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# The Interplay Between Thyroid Hormone Function and the Risk of Type 2 Diabetes Mellitus

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# الترابط بين وظيفة هرمونات الغدة الدرقية وخطر الإصابة بداء السكري من النوع الثاني

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**Abstract:**
Diabetes mellitus and thyroid dysfunction constitute the two predominant endocrine disorders. Individuals diagnosed with type 2 diabetes mellitus exhibit an increased likelihood of developing both hypothyroidism and hyperthyroidism relative to non-diabetic individuals. Uncontrolled type 2 diabetes mellitus can lead to insulin resistance that affects the Thyroid autoimmunity arises due to the existence of autoantibodies. Blood specimens were obtained from Al-Aseel Hospital. The research encompassed 30 patients (15 females and 15 males) alongside 30 healthy participants (15 females and 15 males). Their ages range from 50 to 90. After a 12-hour fasting interval, a segment of the blood specimens was transferred to EDTA-free containers. The tubes were later subjected to centrifugation to isolate the serum for quantifying the concentrations of the thyroid hormones T3 and T4. The leftover samples were transferred to tubes containing EDTA, subsequently centrifuged to isolate the plasma for assessing fasting blood glucose levels. The results of the research suggest The notable increase in fasting blood glucose (FBG) and serum triiodothyronine (T3) detected in both diabetic males and females, whereas in males, there was no considerable rise in T4 levels in the unhealthy cohort relative to the healthy cohort. Conclusion : This research illustrates that type 2 diabetes mellitus correlates with a notable, sex-specific increase in thyroid hormone concentrations, marked by elevated fasting blood glucose and serum triiodothyronine (T3) in both genders, in addition to a significant rise in thyroxine (T4) exclusive to females.

**Keywords:** Thyroxine (T4) , triiodothyronine (T3), Type 2 Diabetes Mellitus.

**الملخص:**
يُعدّ داء السكري واضطرابات الغدة الدرقية من أبرز اضطرابات الغدد الصماء. يُظهر الأفراد المصابون بداء السكري من النوع الثاني احتمالية متزايدة للإصابة بقصور الغدة الدرقية وفرط نشاطها مقارنةً بالأفراد غير المصابين بالسكري. قد يؤدي داء السكري من النوع الثاني غير المُسيطر عليه إلى مقاومة الأنسولين التي تؤثر على المناعة الذاتية للغدة الدرقية نتيجة وجود الأجسام المضادة الذاتية. تم الحصول على عينات الدم من مستشفى الأسيل. شمل البحث 30 مريضًا (15 أنثى و15 ذكرًا) إلى جانب 30 مشاركًا سليمًا (15 أنثى و15 ذكرًا). تراوحت أعمارهم بين 50 و90 عامًا. بعد صيام لمدة 12 ساعة، نُقل جزء من عينات الدم إلى أنابيب خالية من مادة EDTA. خضعت الأنابيب لاحقًا للطرد المركزي لعزل المصل من أجل تحديد تركيزات هرمونات الغدة الدرقية T3 وT4. نُقلت العينات المتبقية إلى أنابيب تحتوي على EDTA، ثم خُضعت للطرد المركزي لعزل البلازما لتقييم مستويات سكر الدم الصائم. تشير نتائج البحث إلى ارتفاع ملحوظ في سكر الدم الصائم (FBG) وهرمون ثلاثي يودوثيرونين (T3) في مصل الدم لدى كل من الذكور والإناث المصابين بالسكري، بينما لم يُلاحظ ارتفاع ملحوظ في مستويات هرمون الثيروكسين (T4) لدى الذكور في المجموعة غير الصحية مقارنةً بالمجموعة الصحية. الخلاصة: يُبين هذا البحث أن داء السكري من النوع الثاني يرتبط بارتفاع ملحوظ في تركيزات هرمونات الغدة الدرقية، يختلف باختلاف الجنس، ويتمثل هذا الارتفاع في ارتفاع سكر الدم الصائم وهرمون ثلاثي يودوثيرونين (T3) في مصل الدم لدى كلا الجنسين، بالإضافة إلى ارتفاع ملحوظ في هرمون الثيروكسين (T4) لدى الإناث فقط.

**الكلمات المفتاحية:** الثيروكسين (T4)، ثلاثي يودوثيرونين (T3)، داء السكري من النوع الثاني .



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## Introduction

Thyroid hormone (TH) regulates metabolic functions essential for ideal growth and development, along with metabolism (Chaker et al., 2016). Thyroid-stimulating hormone (TSH) is produced by the hypothalamus-pituitary-thyroid axis and is recognized to fluctuate in alignment with the circadian rhythm (Fleiner et al., 2016). thyroxine (T4) and triiodothyronine (T3), two thyroid hormones crucial for regulating growth and metabolism, are generated and secreted following the hypothalamus's release of thyroid-releasing hormone (TRH), which subsequently stimulates the anterior pituitary gland to secrete TSH. Elevated levels of T3 and T4 inhibit TRH secretion in the hypothalamus and reduce TSH production in the anterior pituitary, and conversely, as per a negative feedback mechanism governing this process. Thyroid hormone has been shown to govern carbohydrate metabolism and pancreatic activity. On the other hand, diabetes may differentially affect thyroid function. The reaction of TSH to thyrotropin-releasing hormone has been demonstrated to be compromised in diabetes, resulting in hypothyroidism and subsequently reduced T3 levels. Research indicates that diminished T3 levels might be attributed to a reduced conversion of T3 from T4 in diabetes, as evidenced by studies showing hyperglycemia-induced reversible declines in deiodinase activity and hepatic thyroxine concentration. Alternative research has indicated that transient T3 surplus may provoke insulin resistance, consequently resulting in T2DM (Hussein et al., 2021; Khatiwada et al., 2016). Nonetheless, the association between thyroid hormone concentrations and the risk of T2DM is still fiercely debated, with human studies yielding contradictory results. Numerous studies have indicated a beneficial impact of elevated TSH and diminished free thyroxine levels on hyperglycemia and insulin resistance (Al-Geffari et al., 2013; Chaker et al., 2016; Fleiner et al., 2016; Papazafiropoulou et al., 2010), while others have asserted that no correlation was observed in their investigations (Khatiwada et al., 2016). Consequently, it is evident that a thorough assessment of the relationship among TSH, free thyroxine, and T2DM is necessary. Furthermore, nearly all prior investigations have concentrated on assessing the impact of baseline TSH and free thyroxine levels on the likelihood of developing T2DM (Chaker et al., 2016; Fleiner et al., 2016; Wang et al., 2021), yet limited research has explored the dose-response relationship of thyroid hormone levels concerning T2DM risk, prompting this analysis.

## Materials and methods :

The research period spanned from April 2025 to September 2026, during which blood samples were collected from Al-Aseel Hospital. The study included 30 patients (15 females and 15 males) and 30 healthy individuals (15 females and 15 males). their ages between 50 -90 , Following a 12-hour fasting period, a portion of the blood samples was placed in EDTA-free tubes. These tubes were subsequently centrifuged to separate the serum for measuring the levels of the thyroid hormones T3 and T4. The remaining samples were placed in EDTA-containing tubes, then centrifuged to separate the plasma for measuring the levels of fasting blood glucose.

## Analysis of data:

By minitab 17, and a significant level of $p < 0.05$.

## The result :

Table 1 presents data for a group of men consisting of 30 subjects: 15 in the healthy group and 15 in the non-healthy group. The healthy group had a mean age of 71.73 ± 8.96 years, while the non-healthy group had a mean age of 68.27 ± 8.53 years. The p-value is 0.287, indicating no significant difference.

**Table 1: Data for the Non-Diabetic Group and Diabetic Group of Men group**

| parameters | Healthy (n=15) | Non healthy (n=15) |
|---|---|---|
| **Male** | n=15 | n=15 |
| **Age (50-90) years** | 71.73 ±8.96 | 68.27± 8.53 |

**Pvalue =0.287 non significant**



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The results for the male subjects are presented in Table 2, which compares the healthy group and the non-healthy group. In the healthy group, the mean fasting blood glucose (FBG) level was 89.67 ± 6.81, while the mean levels of T3 and T4 were 1.346 ± 0.290 and 6.88 ± 1.45, respectively. In the non-healthy group, the mean FBG level was 193.7 ± 57.5, and the mean levels of T3 and T4 were 1.703 ± 0.467 and 15.8 ± 20.0, respectively. There was a significant elevation in the levels of FBG and T3 in the non-healthy group compared to the healthy group; however, there was no significant elevation in the levels of T4 in the non-healthy group compared to the healthy group .

**Table 2: FBC, T3, and T4 levels in healthy and non-healthy male groups**

| | Heathy 15 | Non heathy 15 | P – value | Significant level |
|---|---|---|---|---|
| **(FBG) (mg/dl)** | 89.67 ± 6.81 | 193.7 ± 57.5 | 0.000 | P < 0.05 significant |
| **T3 (mg/dl)** | 1.346 ± 0.290 | 1.703 ± 0.467 | 0.0**19** | P < 0.05 significant |
| **T4 (mg/dl)** | 6.88 ± 1.45 | 15.8 ± 20.0 | 0.**1** | P > 0.05 Non significant |

**Table 3: Data for the Non-Diabetic Group and Diabetic Group of female group**

| parameters | Healthy (n=15) | Non healthy (n=15) |
|---|---|---|
| **Male** | n=15 | n=15 |
| **Age (50-90) years** | 66.07 ±9.25 | 65.73± 7.99 |

**Pvalue =0.917 non significant**

The results for the female subjects are presented in Table 4, which compares the healthy group and the non-healthy group. In the healthy group, the mean fasting blood glucose (FBG) level was 85.6 ± 24.0, while the mean levels of T3 and T4 were 1.483 ± 0.274 and 8.20 ± 1.46 respectively. In the non-healthy group, the mean FBG level was 183.5 ± 60.7, and the mean levels of T3 and T4 were 1.893 ± 0.374 and 53.3 ± 54.8 respectively. There was a significant elevation in the levels of FBG , T3 and T4 in the non-healthy group compared to the healthy group .

**Table 4: FBC, T3, and T4 levels in healthy and non-healthy female groups**

| | Heathy 15 | Non heathy 15 | P – value | Significant level |
|---|---|---|---|---|
| **(FBG) (mg/dl)** | 85.6 ± 24.0 | 183.5 ± 60.7 | 0.000 | P < 0.05 significant |
| **T3 (mg/dl)** | 1.483 ± 0.274 | 1.893 ± 0.374 | 0.002 | P < 0.05 significant |
| **T4 (mg/dl)** | 8.20 ± 1.46 | 53.3 ± 54.8 | 0.007 | P < 0.05 significant |

## Discusion

The research encompassed 30 patients (15 women and 15 men) alongside 30 healthy participants (15 women and 15 men). The objective is to ascertain the relationship between type 2 diabetes in both genders aged 50 to 90 and the hormones thyroxine (T4) and triiodothyronine (T3). Their ages range from 50 to 90. The findings of the study indicate The significant elevation in fasting blood glucose (FBG) alongside serum triiodothyronine (T3) observed in both diabetic men and women, coupled with a female specific significant elevation in thyroxine T4, demonstrates a complex endocrine interplay where hyperglycemia and insulin resistance disrupt the hypothalamic pituitarythyroid axis and peripheral hormone regulation. High blood glucose levels and compensatory hyperinsulinemia stimulate thyroid tissue proliferation and augment hepatic gluconeogenesis and intestinal glucose absorption, which can temporarily elevate circulating thyroid hormone levels and exacerbate peripheral insulin insensitivity. The distinct sexual dimorphism—wherein females exhibit marked elevations in T4 whereas males show no statistically significant change—aligns with broader clinical evidence indicating that postmenopausal hormonal changes, higher adiposity, and altered estrogen-to-androgen ratios



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increase female vulnerability to thyroid axis dysregulation under metabolic stress. These empirical findings are strongly corroborated by Hussein et al. (2021), who highlighted how type 2 diabetes differentially impacts thyroid hormone conversion and sensitivity through insulin resistance, as well as a comprehensive study by Wang et al. (2021) demonstrating sex-stratified associations between circulating thyroid hormone concentrations T3 and T4 and the risk and severity of type 2 diabetes mellitus. The observed positive correlation and significant elevation of serum T3 in both diabetic sexes, alongside the female-specific increase in T4 levels, find strong support in several clinical investigations. Khatiwada et al. (2016) similarly reported elevated thyroid hormone levels among poorly controlled type 2 diabetic patients, suggesting that chronic hyperglycemia and compensatory hyperinsulinemia exert a trophic effect on the thyroid tissue, thereby enhancing hormone release. Furthermore, Chaker et al. (2016) demonstrated that higher levels of circulating thyroid hormones exacerbate insulin resistance through enhanced hepatic glucose output and altered intestinal absorption. The gender dimorphism identified in this study—where females exhibited marked T4 elevations while males did not—is corroborated by Al-Geffari et al. (2013) and Fleiner et al. (2016). Their findings highlighted that postmenopausal female patients present higher susceptibility to thyroid axis dysregulation under metabolic stress, largely mediated by altered estrogen-androgen dynamics and variations in thyroxine-binding globulin concentration. Conversely, earlier literature such as Papazafiropoulou et al. (2010) observed reduced T3 levels due to impaired peripheral T4 to T3 conversion caused by suppressed deiodinase activity in severe hyperglycemia. However, the compensatory surge in T3 and T4 observed in our cohort aligns with the findings of Hussein et al. (2021) and Wang et al. (2021), emphasizing that insulin resistance and disease progression can dynamically alter thyroid hormone synthesis and peripheral sensitivity in a sex-dependent manner.

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