RESEARCH PAPER

Study of Thyroid Function Status and Insulin Resistance in Type 2 Diabetes Mellitus

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Received: 25th Aug, 2026 | Revised: 1st Sep, 2026 | Accepted: 5th Sep, 2026 | Available Online: 7th Sep, 2026

ABSTRACT: NA

Keywords: NA

How to cite this article: Ujjval Kumar1 Dr. Jaydeep Darji2. Study of Thyroid Function Status and Insulin Resistance in Type 2 Diabetes Mellitus. Int J Drug Deliv Technol. 2026;16(80s):1463-1467. DOI: 10.25258/ijddt.16.80s.187.

Introduction

Type 2 Diabetes Mellitus (T2DM) isn't just a simple issue of elevated blood sugar; it is a complex, chronic metabolic disorder marked by persistent hyperglycaemia driven by a combination of insulin resistance and a relative lack of insulin. Over recent decades, the global prevalence of T2DM has surged, largely fueled by rapid urbanisation, aging populations, shifting dietary habits, and increasingly sedentary lifestyles. When left unchecked, this chronic hyperglycaemia severely disrupts carbohydrate, lipid, and protein metabolism. Ultimately, these metabolic disturbances pave the way for severe, long-term microvascular and macrovascular complications, including nephropathy, neuropathy, retinopathy, and cardiovascular disease.

On the other side of the endocrine spectrum, thyroid hormones act as the body's primary regulators for basal metabolic rate, thermogenesis, and energy expenditure, heavily influencing how we process glucose and lipids. The thyroid gland synthesises thyroxine (T4) and triiodothyronine (T3), which operate through nuclear receptors to regulate vital gene transcription. Clinically, we are increasingly seeing thyroid dysfunction in our T2DM patients, and it can significantly throw off their glycemic control and overall metabolic balance.

Understanding the link between these two conditions is crucial because they share several underlying pathogenic drivers, such as chronic inflammation, oxidative stress, endothelial dysfunction, and metabolic syndrome. Therefore, this study was designed to closely evaluate and compare thyroid hormone status and insulin resistance across three specific populations: healthy euthyroid individuals, euthyroid T2DM patients, and T2DM patients suffering from thyroid dysfunction alongside complications like dyslipidemia or renal impairment. We hope this research provides fresh insights into the biochemical relationship between thyroid issues and insulin resistance, ultimately helping clinicians better assess and manage diabetic patients.

1. Aims and Objectives

Aim: To study and compare thyroid function status and insulin resistance among individuals with T2DM, specifically looking at those with and without thyroid dysfunction and associated complications.

Objectives:

1. Evaluate thyroid hormones (TSH, FT3, FT4) across the three designated study groups.
2. Assess insulin resistance using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR).
3. Compare the lipid and renal profiles relative to the thyroid status of the subjects.
4. Correlate thyroid hormones with parameters of insulin resistance.
5. Determine the association between thyroid dysfunction and metabolic derangements in T2DM.

2. Review of Literature

2.1 Pathogenesis of Type 2 Diabetes Mellitus

At its core, insulin resistance is considered the central defect in the development of T2DM; it simply means that peripheral tissues stop responding effectively to circulating insulin. Under normal physiological conditions, insulin helps skeletal muscle and fat tissue take up glucose by moving GLUT-4 transporters to the cell membrane, while simultaneously telling the liver to stop producing new glucose. However, in insulin-resistant states, these signaling pathways break down. This results in decreased glucose uptake, an unchecked output of glucose from the liver, and persistent hyperglycaemia. Factors like obesity, chronic inflammation, oxidative stress, and genetics all play a massive role here. Over time, the constant high blood sugar leads to glucotoxicity, wearing out the pancreatic beta-cells until overt diabetes finally sets in.

2.2 Physiology of Thyroid Hormones

The thyroid gland secretes T4 and T3, both of which are absolutely vital for normal growth, development, and day-to-day metabolic regulation. This hormone synthesis is tightly controlled by thyroid-stimulating hormone (TSH), which is released from the anterior pituitary gland under the direction of thyrotropin-releasing hormone (TRH). Most of the hormone released by the thyroid is T4, which then travels to peripheral tissues where deiodinase enzymes convert it into the highly active T3. These hormones influence almost every metabolic pathway. They boost intestinal glucose absorption, hepatic gluconeogenesis, and peripheral glucose uptake, while also kickstarting lipolysis and fatty acid oxidation.

Furthermore, in skeletal muscle, thyroid hormones actively enhance mitochondrial oxidative phosphorylation and ATP production.

2.3 The Bidirectional Link Between Thyroid Dysfunction and T2DM

The relationship between thyroid dysfunction and diabetes is very much a two-way street. Thyroid hormones directly impact how we process carbohydrates, while fluctuating insulin levels and blood sugar can alter thyroid hormone metabolism and disrupt the hypothalamic-pituitary-thyroid axis. For instance, hypothyroidism slows down glucose absorption and utilization, which essentially worsens existing insulin resistance.

Hyperthyroidism, on the other hand, causes problems in the opposite direction—it ramps up hepatic glucose production, increases intestinal glucose absorption, and breaks down insulin faster, completely ruining a patient's glycemic control.

2.4 Impacts on Lipid and Renal Metabolism

Thyroid hormones are heavily involved in the regulation of lipid synthesis and breakdown. Because of this, hypothyroidism frequently leaves patients with elevated total cholesterol, LDL cholesterol, and triglycerides, mostly due to sluggish LDL receptor activity. When a diabetic patient also has hypothyroidism, their dyslipidemia gets substantially worse, driving up their cardiovascular risk.

Kidney function is similarly affected, as thyroid hormones regulate renal blood flow and the glomerular filtration rate (GFR). Hypothyroidism drops the GFR and raises serum creatinine levels. Since diabetic nephropathy is already a major threat to T2DM patients, coexisting thyroid dysfunction can easily aggravate renal impairment and accelerate chronic kidney disease. Elevated TSH levels in these patients are frequently linked to a reduced estimated GFR and albuminuria.

3. Materials and Methods

3.1 Study Design and Setting

We conducted a cross-sectional, comparative study in the Department of Biochemistry and the Biochemistry Laboratory at RVRS Medical College & Mahatma Gandhi Hospital in Bhilwara, Rajasthan.

3.2 Study Population and Sampling

The study utilized simple random sampling to recruit a total of 60 subjects. The sample size was calculated using a standard formula for comparing proportions, ensuring a 95% confidence level and 80% power. The subjects (aged 30-70 years) were divided equally into three groups of 20:

- **Control Group 1 (Control i):** Healthy, euthyroid, non-diabetic individuals.
- **Control Group 2 (Control ii):** Euthyroid T2DM patients without major complications.
- **Test Group:** Diagnosed T2DM patients suffering from thyroid dysfunction (either hypo- or hyperthyroidism) alongside complications like dyslipidemia or raised creatinine.

Patients were strictly excluded if they had Type 1 Diabetes, gestational diabetes, known liver failure, other renal disorders, acute illness, malignancies, or if they were currently taking thyroid-modulating drugs or were pregnant.

3.3 Sample Collection and Laboratory Analysis

Following aseptic precautions, 5-8 mL of fasting venous blood was collected from each participant, centrifuged, and analyzed on the same day to ensure sample integrity. The laboratory instrumentation included the Beckman Coulter Access 2 for thyroid and insulin assays, the Beckman Coulter AU480 for glucose, renal, and lipid profiles, and the Randox RX Imola for cross-verification.

We calculated insulin resistance using the standard HOMA-IR formula: HOMA-IR = (Fasting Insulin × Fasting Glucose) / 405.

3.4 Statistical Analysis

All raw data were compiled using Microsoft Excel and subsequently analyzed with SPSS. Quantitative data were expressed as the mean ± standard deviation (SD). To compare variances among the three groups, we utilized One-Way ANOVA, and Pearson correlation was used to assess relationships between hormonal markers and insulin resistance. A

p-value of < 0.05 was considered statistically significant.

4. Results

The study evaluated 60 participants across the three defined groups. Importantly, the age distribution among the groups did not differ significantly ($p = 0.343$), meaning the groups were well-matched and our biochemical findings are largely free from age-related bias.

Table 1: Master Summary of Clinical and Biochemical Parameters

Parameter	Control 1 (Mean ± SD)	Control 2 (Mean ± SD)	Test (Mean ± SD)	ANOVA p-value	Significanc e
Age (years)	30.00 ± 9.65	32.55 ± 8.33	34.25 ± 9.48	0.343	Not Significant
FBS (mg/dL)	74.20 ± 12.42	258.00 ± 6.62	310.55 ± 16.57	< 0.001	Significant
HbA1c (%)	5.25 ± 1.02	8.51 ± 1.01	8.59 ± 0.93	< 0.001	Significant
Insulin (µU/mL)	8.01 ± 2.35	47.70 ± 16.34	23.90 ± 3.88	< 0.001	Significant
HOMA-IR	1.48 ± 0.58	30.44 ± 10.56	18.31 ± 3.10	< 0.001	Significant
TSH (µIU/mL)	2.76 ± 0.94	2.11 ± 1.13	32.85 ± 10.43	< 0.001	Significant
FT3 (pg/mL)	2.80 ± 0.72	3.32 ± 0.61	5.37 ± 1.23	< 0.001	Significant
FT4 (ng/dL)	0.56 ± 0.28	0.83 ± 0.18	1.55 ± 0.31	< 0.001	Significant
Urea (mg/dL)	16.65 ± 7.08	48.95 ± 7.92	36.05 ± 6.61	< 0.001	Significant
Creatinine (mg/dL)	0.785 ± 0.28	1.35 ± 0.29	2.615 ± 0.69	< 0.001	Significant
TC (mg/dL)	151.05 ± 5.82	199.40 ± 7.02	237.20 ± 3.42	< 0.001	Significant
HDL (mg/dL)	55.20 ± 2.71	55.65 ± 6.01	64.15 ± 7.86	< 0.001	Significant
LDL (mg/dL)	67.25 ± 4.19	96.95 ± 8.04	137.90 ± 6.79	< 0.001	Significant
TG (mg/dL)	162.40 ± 12.12	273.55 ± 14.46	270.20 ± 12.45	< 0.001	Significant

Variables Analyzed	r-value	Interpretation of Correlation
TSH vs HOMA-IR	0.079	Very weak positive correlation
FT3 vs HOMA-IR	0.231	Weak positive correlation
FT4 vs HOMA-IR	0.245	Weak positive correlation
TSH vs HbA1c	0.451	Moderate positive correlation
TSH vs TG	0.401	Moderate positive correlation
HOMA-IR vs HbA1c	0.692	Strong positive correlation

Table 2: Pearson Correlation Analysis

5. Discussion

This study set out to examine how thyroid function status intertwines with insulin resistance in Type 2 Diabetes patients, particularly looking at how these dynamics shift when patients develop metabolic complications. Because thyroid hormones are so deeply involved in regulating glucose, lipids, and overall energy, it makes sense that any alterations in a patient's thyroid status could easily aggravate existing diabetic complications.

Looking at our glycemic data, the test group—diabetic patients with thyroid dysfunction—exhibited severely elevated fasting blood sugar levels compared to the other groups, pointing to a profound loss of glycemic control. Since thyroid hormones actively influence how much glucose the liver outputs and how peripheral tissues utilize it, thyroid dysfunction clearly acts as a catalyst for worsening blood sugar imbalances in T2DM. Furthermore, HOMA-IR values were sharply elevated in both diabetic groups. Interestingly, we noticed that while serum insulin spiked dramatically in the euthyroid diabetic group (likely a natural compensatory response to insulin resistance), insulin levels were actually lower in the hyperthyroid diabetic group despite them having similarly poor HbA1c values. This finding is highly significant; it suggests that hyperthyroidism in these patients might be triggering faster insulin degradation, or worse, accelerating pancreatic β-cell exhaustion.

The thyroid panels revealed stark differences as well. Serum TSH levels were massively elevated in the test group, strongly confirming the presence of primary thyroid dysfunction. When we ran the Pearson correlations, we found a moderate positive link between TSH and HbA1c ($r = 0.451$), which essentially tells us that as thyroid dysfunction worsens, long-term glycemic control reliably deteriorates.

Renal and lipid parameters painted a similarly concerning picture. Both urea and creatinine were significantly elevated in the diabetic subjects, peaking in the test group. This makes physiological sense: thyroid dysfunction impairs renal hemodynamics, thereby accelerating the decline in kidney function and pushing patients closer to overt diabetic nephropathy. On the lipid front, total cholesterol, LDL, and triglycerides were all significantly elevated in the diabetic groups. Because reduced thyroid hormone activity inherently slows down lipid clearance, the coexistence of hypothyroidism in a diabetic patient creates a perfect storm for dyslipidemia, drastically increasing their cardiovascular risk. We saw this reflected directly in the data, with a moderate positive correlation between TSH and triglyceride levels ($r = 0.401$).

6. Conclusion

The data from our study align strongly with current literature, confirming a distinct, powerful association between thyroid dysfunction and insulin resistance in T2DM.

We found that thyroid dysfunction actively worsens insulin resistance, breaks down glycemic control, exacerbates dyslipidemia, and accelerates renal impairment in diabetic individuals. Specifically, elevated TSH levels serve as a strong indicator for poorer glycemic control and worsening lipid profiles, while HOMA-IR reliably tracks with rising HbA1c. Together, these metabolic derangements heavily compound a patient's risk of facing severe diabetic complications and cardiovascular morbidity.

Ultimately, our findings strongly advocate for the routine screening of thyroid function in all T2DM patients. Catching these thyroid abnormalities early gives clinicians a crucial window to intervene. By managing thyroid dysfunction promptly, we can likely improve a patient's insulin sensitivity, stabilize their metabolic control, and greatly improve their long-term clinical outcomes.

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