lipid levels in obese women compared with healthy women in Samarra city, suggesting a strong relationship between obesity, thyroid dysfunction, disturbed iron metabolism, and disordered lipid metabolism. The changes observed in lipid levels in obese women with thyroid dysfunction can be explained by several physiological mechanisms. The results in Table (2) showed an increase in TSH and a decrease in both T3 and T4 in the obese women, indicating impaired thyroid function in this group. A high TSH level is usually associated with hypothyroidism, whereas a low level indicates an overactive thyroid. TSH secretion is regulated by a negative feedback mechanism whereby T3 and T4 inhibit the release of both TSH and thyrotropin-releasing hormone (TRH) when their blood levels rise, while low levels stimulate TSH secretion. TSH is also the main stimulator of the thyroid gland for the synthesis and release of T3 and T4, as it binds to its receptors on thyroid follicular cells and activates the biological processes responsible for the synthesis of these hormones [18]. On the other hand, a high TSH may be associated with a decrease in the level of thyroxine-binding globulin (TBG) or a disturbance in the binding of thyroid hormones to it, which alters the levels of free hormones and thus activates compensatory hormonal mechanisms [19, 20]. Low levels of T3 and T4 reflect thyroid dysfunction [21]. T3 is the main hormone responsible for regulating metabolic processes in the body, so its decrease reflects reduced metabolic activity in the obese group. Recent studies also suggest that a low T3 level may be associated with “low T3 syndrome”, which appears in chronic and inflammatory diseases, including obesity, and is considered an indicator of disease severity and poor health status; decreased T3 has been linked to increased complications and poor clinical outcomes [22, 23]. T4 is the main circulating storage form of thyroid hormone and acts as a prohormone that is converted within the tissues into the more active hormone T3; its decrease therefore reflects a reduction in the functional capacity of the thyroid gland or a disturbance of the hypothalamic–pituitary–thyroid axis. This agrees with Nassar *et al.* [24], who reported that a low T4 level in the patient group is an indicator of hypothyroidism. The results of the present study are also consistent with the recent studies of Hafeez *et al.* [25] and Abha *et al.* [21], which demonstrated a relationship between obesity and thyroid function, with high TSH and low T3 and T4 levels in obese women, indicating the presence of hypothyroidism in the patient group.

The results in Table (3) showed that the elevated TC, TGs, VLDL-C, and LDL-C were accompanied by a lower HDL-C in the obese group compared with the control group. This increase reflects a pronounced disturbance in lipid metabolism in the obese group and may be related to hormonal or metabolic changes. High total cholesterol is an important indicator of the effect of thyroid hormone disturbance on lipid metabolism, since these hormones regulate the activity of the enzymes responsible for cholesterol synthesis in the liver, the most important of which is 3-hydroxy-3-methylglutaryl coenzyme A reductase (HMG-CoA reductase), the rate-limiting enzyme of the cholesterol biosynthetic pathway. Thyroid hormones, especially T3, play an important role in regulating this enzyme and thus in determining the cholesterol level in the body. In addition, thyroid hormones increase the expression of LDL receptors in the liver, which contributes to the removal of cholesterol from the bloodstream. When hypothyroidism occurs, the decline in these hormones reduces the activity of these receptors, leading to the accumulation of cholesterol in the blood [19]. This indicates a clear disorder of lipid metabolism associated with impaired thyroid function. Elevated triglycerides are a common alteration associated with hypothyroidism, owing to the direct effects of thyroid hormones, especially T3, on the regulation of lipid metabolism. In hypothyroidism, the low T3 level reduces the activity of lipoprotein lipase (LPL), the enzyme responsible for the hydrolysis of triglycerides in lipoproteins such as chylomicrons and VLDL, resulting in reduced clearance and accumulation of triglycerides in the blood [26]. Hepatic lipase activity is also reduced, slowing triglyceride metabolism and prolonging its residence in the circulation [26].

On the other hand, hypothyroidism increases the hepatic production of VLDL, the main transporter of triglycerides, which contributes to their elevated level in the blood [27]. A decrease in thyroid hormones also reduces the oxidation of fatty acids, leading to their accumulation and re-esterification into triglycerides within the liver and their subsequent release into the blood [27]. Conversely, the decrease in HDL-C can be explained by several pathological mechanisms: hypothyroidism disrupts the activity of the enzymes responsible for HDL metabolism, such as lecithin–cholesterol acyltransferase (LCAT) and hepatic lipase, which affects the maturation and remodelling of HDL particles [19]. In addition, elevated triglyceride levels and insulin resistance, which are

common in hypothyroidism, accelerate the removal of HDL from the blood and reduce its concentration [28]. Changes in HDL composition may also impair its functional efficiency even when its level is not markedly reduced. Accordingly, the low HDL-C in this study reflects an imbalance in lipid homeostasis and an increased risk of cardiovascular disease in patients with hypothyroidism, underscoring the importance of early and comprehensive assessment of the lipid profile in this group of patients [28]. Our results agree with a study conducted in Jordan by Al-Odat *et al.* [28], which showed a relationship between thyroid disorders and elevated lipids, especially total cholesterol and LDL-C, and with the study of Hafeez *et al.* [25], which found significantly higher blood lipid levels, including triglycerides and total cholesterol, in obese compared with non-obese individuals, supporting the relationship between obesity and lipid disorders. However, our findings differed from those of Samananda *et al.* [29], who found no association between BMI and the levels of lipids or thyroid hormones, whereas the present study showed a clear relationship between obesity and elevated lipids. This difference may be explained by differences in lifestyle, dietary habits, and other factors between the studied populations.

The results in Table (4) showed a decrease in both ferritin and hemoglobin in the obese group, indicating depleted iron stores, since ferritin is the main indicator of iron stores in the body. Iron plays an essential role in the synthesis of thyroid hormones through its involvement in the activity of the enzyme thyroid peroxidase (TPO); when iron levels are low, the efficiency of this enzyme decreases, leading to reduced T3 and T4 production and a compensatory elevation of TSH. Obesity, on the other hand, is associated with chronic low-grade inflammation that increases the secretion of the hormone hepcidin, the main regulator of iron absorption. Hepcidin inhibits the absorption of iron from the intestine and prevents its release from body stores, leading to what is known as functional iron deficiency and low ferritin [30]. Chronic inflammation associated with obesity also affects the distribution of iron within the body, trapping it within immune cells and reducing its bioavailability; inflammation has been shown to increase hepcidin levels and to reduce iron availability and ferritin [31]. In addition, thyroid disorders, especially hypothyroidism, may impair iron absorption as a result of decreased gastrointestinal activity, leading to a further decrease in ferritin. These results are consistent with the systematic review and meta-analysis of Garofalo *et al.* [32], which found that iron deficiency is associated with impaired thyroid function and decreased production of thyroid hormones, and suggested that thyroid dysfunction may in turn lead to deficiencies of micronutrients, including iron.

The low hemoglobin observed in the obese group indicates the presence of anemia or a disorder of red blood cell formation. Our results agree with those of Santosh *et al.* [33], who showed that thyroid disorders, whether hypothyroidism or hyperthyroidism, are associated with a significant decrease in hemoglobin levels, since thyroid hormones play an important role in regulating erythropoiesis, so that any imbalance in these hormones leads to a decrease in hemoglobin production. This is consistent with the relationship between thyroid dysfunction and low hemoglobin found in the present study. Iron is an essential component of hemoglobin synthesis, so any depletion of iron stores leads to a decrease in hemoglobin production. Since our study showed a decrease in ferritin, this indicates a true iron deficiency that is directly reflected in hemoglobin synthesis; the World Health Organization has confirmed that iron deficiency is the most common nutritional cause of anemia worldwide [34].

5. CONCLUSION

This study demonstrated a relationship between thyroid dysfunction, lipid abnormalities, and blood and iron indices in obese women. High TSH levels and low T3 and T4 levels were associated with increased levels of total cholesterol, triglycerides, LDL-C, and VLDL-C and with lower levels of HDL-C. These results suggest an important role of thyroid hormones in regulating lipid metabolism, and their disturbance may contribute to an increased risk of cardiovascular disease in obese individuals. The low ferritin and hemoglobin levels indicate the presence of iron-deficiency anemia in the obese group. Regular monitoring of thyroid function and iron status in obese women may therefore be necessary for the early detection and prevention of metabolic complications.

REFERENCES

1- Bullock, J. (2001) ‘Endocrine physiology’, in *Physiology*. 4th edn. Philadelphia: Lippincott Williams & Wilkins, pp. 693–706.

2- Malik, R. and Hodgson, H. (2002) 'The relationship between the thyroid gland and the liver', *QJM: An International Journal of Medicine*, 95(9), pp. 559–569.

3- Bertin, F.R. and Fraser, N.S. (2020) 'Calcium regulation and diseases of the parathyroid gland', in *Equine Endocrinology*. Wallingford: CABI, pp. 28–39.

4- Shen, C.L., Wu, Y.H., Zhang, T.H. and Tu, L.H. (2022) 'Dihydrocaffeic acid decorated iron oxide nanomaterials effectively inhibit human calcitonin aggregation', *ACS Omega*, 7(35), pp. 31520–31528.

5- Li, Z., Gao, Z., Sun, T., Zhang, S., Yang, S., Zheng, M. and Shen, H. (2023) 'Meteorin-like/Metrnl, a novel secreted protein implicated in inflammation, immunology, and metabolism: a comprehensive review of preclinical and clinical studies', *Frontiers in Immunology*, 14, 1098570.

6- Brent, G.A. (2023) 'A historical reflection on scientific advances in understanding thyroid hormone action', *Thyroid*, 33(10), pp. 1140–1149.

7- Strikić, Đ., Pleić, N., Babić Leko, M., Gunjača, I., Torlak, V., Brdar, D. *et al.* (2022) 'Epidemiology of hypothyroidism, hyperthyroidism and positive thyroid antibodies in the Croatian population', *Biology*, 11(3), 394.

8- Mousa, H. and Zoori, A. (2023) 'The prevalence of thyroid disorders in Nasiriya city, Iraq', *University of Thi-Qar Journal of Science*, 10(1).

9- American Thyroid Association (2017) *General information/Press room*. Falls Church, VA: American Thyroid Association.

10- Syed, W., Samarkandi, O.A., Alsadoun, A., Harbi, M.K.A. and Al-Rawi, M.B.A. (2022) 'Evaluation of clinical knowledge and perceptions about the development of thyroid cancer—an observational study of healthcare undergraduates in Saudi Arabia', *Frontiers in Public Health*, 10, 912424.

11- Kayande, S.M., Zingade, U., Munde, S.M. and Khare, A.S. (2023) 'A cross-sectional comparative study of T3, T4 and TSH levels in altered thyroid status in premenopausal women', *European Journal of Cardiovascular Medicine*, 13(2).

12- Jameson, J.L., Fauci, A.S., Kasper, D.L. *et al.* (2018) *Harrison's Principles of Internal Medicine*. 20th edn. New York: McGraw-Hill Education.

13- Satman, I., Omer, B., Tutuncu, Y., Kalaca, S., Gedik, S., Dinccag, N. *et al.* (2013) 'Twelve-year trends in the prevalence and risk factors of diabetes and prediabetes in Turkish adults', *European Journal of Epidemiology*, 28(2), pp. 169–180.

14- Valdés, S., Maldonado-Araque, C., Lago-Sampedro, A., Lillo, A., Garcia-Fuentes, E., Perez-Valero, V. *et al.* (2017) 'Population-based national prevalence of thyroid dysfunction in Spain and associated factors: Di@bet.es study', *Thyroid*, 27(2), pp. 156–166.

15- Yadav, A., Yadav, G.A., Narsingrao, K.K., Kumar, L.N. and Yadav, G.S. (2021) 'Prevalence of thyroid disorders among patients with diabetes in rural South India', *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, 15(3), pp. 885–889.

16- Verma, A., Jayaraman, M., Kumar, H.K. and Modi, K.D. (2008) 'Hypothyroidism and obesity: cause or effect?', *Saudi Medical Journal*, 29(8), pp. 1135–1138.

17- Centers for Disease Control and Prevention (2024) *BMI frequently asked questions*. Atlanta, GA: U.S. Department of Health and Human Services. Available at: https://www.cdc.gov/bmi/faq/index.html (Accessed: 23 June 2026).

18- Shpakov, A.O. (2022) 'Thyroid hormones and their regulation', *Biochemistry (Moscow)*, 87(9), pp. 1021–1036.

19- Liu, H. and Peng, D. (2022) 'Update on dyslipidemia in hypothyroidism: the mechanism of dyslipidemia in hypothyroidism', *Endocrine Connections*, 11(2), e210002.

20- Mullur, R., Liu, Y.Y. and Brent, G.A. (2014) 'Thyroid hormone regulation of metabolism', *Physiological Reviews*, 94(2), pp. 355–382.

21- Abha, P., Keshari, J.R., Sinha, S.R., Nishant, K., Kumari, R. and Prakash, P. (2023) 'Association of thyroid function with lipid profile in patients with metabolic syndrome: a prospective cross-sectional study in the Indian population', *Cureus*, 15(9), e44745.

22- Salas-Lucia, F. and Bianco, A.C. (2022) 'Physiology and pathophysiology of deiodinases in thyroid hormone metabolism', *Endocrine Reviews*, 43(5), pp. 665–706.

23- Chen, Y., Liu, X., Yu, X. *et al.* (2020) 'Low T3 syndrome as a predictor of poor prognosis in patients with severe illness', *Journal of Clinical Endocrinology & Metabolism*, 105(6), dgaa190.

24- Nassar, M., Hassan, A., Ramadan, S., Desouki, M.T., Hassan, M.A. and Chaudhuri, A. (2024) 'Evaluating the effectiveness of combined T4 and T3 therapy or desiccated thyroid versus T4 monotherapy in hypothyroidism', *BMC Endocrine Disorders*, 24(1), 90.

25- Hafeez, R., Zafar, A., Bashir, S., Abideen, Z.U., Ejaz, M., Zahid, N. and Haque, M.U. (2024) 'Thyroid and lipid profile dysfunction among obese versus non-obese individuals', *Pakistan Journal of Health Sciences*, 5(3), pp. 115–120.

26- Mavromati, M. and Jornayvaz, F.R. (2021) 'Hypothyroidism-associated dyslipidemia: potential molecular mechanisms leading to NAFLD', *International Journal of Molecular Sciences*, 22(23), 12797.

27- Duntas, L.H. and Brenta, G. (2012) 'The effect of thyroid disorders on lipid levels and metabolism', *Endocrine Reviews*, 33(6), pp. 933–960.

28- Al-Odat, I., Al-Fawaeir, S. and Al-Mahmoud, M.H. (2024) 'Study of the association between thyroid dysfunction and serum lipid abnormalities', *Biomedical Reports*, 21(4), 138.

29- Samananda, L., Govindaraj, Y., Devi, K.G. and Kanan, W. (2021) 'Correlations of thyroid hormones and lipid profile in relation to body mass index', *International Journal of Medical Research & Health Sciences*, 10(10), pp. 43–49.

30- Insani, D.A., Subagio, H.W. and Hendrianingtyas, M. (2019) 'Iron status and hepcidin levels as potential regulators of haemoglobin homeostasis in overweight and obese women of childbearing age', *Journal of Taibah University Medical Sciences*, 14(6), pp. 531–537.

31- Krygier, A., Szczepanek-Parulska, E., Filipowicz, D. and Ruchała, M. (2020) 'Changes in serum hepcidin according to thyrometabolic status in patients with Graves' disease', *Endocrine Connections*, 9(3), pp. 234–242.

32- Garofalo, V., Condorelli, R.A., Cannarella, R., Aversa, A., Calogero, A.E. and La Vignera, S. (2023) 'Relationship between iron deficiency and thyroid function: a systematic review and meta-analysis', *Nutrients*, 15(22), 4790.

33- Santosh, U.P., Rajesh, P., Kumar, A.S., Chandra, I., Prabhakaran, V. and Nasser, A. (2025) 'A prospective study on anemia in hypothyroidism and hyperthyroidism', *Indian Journal of Otolaryngology and Head & Neck Surgery*, 77(2), pp. 779–783.

34- World Health Organization (2025) *Anaemia*. Fact sheet. Geneva: WHO. Available at: https://www.who.int/news-room/fact-sheets/detail/anaemia (Accessed: 23 June 2026).