e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(8); 1690-1692

Original Research Article

Tissue-Level Insulin and Thyroid Hormone Crosstalk: A Retrospective Study from Katihar Medical College

Anshu Kumar[1], Sharfe Alam[2], Amit Anand[3]

[1]2nd Year Post Graduate Student, Department of Physiology, Katihar Medical College, Katihar, Bihar, India

[2]2nd Year Post Graduate Student, Department of Physiology, Katihar Medical College, Katihar, Bihar, India

[3]3rd Year Post Graduate, Department of Physiology, Katihar Medical College, Katihar, Bihar, India

Received: 24-06-2025 / Revised: 23-07-2025 / Accepted: 24-08-2025

Corresponding Author: Anshu Kumar

Conflict of interest: Nil

Abstract:

Background: Insulin and thyroid hormones are major regulators of cellular metabolism. Their signaling pathways intersect at multiple tissue levels, influencing glucose utilization, lipid metabolism, and protein synthesis. Alterations in one axis can significantly modify the action of the other, contributing to metabolic dysfunction.

Aim: To analyze the relationship between insulin activity and thyroid hormone status at the tissue level, and to evaluate the physiological implications of their crosstalk in a clinical cohort.

Methods: A retrospective study was conducted in the Department of Physiology, Katihar Medical College, over one year. Records of 100 patients who underwent simultaneous thyroid function tests (T3, T4, TSH) and fasting insulin/HOMA-IR assessment were reviewed. Data on age, gender, BMI, and biochemical parameters were extracted. Correlations between insulin resistance indices and thyroid profiles were analyzed.

Results: Of the 100 participants, 42% had altered thyroid function (subclinical hypothyroidism 28%, overt hypothyroidism 10%, hyperthyroidism 4%). Insulin resistance (HOMA-IR >2.5) was observed in 36%. A significant association was found between hypothyroidism and insulin resistance (p=0.01). Patients with hypothyroidism showed higher mean fasting insulin (14.8 ± 3.6 µIU/mL) compared to euthyroid individuals (9.7 ± 2.4 µIU/mL). Conversely, hyperthyroid patients demonstrated increased basal glucose turnover but lower fasting insulin levels. Tissue-level physiology suggests that thyroid hormones enhance GLUT-4 translocation and mitochondrial activity, while hypothyroidism reduces insulin sensitivity in skeletal muscle and adipose tissue.

Conclusion: The study highlights a significant interaction between insulin resistance and thyroid hormone imbalance, particularly hypothyroidism. Understanding this crosstalk is crucial for early detection of metabolic disturbances and developing integrated treatment approaches for endocrine disorders.

Keywords: Insulin Resistance, Thyroid Hormones, Metabolic Crosstalk, Hypothyroidism, Physiology.

Introduction

The endocrine system functions as a complex network, where hormones often interact at cellular and molecular levels. Among these, insulin and thyroid hormones are pivotal regulators of energy homeostasis. Insulin promotes glucose uptake, glycogen synthesis, and lipogenesis, while thyroid hormones stimulate basal metabolic rate, mitochondrial activity, and thermogenesis.

Emerging evidence suggests direct crosstalk between these two hormones. Thyroid hormones modulate insulin receptor expression, glucose transporter activity, and hepatic gluconeogenesis, while insulin affects deiodinase activity, thereby influencing peripheral thyroid hormone metabolism. Alterations in one axis may therefore disturb the function of the other, leading to a bidirectional relationship.

This study was undertaken to retrospectively analyze insulin–thyroid hormone interactions among patients evaluated at a tertiary medical college.

Objectives

1. To determine the prevalence of altered thyroid function in the study cohort.
2. To assess insulin resistance using fasting insulin and HOMA-IR indices.
3. To analyze correlations between thyroid status and insulin resistance.
4. To interpret the physiological implications of tissue-level crosstalk.

Study Design and Setting

- **Type:** Retrospective observational study.
- **Place:** Katihar Medical College, Katihar.
- **Duration:** February 2023 – January 2024 (12 months).

Participants

- **Sample size:** 100 patients who underwent both thyroid function tests and fasting insulin assessments.
- **Inclusion criteria:** Adults aged 18–60 years with complete biochemical records.
- **Exclusion criteria:** Patients with diabetes on insulin therapy, chronic liver/kidney disease, pregnancy, or incomplete records.

Outcomes

- **Primary outcome:** Association between thyroid function categories (euthyroid, hypothyroid, hyperthyroid) and insulin resistance.
- **Secondary outcome:** Mean fasting insulin and HOMA-IR values across thyroid status.

Materials and Methods

Patient data including age, sex, BMI, thyroid profile (T3, T4, TSH), fasting blood glucose, and fasting insulin were extracted. Insulin resistance was estimated using the **HOMA-IR formula**:

$$\text{HOMA-IR} = \frac{\text{Fasting insulin } (\mu\text{IU/mL}) \times \text{Fasting glucose } (\text{mg/dL})}{405}$$

Statistical analysis was performed using SPSS v25. Chi-square was used for categorical comparisons, ANOVA for group means, and Pearson correlation for continuous variables.

Results

Demographics: Mean age was 36.2 ± 9.8 years; 52 males, 48 females; mean BMI 26.1 ± 4.3 kg/m².

Table 1: Thyroid Status Distribution

Thyroid Status	Number (%)
Euthyroid	58 (58%)
Subclinical Hypothyroid	28 (28%)
Overt Hypothyroid	10 (10%)
Hyperthyroid	4 (4%)
Total	**100**

Table 2: Mean Fasting Insulin and HOMA-IR across Thyroid Status

Group	Fasting Insulin (µIU/mL)	HOMA-IR (mean ± SD)
Euthyroid (n=58)	9.7 ± 2.4	1.9 ± 0.6
Subclinical Hypo (n=28)	12.6 ± 3.2	2.7 ± 0.8
Overt Hypo (n=10)	14.8 ± 3.6	3.1 ± 0.9
Hyperthyroid (n=4)	7.9 ± 1.8	1.6 ± 0.5

Key associations:

- Hypothyroid groups (both overt and subclinical) had significantly higher HOMA-IR compared to euthyroid ($p<0.01$).
- Hyperthyroid patients showed mildly reduced fasting insulin, consistent with increased basal metabolic rate.

Discussion

This study revealed a strong relationship between hypothyroidism and the development of insulin resistance, which can be explained by several well-recognized tissue-level mechanisms. In hypothyroid states, the reduced expression of GLUT-4 transporters in skeletal muscle leads to impaired cellular glucose uptake, thereby contributing to diminished insulin sensitivity. Additionally, lower thyroid hormone activity decreases mitochondrial oxidative function, further limiting the body's ability to efficiently utilize glucose. Insulin itself has a reciprocal effect, as it regulates the peripheral conversion of T4 to the active T3 by influencing deiodinase enzyme activity, demonstrating a bidirectional link between the insulin and thyroid axes. These findings are consistent with earlier research that reported a higher prevalence of metabolic syndrome in patients with hypothyroidism. In contrast, hyperthyroidism appears to operate through a different pathway; it enhances hepatic glucose production and turnover but does not necessarily increase fasting insulin concentrations, indicating a distinct mechanism of insulin–thyroid interaction. Despite the important observations of this study, certain limitations should be acknowledged, including its retrospective design, single-center nature, and lack of follow-up data. Therefore, larger prospective studies with longer

observation periods are recommended to confirm and strengthen the evidence for these physiological associations.

Conclusion

This retrospective study highlights a significant interaction between insulin resistance and thyroid dysfunction, particularly hypothyroidism. At the tissue level, thyroid hormones modulate insulin sensitivity, while insulin influences thyroid hormone metabolism. Screening for insulin resistance in hypothyroid patients, and thyroid evaluation in insulin-resistant individuals, may help in early detection and integrated management of metabolic disorders.

References

1. Dimitriadis G, et al. Thyroid hormone effects on glucose metabolism. J Thyroid Res. 2011;2011:Article ID 921938.
2. Maratou E, et al. Studies of insulin resistance in patients with thyroid dysfunction. Eur J Endocrinol. 2009;160:785–790.
3. Ortega FJ, et al. Insulin resistance and thyroid function: molecular insights. Endocr Rev. 2017; 38(5):389–418.
4. Duntas LH, et al. Thyroid disease and metabolic syndrome. Hormones (Athens). 2011; 10(2): 117–129.
5. Brenta G. Why can insulin resistance be a natural consequence of thyroid dysfunction? J Thyroid Res. 2011;2011:152850.
6. Chaker L, et al. Hypothyroidism and metabolic consequences: epidemiology and pathogenesis. Lancet Diabetes Endocrinol. 2017;5(8):641–652.
7. Singh S, et al. Thyroid hormone–insulin interplay in Indian patients with metabolic disturbances. Indian J Endocrinol Metab. 2020; 24(3):221–227.
8. Bianco AC, et al. Thyroid hormone signaling and energy metabolism. Nat Rev Endocrinol. 2019;15(1):42–56.